The Brain Behind Myofascial Trigger Points

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This thesis is my original work with no material jointly-authored, previously published or used for other studies, with all contributions and references clearly cited. It is the result of work I carried out since diving into this topic during my master's studies in early 2015. The resulting synthesis contains much inference, upon which I hope other researchers build. I acknowledge that copies of this thesis must be lodged at the University of Thessaly library, and that copyright of all material in it resides with its original holders. Otherwise, this full work is released under the CC0 license, with all rights waived and further work stemming from it actively encouraged.

I must thank my sister, **Joanna Sulzycki**, for modeling in the *Methods* section, and my colleagues, **Lois Reyes** and **Jana Konkin**, for acting in my trigger point treatment video and for the "melting" script, respectively. I am indebted to **Vassilis Bouglas** at the University of Thessaly library for his truly above-and-beyond help in my research, and to my practicum supervisor, **Marek Graczyk**, and the physiotherapy department director, **Małgorzata Kawa**, at Gdańsk Sport University who gave me the means and participants to run my study on campus, as well as **Agnieszka Lasota** and **Dagmara Budnik-Przybylska**, for helping recruit further participants from local actors and neighbouring Gdańsk University sport science students. Most importantly, however, were the finances that made all this possible through my working a *very* irregular schedule at Queen's Park Massage Therapy in New Westminster just outside Vancouver, something the owners, **Kelly Baxter** and **Sharon Nagle**, not only allowed but actively supported.

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shifting then but that are so clear and obvious now. And to my family — extended throughout Europe (mostly Poland) and Canada (mostly Hamilton): knowing that you are always there with a warm meal, a place to sleep, a hot shower, and a drive to the station is a godsend… a sincere thank-you to my aunt, **Lucy**, for inspiring me to become a massage therapist, my uncle, **Wojtek**, for driving me to massage college ~100km from Hamilton to Toronto for months on end, rain or shine, and my cousins **Ewa**, **Mateusz**, and **Piotruś**, for all the love in Gdańsk, providing airport rides, restful nights, and everything in between. This security was what really freed me to focus on these studies. And particularly my mother, **Barbara**, for always being there. Always. We never wanted for anything because of her now calloused hands. And my father, **Janusz**, for coaching our minds and bodies, for introducing me to sport, to philosophy, and to ethology — leading to biology studies that ultimately started me on this privileged path of learning. And to my inspiring sisters, **Magdalene**, the other half of my mind, and **Joanna**, the other half of my smile. I love you all. So very, *very* much. There is a piece of each and every one of you in this paper. I do not have room enough for a proper outpouring of thanks and emotion for all the good that has met me in life, but here's hoping this paper pays some of that forward by reducing pain for others. And in closing, of course, an exceptional thank-you to the stoic face of patience itself that was my supervisor, **Marios**. He waited his while to receive this completed document, but now has, in his own words, a "good thesis".

I thank you all for your support and especially space to think while I sometimes chipped, sometimes hacked, sometimes stumbled but always fell forward in this scope creep of a Sisyphean effort.

Ideas need space. This one, not anymore.

Thank you.

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April 4th, 2018

A good thesis is a done thesis

– Marios Goudas, supervisor

Abstract

Despite imagery's regular use in massage therapy there are few studies examining its effectiveness. This experimental study examined the effect of imagery on manual trigger point release. Forty individuals (11 male and 29 female), aged 19-51 (M = 26.13, SD = 8.05) were divided into two groups; one focusing on deep, diaphragmatic breathing and the second on a rich imagery script, imagining their trigger point being a "ball of butter melting". Manual trigger point release was applied four successively deeper times to the same point in the dominant upper trapezius muscle following the common "searching for 7" pain scale protocol. Significant differences were found in self-reported pain tolerance scores for participants using the breathing (M = 2/10, SD = 1.8/10) versus imagery scripts (M = 1/10, SD = 1.2/10), t(37) = 2.10, p = .043. A trend only approaching statistical significance was observed between treatments in both average press release (imagery: M = 25.6 s, SD = 15.5 s, breathing: M = 30.0 s, SD = 27.3 s, t(38) = 0.62, p = .540) and total treatment time (imagery: M = 2:15 m, SD = 1:08 m, breathing: M = 2:36 m, SD = 2:04 m, t(38) = 0.67, p = .507), with largest differences in the final two presses.

However, analyzing the above while taking local *v.* referred pain presence into account showed significant main effects of pain on the first press (local/breathing: M = 43.2 s, SD = 24.8 s; local/imagery: M = 31.5 s, SD = 16.1 s; referred/breathing: M = 23.2 s, SD = 12.5 s, referred/imagery: M = 28.1 s, SD = 12.2 s; $F_{1,30}$ = 4.53, p = .042) and significant interaction effects of both pain and treatment type on total treatment time (local/breathing: M = 3:32 m, SD = 2:34 m; local/imagery: M = 2:07 m, SD = 1:12 m; referred/breathing: M = 1:50 m, SD = 1:11 m, referred/imagery: M = 2:22 m, SD = 1:08 m; $F_{1,30}$ = 4.64, p = .039). Based on this study, therapists should include breathing techniques in manual trigger point release for patients experiencing referred pain and imagery scripts for patients experiencing only local pain.

Keywords: *massage therapy, myofascial trigger point, imagery, PETTLEP, gate control, neuromatrix, neural modulation, sensitization, referred pain, pain, muscle, neurology*

Contents

CONTENTS *(cont'd)*

Lists of Figures & Tables

Introduction

Background

Massage therapists across Canada often use psychological techniques during treatment, even if they are rarely labelled as such. From a warm clinic decor to the hushed tones and gentle music used in treatment, the field is constantly seeking new ways of fostering mind-body harmony. The process begins in school with an introduction to the "principles of massage": a set of gold standards outlining a slow, progressive approach to treatment.[1] These are to be followed regardless of technique or style with few exceptions, ensuring an easy, supportive progression that builds trust and confidence into the vital patient-therapist relationship. The ultimate goal, of course, is the delivery of a treatment that is maximally effective and long-lasting… something that is nearly impossible unless the patient is relaxed. It is logical, therefore, that certain psychological elements would have seeped into common massage therapy practice, from reframing pain, to goal-setting across treatment plans, to even harnessing imagery during the more painful moments in treatment. It is on this last tendency that this paper focuses.

As massage therapy develops formal protocols, a common sense/no-harm philosophy with little scientific backing has prevailed, kept alive through misunderstanding and uncertainty. A good example is the fear that massage may metastasize cancer in patients through increasing blood flow and thereby physically "moving" the cancer to other body regions. In fact the opposite is

true: massage boosts the immune system through upping the production of NK cells, known for combating cancer.[2]

Since rigorous, peer-reviewed studies on the mechanisms and efficacy of massage are still few and far between[2-4] — with those exploring trigger point pathology rarer still[5,6] — interdisciplinary research is a good springboard for key findings to emerge. Sport psychology has much to offer in this regard, especially considering its strong history of training perception to reach certain performance levels. Because of all this, research for this paper extended beyond the traditional literature or even media. It drew on web forums, conversations with therapists, university tutorials and course notes, and several clinicians' videos explaining various phenomena for their patients and students. For this reason, what is presented is more a launchpad into the area straddling sport psychology and massage therapy than it is any definitive text on either subject. It is also why it begins with a series of separate concepts that only reach full synthesis in the discussion.

Even so, this paper is an attempt at bridging these fields beyond the usual anecdotal, "common sense" approach criticized earlier. Recent calls have been made for closer psychosomatic collaboration from both physiological[7,8] and psychological[4,9-12] fronts. Benefits of such integration include increased professional growth for therapists as well as improved treatment effectiveness for patients.[4] Clear applications of massage have already been found in improving anxiety, depression,[4] perception/sensation, sleep, learning, and alertness.[10,13] Indeed, connecting psychology with massage therapy is not only recommended[14] but required when it comes to the phenomenon of pain, which is modulated by both physiological and psychological processes.[7,8,11,12,15-26] There currently exist some manual treatment modalities tying psychological theories into their application[4,27-30] and some forays have already been made to integrate massage therapy into psychotherapy sessions — with great effect.[31] What were once purely anecdotal cases tying these fields together are quickly gaining traction as scientifically-sound research.[32-35] Further, since pain is both a sensory and emotional experience, it makes sense to use both pathways in dealing with such a complex phenomenon.[36] Past studies have linked both massage therapy and imagery with muscle pain relief;[37,38] it is the aim of this paper to continue building upon these psychosomatic connections in the field of rehabilitative therapy.

Problem

Imagery use has naturally evolved alongside massage therapy's development into a serious health profession worldwide. Therapists often create scripts spontaneously when working through particularly stubborn muscle knots; generally simple, relatable, relaxing themes rooted in local culture. These are inevitably shared throughout clinics, naturally paired with anecdotal stories of their effectiveness. I have worked over a decade as a massage therapist in spas and in chiropractic, physiotherapy, massage, and sports medicine clinics in five distinct regions across Canada. Each of these was set on vast bodies of water: Toronto (Great Lakes), Cape Breton (Atlantic Ocean), Yellowknife (Great Slave Lake), Vancouver (Pacific Ocean) and Montreal (St. Lawrence River). During this time, I would often note therapists using imagery scripts with coastlines and water as main themes (eg: "undulating waves hugging the shore", "golden sand baking in the sun", "the call of seagulls and the swells of approaching tides").

Though nice and relaxing, I wanted to know what imagery's actual effectiveness was in treatment, specifically regarding trigger point release. Should it prove to be ineffectual, then it could be abandoned and we could shift our focus to other, better techniques for pain relief. However, should the opposite be true then imagery could be officially incorporated into the trigger point treatment protocol slowly being developed in physical therapy clinics worldwide. A second reason I was interested in this topic was the complete absence of any comprehensive theory explaining the mysterious trigger point referred pain phenomenon I would deal with almost daily in clinic. This pain didn't seem to follow any known neural routes… so where was it coming from?

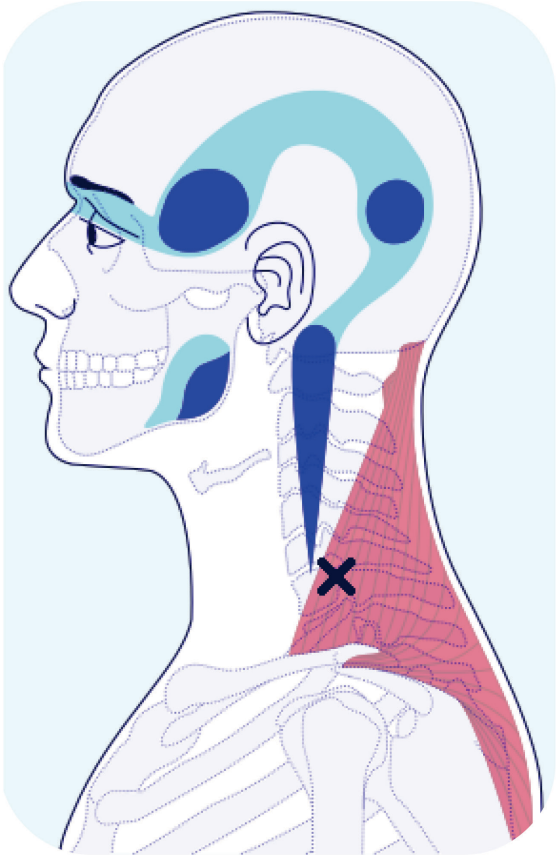


Figure 1: Upper trapezius referred pain. *Classic "question mark" pain brought about through pressing its trigger point (**X**).*[39]

The muscle chosen for this study was the upper trapezius since it is here that trigger points are most often found,[39] causing what is probably the most

classic of all muscle tension headaches: a "question mark" pain referral looping around the ipsilateral ear, starting at the neck and mastoid process up to the temple and even sometimes including the jaw below (*Figure 1*).[40-42] Headaches of this type have a very high incidence rate, ranking along with migraines as the world's 8th most burdensome health issue (ahead of diabetes, heart disease, cancer, and malaria),[22] with 30-40% of the global population experiencing at least one annually[43,44] (though rates do vary across cultures),[45] and costing billions of dollars in combined employee inefficiency and outright absence.[46] As shorter healing times have direct implications for a nation's health and economy, and as myofascial disorders like tension headaches consistently rank as one of the most prevalent conditions in the world,[47-53] the results from this study present applications for a large segment of the world population.[39,54]

The main theoretical framework used in this study is Melzack and Wall's gate control model of pain perception (*Figure 4*).[55] This theory is the one most frequently taught in Canadian massage colleges to explain why massage is so effective in alleviating pain.[2,56] Further, according to the gate control theory, negative cognitions such as expectations or worries have the power to intensify pain perception while positive cognitions like distraction, relaxation, and positivity have the power to lessen or even completely deactivate pain perception.[55,57,58] Since imagery delivers these very positive cognitions,[59] it seemed a logical addition to the otherwise purely mechanical trigger point treatment.

Hypothesis

Based on the following literature review and on my own clinical experience, including anecdotal evidence from many physiotherapists, chiropractors, and other massage therapists along the way, releasing trigger points with traditional massage therapy combined with a relaxing imagery script was expected to result in faster and more effective treatment times than would coupling the treatment with a generic breathing script.

Literature Review

Massage therapy, psychology, and pain

In addition to the many studies written on the physiological effects of massage therapy, there have been several on the psychological and behavioural effects it has on patients. Findings relate a wide variety of improvements: reductions in stress, anxiety, depression, cortisol levels, ADHD symptoms, and pain, as well as in activity limitations caused by pain. Further, massage improves mood, sleeping, eating habits, body image perception, relaxation, performance, alertness, and concentration.[2,4,10,56,60-62] But this is not without reproach. Criticism always returns to inconsistent findings, methodological flaws, and to a simple lack of studies in the field.[2,61]

However, the main gap this paper fills compared to the studies mentioned is that nearly each studied the unidirectional effects of massage on a person's psychology; rarely was the reverse ever examined. Indeed, this has been the main stance of Western medicine since the scientific revolution: that the body affects the mind is clear. The reverse pathway is coming to light in mainstream science only as of recently.[63] Articles on the psychology of trigger point treatment were even scarcer, despite the treatment having clear psychological ties regarding pain perception and despite this therapy being one of the most painful and most prevalent massage modalities currently used in across Canada.[14]

Current theories explaining why massage alleviates pain focus primarily on physiological phenomena, including its increasing blood and decreasing cortisol circulation, its activation of the parasympathetic nervous system via relaxation, and its modulation of incoming nerve signals as

outlined in the gate control model (*Figure 4*). In addition to all these factors, there also exist the hypotheses that massage improves affect via social support and touch,[3] and that people's personalities — broadly described as pain-adaptive or -nonadaptive — can predict pain response (with the former inherently dealing better with managing pain than the latter).[20,21,64] Such integrated, personalized treatment is already underway for low back pain, with patients being reliably divided into several subgroups based on psychosocial risk factors for specialized treatment instead of treating every incidence of the condition as part of a single batch phenomenon.[65]

Pain: sensation, modulation, and perception

Pain is particular among sensations in two ways. First, it is not always subject to habituation. Indeed, it often undergoes the opposite, intensifying with increased or maintained stimuli in what is called "sensitization".[22,23,66-70] Secondly, it has an emotional element: unpleasantness, consisting first of sensation and then only sometimes perception (*Figure 7*). This first is the reception of all the physical elements — pressure, registration of touch, *etc.* The second is the cerebral labeling of these as unpleasant, and is heavily dependent on the modulation the nociceptive signal undergoes from peripheral stimulation to cerebral processing.[71]

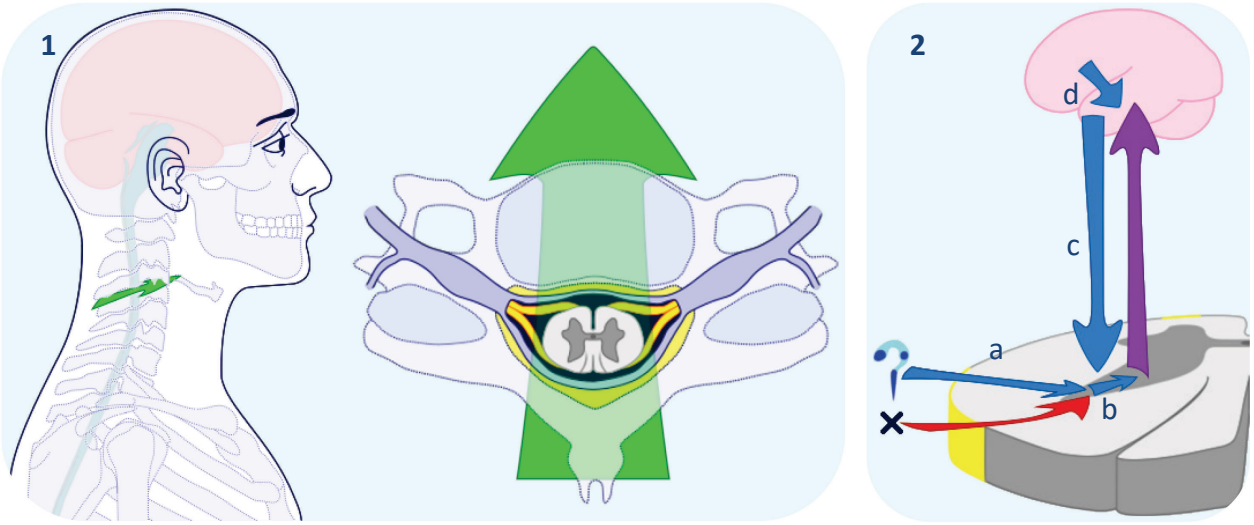


Figure 2: Main pain theories in context. Image 1 *contextualizes cerebrospinal neuroanatomy, zooming in on a C3/4 transverse section where many trapezius nerves enter and exit the spinal cord.* ***Image 2*** *zooms in on the spinal cord, showing the pain stimulus (**X**), the site of referred pain (**?**), and all pain modulation models outlined in this paper: original ascending pain (**red**), potential modulation (**blue**, including referred pain (**a**), gate control (**b**), and descending modulation (spinal: **c**, cerebral neuromatrix: **d**)), as well as the resulting signal perceived (**purple**).*

Modulation happens in a few spots along pain's path to the brain, where it may intensify or diminish, or even disappear altogether,[23,57,67,72] explaining why pain may occur without injury (*eg*: phantom limb pain), may remain long after an injury has healed, or may even not occur at all when expected (*eg*: battle wounds). Pain is a bidirectional process, undergoing modulation through both ascending and descending pathways (*Figure 2*). Ascending modulation occurs when pain is modified by a competing signal arriving from the periphery or by an interneuronal signal still in the spinal cord, as with referred pain (*Figure 3*) or in the gate control model (*Figure 4*). Descending modulation occurs in the spinal cord where a cerebral signal modulates peripheral pain (*Figure 8*) as in the pain-tension cycle[67,73] or inside the brain as per the neuromatrix theory[16,72,74,75] through release of analgesic neurotransmitters (*eg*: serotonin).[76]

Ascending modulation

Referred pain

The most pertinent diagnostic tool for this study was the referred pain pattern elicited through point compression of the tender trigger point (*Figures 1 & 18*).[77] Though not following any known nerve pathways or myotomes,[78,79] these patterns are very consistent across patients.[5] Because of this, when a patient is experiencing referred pain in a known pattern, their therapist can often quickly deduce which muscle is behind it.[39,46,78,80-82] These patterns are especially prevalent in muscle tension headaches (muscular in origin and characterized by their gradual onset, accompanying sensations of pressure and tension, and sustained triggering of generally unconscious contractions in head and neck musculature),[57,83] while also being an important factor in the etiology and maintenance of chronic migraines.[84] However, there is no one clear mechanism yet explaining their phenomenon completely.[85]

