MYOFASCIAL PAIN AND DRY NEEDLING FOR THE TRAPEZIUS MUSCLE

by

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Abstract

Purpose: The purpose of this project is to first conduct an extensive review of recent literature on myofascial pain syndrome and trigger point dry needling, and the clinical evidence for the use of dry needling for myofascial pain in the trapezius muscle. Second, to write a manuscript for publication to disseminate the knowledge to nurse practitioners.

Background: Musculoskeletal pain is a common complaint in the general medical clinic and can have large implications on quality of life. Myofascial pain can be difficult to diagnose and manage.

Results: There are many different theories about the physiology behind myofascial pain and myofascial trigger points. These theories include peripheral and central sensitization components. Studies have shown that patients with myofascial pain have more inflammatory mediators and sensitizing substances present, and show changes in spontaneous electrical activity in end plate zones. There also are many theories on how dry needling physiologically addresses myofascial pain but no basic research has proven these theories recently that the author is aware of.

Conclusion: Trigger point dry needling is a good therapeutic option for addressing and managing myofascial trigger points and myofascial pain in the trapezius muscle. Further research and replication of results is needed on this topic to strengthen the evidence supporting use of dry needling. Additionally, a clear, agreed upon, defined procedure for using needling will be needed to further study and promote it as a therapeutic option.

Keywords: myofascial pain, trigger point dry needling, trapezius
Myofascial Pain and Dry Needling for the Trapezius Muscle

Musculoskeletal pain is a major cause of morbidity in today’s societies (Ziaeifar, Arab, Karimi & Nourbakhsh, 2014). Approximately one third of those experiencing musculoskeletal pain have myofascial pain syndrome (MP) (Ziaeifar et al., 2014). MP accounts for approximately 30% of visits in general medical clinics and 85-93% of patients in specialty pain management clinics (Ziaeifar et al., 2014). It can be difficult and controversial to diagnose and manage MP (Ziaeifar et al., 2014). Additionally, there is a lack of knowledge and understanding of MP, and what the best treatment and management approach for it is. Trigger point dry needling (DN) is one treatment option which has been growing in popularity. However, as with MP, there is not a good understanding of DN, how it works and if it is a good therapeutic treatment option for MP, which further compounds the challenge of addressing and successfully managing MP. By way of a thorough literature review on MP and DN, this paper and project aims to address a knowledge gap for primary health care professionals, particularly Nurse Practitioners (NPs), that will lead to a better understanding of MP and how to treat it using DN. This paper will discuss MP, DN, and myofascial trigger points (MTPs), taking into account biochemical, central and peripheral sensitization theories, how to approach diagnosis of MP, and the different management strategies, focusing on DN. Finally, a review of recent clinical evidence for use of DN specifically in the trapezius muscle for MP will be discussed.

Description of Problem

Nature of the Problem – Myofascial Pain and Dry Needling

The skeletal muscle is the largest organ in the human body, accounting for almost 50% of our body weight (Yap, 2007). MP is a common articular musculoskeletal pain, and is often under diagnosed and under treated (Borg-Stein, 2004; Shah et al., 2008; Yap, 2007). It is one of the
most common causes of neck and back pain (Yap, 2007). The prevalence of musculoskeletal pain increases with age and can have a significant impact on the individual’s quality of life (Yap, 2007). It also has a significant financial impact on the health care system in particular, and society on the whole (Yap, 2007). It affects up to 95% of people with chronic pain disorders, is common in patients at specialty pain management centers and contributes to approximately 30% of patients visiting a general medical clinic (Borg-Stein & Simons, 2002; Borg-Stein, 2004; Shah et al., 2008).