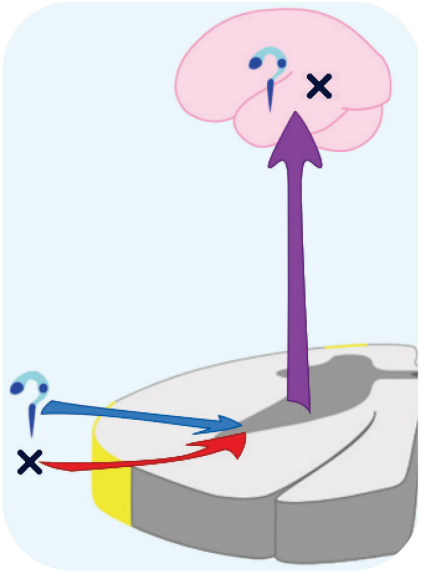


Figure 3: Referred pain. *Trigger point (**red**) and referral pain (**blue**) nerves converging as pain (**purple**) "felt" at both injury (**X**) and referral (**?**) sites.*[39]

The current model posits pain as converging at spinal synapses,[78,86] a process identified over fifty years ago and consisting of "convergent projection" — *ie*. spinal nerve neural overlap of the injury and referral zones — and "central sensitization" — increased spinal neuron response with repeated peripheral neuron stimulation, enabling smaller and stimuli to trigger a response (*Figure 3*).[87-101]

This happens over several stages. First, a muscle suffers a mild injury (*eg*: repetitive stress in sustained poor posture as in *Figure 10*), sending painful neural signals to the spinal cord. These are ignored by the brain owing to their mildness, but converge with signals arriving from unrelated nerves, perhaps even synapsing with them over time. If this is the case, chemicals released from the first nerve could activate several nonproblematic nerves nearby, making it seem to the brain as if these nerves are now delivering painful signals as well.[46,74,89-91,102] These convergence and sensitization processes would strengthen over time, allowing less intense stimuli of the injury site to result in the same or even more intensified referred pain responses,[39,67,102] similar to the phenomenon at the root of arm/chest/jaw referred pain common during a heart attack.[103]

Gate control theory

A second model explaining how massage releases both local and referred pain is that of "gate control".[55,104] Pain was initially thought to correlate directly with the degree of injury (*ie*. being *objectively* painful, as in *Figure 5*).[12,16,105] Gate control theory, however, showed that pain is not a simple linear process starting with a stimulus and ending at perception. Rather, it is controlled by "gates" modulated by competing nerve signals. This happens in the spinal cord where primary sensory nerves synapse with interneurons, as well as in the brain itself, where sensation, emotion, and memory combine with other cognitions to form perception (*Figure 4*).[55,57,74,75]

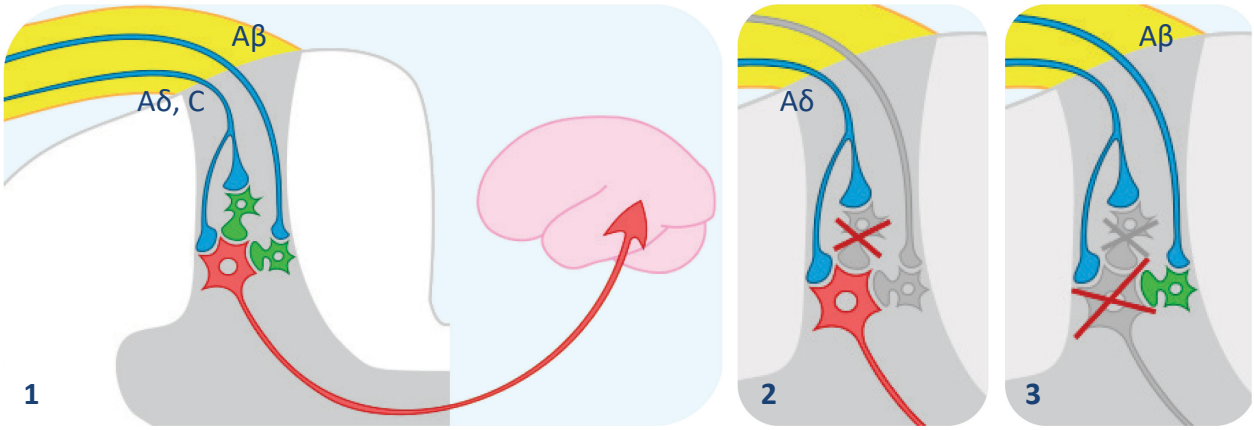
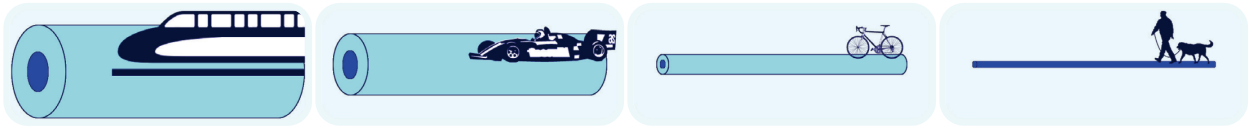


***Figure 4: Gate control theory. Image 1** shows the neurology involved: peripheral sensory nerves (**blue**), inhibitory spinal interneurons (**green**), and the projection neuron bringing final modulated signals to the brain (**red**). **Image 2** shows **Aδ** firing from a painful signal, stimulating pain directly as well as indirectly through inhibiting inhibitory interneurons. **Image 3** shows **Aβ** firing from pressure or motion. This stronger mechanical signal immediately overrides **Aδ** nociception via spinal interneurons, canceling the perception of pain. Pain cannot be perceived without the activation of interneurons carrying this signal to neurons that ultimately arrive in the brain.*[55,106-112]

This modulation is possible because different sensations travel along different nerves at different speeds. Proprioception travels fastest, through thick, myelinated Aα fibres, while touch travels more less quickly through thinner, myelinated Aβ fibres. Sharp, acute pain travels slowly through thin, myelinated Aδ fibres, and sustained, "burning" pain travels slower still, through thin, unmyelinated C fibres.[46,106,114-116] To put this all in perspective, Aα signals travel at the speed of a bullet train and Aβ almost as fast as Formula One racing cars, while Aδ signals could only keep pace with fast cyclists and C signals would fall behind even a brisk walker (*Figure 6*).[36,67,117,118]

Figure 5: Original pain model. *Descartes' 1664 pain pathway is linear and unidirectional.*[113]

The majority of Aδ signals (90%) quickly reach the brain, precisely locating the injury. However, only about 10% of the C fibre signals do the same, making the source of this duller pain much harder to locate, partially explaining why some chronic pain becomes very difficult to pinpoint.[116]



Aα: proprioception, 13-20 µm
80-120 m/s (avg. ~360 km/h)

Aβ: touch, 6-12 µm
35-75 m/s (avg. ~200 km/h)

Aδ: sharp pain, 1-5 µm
5-35 m/s (avg. ~70 km/h)

C: sustained pain, 0.2-1.5 µm
0.5-2.0 m/s (avg. ~5 km/h)

Figure 6: Afferent nerve comparison: *diameter, perception, myelination (light blue), and conduction velocities.*

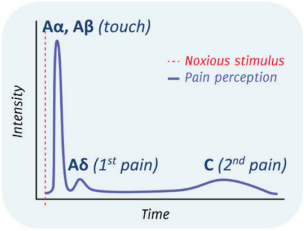


Figure 7: Pain timeline.[72,117]

These differences explain why acute injury subjects generally first report an immediate shock — registering the sensation of touch — followed by an almost-immediate jolt of intense, stabbing pain ("first" pain), ending in a sustained, throbbing pain ("second" pain) that appears slowly and remains for some time (*Figure 7*).

All these fibres converge in the spinal cord,[55] from which pain travels to the brain from the Aδ and C fibres unless inhibited through a much faster signal traveling via Aβ fibres, like pressure or motion (*Figure 4*). Noxious thermal stimuli (>43°C or <25°C) may also override nociception: these are registered by both Aδ and C fibres,[114] with the latter likely being the primary afferent.[119] This ascending gate control modulation explains both why we reflexively rub, shake, and ice injuries immediately, as well as why massage brings almost immediate pain relief to sore muscles.[117,120]

Descending modulation

Nociception is also modulated via descending pathways through direct cortical or thalamic neurotransmitter release (*eg*: serotonin, noradrenaline, endogenous opioids),[67,76] through rapid, cognition-induced brain activity (*eg*: beliefs, experiences, expectations, fear),[55] and through behavioural changes like increased sleep or exercise.[12,18,26,36,55,121,122]

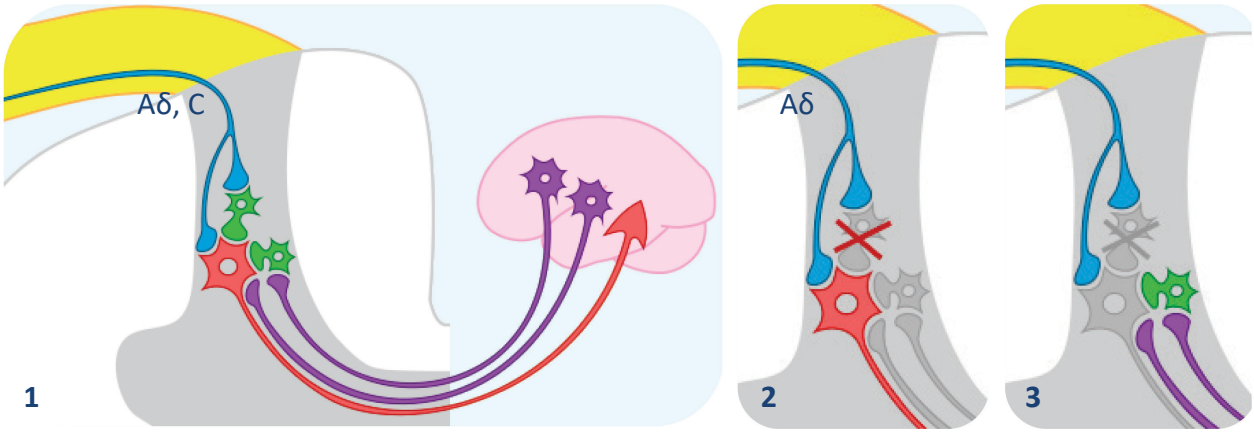


***Figure 8: Descending modulation. Images 1** and **2** are as in **Figure 4**, with the focus now on modulatory nerves (**purple**) descending from the brain.[16] **Image 3** shows this modulation overriding **Aδ** nociception, eliminating pain.[55,57,74,107-109,114,123] Prescription opioids like morphine act similarly and so are used in cases of chronic pain.[123]*

The brainstem bridges cognitions with top-down pain modulation (*Figure 8*). Nociception enters the brain from the spinal cord (*Figure 9.3 violet track*) where some neurons — mostly Aδ[116] — transmit information on location and intensity to the somatosensory cortex via the thalamus[124] (*Figure 9.3 red track*). This branch meets with descending analgesic tracts in the midbrain's periaqueductal grey matter,[125,126] modulating pain based on fear, attention, and expectation,[11,123,127,128] and in the rostroventral medulla oblongata's nucleus raphe magnus (NRM)[114,123] is close to the retrotrapezoid nucleus which itself regulates breathing,[129-131] perhaps explaining why focused breathing consistently delivers analgesic effects.[132-135] Other neurons travel to the cingulate and insular cortices via the pons' parabrachial nucleus and the amygdala, modulating pain through emotion (*Figure 9.3 yellow track*). The amygdala fires more in situations involving fear[136] while the insular cortex serves to identify pain magnitude[122] and is more active with visual input,[137] even if imagined (as is true for the anterior cingulate cortex).[136] This anatomy explains why cognitions like anxiety, worry, depression, and focusing on pain could "open" the gate mentioned earlier, while distraction, relaxation, and positive emotions could "close" it.[11,57,104]

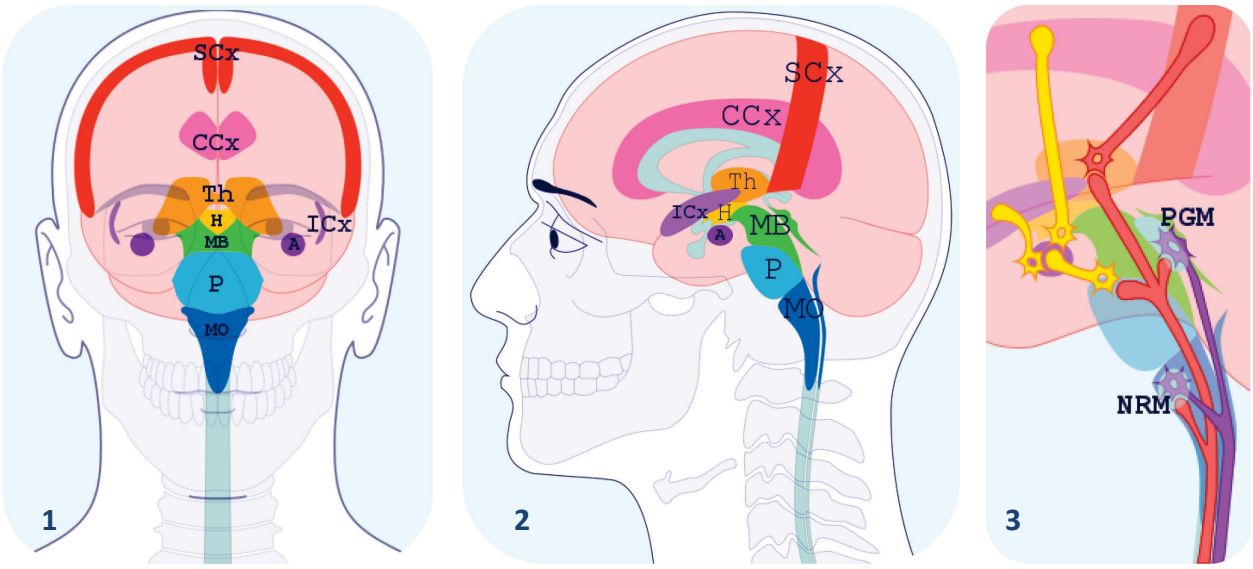


***Figure 9: Pain in the brain. Images 1** and **2** show the brainstem's medulla oblongata, pons and midbrain; the amygdala and the cingulate, insular and somatosensory cortices; and the thalamus and hypothalamus. Nociception ascends (**red**) in **Image 3**, and is modulated by cerebral affects (**yellow**) and direct brainstem input (**violet**), perhaps via the medulla oblongata's nucleus raphe magnus and the midbrain's periaqueductal grey matter.*[23,30,114,123,138,139]

Clinicians are now teaching patients to fear pain less and to not catastrophize their state or future,[16-26,140] while also teaching them to better deal with day-to-day occupational, mood, and social stressors, knowing that mastering these leads to more effective pain resolution.[21,23,138,140]

The detrimental mind-body feedback loop of a physical injury causing psychological stress — itself increasing risk of re-injury — has been extensively studied.[69] Tension, anxiety, and panic cause muscle spasms, vasoconstriction, and blood flow shunting from the extremities to the core, causing a drop in oxygen availability and bringing alertness as well as muscle and brain activity down with it. Once this loop is understood — as well as the patient's own power over it — the patient is better able to put a stop to it and take an active role in their own recovery, reducing anxiety and thereby allowing for healing to begin.[68,73]

Of special importance to this paper is that trigger points worsen with psychological stressors. In one study, trigger point activity was shown to increase with both expressed and inhibited anger, with significant correlation with the latter in particular.[141] This may be due to muscle guarding[141] or to sympathetic neurological involvement at muscle spindles (capsular sensory receptors embedded in muscle bellies)[142] seen in pharmacological studies showing decreased electromyographic activity in the trigger point with sympathetic neuron blockers but not with

motor neuron blockers.[143] Other studies showed that recalling emotional events increased trigger point activation[144] while relaxation techniques did the opposite.[145,146] Ties were later found between individuals with internalizing traits (high worry, fear of criticism, low assertiveness) and feeling more pain in trigger points than individuals without these traits.[144] Lastly, biopsychosocial interventions — physiological interventions with psychsocial support systems — have been found to decrease the overall experience of chronic pain,[14] while a lack of social support relates to a marked increase in its perception.[3,12,21] Therefore, interventions including muscle relaxation, biofeedback, social support, and imagery are recommended as a major goal of treatment.[147]

Neuromatrix theory

All this is being distilled into a theory that it is a widespread cerebral network — a "neuromatrix" — that perceives pain,[72,74,75,104] modulating its sensation starting with cognitions that were traditionally written off as mere reactions (*eg*: apprehension, fear).[74,75] It shows pain to be a very complex process governed by a convergence of influences, including genetic determinants, cognitive events, and somatosensory input.[67,75] It underlines that pain management may be more effective when cognitive processes are taken into account, explaining why cognitions like imagery have mitigated pain consistently in both the literature as well as in clinical practice.[148]

Trigger point pathophysiology

Trigger points were described as early as the 16th century and have remained a very controversial topic since their rediscovery in the 20th,[84] with even their very existence currently being questioned.[149-153] That said, the focus of this paper isn't so much on their physiology or etiology as it is on the mediation of the pain they cause, both locally and via referral, which is very real indeed. Still, their commonly-accepted pathogenesis follows for context.

Formation

From the seminal text on the subject, a trigger point is a hyperirritable spot in skeletal muscle often palpable as a nodule in a taut band.[39,84,86,154,155] It forms when performance intensity outmatches the body's normal ability to sustain it and normal recovery is prevented.[5,156-160] This can be the result of sudden muscle load increases, as with intense movements or trauma (*eg*: whiplash, muscle strain),[84] of slowly-mounting strains as in repetitive stress injuries (chronically sustained, submaximal contractions),[5,6,46,84,161,162] or even of the mere addition of biomechanical stress to cooled muscles (these are less supple and therefore more prone to tearing).[102]

Trigger points occur in 20-70% of the general population,[39,53,163-165] most often in, again, the upper trapezius muscle.[39] This is likely due to its frequent flexion, both in the classic shoulder-hunching, defensive posture typical in response to stress, as well as in sedentary postures increasingly prevalent throughout society (*eg*: anterior head carriage and accompanying shoulder rounding characteristic of Upper Crossed Syndrome, as in *Figure 10*). Our head weighs 10 lbs, with another 10 lbs of stress added to our neck musculature every inch it moves forward of the midline (the upper trapezius being a key player).[166,167] It is not surprising, therefore, that these muscles may respond with injury over time, potentially causing trigger point formation.

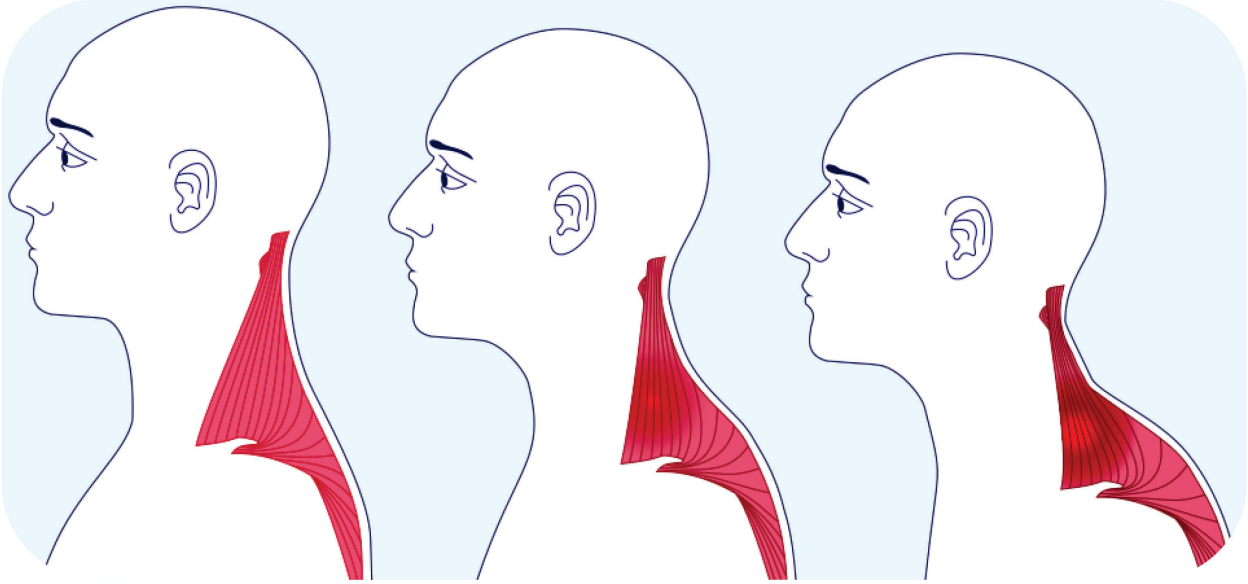


Figure 10: Trigger point formation. *Posture-induced via anterior head carriage and muscle guarding: as the head leans forward, tension on the upper trapezius muscle increases with its fibres hardening in response.*[46,166-168]

Key to this study is the emotional activation of trigger points. Intense emotions sustain muscle tension via "muscle guarding",[160] reducing tension via muscle shortening in the short-term but creating a chronic pain-spasm-pain cycle in the long run (*Figure 10*).[169,170] This tension decreases blood flow and distorts posture, further exacerbating the condition and its associated pain.[78,80,86]

Anatomy

Trigger points are often found near the point of motor innervation in muscle, with excessive nerve activity in — or direct stress or trauma to — this area triggering irritation (*ie*. flexing, stretching, or pressing it).[80] When a series of such hyperirritable loci remain contracted within a muscle, they form a raised, palpable nodule — a trigger point — while pulling the rest of their fibres into a taut band (*Figure 11*). This sustained contraction could be the result of excessive acetylcholine release.[5]

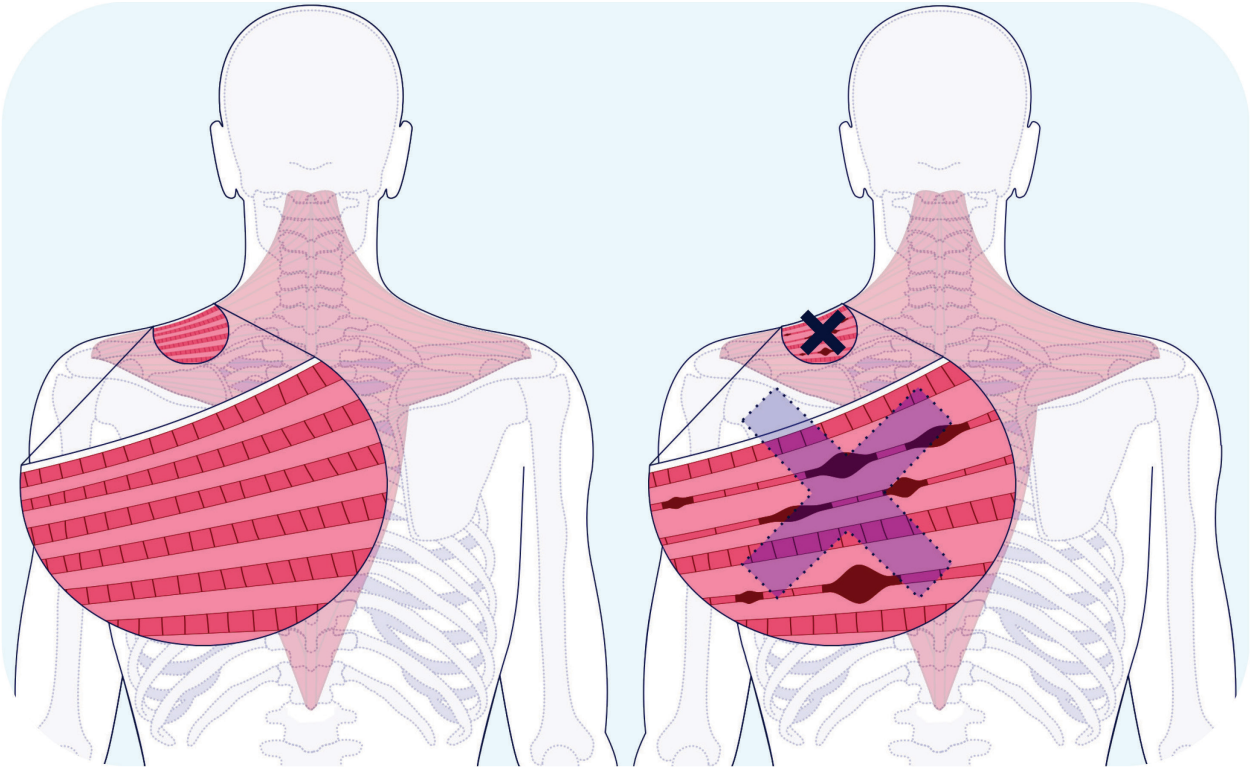


Figure 11: Proposed trigger point anatomy. *Normal (**left**) v. tense (**right**) muscle fibres show the formation of nodules and their associated taut bands.[86] A trigger point is hypothesized to be a concentration of such nodules (**X**).[5,154,171-173]*

The increase in energy consumption this causes, and its associated reduced energy supply via severe local hypoxia due to sustained muscle contraction, creates an energy crisis that further contributes to the trigger point pain cycle.[86,102,174] The point may be painful locally and can elicit predictable referred pain or muscle twitch responses into otherwise unassociated muscles (*Figures 1 & 3*).[77,175] These are distal effects are suspected of being mediated by mechanisms in the spinal cord.[86] Additional symptoms may include limited, painful range of motion as well as weakness in the affected muscle. Further, a trigger point may be active — currently producing pain, or latent — asymptotic until activated (again: flexed, stretched, or pressed).[39,47,53,84,158]

As different from acupuncture points

Due to their similarity, a distinction must be made between trigger and acupuncture points. The first are a muscular pathology and can occur anywhere inside the muscle belly,[164] though usually at large groupings of dysfunctional neuromuscular junctions.[102] Conversely, the second seem to lack any uniform morphology[86] and occur only on predefined lines throughout the body, termed "meridians" or "energy flows". These are based on traditional Chinese medicine and have no clear biomedical basis thus far.[85,160] Interestingly, one study found a 71% overlap between the

two in their distribution and referred pain.[176] Both phenomena also present with local twitch responses as well as possible spinal cord mechanisms,[86] suggesting they share at least some underlying physiological underpinnings.[160,176]

As different from muscle "knots"

In a similar vein, trigger points are not "muscle knots", a very common confusion. Just noting muscle tension is not grounds enough to suspect trigger point presence.[152,153] First, bony prominences (*eg*: ribs, hips) and tendinous muscle attachment sites (*eg*: levator scapulæ) are often mistaken for "knotted" muscle. Secondly, some muscles are naturally stiffer owing to consistent use in posture (*eg*: solei, erector spinæ). Lastly, even when a muscle is tight or hard, it is not always painful itself or referring pain anywhere else. In one study of the trapezius muscle almost 30% of tender sites did not share comorbidity with tissue hardness.[6]

As different from healthy muscle

Lastly, trigger points have been shown to differ biochemically from healthy muscle[177-179] and to undergo thermal[180] and biochemical[181] changes when released. However, a lack of agreement regarding accepted diagnostic criteria has been a serious handicap in both recognizing myofascial trigger point pain as a distinct phenomenon and in studying the topic and comparing treatment effectiveness across different techniques.[39,53,85,157,165,182-184] That said, treatment standardization,[53,185,186] increased clinical experience,[187] and improved technological diagnostic tools[180,188,189] have already started clearing these issues up.

Trigger point treatment

As explained by the gate control model (*Figure 4*), individuals often shake and then rub affected areas immediately following injury.[115] Similar reactions are found in people suffering from trigger points: there is a common tendency to self-massage or stretch the area of referred pain.[158] This is ineffective as the pain's source — the trigger point — is almost always elsewhere. However, compressing this point, once found, is one of the most effective, non-invasive ways of resolving the issue,[84,156,190,191] with its application becoming something of a specialty in itself.[157]

Common protocol includes first massaging the referral site to increase the patient's confidence in the procedure and to harness any placebo effect possible. This is followed by pressing the trigger point, recreating the pain (referred and/or local) and holding the pressure until the pain disappears or diminishes greatly, then "flushing out" the area through general massage. Though

deep finger pressure was advocated early on, current protocol suggests a gentler approach, first focusing on accurately locating and confirming the trigger point and then pressing it gently, going deeper to match the patient's subjective rate of pain relief. This takes into account that trigger points are hypoxic, energy-crisis zones, where adding deeper pressure could potentially exacerbate the problem. Pressure treatment should be followed by a stretch resolving any remaining neuromuscular junction contractures.[102] Slow, regular breathing is an effective method of continuing chronic pain management,[135] especially breathing "into" the trigger point during release.[158] Again, since regions in the medulla are both responsible for breathing (the respiratory centre) as well as for pain modulation (the nucleus raphe magnus),[127,128] activation of the first system could also have effects on the second (*Figure 9*).

Hot and cold therapy

Icing referred pain provides a strong signal along Aδ and C fibres,[192] possibly overriding the sensitized "false" pain signal at the spinal convergence[115] (*Figure 12*). Heat at the trigger point eliminates pain and brings in blood via vasodilation, resolving any energy crises. It also relaxes the area through facilitating release of any unconscious muscle guarding.

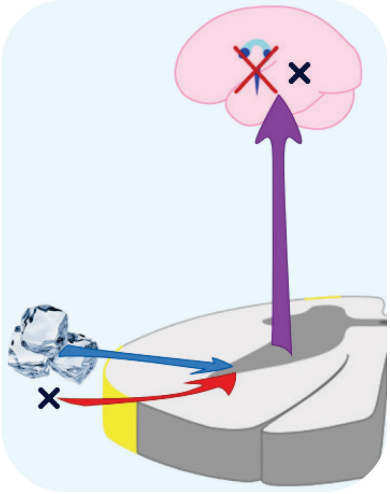


Figure 12: Trigger point therapy and ice. *How cooling may override spinal sensitization.[39,115]*

Homecare includes stretching and applying heat to the trigger point zone to release any remaining adhesions and flush it of any biochemical aftereffects.[158] Flare-ups are dealt with by icing the referred pain zone while applying heat to the trigger point (*Figures 24 & 30*). Relief lasts at least a few weeks following treatment,[186] if not several months or until the next stressor.

Other approaches

Needling has been very successful,[155,193-195] especially if a local twitch response is elicited before the needle is applied.[86,175] It functions through mechanically disrupting the point itself,[156] though it is important to, again, distinguish trigger point needling from acupuncture.[85] A stretch-and-spray technique pioneered by Travell is also effective, stretching the affected muscle while spraying it with a coolant and distracting the patient from the sudden pain.[38,39] Chiropractic cervical spine adjustments have also met with success,[196,197] possibly through unblocking spinal nerve pathways or through the reflexive muscle relaxation process such adjustments naturally

produce.[161] Other treatments include ultrasound,[198-202] transcutaneous electrical nerve stimulation (TENS)[38] and low level laser therapy,[203] various injections,[204] and even mud bath or magnetic field immersion. It is not yet certain how effective these last approaches are.[47,156]

Imagery neuroanatomy

Perception

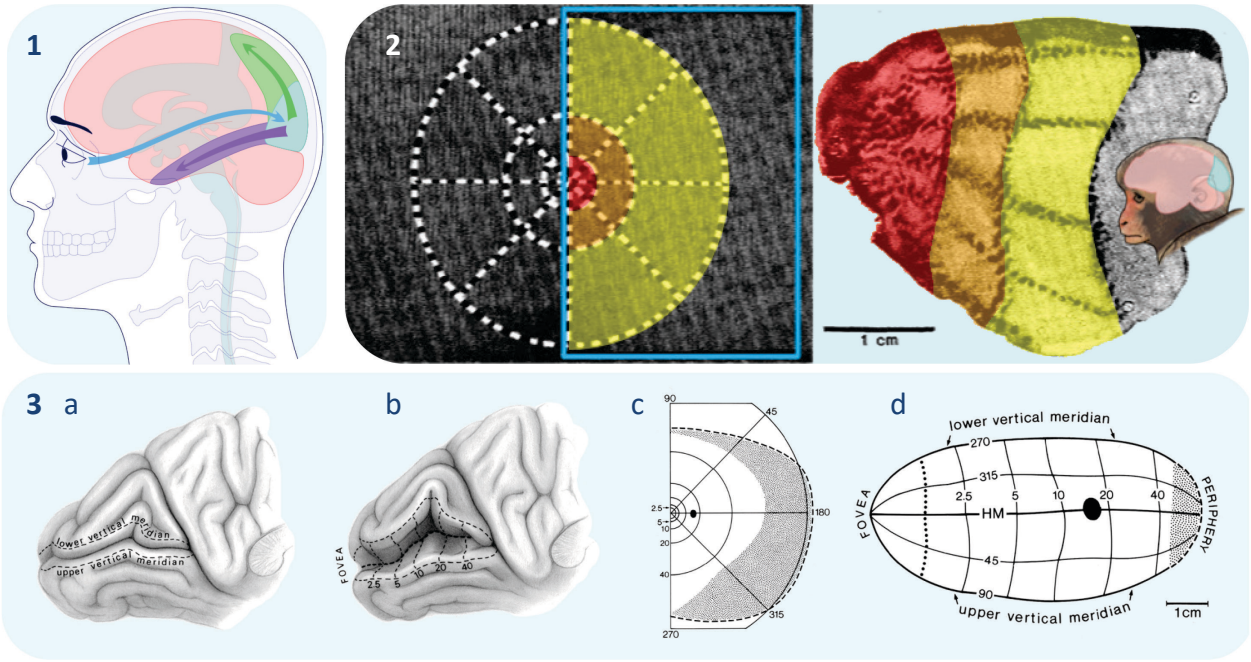


***Figure 13: Perception neuroanatomy. Image 1** shows visual cortex image arrival (**blue**) and dorsoventral splitting into action- (**green**) and description-oriented (**violet**) streams. **Image 2** shows macaque visual cortex retinotopic organization its field of vision (**box**) and corresponding cortex activity (**slice**, from **blue** section of macaque head inset).[205,206] **Image 3** shows the corresponding human anatomy (**a-b**), visual field (**c**), and visual cortical map (**d**).[207]*

To understand imagery, it helps to first understand perception. When eyes see, neural signals reach the brain's posterior visual centre, retinotopically mapping the stimulus to resemble the visual field.[205] This is seen with eerie accuracy in a macaque's occipitocerebral energy (glucose) consumption in its visual cortex matching its visual field (*Figure 13.2*). Compartmentalization continues as the brain processes an image, passing signals further ventrally and dorsally to memory, emotion, and sensation centres, making sense of what is being seen.

Imagery

Imagery differs from perception in that it recreates the same without visual input,[208,209] bridging high-level information (thoughts, emotions, memories) with low-level sensations.[210] All this

information is processed in occipital visual centres,[209,211-224] then emerging through two major nerve bundles: the superior and inferior longitudinal fasciculi. The first carry spatial, action-oriented details dorsally through the parietal and frontal lobes (*eg*: where an object is, how to use it), while the second carry description-oriented object details ventrally and into the temporal lobe (*ie*. what an object is).[209,225-229]

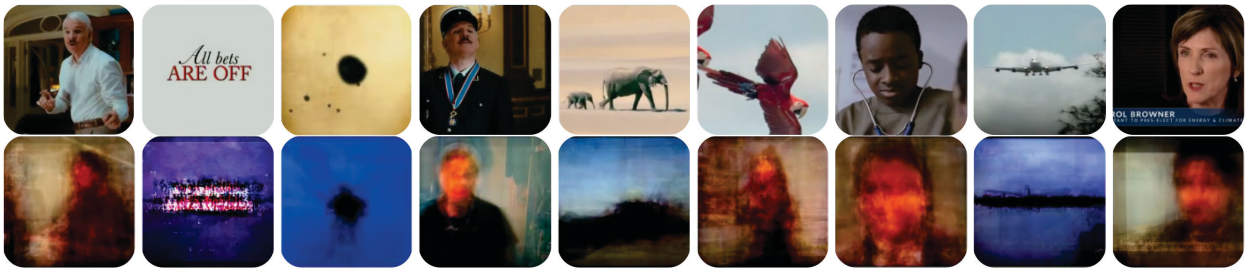
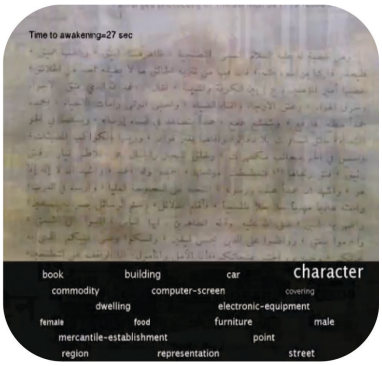


Figure 14: Decoding brain imagery. *Video snippets seen (**top**) and then reconstructed via fMRI (**bottom**).*[230,231]

Certain objects or details have distinct representations along these streams, with their imagery evoking very clear neural signatures. Surfaces, faces, outdoor scenes, houses, chairs, animals, and tools all light up unique patterns along the ventral stream,[209,232] with characteristics like image vividness acting to further distinguish the signature.[233] Recent studies have even been able to decode what a person is seeing or imagining through retinotopic reconstruction of ventral stream activity (*Figures 13.3 & 14*),[230,234-237] and are now decoding even technique now decoding even dreams (*Figure 15*).[231,235,238-241]



***Left**: "… There was something like a writing paper for composing an essay, and I was looking at the characters from the essay…"*

***Left**: "… there were persons, about 3 persons, inside some sort of hall. There was a male, a female, and maybe like a child… a boy, a girl, and mother…"*

Figure 15: Decoding dreams. *Video stills of reconstructed dreams and descriptor keywords chosen by the computer, alongside participant descriptions of their dreams immediately upon awakening.*[238,239,242]

Virtually all pathways traveling between visual areas are bidirectional.[215,243,244] Parieto- and temporo-occipital regions are especially active during imagery,[211,219,245-251] with regions in the frontal and parietal cortex activated during both imagery and perception,[252-254] particularly in the left precuneus[209,255] and other structures in the left hemisphere nearby,[254,256-258] underlining their importance in creating and maintaining mental images.[209] Perception also receives input from memory[259] so imagery may have some ties to temporal memory areas,[209,218,254,260,261] providing a reason for this bidirectionality (*Figure 16*).