The traditional definition of MP has it originating from MTPs which are discrete hyperirritable, palpable nodules. MTPs have been theorized to develop from heavy eccentric and, or concentric loading (Boyles, Fowler, Ramsey & Burrows, 2015). MP can also occur in combination with other underlying pain generating conditions such as disc herniation or radiculopathy (Borg-Stein & Simons, 2002). Symptoms of MP can begin after a distinct injury or trauma, or be gradual and insidious in onset (Borg-Stein & Simons, 2002). MP has been described as a deep, regional ache that is often accompanied by a sensation of tightness or pulling (Borg-Stein, 2004). Some of the symptoms associated with MP and MTPs are autonomic dysfunction such as abnormal sweating, lacrimation, dermal flushing, vasomotor and temperature changes; functional complaints such as decreased work tolerance, impaired muscle coordination, stiff joints, fatigue and weakness; and other neurologic symptoms such as paresthesia, numbness, blurred vision, twitches, trembling; additionally, sleep disturbance, mood changes and stress are sometimes experienced (Borg-Stein & Simons, 2002; Borg-Stein, 2004). More insidious causes of MP are exercise, low level repetitive stress on the muscle, acute and chronic mechanical trauma, electrical trauma, sustained and prolonged stress, and ischemia which can all lead to muscle damage and MTP formation (Ge & Arendt-Nielson, 2011).
Precipitating factors of MP are trauma, mechanical factors, degeneration, nerve root compression, emotional and psychological stress, endocrine and metabolic deficiencies, nutritional deficiencies and chronic infections (Yap, 2007). Mechanical factors could be internal such as poor posture or scoliosis, or external such as poor ergonomics (Yap, 2007). Structural degeneration of the bones and joints, and degeneration from aging can cause gradual loss of myofascial flexibility and contribute towards MP and MTPs (Yap, 2007). Emotional stress such as anxiety, along with increased sympathetic output and sleep deprivation can lead to increased muscle tension and fatigue. In particular, chronic muscle imbalance is very prevalent in modern society as there has been a trend towards a more sedentary lifestyle. The skeletal muscles can be divided into dynamic and postural muscles. In a sedentary lifestyle, more time is spent in static postures causing the dynamic muscles to become lax, while the statics ones become progressively tighter and inflexible. The imbalance between the two groups of muscles can lead to MP (Yap, 2007).

The trapezius muscle is the muscle most affected by MTPs, and active MTPs in this muscle can cause headaches, and pain and functional disability to the neck and shoulder (Pecos-Martin et al., 2015; Priyanki & Tilak Francis, 2017; Ziaeifar et al., 2014, Ziaeifar et al., 2014). The most common places for MTPs is within posture muscles such as the trapezius muscle, as they are often weak muscles that are used all day and are prone to develop trigger points (Priyanki & Tilak Francis, 2017). The trapezius in particular can be activated by working long hours and poor posture from activities such as sitting in front of the computer, writing and TV watching without adequate breaks (Priyanki & Tilak Francis, 2017). Many studies have reported that patients with symptoms of neck pain, tension headaches, dizziness, vertigo, taut and painful
trapezius muscle, and limited neck and shoulder range of motion, had MTPs in the trapezius muscle (Pecos-Martin et al., 2015; Ziaeifar et al., 2014).

There are several different invasive and non-invasive methods to address MP and MTPs. DN therapy is the single most effective method to deactivate a MTP (Priyanki & Tilak Francis, 2017). DN of MTPs is an invasive therapy where a solid filament needle is inserted into a MTP to reduce pain (Pecos-Martin et al., 2015). The theories behind how DN addresses MTPs and MP will be discussed further in this paper.

Relevancy of the Problem

Treatment of MP can be a daunting challenge and there is no standard treatment for soft tissue pain (Shah, 2008; Borg-Stein, 2004). Additionally, clinical and scientific research on MP and MTPs is lacking and lags behind when compared to other pain conditions such as arthritis or neuropathic pain (Shah, 2008, Borg-Stein, 2004). With the high prevalence of MP in general primary health care clinics, its increased incidence in our aging society, the fact that it is often under diagnosed and under treated, and the increasingly sedentary lifestyle of our population which contributes to the development, these factors make MP a very relevant yet unknown health topic for NPs.

As stated above, many patients that NPs encounter in primary care will experience musculoskeletal pain (Creech, 2011). Data has shown that patients with chronic pain will have a higher average number of visits per year compared to all types of patients with any other diagnosis (Creech, 2011). Nearly 60% of adults have had pain persistently for greater than one year (Arnstein & St. Marie, 2010). Poor pain management can lead to many individual and societal impacts, both financially and socially (Arnstein & St. Marie, 2010). One of the barriers to pain management is inadequate professional education and knowledge (Arnstein & St. Marie,
2010; Creech, 2011). There are many theories as to why health care providers and NPs are not adequately managing pain, such as 1) fear of opioid use leading to addiction, 2) beliefs about drug seeking behaviors, 3) lack of education, and 4) concerns about serious adverse reactions (Creech, 2011). This paper and project will provide information on MP and DN to address the lack of education and existing knowledge gap which are barriers to NPs adequately managing pain.