However, the phenomena differ in key ways[262] (*eg*: perception activates posteroanteriorly along the ventral stream while imagery does the reverse,[234] suppressing primary sensory regions[263] and hinting at memory input).

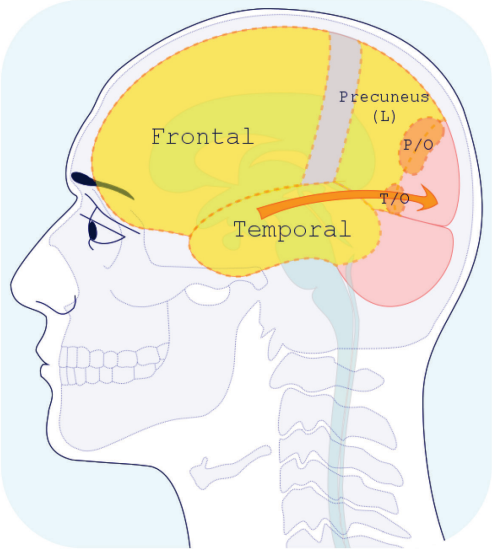


Figure 16: Imagery neuroanatomy. *Areas associated with imagery forming and storage (**yellow**, including left precuneus), as well as parieto- and temporo-occipital regions and the reverse ventral stream (**orange**) highlighted.*

Imagery in rehabilitation

Past literature has showed imagery's effectiveness in improving conditions as varied as cancer,[264] psoriasis,[265] stress,[266] ulcers, paraplegia, fractures, hip disarticulations, intraabdominal lesions,[267] phantom limb pain,[268] and chronic pain.[269] It has also been successful in injury rehabilitation, resulting both in faster recovery times[270] as well as in improved pain management associated with healing.[59,267,271-275] It is unsurprising therefore, that many imagery scripts have been developed over the years to treat a wide array of conditions, often emerging from the fields of psychiatry and psychotherapy.[63] In the case of injury, imagery scripts are most effective when they are clear, accurate, strong, positive, and meaningful to the patient, and if the patient understands and can accurately imagine the entire healing process (*eg*: anatomy, treatment modality, end goal).[59] Such detailed imagery requires the patient to be relaxed, facilitating enhanced mental reception and muscle relaxation. Only then can proper healing take place.[270]

Injury imagery has three main focuses: pleasant imagining (comfort-inducing, reducing sympathetic nervous activity and muscle tension, useful especially in early stages of rehabilitation), pain acknowledgement (assigning pain physical properties for later mental manipulation) and dramatized coping (reframing, where pain is now part of the challenge of goal

achievement). Injury imagery may also take one of two directions: associative or dissociative — focusing "towards" or "away from" the pain. Dissociative imagery tends to be more effective, though associative approaches are useful in reducing pain while providing the athlete with a sense of control over their condition.[59] Lastly, imagery may be motivational (energizing), cognitive (helping first plan and then practice a technique or strategy),[276] or healing (visualizing positive, healthy physiological processes or imaging oneself in a state of health).[273,277] Initially, motivational imagery was suspected as being used more often in athletic rehabilitation, particularly with elite athletes.[278] A follow-up study, however, showed athletes in rehabilitation using all forms of imagery mentioned, only in lower amounts than those in training or competition.[7,277] Healing imagery on its own was found to increase athletes' satisfaction in the rehabilitation process, though without decreasing the time required to return to sport.[279] Its application generally lapsed in the beginning until a few days post-injury because athletes needed to let their minds clear of negativity. Healing and cognitive (but not motivational) imagery was then generally applied until the injury had healed.[280,281]

The PETTLEP model

One effective approach for rich, relevant imagery is the PETTLEP model,[282] traditionally used for motor imagery (*ie*. the mental rehearsal of an action). This was one of the first approaches backed by neuroimaging technology and providing a concrete, comprehensive method for systematically enriching imagery. It states that imagery ought to be **physical** (*eg*: done while wearing a game-day uniform, or in the arena where the imagined performance will take place), take place in the same **environment** as the upcoming event, and revolve around details relevant to the **task** in real-**time** (*eg*: appropriate skill level, matching personal preferences). It should also be re-**learned** as needed and be packed with the same **emotions** felt as when performing the task imagined. Lastly, it can take one of two **perspectives**: first-person is recommended for open-skilled tasks with a focus on timing (*eg*: karate) while third-person is recommended for aesthetic tasks where form and positioning are important (*eg*: surfing). Such rich imagery results in fewer distractions, though, should these hit, the patient should focus first on re-establishing imagery through the senses they respond to most easily (generally visual), which quickly sweeps in with it other, less-used senses. Looking at images of natural settings beforehand clears the mind well, and for those who tend to verbalize what they see, it is recommended to spend a few minutes before treatment letting their gaze wander the room, consciously not putting a label to anything spotted.[63]

Physiological effects of imagery

Other studies have also shown imagery to change the body's physiology, from causing similar brain activity as performing the imagined motion would,[283] to raising heart and respiration rates,[284] to increasing muscle strength,[285-287] activation,[288,289] and relaxation — this last both at the motor neuron level[290] as well as via fatigue induced through depleted self-control.[291] It has also been found that personalised, emotion-laden imagery scripts result in increased muscle activity, likely because of the increased image vividness brought about with this approach.[292] Of specific importance to this study, imagery has been helpful in resolving most major classifications of headaches,[293,294] with hand-warming biofeedback exercises showing particular effectiveness and widespread use.[295-301] Other studies showed that imagery can be used to increase skin temperature,[302] of which a key one showed that focusing skin-warming imagery to trigger point sites — in combination with implementing a general relaxation and thermal biofeedback training program — increased skin temperature and muscle relaxation at these sites while also decreasing subjects' overall pain sensitivity.[303] A potential explanatory mechanism for all these physiological changes is that imagery eliminates the stressful, destructive, vasoconstriction-causing mental images.[304]

Methods

Population

Forty participants, 11 male and 29 female, were recruited from Gdańsk University and Gdańsk Sport University on the Baltic coast in northern Poland. Thirty were students or their friends and ten were professors or their friends, aged 19-51 (M = 26.13, SD = 8.05). There were no exclusion criteria. Testing took place in Polish at Gdańsk Sport University's sport psychology office with a massage table from the physiotherapy department and with the Smart Voice Recorder app on an LG Nexus 4 cell phone recording audio throughout treatment. Only the therapist and the current study participant were in the room during testing.

Groups and scripts assigned

On entering treatment participants freely consented to participate in the study (*Appendix*), were then randomly assigned into control or imagery groups, and then were read the treatment protocol, trigger point description, rating measures,[305] and their group-specific script. The control group (n = 20) was given a breathing-focused relaxation script, below, then had it demonstrated and tried it prior to treatment (*Appendix*):

> *"Concentrate on breathing, first filling up your stomach, then your ribs from the sides, and finally your chest. Exhale in the reverse order. Breathe in with your nose and out with your mouth. Breathe slowly. If you lose focus, try to return to it, concentrating on breathing."*

Meanwhile, the experimental group was given an associative, pleasant, PETTLEP-based imagery script infusing warm feelings of melting into the trigger point (*Appendix*). Elements not usually associated with imagery were emphasized in the script, as follows:

> *"Imagine that this tight knot is a ball of butter, melting on the beach under a hot, afternoon sun. Use all your senses: smell (salty air), sight (sand, gold and blue, water),* ***sound*** *(surf, seagulls),* ***touch*** *(grains of sand, warm wind, dripping sweat),* ***taste*** *(salty water, sweat, butter),* ***movement*** *(the butter melting, waves),* ***temperature*** *(hot, nice), and* ***emotions*** *(melting, relaxation). Ensure your image is physically accurate (sizes, based in reality), that the focus is the ball of butter, that time is moving normally (not too fast, not too slow), and perspective (that the muscle knot actually is this ball of butter). If you get sidetracked, try to return to concentrating on this image."*

Positioning and treatment

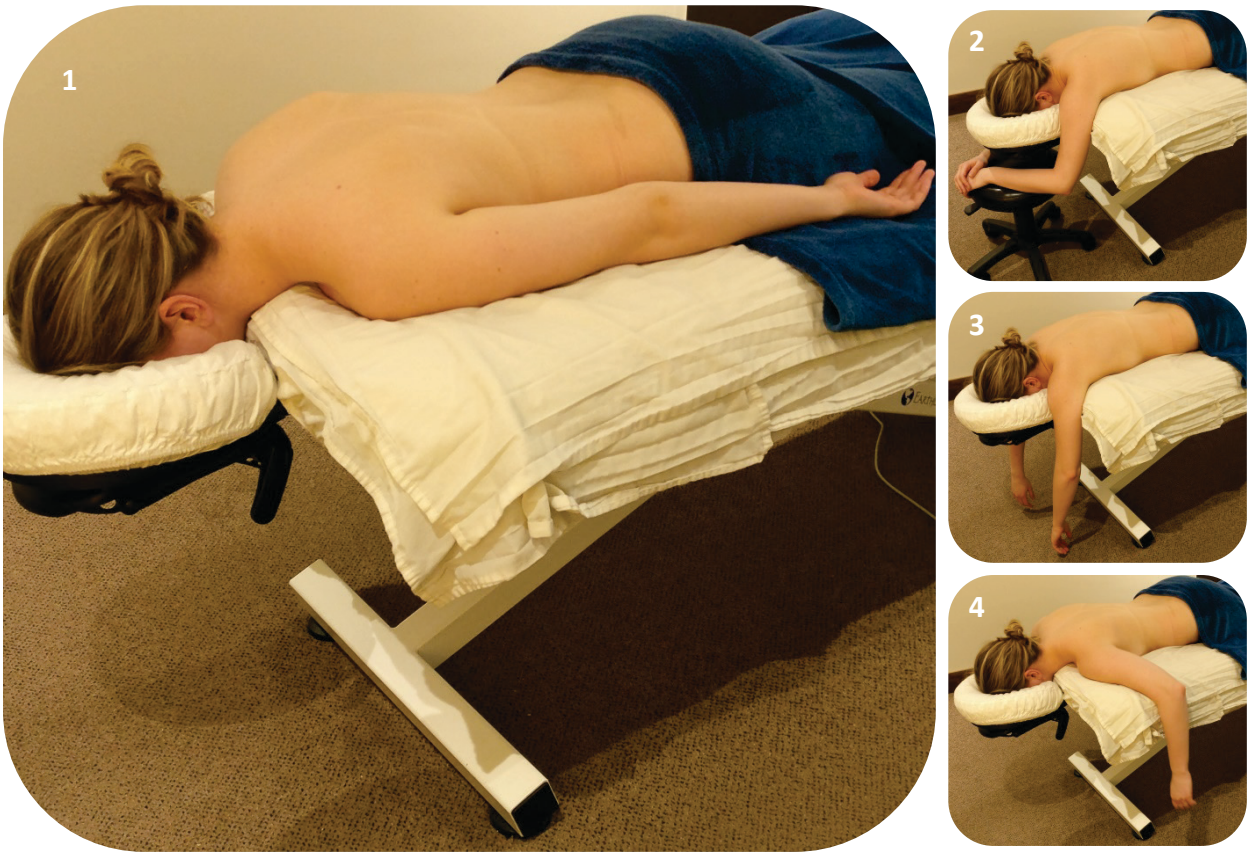


Figure 17: Participant positioning. Image 1 *shows an upper trapezius positioned for best access during treatment.*

The therapist would then leave to wash his hands, letting the participant disrobe and lay prone on the massage table, arms comfortably at their waist (*Figure 17.1*). This position maximally stretches the upper trapezius muscle, facilitating trigger point targeting and simultaneously encouraging muscle relaxation (as compared to their laying with shoulders near the head or arms hanging off the table's sides, *Figure 17.2-4*). Treatment would then begin.

The therapist first warmed the upper trapezius up with general Swedish massage techniques (*eg*: effleurage, petrissage) for about 30s before palpating for a trigger point for another 30s. Due to the lack of consensus defining a trigger point in the literature, and to their being several symptoms possibly suggesting its presence, a trigger point in this study was defined simply as "a tender point", with a concerted effort made to create referred pain (n = 21).[86] Once found, the therapist would sink into the point using a point-press technique with his thumb, fingertips, knuckles (*Figure 18.1-4*), or pisiform wrist bone (*Figure 18.5*), striving to create a standard experience across all presses and participants differing only in pressure. He would sometimes move to the more intense pincer-press technique when it was clear the muscle would not slip from under his fingers (*Figure 18.6*). If the participant showed no pain at all or had particularly tense upper back musculature, the therapist would use his elbow, sometimes pulling the edge of the table in with his free hand to increase pressure (*Figure 18.7-8*). This was a rare occurrence and not systematically recorded. Minimal lotion was used to avoid slipping.

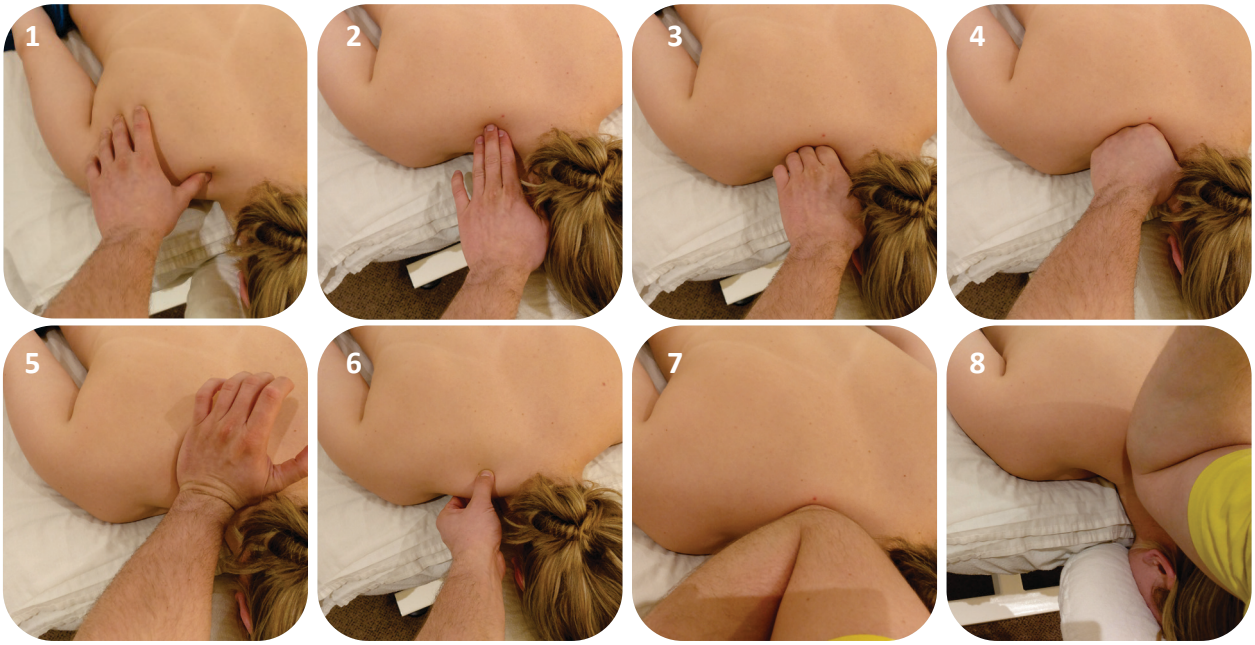


Figure 18: Various point-press techniques: *starting with the most standard to those delivering more pressure.*

The upper trapezius trigger point was chosen because it is easy to locate and treat, and because it is the most prevalent trigger point in humans.[39] The dominant arm was always treated (36 R and 4 L). The therapist would locate and press on the trigger point, stopping when the participant reported pain at a level of 7/10 on the predetermined pain scale, or "just before it became unbearable". Pressure would then be sustained and pain would slowly ease off, usually after 30-60 s. The participant would tell the therapist when pain dropped to 0-1, at which point the therapist would press deeper into the same spot until a 7 was reached once again. A total of four presses was applied, each building on the depth of the previous one. If the trigger point released completely, such that further presses evoked "pressure, but no pain", it was said to have been released and testing complete, with all further presses being automatically assigned a value of "0 seconds" (n = 3). Otherwise the protocol was continued but not beyond four presses to avoid soft tissue injury (n = 37). After testing, the participant received 15-30 minutes' free massage therapy in appreciation for their participation.

Data collection

All audio during treatments was recorded so revisiting and finding the e "0-1" and "7" time points was possible for analysis. This was done using Power Sound Editor Free on a Samsung Series 5 laptop running Windows 8.0. Finally, participants were given a post-treatment manipulation check questionnaire with six questions on trigger point symptoms, pain tolerance, and focus ability (*Appendix*):

1. *What was your most intense pain ever, and how would you rate it (0 = nil, 10 = max)?*
2. *How painful was the full trigger point treatment, from start to finish (0 = nil, 10 = max)?*
3. *What were you concentrating on exactly during treatment?*
4. *To what degree were you focused on your image/breath (0 = nil, 10 = max)?*
5. *Did you lose your concentration at all? If so, how many times?*
6. *Where did you feel the pain in your body?*

Results

Main findings

Independent-samples t-tests comparing time for pain resolution across all presses, mean press time, total treatment time, distractibility, focus ability, pain tolerance, and treatment pain between breathing and imagery conditions. The only significant result observed was the breathing group's doubled pain tolerance (M = 1/10, SD = 1.2) to that of the imagery group (M = 2/10, SD = 1.8); $t(37) = 2.10$, $p = .043$ (highlighted in *Table 1*).

Table 1: Result summary. *Group differences in pain resolution, focus ability, distractibility, and pain tolerance.*

	Breathing			Imagery			Δ		t	*p*
	M	SD	*n*	M	SD	*n*	M	SD		
Press 1 (s)	32.2	21.1	20	29.8	14.0	20	2.4	7.1	(38) = 0.42	.674
Press 2 (s)	22.3	17.9	20	25.8	16.8	20	3.5	1.1	(38) = -0.63	.533
Press 3 (s)	32.1	37.9	20	22.6	14.6	20	9.5	23.3	(38) = 1.05	.303
Press 4 (s)	33.5	41.0	20	24.4	22.1	20	9.1	18.9	(38) = 0.87	.388
Press M $_{1\text{-}4}$ (s)	30.0	27.3	20	25.6	15.5	20	4.4	11.8	(38) = 0.62	.540
Σ *Treatment time* (m)	2:36	2:04	20	2:15	1:08	20	0:21	0:56	(38) = 0.67	.507
Focus ability (/10)	7.5	1.8	20	7.1	2.4	20	0.4	0.7	(38) = 0.56	.580
Distractibility (/10)	2.4	2.5	20	3.4	3.0	20	1.0	0.4	(38) = -1.12	.270
Pain Tolerance (/10)	2.0	1.8	20	1.0	1.2	19	1.0	0.6	(37) = -2.10	.043
Treatment Pain (/10)	5.8	1.6	18	5.0	2.4	17	0.9	0.9	(33) = 1.09	.283

Still, pain resolution diverged between the two groups with subsequent presses, rendering results 27% faster with imagery than with breathing by the final press (*Figure 19*).

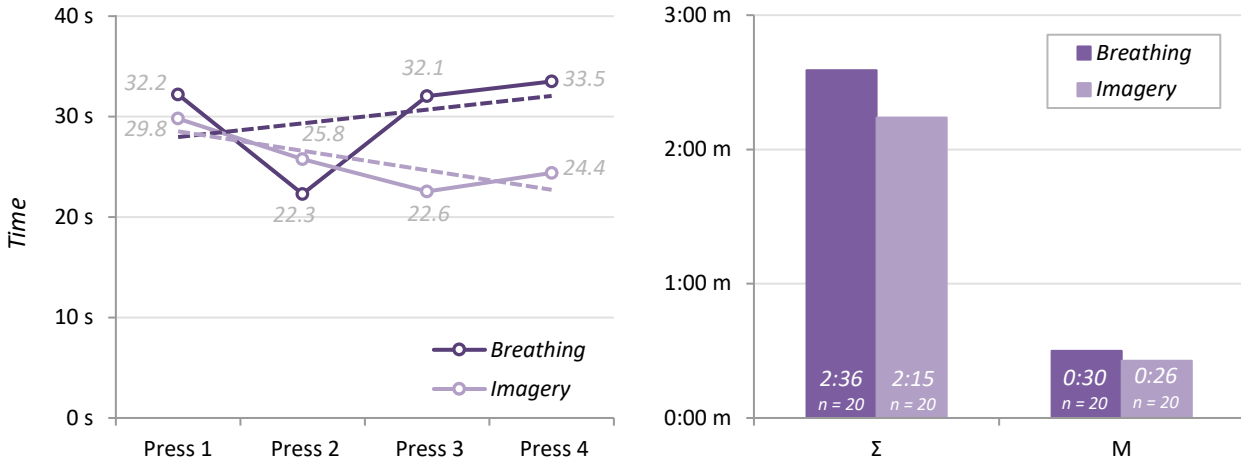


Figure 19: Main results. *Treatment times across presses (**left**), and total treatment and mean press times (**right**).*

Referred versus local pain

Factorial ANOVAs comparing breathing *v.* imagery treatments between subjects experiencing only local pain (OLP) *v.* referred pain (with or without local pain, RP) showed significant main effects of treatment on pain tolerance, $F_{1,29} = 4.39$, $p = .045$ (*Table 1*), significant main effects of pain on the first press, $F_{1,30} = 4.53$, $p = .042$ (*Table 2*), as well as significant interaction effects of both pain and treatment on total treatment time, $F_{1,30} = 4.64$, $p = .039$ (*Table 2*).

Table 2: Results summary: referred v. local pain groups. *Significant results and preferred treatments highlighted.*

	Only Local Pain *(n = 19)*								Referred (±Local) Pain *(n = 21)*							
	Breathing			Imagery			Δ		Breathing			Imagery			Δ	
	M	SD	*n*	M	SD	*n*	M	SD	M	SD	*n*	M	SD	*n*	M	SD
Press 1 (s)	43.2	24.8	*9*	31.5	16.1	*10*	11.7	8.8	23.2	12.5	*11*	28.1	12.2	*10*	4.9	0.3
Press 2 (s)	31.3	21.5	*9*	25.7	19.0	*10*	5.6	2.6	14.9	10.2	*11*	25.8	15.3	*10*	10.9	5.1
Press 3 (s)	48.4	50.3	*9*	21.6	16.5	*10*	26.8	33.8	18.6	16.4	*11*	23.5	13.3	*10*	4.9	3.2
Press 4 (s)	46.8	46.2	*9*	22.3	25.2	*10*	24.5	21.0	22.6	34.7	*11*	26.5	19.7	*10*	3.9	14.9
Press M $_{1\text{-}4}$ (s)	42.4	33.5	*9*	25.3	18.0	*10*	17.2	15.5	19.8	16.3	*11*	26.0	13.6	*10*	6.1	2.7
Σ_t (m)	3:32	2:34	*9*	2:07	1:12	*10*	1:25	1:22	1:50	1:11	*11*	2:22	1:08	*10*	32.5	3.1
Focus (/10)	7.6	0.8	*9*	7.0	2.5	*10*	0.6	1.7	7.4	2.3	*11*	7.2	2.5	*10*	-0.2	0.2
Distract. (/10)	2.5	1.5	*9*	3.7	3.2	*10*	1.2	1.8	2.3	3.2	*11*	3.0	2.8	*10*	0.7	0.4
P. Tol. (/10)	8.1	1.6	*9*	9.2	1.3	*10*	1.1	0.3	7.8	2.0	*11*	8.7	1.0	*9*	0.9	1.0
Tx. Pain (/10)	6.2	1.6	*9*	4.5	2.5	*10*	1.8	0.8	5.3	1.5	*9*	5.9	2.3	*7*	0.5	0.8

This last interaction effect, though statistically insignificant but for total treatment time, played a large role in the study. Patients with only local pain experienced relief almost twice as quickly with imagery while those with referred pain did so a fifth faster focusing on breath (*Figure 20*).

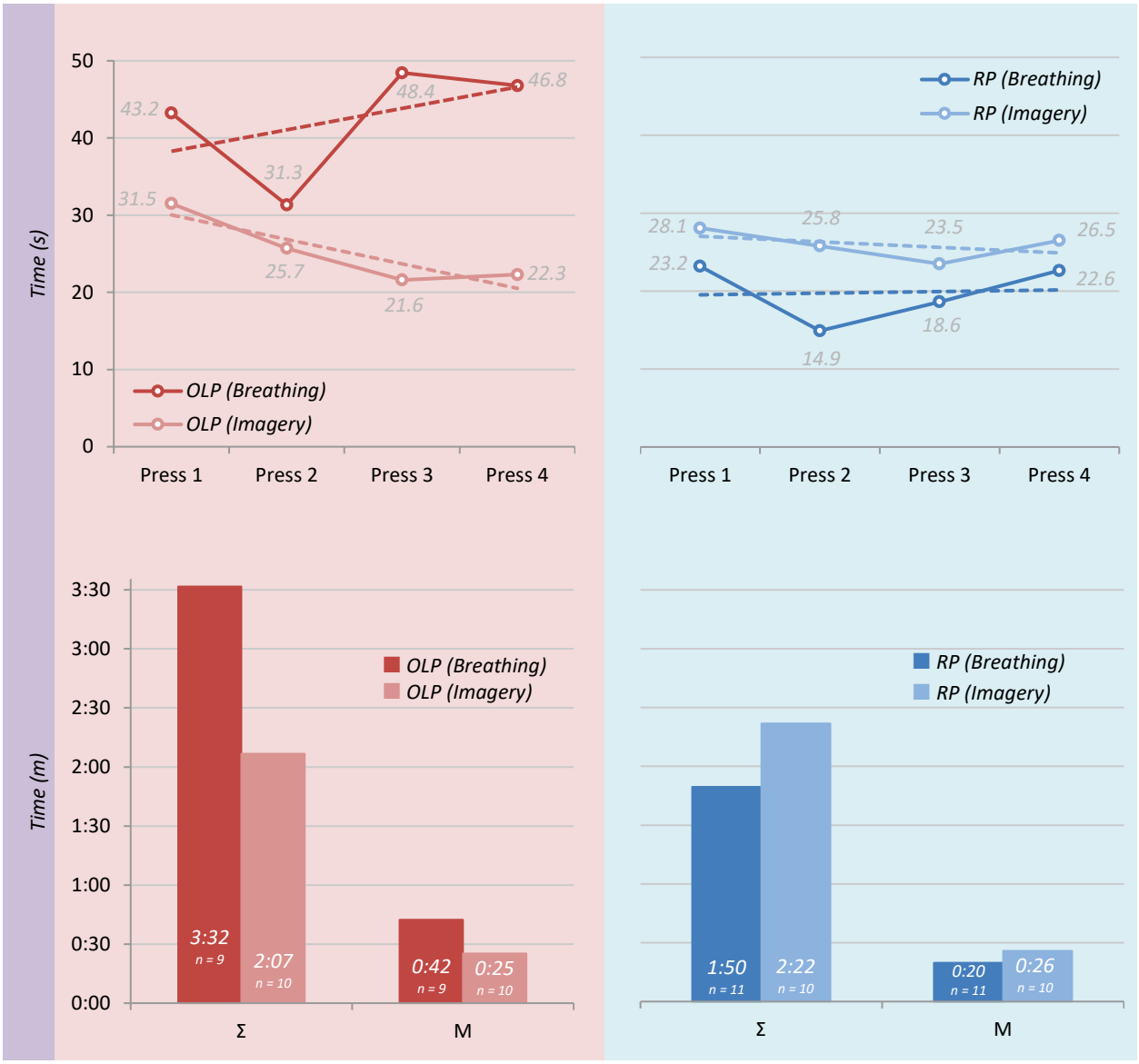


Figure 20: Treatment efficacy: referred versus local pain. ***Figure 19*** *parsed into OLP (**left**) and RP (**right**) results.*

Individual variance

Individual variance was high and increased throughout treatment, as seen in standard deviation differences between the two groups. The imagery group generally saw smaller deviations in treatment, especially throughout the first and third presses (*Table 1* & *Figure 21*).

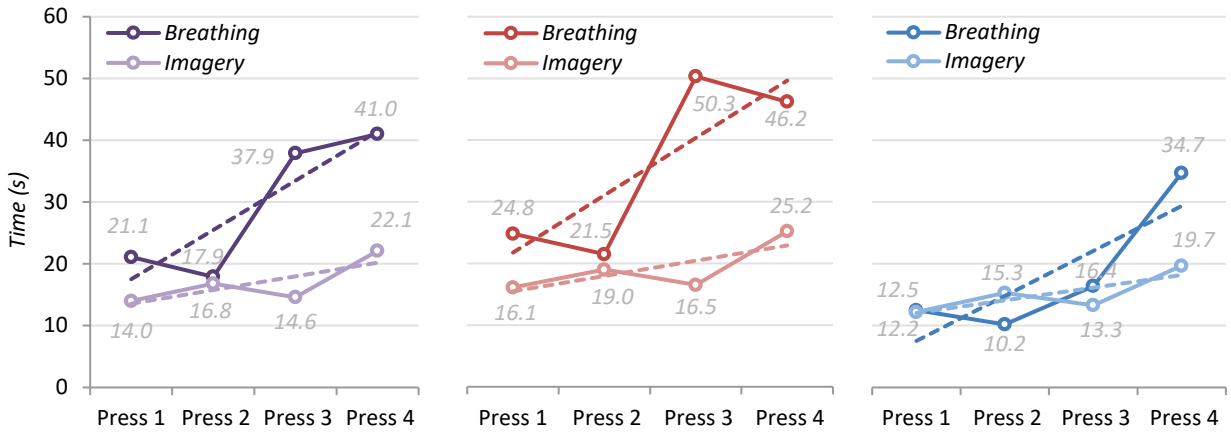


Figure 21: Individual variance. *Press time standard deviations in all (**left**), OLP (**centre**) and RP (**right**) participants.*

Focus ability and distractibility

Data was collected on participants' ability to focus through the question *"To what degree were you focused on your image/breath (0 = nil, 10 = max)?"* (n = 40) and on their distractibility with "*Did you lose concentration? If so, how many times?*" (n = 40) (*Table 2*). Where distraction was so high that a participant was unable to assign it a number, a "10" was automatically assigned (n = 3: one control and two imagery, *Table 5*). Imagery intervention participants were more easily distracted and less able to focus, independent of their pain location (*Figure 22*).

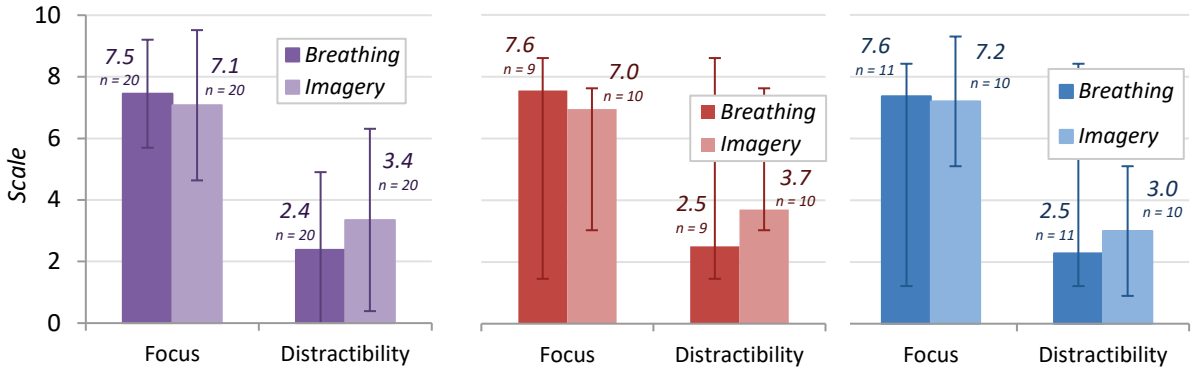


Figure 22: Focus and distractibility. *All (**left**), OLP (**centre**) and RP (**right**) participants.*

Of the 20 breathing group participants, 14 focused on breathing: five on its process (*eg*: proper form, counting rhythm), and one also on talking with the therapist. Two of these 14 also focused on "spreading" the treatment through their bodies. Of the remaining six, five focused just on the pain (*eg*: defining its location) and one on the therapist's voice and hand warmth. The last participant focused just on treatment location, *ie*. on her neck (*Table 3*).

Table 3: Participant focus throughout treatment. *Groups divided into* ***focused*** *and* ***distracted*** *participants.*

Participants	*Focused*	*Distracted*
Breathing	14 breathing *(inc. 5 process (inc. 1 pain), 1 therapist, 1 pain)*	5 pain *(inc. 1 therapist)*, 1 treatment
Imagery	16 imagery *(inc. 11 rich, 2 environment, 1 pain)*	3 pain, 1 treatment

Of the 20 imagery participants, 16 focused on imagery, 11 reporting imagery including more than just butter (*eg*: "hearing seagulls", "focusing on the act of melting", *etc.*) and two not seeing butter at all but rather a seaside environment ("ocean waves", "the sun"). Only one participant of these 16 also focused on the pain being felt. Of the four unable to focus on imagery, three focused instead on treatment pain while the remaining participant focused on neither pain nor imagery but on the relaxing treatment environment (*Table 3*).

Pain tolerance

Incomplete data was collected on general pain tolerance through asking "*What was your most intense pain ever and how would you rate it (0 = nil, 10 = max)?*" (n = 39) and on treatment pain through asking "*How painful was the full trigger point treatment from start to finish (0 = nil, 10 = max)?*" (n = 35). Pain tolerance was estimated by inverting the first scores while scores from the second remained as recorded to gauge pain felt during treatment. (*Figure 23*).

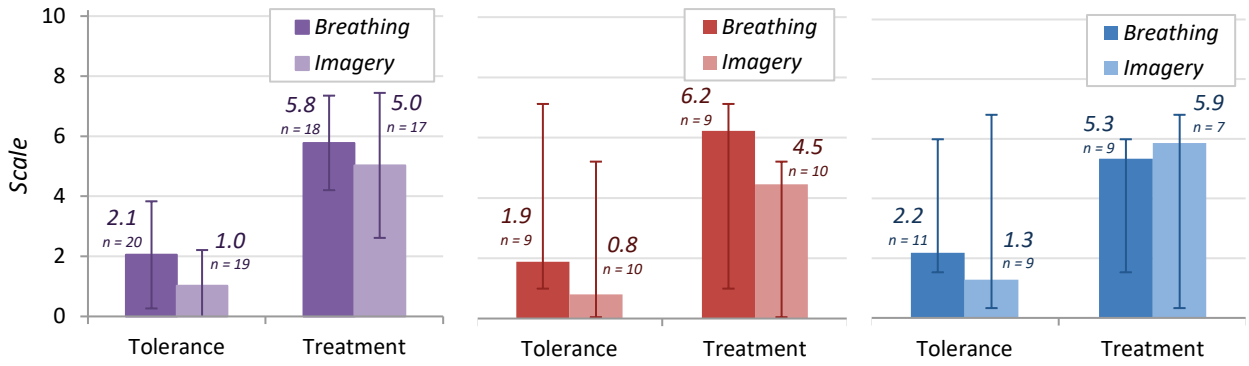


Figure 23: Pain tolerance. *Pain tolerance and treatment pain in all (**left**), OLP (**centre**) and RP (**right**) participants.*

Pain tolerance was about twice as strong in imagery versus breathing interventions, irrespective of the pain being felt locally or as a referral. In-treatment pain hovered around mid-range, with the only-local-pain group experiencing less pain with imagery and the referred-pain group experiencing a little less pain when focusing on breathing. It is important to note these subjective measures were obtained *after* treatment.

Trigger point symptoms

Trigger points were identified primarily through referred pain patterns. Of the forty participants, nineteen felt pain only locally at the press site. Eleven felt only referred pain: ten only in their mastoid process and one partway up her neck in the beginnings of the classic, "question mark" pattern. The last ten felt both local and referred pain in at least their mastoid process, with three feeling parts of the question mark emerging: the first in her temple, the second in her neck, and the third in both (*Table 4*). Nodules, taut bands, muscle twitching, and profuse sweating were only noticed later in some participants and so were not systematically recorded.

Table 4: Trigger point pain distribution among subjects. *Each subject counted only once. "Question mark" refers to any pain across the classic pattern circling the ear as in* ***Figure 1****.*

Pain	no referred pain	+ only mastoid process referral	+ "question mark" referral
Local	19	7	3
Referred	-	10	1

Exceptional treatments

Three participants experienced full trigger point release before reaching their fourth press, receiving scores of "0 seconds" for remaining presses, and three required much more time for treatment. All but one felt just local pain, that one feeling pain in his mastoid process (*Table 5*).

Table 5: Exceptional treatments. *Average (**grey**) versus above- (**pink**) and below-average (**blue**) results, including general pain tolerance, treatment pain, and pain location (only local, only referred, or both).*

Group	Sex *(F:M)*	Age	Press *(m:ss)*						Focus ability	Distract-ibility	Pain		
			1	2	3	4	M	Σ			Tol.	TrP	Location *(OL:OR:B)*
Avg Total	29:11	26.54	0:31	0:24	0:27	0:29	0:28	2:25	7.3	2.9	8.5	5.4	19:11:10
Avg Imagery	12:8	25.96	0:32	0:22	0:32	0:34	0:20	2:36	7.1	3.4	9.0	5.0	10:5:5
Avg Breathing	17:3	27.12	0:30	0:26	0:23	0:24	0:26	2:15	7.5	2.4	8.0	5.8	9:6:5
Breathing	M	20	0:09	0:07	0:10	-	0:07	0:43	9	0	5	4	Ref: MP
Imagery	F	38	0:34	0:17	0:08	-	0:15	1:28	0	10	10	3	Only Local
Imagery	F	21	0:11	0:09	-	-	0:05	0:28	8	0	10	4	Only Local
Breathing	F	29	0:38	1:02	2:22	2:20	1:36	7:19	8	2	10	7	Only Local
Breathing	M	26	1:26	1:06	1:49	1:31	1:28	7:03	8	2	6	3	Only Local
Breathing	F	51	1:26	0:47	1:23	1:15	1:13	6:16	6.5	3.5	6.5	7	Only Local

Discussion

Summarizing ascending modulation: referred pain and massage

Proposed explanations of ascending trigger point pain modulation are outlined below in the same order of typical experience before, during, and in follow-up care after massage therapy.