This topic is relevant to NPs as MP has been shown to be a common complaint in the primary care setting, and it has been shown to be a poorly managed condition. Therefore, it is important for NPs to gain a better understanding about MP, and its pharmacologic and non-pharmacologic treatment options to be able to better manage it. Additionally, with the current opioid crisis it is important to have knowledge on appropriate non-pharmacologic therapeutic options to address MP, especially as many patients are searching for holistic or non-pharmacological treatment options.

Project Aim, Goals and Scope

This project will provide knowledge about MP and DN to address the under diagnosis of MP and the appropriate use of DN as a treatment therapy. This will be achieved through a literature review to synthesize the current research generally about MP and DN, and specifically with regards to MTPs in the trapezius muscle. The information will then be disseminated through an article to be submitted for submission with The Journal of Nurse Practitioners. This publication was chosen because the target audience is NPs to whom this project is aimed. The goal of this project is to address and fill the knowledge gap that exists for NPs on MP as a disease process, and DN as a potential therapeutic option. This will include addressing concepts such as what MP and MTPs are, how to diagnosis MP, the theoretical physiology of MTPs, what
The aim is to fill the knowledge gap so that NPs can feel confident in their knowledge on MP and diagnosing it along with suggesting, discussing and answering questions about DN as a therapeutic option for appropriate MP patients. This project will benefit individual NPs in their own, specific practice; it will benefit the NP profession as a whole through the development of competent, up to date and evidence based NPs; and it will benefit patients and society by having knowledgeable NPs who can discuss and diagnosis MP and utilize DN therapy as an option to meet patients’ health care needs.

**Background**

**Myofascial Pain**

**History.**

The concept of MP and MTPs was first identified in the Western world by Guillaume de Baillou in the 16th century, the Dean of Medicine at the University of Paris, who coined the term muscular rheumatism (Cummings & Baldry, 2007). Balfour and Scudamore, two British physicians at the beginning of the 19th century, expressed that pain arises as a result of inflammation in the fibrous connective tissue of the skeletal muscle (Cummings & Baldry, 2007). This led Sir William Gowers to conclude in 1904 that muscular rheumatism develops as a result of inflammation of the muscular fibrous tissue (Cummings & Baldry, 2007). In 1841, French physician Francois Valleix observed that pain in muscular rheumatism arises from well-defined focal points of tenderness. German physician, Cornellus agreed and added that the nerve endings at these sites were in hyperactive states which can be precipitated by altered weather
conditions, physical exertion and emotional situations (Cummings & Baldry, 2007). In the 1930s, experiments confirmed that pain arose from nerve hyperactivity at these tender points, but it could be alleviated by injecting local anesthetic into these tender sites (Cummings & Baldry, 2007). Finally, Janet Travell continued to study this pain and realized that the pain arises not from the skeletal muscle itself but from the fibrous connective tissue, terming the disorder myofascial trigger point pain syndrome in 1950 (Cummings & Baldry, 2007). In further research with David Simons, they realized there also was a specific pain referral pattern (Cummings & Baldry, 2007).

**Trigger points.**

MTPs are easily identified through physical examination as hyperirritable nodules within taut bands of skeletal muscle that when palpated produce a muscle twitch, referred pain or reproduce the patient’s symptoms (Borg-Stein & Simons, 2002; Shah et al., 2008). They are also identified in the endplate zone of the muscle, comparable to acupuncture points which can also be in the end plate zone or in other areas of the muscle (Hong, 2000). There is a high degree of correspondence (71%) between MTPs and acupuncture points (Hong, 2000). Evidence now shows that MTPs are found where a muscle’s motor nerve branch enters the muscle and terminates in a number of endplates (Resteghini, 2006). These sites also have a neurovascular bundle that have large and small sensory nerves (Resteghini, 2006). The smaller sensory nerves have terminal nociceptors and blood vessels that are closely associated with autonomic nerve fibres (Resteghini, 2006). The proposed theoretical physiology behind MTPs will be discussed further in this paper. Some authors have laid out characteristics associated with MTPs such as 1) compression may elicit local or referred pain that is often familiar to the patient’s symptoms, 2) rapid compression or needling of the muscle taut band may elicit a local twitch response, 3)
restricted range of motion and increased sensitivity to stretch may cause tightness or shortening of the involved muscle, 4) the muscle involved may be weak due to pain and have little noticeable atrophy, 5) may be associated autonomic phenomena, and 6) the MTP can be active or latent (Resteghini, 2006).