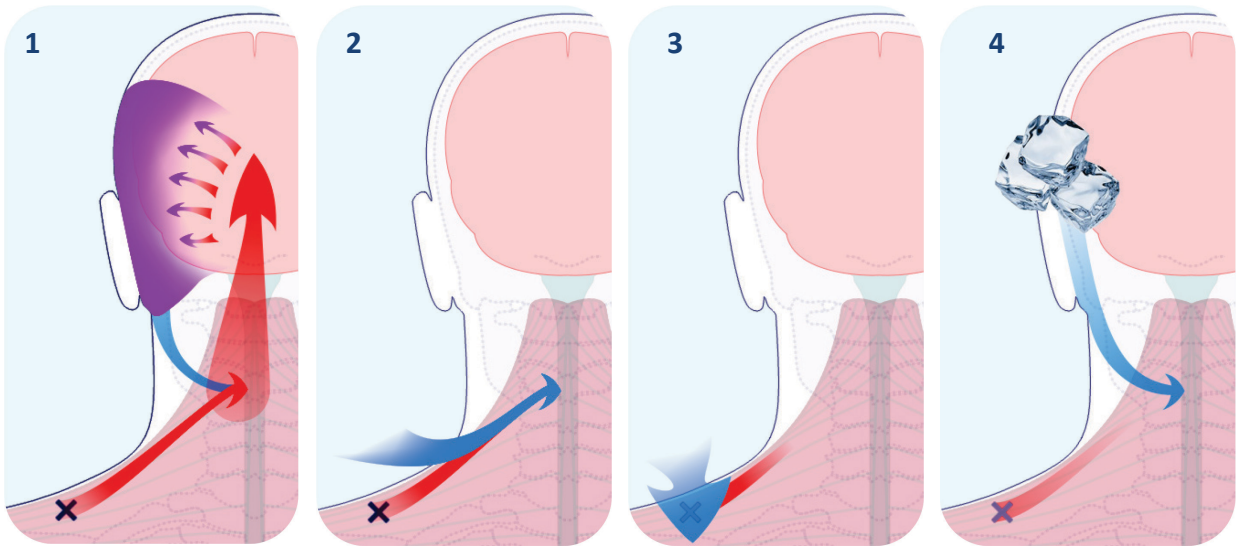


Figure 24: Ascending modulation: *trigger point (**X**), primary pain (**red**), referred pain (**purple**), and modulation (**blue**).*

A tender point sends a painful signal to the brain, sensitizing an otherwise unaffected nerve and creating a brain-mediated illusion of pain in its associated referral area (*Figure 24.1*). Massage initiates immediate gate control suppression of this signal, leading to muscle healing and eventual complete trigger point resolution (*Figure 24.2-3*). Finally, applying ice to the site of referred pain overrides any residual signals from the now-resolved trigger point (*Figure 24.4*).

Summarizing descending modulation: pain in the brain

Cerebral pain modulation is a particularly comprehensive, many-faceted process only starting to be explored in the neuromatrix model.[16,72,74,75,104] In reviewing the literature for this paper many key elements of this process began to converge, the synthesis of which follows.

Ascending pain is modified by analgesic tracts from the brainstem's descending periaqueductal grey matter[125,126] and rostroventral nucleus raphe magnus, which modulates pain based on fear, attention, and expectation[19,123,127,128,306] (*Figure 25.2: purple tract*). The retrotrapezoid nucleus near the latter holds a breathing centre,[129-131] hinting that focused breath may lighten perceived pain[132-135] via Hebb's rule: "cells that fire together wire together".[307,308] The physical information in the signal like the pain's location and intensity then travels to the somatosensory cortex via the thalamus, a hub for almost all information travelling to the cortex[124] (*Figure 25.2: red tract*), while a second pathway enters the cingulate and insular cortices via the pons and amygdala, modulating pain through emotion (*Figure 25.2: yellow tract*). The amygdala's firing rate increases with fear[136] while the insular cortex identifies the magnitude of the pain[122] and is more active with visual input,[137] even if only imagined (as in the anterior cingulate cortex).[136]

Creating a similar model for imagery/perception is now useful. A visual signal splits into dorsal "spatial" and ventral "description" streams, passing through many of the structures mentioned earlier from the eyes to the occipital visual centre. Information on interacting with an object travels through the dorsal visual stream toward the somatosensory cortex, itself receiving location and intensity information from the pain signal. Information on what an object is travels through the ventral visual stream into the temporal lobe, near where pain's emotional layer is determined (and where long-term visual associative memories are processed and stored).[309] Hebb's rule may apply again in the resulting signal overlap (*Figure 25.3-4*).

The beginnings of a cerebral imagery map are presented in *Figure 25.4*. Interesting points to note are the general activity in bilateral temporal lobes, the parieto- and temporo-occipital regions (near the ventral visual stream source), and the left precuneus. The main suggestion here is that the reverse ventral stream at the heart of imagery is passing near — if not through — key structures of pain modulation: the amygdala, the insular cortex, and even perhaps the brainstem. This hazy concept is where further research is needed: in determining the neurosignature of imagery and its effect on that of pain, as well as all the brain structures involved (particularly the contributions of higher-order functions located in the frontal lobe).

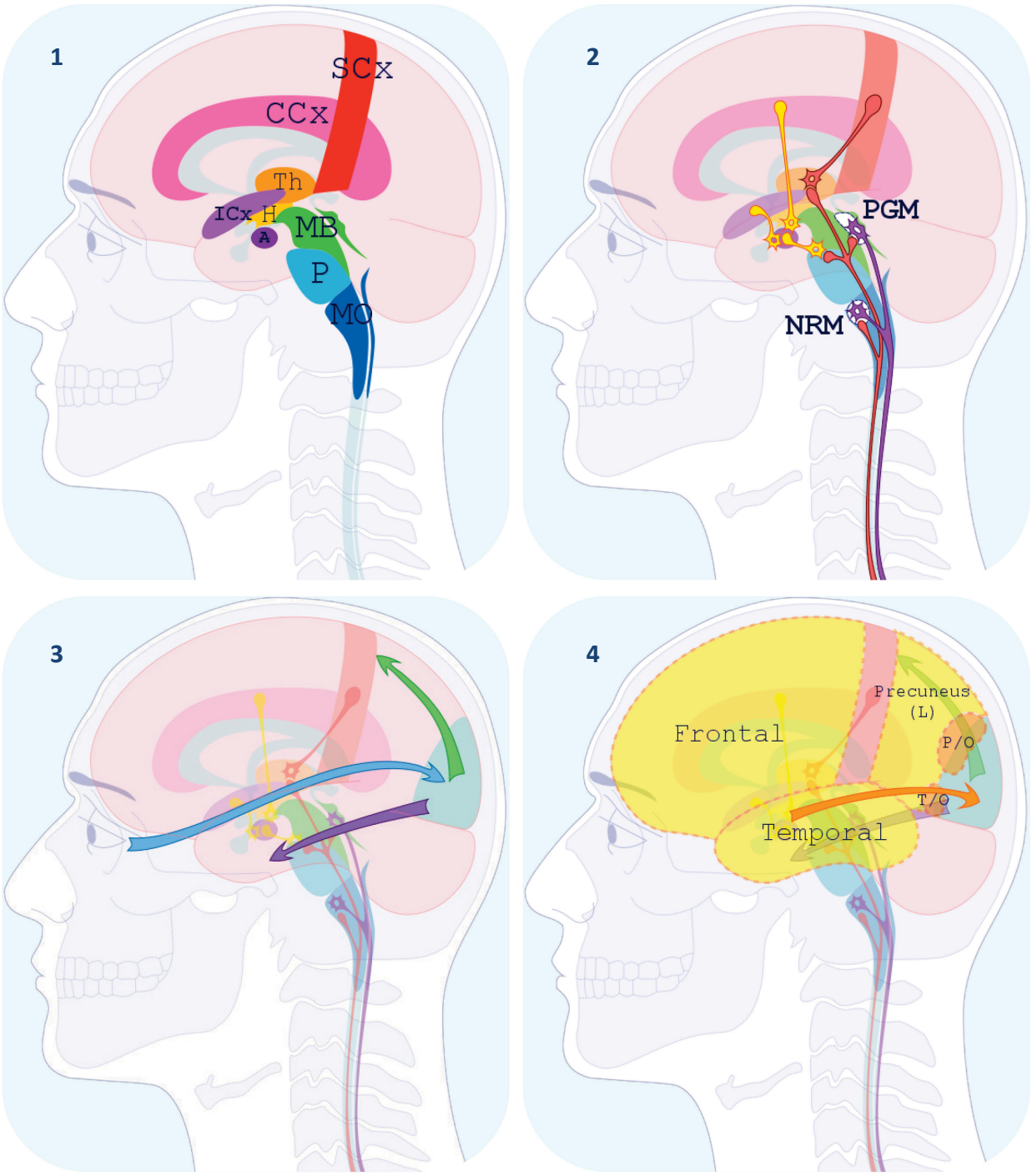


Figure 25: Descending modulation: Image 1 *shows cerebral regions involved in pain processing: the brainstem's medulla oblongata, pons, and midbrain; the amygdala and the cingulate, insular and somatosensory cortices; and the thalamus and hypothalamus.* ***Image 2*** *shows ascending nociception (**red**) as well as brainstem (**violet**) and affective cerebral modulation (**yellow**), and their overlap with the rostroventral medulla oblongata's nucleus raphe magnus and the midbrain's periaqueductal grey matter.* ***Image 3*** *shows the dorsal (**green**) and ventral (**violet**) visual streams activating the cortex via vision and perception (**blue**), substituted by imagery in* ***Image 4****, highlighting the many areas of cerebral modulation: the reverse ventral stream, the parieto- and temporo-occipital regions, the frontal and temporal lobes, and the left precuneus.*[30]

Moving towards a comprehensive, neurological trigger point model

Tying these visuo-imagery activity overlaps in with the results of the current study present exciting applications for both research and clinical work. Bypassing the current debate of trigger points' factual, physiological existence,[149-153,310-312] these findings not only confirm the presence of muscle-based referred pain in over half of an otherwise healthy population (*Figure 20; Tables 2 & 4*), but also its distinction from local pain on a neurological basis (*Figures 2, 24-25*).

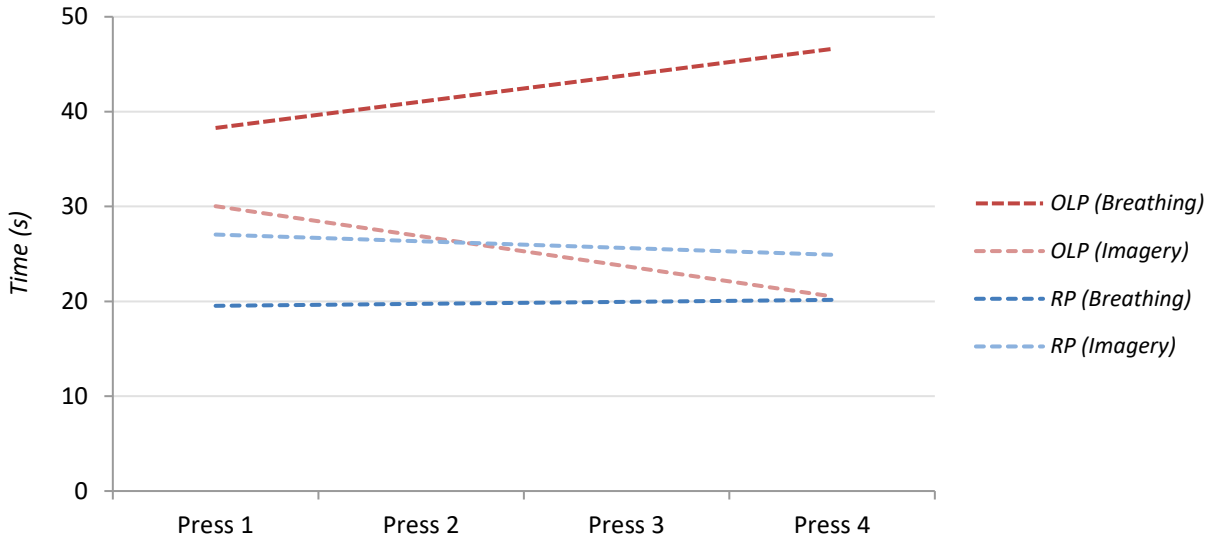


***Figure 26: Pain release trendlines across presses**. Groups are divided based on intervention type and only local pain (**OLP**) v. referred pain (**RP**) presence.*

Patients experiencing only local pain found focusing on imagery much more beneficial. Average press and total treatment times were 42.4s and 3:32m with breathing, but 25.3s and 2:07m with imagery: an average of 40% faster with the latter. Patients experiencing referred pain (with or without local pain) found focusing on breathing much more beneficial, with average press and total treatment times of 19.8s and 1:50m with breathing, but 26.0s and 2:22m with imagery: an average of almost 25% faster with the former (*Table 2, Figures 20, 26-27*).

These findings should shake up physical rehabilitation approaches, where deep diaphragmatic breathing is consistently applied indiscriminately to "ground" patients and reduce their pain irrespective of its nature or their condition.

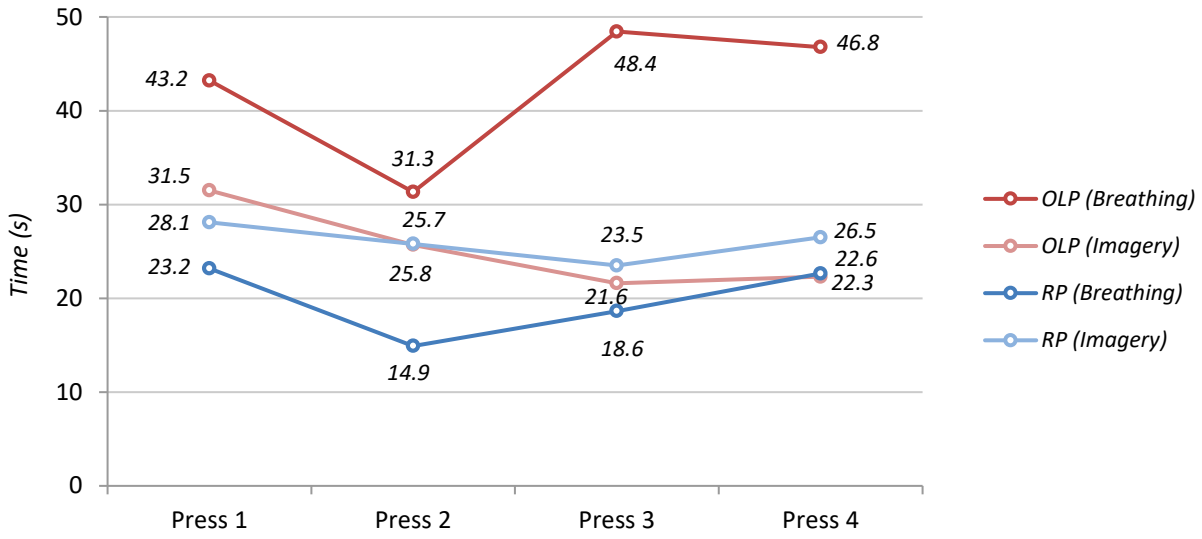


Figure 27: Original pain release time differences between groups. *Note the U-shaped resolution patterns.*

Noting all the above, it is recommended to integrate imagery in treating local tender spots but breathing for referred pain, and *not* to rely on breathing when massaging through local muscle pain — this actually increases nociception. That said, I have had success mixing the two approaches in clinic since making these discoveries, starting with the recommended approach and then breaking through plateaus with the alternative, generally after the second press. This plateauing effect is supported by the data with all treatments undergoing a U-shaped process, with initial and final presses generally lasting longest and middle presses resolving quicker (*Figs. 19-20 & 27*). This is perhaps owing to the neurological sensitization phenomenon and is why I rarely apply more than two successive presses anymore in trigger point treatment.

Explaining pain

Local pain and imagery

Previous studies show imagery is a higher-order function, occurring deeper in the brain than the brainstem where breathing is controlled[313,314] (*Figures 9 & 25*). It is reasonable that "true" local pain is also processed by similar higher-order cognitions involving image reconstruction, sensation, perception, memory, emotion, and even conscious thought. Imagery's success here may work through dividing cerebral processes, redirecting an otherwise busy conscious mind toward a non-deleterious outlet and letting the brain deal with the local pain subconsciously. These thoughts intersect with popular findings that cognitive load and pain have been seen to affect each other, even if concise neural pathways haven't yet been fully explored.[18,315-318]

Local pain and breathing

A forced focus on breath confounded the local-pain-only group, as seen by longer treatment times and standard deviations during later presses (*Figures 20 & 21*). Treatments were almost 30% slower in the first two presses and over twice as slow by the final two in this population (*Figure 27*). A brain coping with "true" pain in response to potentially damaging pressure may act in this way. The unnecessary neural activity required in forced breathing could directly interfere with neurological coping processes, or even indirectly affect them through spreading resources too thinly, taking energy away from more direct pain management.

Referred pain, breathing, and imagery

That breathing was so effective in mitigating idiopathic referred pain suggests that this pain may be processed much earlier on in the brain, perhaps not higher than in the brainstem, alongside the brain's breathing command centres. Referred pain may indeed be something more of a broken reflex or a faulty feedback loop stuck in a phase, not making it quite up into the brain for proper processing as normal, "healthy" pain does. Breathing, controlled in the brainstem itself, may therefore be activating nearby nerve clusters and nipping the problem much closer to its source. That imagery wasn't effective at all further suggests higher-order cognitions may not be as involved in this phenomenon (*Figure 30.3*).

Caveat emptor

The above is just the beginning of a neuromatrix-like synthesis of trigger point referred pain. All pathways suggested in this paper are theoretical and not anatomically correct (*Figures 2, 3, 4, 6, 7, 8, 9, 12, 13, 16, 24, 25, & 30*). The resulting model should be used rather as an outset for further research and not as an authoritative, comprehensive review of cerebral pain modulation. Still, it offers treatment modifications already being used in clinic to help patients better understand their pain and receive faster and more effective treatment through it.

Study limitations

The biggest limitation was the lack of a control group. This came about through introducing a breathing script to what was meant to be the control group, intending to offset script presence as a potential mediating factor. When breathing itself resulted in a stark difference in treatment outcomes, especially after dividing the group further along referred/local pain presence, it became clear that the breathing script could be affecting the outcome in its own way.

The second limitation was the uncertainty around proper trigger point targeting. This came about in a simpler way: in clinical massage therapy a trigger point is almost always defined by its referral pattern. Presence of a taut band or tender nodule are only relevant in locating the point that may be triggering the pain, and a local (or, indeed, sometimes quite distal) twitch response serves only to confirm it. However, patients do not come in complaining of taut bands or twitch responses but of referred pain, so dealing with this symptom naturally became the focus of this paper. This novel clinical approach to defining a trigger point as one that elicits a referred pain response when activated (*ie.* is pressed, flexed, or stretched) as opposed to the accepted, minimum criteria of tender point presence in a taut band,[84] speaks to the need of peeling back the term from the umbrella function it is serving.[150,312]

The third criticism concerns applying standard treatments to all participants. This was managed as well as possible, given the circumstances. Still, it was very subjective and based entirely on the therapist. The process could be improved through creating a clear massaging protocol, collecting pre-treatment information to classify patients,[65] and using tools like tissue compliance meters, pressure threshold force gauges,[303] or an algometers to both apply and measure pressure reliably,[319] and electromyography to objectively measure muscle relaxation.

The last criticism is the most difficult to fix: standardizing imagery. Cultural differences make an image relevant in Canada ("butter melting on a beach") less relatable in Poland ("Shouldn't it be in a frying pan?" – one participant), but this is just the beginning. There are also the many personal factors involved. A standard image may be interpreted completely differently based on individual memories, personalities, and even current moods.[64,270,284,320] For example, a calm beach may be perceived as too hot or too empty or on the edge of shark-infested water,[269] confounding its anticipated relaxing effects. Further, the use of such scripts may interfere with spontaneous ones a participant might already be using[269] and that may be more vivid[292] (albeit with smaller analgesic effects than one proposed by a properly trained therapist).[320] Lifestyle and health status also come into play: some study participants were typical university students and others were athletes with potentially higher body awareness, motivation, skill in coping with pain, and experience with psychological interventions and physical therapy. Tied into all this is the subjective nature of pain itself: is it referred or local, muscular or mental, chronic or acute?[321] These answers affect not only the imagery recommendation but also its timeline of application.[59,280,281] These individual narratives must be taken into account on top of any cultural and psychological considerations when designing an objective imagery treatment protocol.

Directions for future research

Were I to run this experiment again, I would keep the above in mind as well as keeping notes regarding techniques used and a subjective gauge of the pressure/effort required for each press, the presence of other symptoms (sweating, twitch responses, nodule/band presence), and the patient's perception of the passage of time during treatment. I would apply the pain tolerance questionnaires and a pre-treatment range-of-motion test, as well as a post-treatment follow-up weeks later to see how long the treatment effects lasted. I would clarify my post-treatment questionnaire regarding concentration: this is not a general term regarding overall concentration but patients' specific focus on their imagery or breathing. Aside from redoing the study with the above fixed, there are a few other directions that may be of interest to explore.

First, a deeper study of trigger point neurology is necessary, tying neuromatrix theory in with gate control and describing neural pathways for all trigger points observed so far. Referred pain consistency across thousands of cases points to an underlying physiology based in neurology or maybe even fascial chains.[162,324,325] Such a comprehensive map would develop richer, more reliable pain charts for clinicians and perhaps begin explaining phenomena like twitch responses or muscle hardening to form bands and nodules (*Figures 28 & 29*).

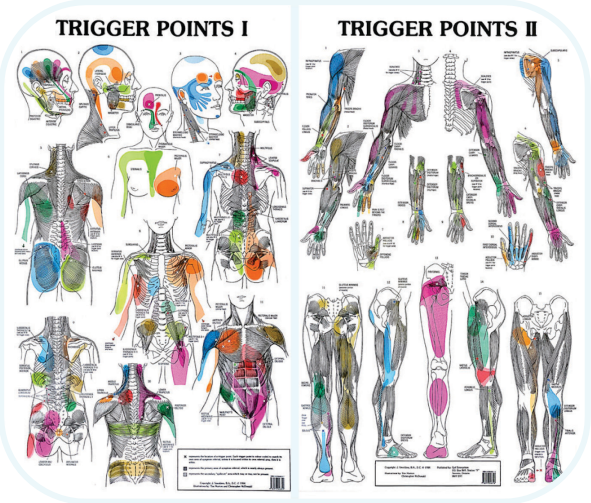


Figure 28: Trigger point posters. *Very popular in clinics throughout Canada, yet not updated since 1984.*[322,323]

Second, a comprehensive brain activity map comparing regions active in imagery with those modulating pain is sorely needed (*eg*: *Figure 25*). Emphasizing different imagery elements or content activates different brain regions[209,213,221] and so may differ in analgesic effect. Comparing results from visual versus auditory, kinesthetic, or even olfactory or gustatory imagery may lead to interesting results, made even richer if taking cultural considerations and the effect of imagery practice (even just a few minutes pre-testing) into account. And the same applies to the breathing protocol: which breathing techniques work best, which least, and why? And are there other, better focuses, like "grounding" oneself (*eg*: sinking into the massage table or placing hot stones on the sacrum or palms), or on maintaining a slow, steady heartbeat?

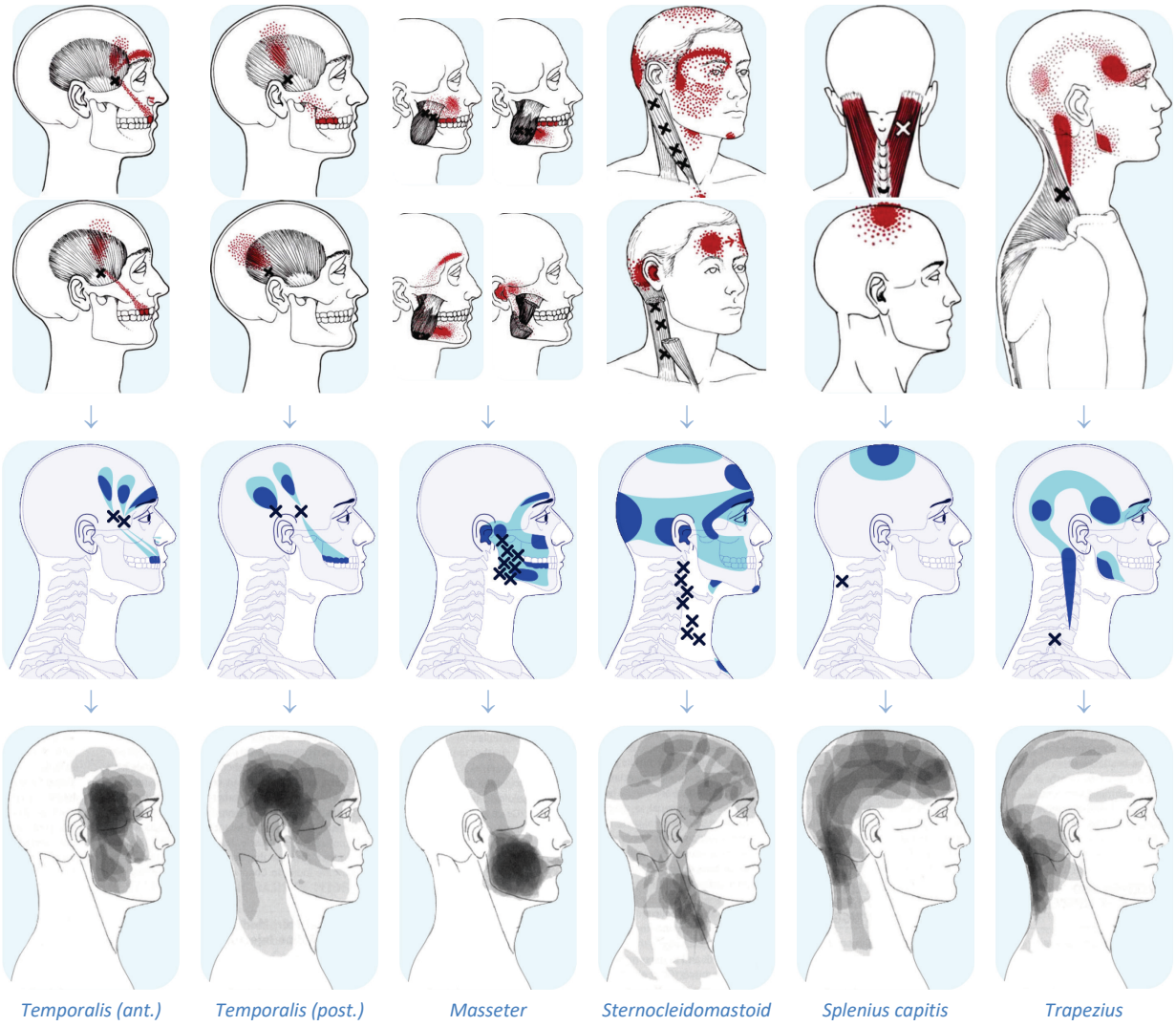


Figure 29: Referred pain pattern evolution. *Travell and Simons first published comprehensive, observed patterns in 1983 (**top**; summarized at **middle**),[39,326-330] since refined by Schmidt-Hansen in 2008 (**bottom**).[46,81,82]*

Third, a comprehensive comparison of muscle "knots" and trigger, tender, and acupressure points is in order. These are used interchangeably, misleadingly suggesting all are the same phenomenon. This study already separates trigger points with idiopathic referral patterns from tender points with normal, localized, "true" pain patterns, and the literature confirms trigger point pain is rarely "burning" but rather "steady, deep, aching",[39,331] and often is sudden, sharp, and clearly defined (suggesting cutaneous[90,175] Aδ activity). Further studies could build on these distinctions,[46] comparing twitch response locations (and patients' consciousness of them — few seem to be aware of them) and diving deeper into understanding hyperirritable spots, palpable nodules, and taut bands, comparing the prevalence and comorbidity of all.[39,154]

Lastly, this study has left me with many follow-up questions. Do imagery or manual trigger point massage therapy release specific neurotransmitters? What is the best hydrotherapy protocol, taking all these pain theories into account? What is the actual mechanism of injury behind trigger points? Does explaining referred pain to the patient increase treatment efficacy? Is entering a trigger point slowly versus quickly better or worse for the overall outcome? Does time seem to drag on or speed up during treatment? Are patients more sensitive to trigger points before or after physical activity, or workdays? How long does it take for the pain return, and why? Are there any adverse effects from the deep manual pressure applied? What imagery scripts are currently in use and do they change based on treatment/patient? How else do patients generally cope with trigger point referred pain (*eg*: massaging the sore spot or the referral pattern, aromatherapy, listening to music, ingesting medication or certain foods, or ignoring it until it subsides)? All of these are great ideas for follow-up studies.

How these findings have changed my practice

Trigger point release now takes me 30-60s, from 2-5m. If someone presents with referred pain (most often from neck muscles into the back, neck, shoulder, arm, or head), I do the following:

1. Put the patient at ease with small talk and jokes while warming up the area containing the suspected trigger point with general Swedish techniques (effleurage, petrissage).
2. After about a minute, palpate the suspected trigger point, pushing into it firmly and quickly to elicit a sharp, immediate referred pain. This is to convince the patient beyond doubt that this is the source of their pain, and not where they feel it. This limits their doubt which otherwise could cause the patient to dismiss this connection, denying successful resolution.
3. Pressing firmly into the point, I get them to breathe "yoga breaths, deep into your stomach and not your chest, inhaling, then exhaling, then remaining with empty lungs for as long as is *comfortable*, not possible. When you need air, breathe in. Keep your airway open during the whole process, at no time closing it as if you're bearing down." I sometimes add an image to make the breath more manageable (*eg*: "Imagine biking over a hill, and then spending some time in a valley."). It is not important if they use their mouth, nose, or a combination of both for their breath. (This breathing was adapted from my practicum at Gdańsk Sport University under Dr. Marek Graczyk, who was using a form of it very successfully to quickly bring Olympic rowers into flow state, and can be seen in action on *youtu.be/gRiDDTTG_hE*).

4. By the second breath, I ask if the referral has subsided; if so, I release the point and "flush" it out with more effleurage. If not, I keep pressing for 2-3 more breaths and explain referred pain as outlined in this paper: "What you are feeling is an illusion, a mix-up of nerve signals from where I'm touching and where you're feeling pain. You had a small injury that sent pain signals to your brain, which it ignored because they were so light. In the spinal cord on their way to the brain, these signals met with signals from another area that took up this painful signal. This is what you are feeling, and we are amplifying it so you can breathe through it and your brain can realize there is no pain there, breaking this faulty loop." I avoid more than 3 subsequent presses because they are ineffective (*Figure 27*), instead teaching the patient the appropriate muscle anatomy and technique to massage themselves as homecare, and directing them to heat the painful muscle and apply ice to the referral to override the illusory pain signal as needed.

   Some patients find the following analogy more useful than the neurological explanation above: "Imagine I am launching Silly String at you from a distance. You ignore me because, though a little annoying, I'm not really hurting you and you're busy with something else. After some time however, you look again and see your friend is standing beside me. 'Why', you wonder, 'is my friend also shooting me with Silly String?' — He isn't. He is only standing nearby so it *appears* that he is at first glance. To stop this illusion of his annoying you, you can do one of two things: take away my Silly String, resolving the original cause of your frustration (*ie*. massaging or applying heat to the trigger point proper), or give your friend a water gun. When he starts spraying you, you know he can't be behind the Silly String because his hands are occupied with something else (*ie*. applying ice to the area of referred pain, breaking the illusion of referral)."

   If, on the other hand, I see the patient has a tender point with true pain and no referral, I immediately coach them through it with relaxing imagery, encouraging thoughts of "melting, or waves lapping the pain away, or lava breaking the muscle up from underneath." I then have them focus on this image until the pain starts subsiding, usually by 2-3 breaths.

CONCLUSION

The brain behind myofascial trigger points

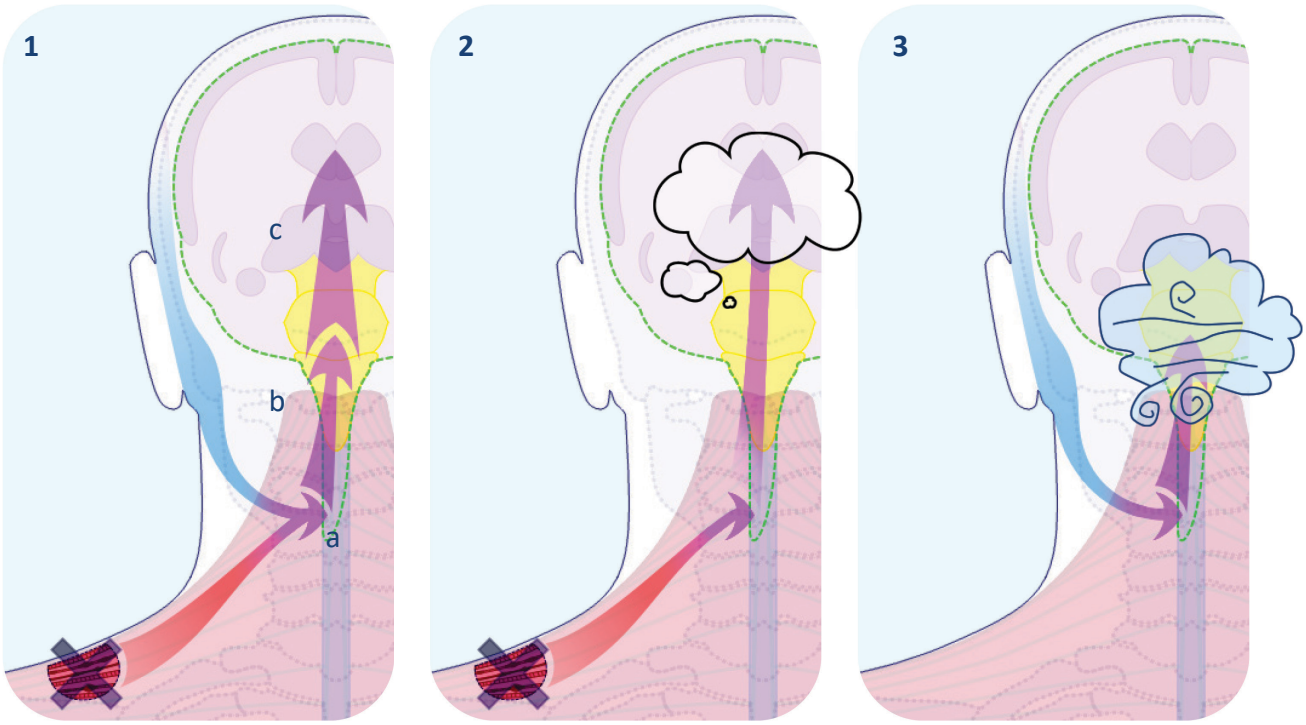


Figure 30: The brain behind myofascial trigger points*: Trigger points as a neurological phenomenon.* ***Image 1*** *shows the trigger point (**X**) initiating pain (**red**) and undergoing modulation (**purple**) at its spinal junction (**a**), in the brainstem (**b**), and in the brain (**c**). Repeated stimulation, as in repetitive stress injuries, result in the illusion that is referred pain (**blue**).* ***Images 2*** *and* ***3*** *show the initial injury and its resulting referred pain as two separate phenomena, the first mitigated through imagery in the brain and the second through breathing in the brainstem during massage.*

Myofascial trigger point referred pain is best explained by Ruch's 1946 convergent-projection theory,[89] specifically through neuroplasticity in the form of central sensitization. Its mitigation may occur to some extent in the spinal cord with massage therapy as per Melzack and Wall's gate control theory[55] (*Figure 4*), but can be greatly sped up by focusing on breathing, perhaps by activating brainstem breathing centres as per the results of this study (*Figures 20, 26-27*).

Massage therapy also relieves local muscle pain, a process that sees improved results with a focus on imagery through perhaps facilitating higher-order brain activity that itself kickstarts innate antinociceptive processes. Further developing rehabilitative imagery scripts and protocols to harness this cerebral antinociception is a recommended future research pursuit as a result of this study (*Figures 20, 26-27*). Focusing on breathing, on the other hand, seems to have adverse effects on local pain mitigation. These findings (*Figures 24-25 & 30*) contribute to Melzack's current neuromatrix theory development.[16,74,75]

Applications

Clinically, the results of this study suggest the use of breathing to mitigate referred pain from myofascial trigger points and imagery to mitigate "true" local pain from tender points. The two phenomena are distinct, seeming to activate overlapping but different neural networks. Further research into disentangling this pain in the brain is needed, particularly in studying pain modulation in the spinal cord, brainstem, and brain proper (*Figures 24-25 & 30*). Until then, a step in the right direction for massage therapists would be to consider treating more than just muscle in what is increasingly looking to be a neurological phenomenon.

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Appendices

Informed Consent Form for Participation in a Research Study

1. *Title of the study:* Imagery Effectiveness in Trigger Point Treatment

2. *Aim of the Study:* To judge the effectiveness of a short guided imagery script on trigger point release.

3. *Description of research activities:* You will be read an imagery or relaxation script to keep in mind during the course of treatment. The therapist will then examine your neck for trigger points: tight knots of muscle that refer pain to the back of the skull, to the forehead or to the face. Upon finding one, the therapist will treat it through pressure while you focus on what was in your script. Once the trigger point is treated, the therapist will flush the area out with general massage. A few measurements will be taken before, during, and after treatment, including audio-recording the treatment itself so the therapist can verbally mark how long it takes to resolve your trigger point. The recording will be destroyed immediately following its analysis and your anonymity will be preserved. **Please do not share your script with other participants**.

4. *Risks/ discomfort involved:* There are no known risks involved in this study outside of the normal, sometimes painful trigger point release therapy. In fact, this study is anticipated to increase the effectiveness of the resolution of your trigger point.

5. *Expected impact:* The outcomes of this research are twofold: first, you may benefit from leading edge physical therapy research to arrive at a healthier state faster. Secondly, the database to which you contribute will lead to a richer, more effective protocol for future trigger point release therapy.

6. *Dissemination of results:* Results gathered will be collected and presented in a thesis paper and possibly in a journal article. Your participation will remain anonymous and any details that might identify you will not be made public. Where names will be required (*eg*, to facilitate reading), pseudonyms will be used.

7. *Further Information:* You are encouraged to inquire about anything regarding this study. The researcher's contact will be provided so you will be able to follow-up on points during or following the study as well.

8. *Freedom of consent:* As a volunteer, you are free to withdraw from this study at any point, no questions asked. If ever you feel uncomfortable with continuing the study—during or after treatment, up until the publication of the study as a master's thesis in June 2015, you retain full rights to having your data erased from all records. In this case, your data will not be used and will be permanently deleted from all places where it was stored. Should you decide to withdraw, please contact me, Paul Sulżycki.