Active MTPs are associated with pain at rest, acutely tender to palpation and may contribute to general motor dysfunction including decreased range of motion and stiffness (Borg-Stein & Simons, 2002; Shah et al., 2008; Shah, 2008). They can be identified through physical examination by gentle palpation across the direction of the muscle fibre and a ‘rope-like’ nodularity can be appreciated in the taut band of muscle (Borg-Stein & Simons, 2002). A local twitch response should always be elicited in active MTPs (Hong, 2000).

Latent MTPs may be associated with motor dysfunction, muscle tenderness, decreased range of motion, stiffness and no spontaneous pain (Borg-Stein & Simons, 2002; Shah et al., 2008; Shah, 2008). Some studies suggest that latent MTPs are present in shoulder-girdle muscles of 45-55% of asymptomatic young adults (Borg-Stein & Simons, 2002). Latent MTPs can be verified objectively using intramuscular electromyography which shows spontaneous electrical activity, or by ultrasound imaging which demonstrates a focal, hypoechoic region with reduced vibration amplitude (Ge & Arendt-Nielson, 2011). Latent MTPs differ from active MTPs in that they are not responsible for spontaneous pain (Ge & Arendt-Nielson, 2011). Latent MTPs may predispose the muscle to further damage and can easily transform into active MTPs under influence of precipitating factors in patients with chronic musculoskeletal pain syndromes (Ge & Arendt-Nielson, 2011). The first change from an unaffected muscle band to a tender, taut band is the development of a contracted, taut group of muscle fibres that can become painful with sufficient stress (Ge & Arendt-Nielson, 2011). It is common knowledge that a significant number
of adults who have latent MTPs only illicit pain with direct compression, not spontaneously (Resteghini, 2006).

**Physiology of MP and MTPs.**

There are a few key differences between muscle and cutaneous pain (Shah, 2008). Firstly, in muscle pain there is activation of muscle nociceptors which causes an aching or cramping pain that is diffuse, difficult to localize and can be referred to deep somatic tissues (Shah, 2008). MP also activates unique cortical structures (Shah, 2008). MP is inhibited more strongly by descending, pain modulating pathways (Shah, 2008). Finally, the activation of muscle nociceptors is more effective in inducing neoplastic changes in dorsal horn neurons (Shah, 2008).

**Peripheral sensitization.**

Muscle nociceptors can be activated in two different ways. The first way is mechanically by deforming the axonal membrane of the nerve endings (Shah, 2008). Or they can be chemically activated by release of sensitizing or pain producing substances from surrounding tissues and immune cells (Shah, 2008). Chemical activation is very interesting and is especially relevant to chronic pain where swelling does not occur (Shah, 2008). Bradykinin, prostaglandins, serotonin are substances that are not known as being sensitizing or activating for muscle nociceptors, but they can cause vasodilation which could lead to mechanical activation by changing or affecting the normal tissue relationship (Shah, 2008). However, when there is adequate amounts of bradykinin and serotonin, nociceptors can be directly activated (Shah, 2008). In a sensitized muscle nociceptor, the stimulation threshold is lowered to a level which could be triggered by every day, normal stimulation such as even light pressure or movement (Shah, 2008).
When a nociceptor is stimulated by a noxious stimulus, it releases stored neuropeptides such as substance P and calcitonin gene-related peptide (Shah, 2008). These substances cause vasodilation, plasma extravasation and release of sensitizing substances from the surrounding tissue (Shah, 2008). More importantly, the release of those neuropeptides in high enough levels causes a cascade which includes the release of histamine from mast cells, bradykinin from kallidin, serotonin from platelets and prostaglandins from endothelial cells (Shah, 2008). All of the above causes a production and release of sensitizing substances (Shah, 2008). Nociceptors play an important and vital role in maintaining normal tissue homeostasis by balancing vasoconstriction by the sympathetic nervous system and sensing the peripheral biochemical milieu (Shah, 2008). When there is injury to the tissue, secretion of substance P and calcitonin gene-related peptide occurs which causes vasodilation, plasma extravasation, local swelling and release of activating substances that could change responsiveness of a nociceptor (Shah, 2008). Activation of a nociceptor ending is not just due to damage of the nerve ending but also due to the binding of specific substances to their receptors on the muscle nociceptors (Shah, 2008). Studies have confirmed that the biochemical such as bradykinin, serotonin, prostaglandins, substance P and inflammatory biochemicals are significantly higher in muscles with MTPs and that this elevation is not limited to areas local to the MTPs (Shah et al., 2008). Overall research has indicated that subjects with active MTPs also have more inflammatory mediators and sensitizing substances present generally (Shah et al., 2008). The elevated levels of these substances support hypothesis that there are areas of relative local ischemia and hypoxia compared to normal muscle (Shah et al., 2008).

*Central sensitization.*
With regards to central sensitization, pain and dysfunction from MTPs occur due to altered responsiveness in the dorsal horn (Shah, 2008). Chronic MTPs may be the source of continuous noxious stimulation that sensitizes the dorsal horn neurons which could lead to more pain and, or the spread of pain (Shah, 2008). Or, the sensitized central nervous system could also lower the activation threshold of the peripheral nociception in the MTP which could cause a latent MTP to become an active one (Shah, 2008).

Once a peripheral nociceptor is sensitized, the group IV afferent nerve endings fire at a lower threshold and induce central sensitization which causes expanding of the receptive field in the dorsal horn (Shah, 2008). The expanded receptive field in the central nervous systems helps to explain why there are such unusual patterns of pain referral in MP, for example affected suboccipital muscles referring pain to the frontal region of the head (Shah, 2008).

In the dorsal horn there are effective and ineffective synapses (Shah, 2008). The effective synapses influence the post synaptic neuron and the ineffective ones do not influence the post synaptic neuron under normal circumstances (Shah, 2008). When there is an intense or continued input, substance P will be released with glutamate and these allow entrance of calcium into the cell which activates enzymes; previous ineffective synapses now have an effect (Shah, 2008). So enough substance P in the dorsal horn will increase the efficacy of synaptic connections in the spinal cord which supports the spread of noxious input (Shah, 2008).

Additionally, it has been suggested that areas with MTPs have multiple dysfunctional endplates and each endplate has associated muscle fibres which can contract to form a tender, taut band (Resteghini, 2006). The connection between the two has been termed the energy crisis theory (Resteghini, 2006). This theory hypothesizes that when there is an increase in acetylcholine production, triggered by precipitating factors, acetylcholine stimulates their receptors in the post
synaptic membrane of the neuromuscular junction and produces an increase in the number of endplate potentials, and the nerves of the neuromuscular junction become super sensitized to acetylcholine (Borg-Stein & Simons, 2002; Resteghini, 2006). The increase in the number of endplate potentials can be detected as spontaneous electrical activity and cause a sustained depolarization of the post synaptic membrane in the muscle fibres (Resteghini, 2006). Some authors do not call this spontaneous electrical activity but refer to it as end plate noise (Resteghini, 2006). Either way, these represent an abnormal end plate activity (Resteghini, 2006). The sustained depolarization causes a continuous release of calcium, causing a sustained shortening of the muscle (Resteghini, 2006). All of the above increase the demand for acetylcholine, cause a sustained depolarization, ongoing release and uptake of calcium, and sustained sarcomere shortening which leads to local increased demand for energy (Resteghini, 2006). In addition, the increased sustained muscle contraction could compress blood vessels and affect blood, oxygen and nutrient supply, further contributing and leading to an energy crisis (Resteghini, 2006). Local hypoxia, breakdown of the calcium pump, and failure to remove calcium could cause a sustained muscle contraction and shortening (Resteghini, 2006).

**Diagnostic approach to myofascial pain.**

MTPs should always be considered as a diagnosis in any musculoskeletal condition (Borg-Stein & Simons, 2002). A thorough history and physical assessment is key in the diagnosis of myofascial pain (Borg-Stein & Simons, 2002). Red flags for more serious musculoskeletal pathologies such as fractures, neurological deficits, malignancies and infection should be excluded (Yap, 2007). A careful medical, neurologic and musculoskeletal assessment should be performed (Borg-Stein & Simons, 2002). The affected areas should be inspected for asymmetry of posture and restriction of active and passive range of motion (Yap, 2007). Posture,
biomechanic and joint function should be assessed for precipitating or affiliated factors that may contribute to the local or regional pain (Borg-Stein & Simons, 2002). Often abnormalities to cervical posture, biomechanics, joint function, muscle strength and imbalances will be noted (Borg-Stein, 2004). The health care provider should be able to identify MTPs by palpation (Borg-Stein, 2004). Palpation is the basic method of diagnosing MP and MTPs (Yap, 2007). The MTP can be identified by gentle palpation across the direction of the muscle fibre and a ‘rope-like’ nodularity can be felt, usually palpation is painful and reproduces the patient’s pain pattern (Borg-Stein & Simons, 2002). To properly feel and locate the MTP and taut bands of muscle it is important to adequately relax the muscles that are in pain and spasm (Yap, 2007). Relaxation can be achieved mechanically by passively approximating the origin of the muscle to its insertion site or through neuromuscular techniques (Yap, 2007). Initially a flat palpation with the finger bellies can be performed as an initial survey for tone, spasm and superficial tenderness (Yap, 2007). The finger tips can be used to palpate across the muscle fibres for taut bands and MTPs (Yap, 2007). The fingers and thumbs can be used for a pincer palpation of the accessory muscle use. Finally, an algometer, which measures force, can be used on identified MTPs to measure minimum pressure needed to induce pain (Yap, 2007).