   Participant's declaration:

   I have read this form and understand the procedures involved. I agree to participate in this study.

Participant:		*Signature:*		*Date:*	dd·mm·yyyy
Researcher:		*Signature:*		*Date:*	dd·mm·yyyy

Formularz zgody na udział w badaniach naukowych

1. *Tytuł badań:* Skuteczność leczenia punktu spustowych przy pomocy obrazowości (EN: *Imagery Effectiveness in Trigger Point Treatment)*

2. *Cel badań:* Aby ocenić skuteczność leczenia punktów spustowych przy pomocy obrazowości.

3. *Opis działalności badawczej:* Dostaniesz polecenie wyobrażenia sobie scenę. Terapeuta przebada Twą szyję i plecy na punkty spustowe – zbite mięsnie które promieniują ból do twarzy, głowy, lub karku. Jak one się znajdą terapeuta je wymasuje, a Ty będziesz skupiał/a się nad swoją sceną w wyobraźni. Jak punkt zostanie wyleczony, terapeuta wymasuje rejon. Wyniki będą zapisane przed, podczas i po leczeniu, w tym audio-nagrywanie samego leczenia. To pozwoli terapeucie by werbalnie zaznaczał jak długo trwa rezolucja punktu spustowego. Nagranie zostanie zniszczone natychmiast po jego analizy i Twoją anonimowość zostanie zachowana. **Proszę nie dzielić się swoją sceną innym ochotnikom**.

4. *Przewidywana ryzyka/dyskomfort:* Nie ma żadnych znanych zagrożeń związanych z tym badaniem poza normalnym bólem punktu spustowego. Wręcz przeciwnie, te badanie przewiduje większą skuteczność leczenia.

5. *Przewidywany wpływ tych badań:* Wyniki tych badań są dwojakie: po pierwsze, Ty skorzystasz z najnowszych teorii badań w terapii masażu, aby dojść do zdrowszego stanu szybciej. Po drugie, baza danych dzięki Twoich wynikom stanie się bogatsza, rozwijając bardziej efektywny protokół na leczenie punktów spustowych.

6. *Upowszechnianie wyników:* Wyniki zostaną zebrane i przedstawione w pracy magisterskiej, lub ewentualnie w czasopiśmie. Twój udział pozostanie anonimowe i wszelkie dane, które mogą Cię zidentyfikować nie będą opublikowane. Gdzie nazwy będą wymagane (żeby wyniki rozróżnić), to pseudonimy będą wykorzystywane.

7. *Więcej informacji:* Zachęcamy żebyś pytał/a się naukowca o wszystkim co ma wspólnego z tą pracą. Kontakt badacza jest Ci dostępny, jeżeli będziesz miał/a pytania po badaniach.

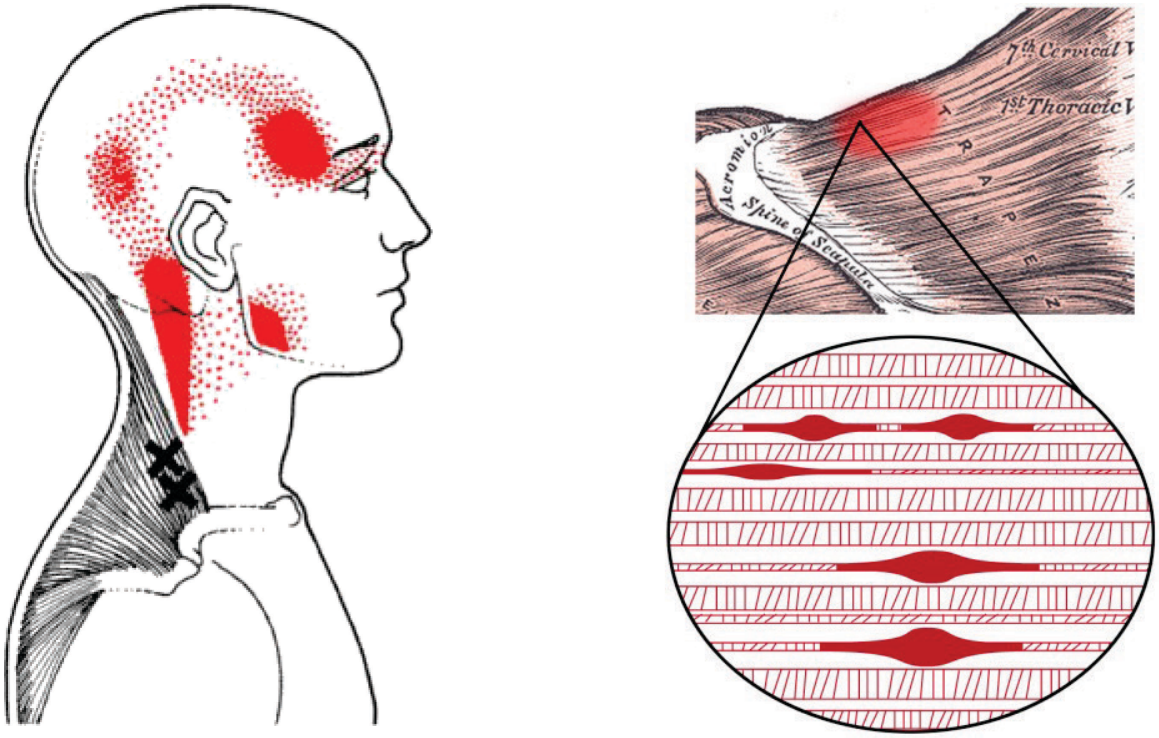
8. *Wolna zgoda:* Jako wolontariusz, jesteś wolny, aby wycofać swoją zgodę bracia udział w tej pracy w dowolnym momencie. Gdybyś kiedykolwiek czuł/a się niewygodnie kontynuując badania – podczas lub po badaniach, aż do publikacji pracy w czerwcu 2015 r., zachowujesz pełne prawa mieć swoje dane usunięte z bazy danych. W tym przypadku, dane Twoje nie będą używane do badań i zostaną usunięte z wszelkich miejsc, gdzie były zapisane. Jeżeli zdecydujesz się wycofać, proszę o kontakt: Paweł Sulżycki.

   Oświadczenie uczestnika:

   Przeczytałe/am i zrozumiałe/am formularz, i procedury. Wyrażam zgodę na udział w badaniu:

Uczestnik:		*Podpis:*		*Data:*	dd·mm·rrrr
Badacz:		*Podpis:*		*Data:*	dd·mm·rrrr

Breathing script: What is a trigger point?



Parmenter D (2014). Trigger Point Complex. Retrieved from commons.wikimedia.org/wiki/File:Trigger_Point_Complex.jpg
Simons, D. G., Travell, J. G., & Simons, L. S. (1999). Travell & Simons' Myofascial Pain and Dysfunction: The Trigger Point Manual (Vol. 1). Baltimore: Williams & Wilkins. p. 279.

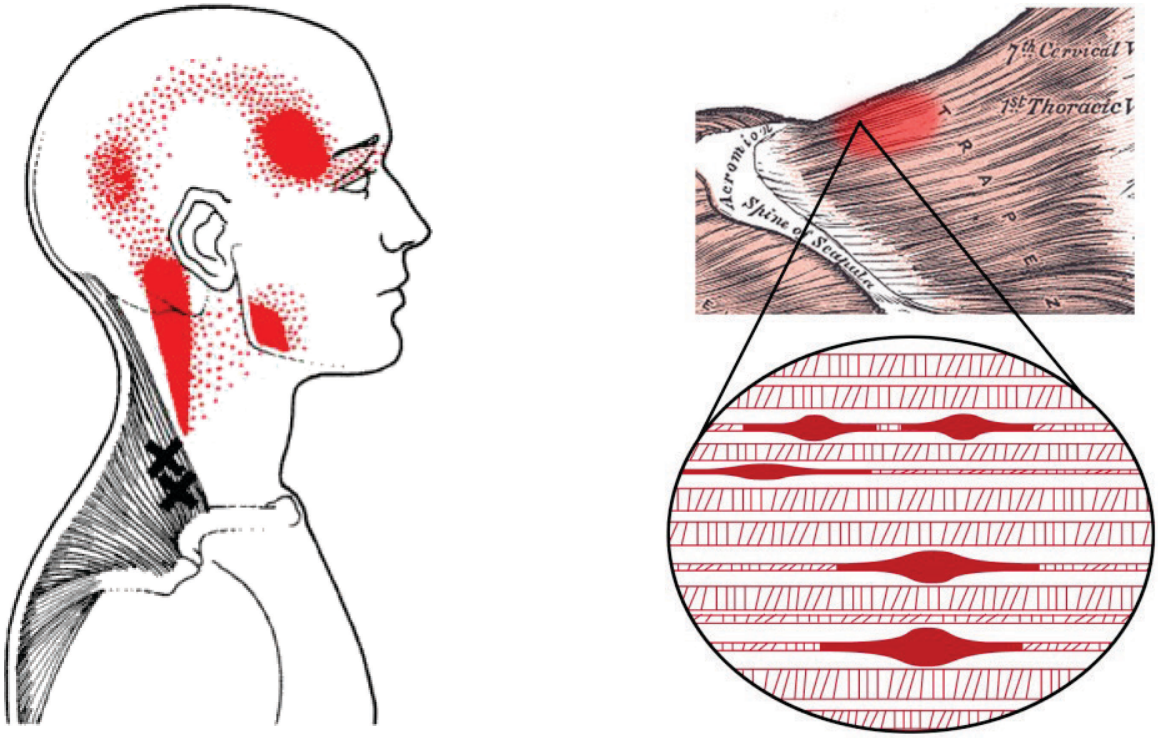
Treatment protocol & introduction to using the pain scale

1. "0" = no pain, "10" maximum pain
2. Therapist finds point, presses until "7" (just before pain becomes unbearable)
3. Pressure is maintained until you reach a "0" or "1" on the scale
4. Pressure is increased, until "7" again
5. Process repeated 4 times, or until pain is gone (*ie*. you feel only "pressure, not pain")

Relaxation script: Focusing on breathing

Concentrate on breathing, first filling up your stomach, then your ribs from the sides, and finally your chest. Exhaling, this order is reversed. Breathe in with your nose and out with your mouth. Breathe slowly. If you lose focus, try to return to it, concentrating on breathing.

Oddech: Co to jest punkt spustowy?



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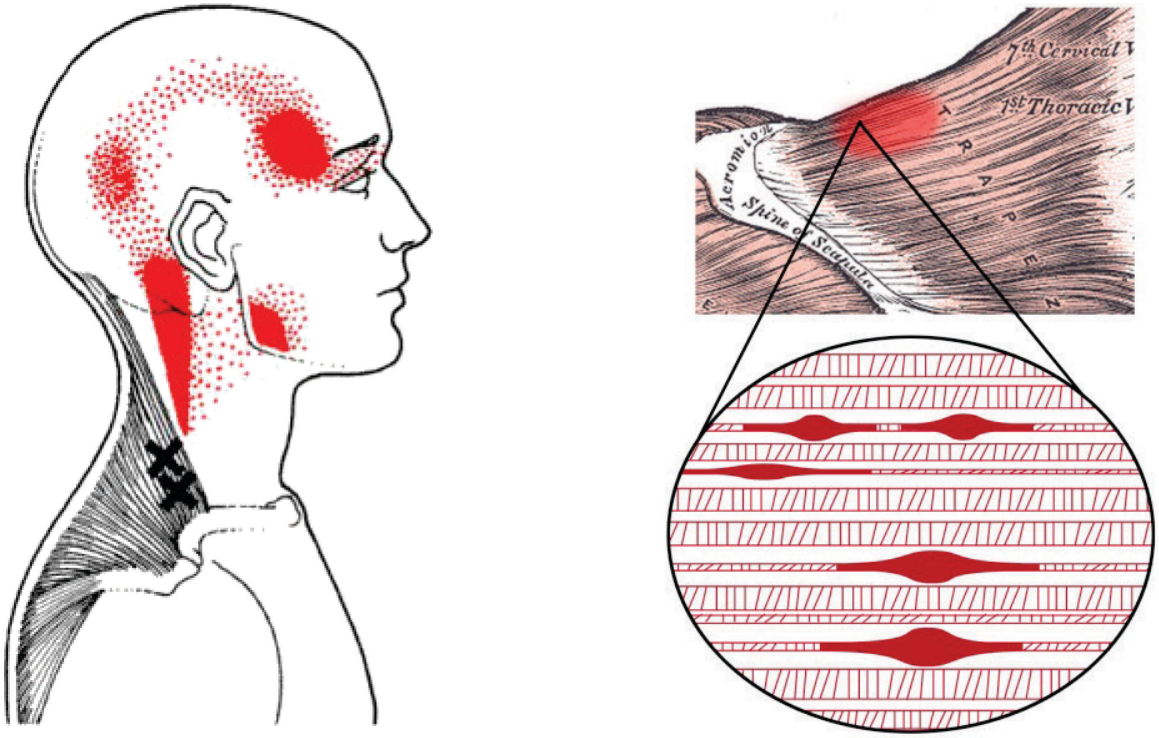
Protokół i skala bólu

1. „0" = brak bólu, „10" maksymalny ból
2. Zlokalizować punkt, nacisnąć aż „7" (tuż zanim ból staje się nieznośny)
3. Trzymać aż zejdzie do „0" lub „1"
4. Przycisnąć mocnej, aż „7"
5. Powtórzyć aż do 4 razy lub aż ból zniknie i czuć „tylko nacisk, a nie ból"

Relaksacja: skupienie się nad oddechem

Skup się na oddechu, wypełniając w pierw brzuch, potem żebra od boku, a ostatnio klatkę piersiową. Na wydechu, to odwrotna kolejność. Wdychaj przez nos, wydychaj przez buzie. Oddychaj powoli. Jeżeli stracisz skupienie, to postaraj się do niego wrócić, jeszcze raz skupiając się na oddechu.

Imagery script: What is a trigger point?



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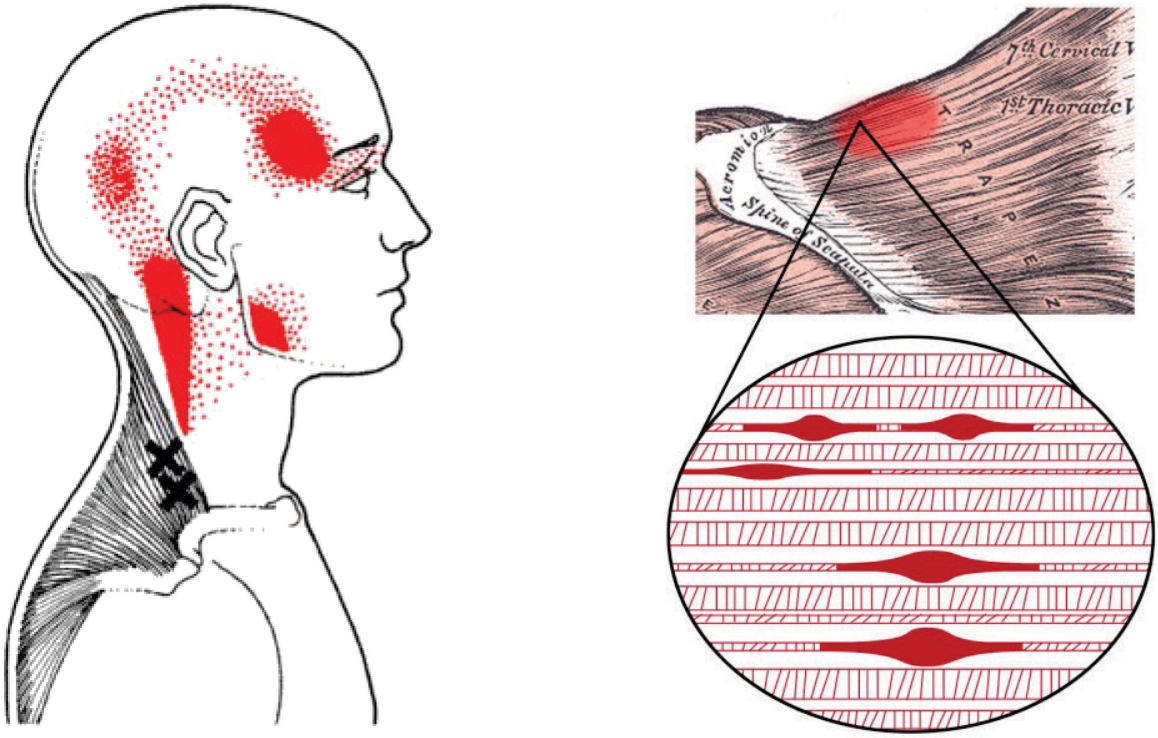
Visualization script: Focusing on imagery

Imagine that this tight knot is a ball of butter, melting on the beach under a hot, afternoon sun. Use all your senses: smell (salty air), sight (sand, gold and blue, water), **sound** (surf, seagulls), **touch** (grains of sand, warm wind, dripping sweat), **taste** (salty water, sweat, butter), **movement** (the butter melting, waves), **temperature** (hot, nice), and **emotions** (melting, relaxation). Ensure your image is physically accurate (sizes, its being based in reality), that the focus is the ball of butter, that time is moving normally (not too fast, not too slow), and perspective (that the muscle knot is actually this ball of butter). If you get sidetracked, try to return to concentrating on this image.

Wyobraźnia: Co to jest punkt spustowy?



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Wizualizacja: skupienie się nad obrazkiem

Wyobraź sobie, że ten punkt bolesny to kuleczka masła, topiąca się na plaży pod wpływem gorącego słońca. Wyobrażaj wszystkie zmysły: zapach (słone powietrze), widok (piasek, złoto i niebiesko, woda), **dźwięk** (fale przybrzeżne, mewy), **dotyk** (ziarnka piasku, ciepły wiatr, pot kapiący), **smak** (słone morze, pot, tłuszcz masełka), **ruch** (topienie się kuleczki, fale przybrzeżne), **temperaturę** (ciepło, przyjemnie), i **emocje** (topienie, relaks, wsiąkanie się). Upewnij się że obraz jest fizycznie poprawny (rozmiary, oparty nad rzeczywistością), że fokus jest te masełko, że czas się porusza normalnie (nie za szybko, ani za wolno), i perspektywę (że punkt bolesny w zbitej mięśni jest jednak tą kuleczką masła). Jeżeli oderwie się uwaga, to powoli przywróć ją do tego obrazu.

Post-treatment questionnaire

1. What was the worst pain you've ever experienced? How would you rate it?

   ("0" = nothing, "10" = maximum pain)

2. How much did the full trigger point treatment hurt?

   ("0" = not at all, "10" = maximum pain)

3. What were you concentrating on?

4. To what degree did you focus on your image/breathing?

   ("0" = none, "10" = maximally)

5. Were you distracted from your image/breathing? If so, how many times?

6. Where did you feel your pain during treatment?

Kwestionariusz po leczeniu

1. Jaki był najbardziej intensywny ból w Twoim życiu? Ile zawierał na skali?

   ("0" = nic, "10" = maksymalny ból)

2. Ile bolało leczenie punktu spustowego?

   ("0" = nic, "10" = maksymalnie)

3. Nad czym dokładnie się skupiła/eś?

4. W jakim stopniu była/eś skupiony/a na obrazie lub oddechu?

   ("0" = nic, "10" = maksymalnie)

5. Czy Ci się uwaga oderwała? Ile razy?

6. Gdzie czuła/eś fizyczny ból podczas masażu?