When considering differential diagnosis for muscle pain, four questions were suggested to gain an understanding of whether MP is contributing to the patient’s condition. 1) Is there regional MP with MTPs? 2) Is the MP the primary pain generator or are there other coexisting or underlying structural diagnosis? 3) Are there any nutritional, metabolic, endocrinological, psychologic, visceral, or inflammatory disorders that are contributing or causing the pain? 4) Is the pain widespread or are there other associated symptoms (Borg-Stein & Simons, 2002)? Differential diagnosis considerations should include joint disorders, inflammatory disorders,
neurological disorders, regional soft tissues disorders, discogenic disorders, visceral referred pain, mechanical stresses, nutritional, metabolic and endocrine disorders, psychologic disorders, infectious diseases and fibromyalgia, or widespread chronic pain (Borg-Stein & Simons, 2002). Some examples of the above are osteoarthritis, loss of normal joint motion, rheumatoid arthritis, bursitis, tendinitis, entrapment neuropathy, metabolic neuropathy, Vitamin B12 deficiency, alcoholic and toxic myopathy, hypothyroidism, electrolyte deficiencies, anxiety, depression, chronic hepatitis, bacterial or viral myositis (Borg-Stein & Simons, 2002). Examples of mechanical stresses are postural dysfunction, scoliosis, and poor body mechanics (Borg-Stein & Simons, 2002). Referred visceral pain can be gastrointestinal, cardiac, pulmonary or renal (Borg-Stein & Simons, 2002).

The work up for MP should include a complete blood count, erythrocyte sedimentation rate, chemistry panel, thyroid function, along with Xray, bone scans, computed tomography, magnetic resonance imaging. None of these diagnostic tools can diagnosis MP and MTPs but they can support clinical impression and it is important to use them to rule out other disease processes and causes (Borg-Stein, 2004). MP, MTPs and taut bands will not show on imaging investigations but they also provide useful anatomical investigations (Yap, 2007).

Some authors have proposed specific diagnostic criteria and suggested seven necessary features for a diagnosis (Shah, 2008). Firstly, there must be localized pain in a taut band of muscle. Second, there must be a local twitch response to cross fiber stimulation of the taut band. Third, pain to deep palpation that is recognized as pain. Fourth, referred pain to characteristic distant regions based on myofascial maps. Fifth, there is restricted movement of joints related to the muscle. Six, weakness is present that is not caused by a neurological component. Seventh, there is anatomic dysfunction.
Pharmacological interventions.

Pharmacologic drug categories which have been used to address MP are nonsteroidal anti-inflammatory drugs (NSAIDs), Tramadol, antidepressants, alpha 2 adrenergic agonists, anticonvulsants and Botulinum toxins (Borg-Stein & Simons, 2002; Borg-Stein, 2004; Yap, 2007). There is a minimal amount of literature considering the use of NSAIDs for MP or chronic muscle pain. Some studies have found that when NSAIDs are used in combination with other drugs (alprazolam, amitriptyline or cyclobenzaprine) there is a small supplemental benefit for fibromyalgia pain (Borg-Stein & Simons, 2002). Interestingly, even though there is scarce amount of evidence for use of NSAIDs it continues to be a very popular choice for patients and clinicians (Borg-Stein & Simons, 2002). Tramadol has several studies supporting its efficacy in fibromyalgia, chronic low back pain and osteoarthritis, which are sometimes seen in co-existence with MP (Borg-Stein & Simons, 2002). Tramadol is a combination of a weak opioid agonist and an inhibitor or reuptake of serotonin and norepinephrine (Borg-Stein & Simons, 2002). However, there are no published randomized control trials of Tramadol use for MP (Borg-Stein & Simons, 2002). With regards to antidepressants, Tricyclic antidepressants have been found to be effective for chronic tension type headache, fibromyalgia, acute lower back pain and intractable pain syndromes with muscle spasms (Borg-Stein & Simons, 2002). Efficacy of select serotonin reuptake inhibitors for fibromyalgia for improving pain, sleep and global sense of well-being has been established, however use of select serotonin reuptake inhibitors specifically for MP has not been studied (Borg-Stein & Simons, 2002). Tizanidine, an alpha-adrenergic agonist, was studied in a mix population of patients with MP and fibromyalgia, and was found to reduce pain. In other studies evaluating efficacy in treating acute low back pain and neck pain, it was also found to have been useful (Borg-Stein & Simons, 2002). However, there are no double-blind placebo
controlled studies of Tizanidine in treatment of MP (Borg-Stein & Simons, 2002). For anticonvulsants, there have been no controlled trials for treatment of MP (Borg-Stein & Simons, 2002).

Botulinum toxin is a promising agent for chronic MP syndromes. One small randomized, double-blind, placebo controlled trial study found that it demonstrated a 30% reduction in pain, palpable muscle firmness and pressure pain thresholds (Borg-Stein & Simons, 2002). Another study found that compared to injections of steroids, Botulinum toxin was found to provide greater improvement for MP (Borg-Stein & Simons, 2002). Other studies comparing botulinum toxin to placebos were unable to find a significant difference in outcomes (Borg-Stein & Simons, 2002). Overall, the clinical evidence studying pharmacological drugs and their efficacy on MP appears to be lacking or do not show significant efficacy in treating MP. However, some of these medications may be useful in addressing impacts of MP such as emotional distress or sleep deprivation, or addressing precipitating or contributing factors such as osteoarthritis, etc. (Borg-Stein & Simons, 2002; Yap, 2007).

**Non-pharmacological interventions.**

There are multiple different non-pharmacological approaches to addressing MP and MTPs such as postural, mechanical and ergonomic modifications; stress reduction; acupuncture; massage, TENS, ultrasound and heat; stretch; and trigger point DN (Borg-Stein & Simons, 2002; Borg-Stein, 2004; Yap, 2007).

Postural, mechanical and ergonomic modification therapeutic approaches to MP have little data to support their efficacy (Borg-Stein & Simons, 2002; Borg-Stein, 2004; Yap, 2007). Occupational medicine literature presents evidence that injuries are more common when there is greater loads of weight and undesirable posture are held (Borg-Stein & Simons, 2002).
Occupational muscle pain syndromes are hypothesized to occur as a result of repetitive microtrauma and myofascial shortening therefore correction of awkward postures is a baseline treatment, but the data regarding long term efficacy is lacking (Borg-Stein & Simons, 2002). Stress reduction strategies are approaches such as cognitive behavioural therapy, meditation, progressive relaxation training and biofeedback (Borg-Stein & Simons, 2002; Borg-Stein, 2004; Yap, 2007). These approaches are usually incorporated into chronic pain management programs (Borg-Stein & Simons, 2002). There are a few studies which analyze their efficacy and often the studies examine combined treatment therapies (Borg-Stein & Simons, 2002).

Acupuncture as a therapeutic approach has growing evidence for its success in MP and fibromyalgia (Borg-Stein & Simons, 2002). The limited, high quality studies which have examined this therapeutic option suggest that real acupuncture is better than sham for relieving pain (Borg-Stein & Simons, 2002). It has been used as an adjunct to treat fibromyalgia, MP, lower back pain, osteoarthritis and lateral epicondylitis (Borg-Stein & Simons, 2002). It was found to be better than NSAID treatment and sham acupuncture (Borg-Stein & Simons, 2002). Future studies need to address the topics of optimal acupuncture techniques, duration of benefit and booster treatments (Borg-Stein & Simons, 2002).

Massage, TENS, heat and ultrasound all show conflicting and mixed evidence for their efficacy in addressing MP (Borg-Stein & Simons, 2002; Borg-Stein, 2004; Yap, 2007). Very few studies on massage suggest it is an effective treatment option for MP or MTPs (Borg-Stein & Simons, 2002; Borg-Stein, 2004; Yap, 2007). One study found that it, in combination with stretch was better compared to the control group (Borg-Stein & Simons, 2002). TENS works to decrease inflammatory byproducts at painful sites by increasing vascular circulation, and also aides with muscle spasm and edema (Yap, 2007). It has also shown mixed results for addressing
MP; some studies have found no benefit while others have found that it reduced MP (Borg-Stein & Simons, 2002). Heat therapy is a commonly used approach, it works by increasing blood flow and tissue distensibility and decreased muscle spasm (Yap, 2007). Ultrasound can provide deep heat, but clinical studies have not shown any benefit (Borg-Stein & Simons, 2002; Yap, 2007).

Stretching is the basis for MP treatment as it can address muscle tightness and shortening which are associated with MP (Borg-Stein & Simons, 2002; Borg-Stein, 2004; Yap, 2007). It works towards gradual improvement of range of motion and return to normal activity (Borg-Stein & Simons, 2002). Slow, sustained stretching during range of motion is the best approach (Borg-Stein & Simons, 2002). Patients should stay active and perform daily activities in a lightly, loaded manner (Borg-Stein & Simons, 2002). If a movement leads to pain, the movement should be stopped and the patient should try to gently extend the movement to help relieve any muscle tightness (Borg-Stein & Simons, 2002). Strengthening exercises are contraindicated (Borg-Stein & Simons, 2002). Electromyographic studies have shown that muscles with active MTPs are fatigued, fatigue more quickly and easily, and recover more slowly (Borg-Stein & Simons, 2002). Strengthening exercise could overload the muscle, cause pain, worsen the MTP and result in substitution of other muscles (Borg-Stein & Simons, 2002). Once the MTP pain has decreased and range of motion is back to baseline, a gradual program of strengthening and stabilization should be started (Borg-Stein & Simons, 2002).

Overall there are no clear treatment guidelines for pharmacological and non-pharmacological therapeutic approaches to MP and MTPs. Clinical evidence is lacking or shows poor efficacy for many of the above proposed non-pharmacological therapeutic interventions. Several of the therapeutic options appear to be used in combination. Trigger point DN is one option that appears to be promising and will be addressed in this paper.
Dry Needling

Dry needling (DN) is a form of therapy used to treat different painful musculoskeletal conditions, usually MP (Zhou, 2015). It is considered to be different than acupuncture. Traditional acupuncture and western medicine acupuncture both have a broader range of indications than DN, because they address more than just musculoskeletal pain such as gastrointestinal and neurologic disorders (Zhou, 2015). An earlier verified study on DN in PubMed, performed by Lewit in 1979, found that DN had a complete, analgesic effect immediately on painful spots (Zhou, 2015). He claimed that this therapeutic effect was not a result of the anesthetic solution injected but from the mechanical stimulation of the needle (Zhou, 2015). DN of the muscle has been found to increase muscle strength and improve range of motion (Zhou, 2015).

What is dry needling.

DN appears to be an effective treatment option for MTP pain relief at various points of time and in various areas of the body (Boyles et al., 2015; Shah, 2008). It has been found to be more effective than stretching, TENS, and equally efficacious as compression and other needling options (Boyles et al., 2015). DN of MTPs provides as much pain relief as wet needling using injection of lidocaine, but DN has been found to result in more post needling soreness (Borg-Stein & Simons, 2002). One of the aims of DN is to break up MTPs and any associated fibrotic core (Yap, 2007). There is conflicting evidence within the literature about the necessity of a local twitch response for successful treatment (Boyles et al., 2015). Theoretically, a local twitch response would be important as it confirms accurate needle placement in a MTP (Boyles et al., 2015).
Travell and Simons have listed 2 key elements of DN. The first is that the needle diameter ranges from 0.4-3.4 mm and the second that aseptic technique is used (Zhou, 2015). They also suggest that DN should be inserted into multiple sites over the whole affected region in order to eliminate tenderness in the whole MTP region (Hong, 2000). This differs from newer studies which focus on eliciting a local twitch response indicating the actual MTP has been found and is the focus. Usually, solid filiform needles or hollow core hypodermic needles are used for DN (Zhou, 2015). Techniques for DN can be divided into superficial and deep (Zhou, 2015). In superficial DN, needles are inserted to a depth of 5-10 mm above the underlying trigger points (Zhou, 2015) and the needles are inserted for 10 seconds to three minutes. Some superficial techniques insert the needle at a 20 to 30-degree angle, without penetrating the muscle (Zhou, 2015). When used over the wrist or ankle, the needles are inserted almost horizontally into the connective tissues between the muscle and the skin (Zhou, 2015). In deep DN, the needles are inserted perpendicularly and deeply towards the MTPs to reach them. The needles are often manipulated in and out to reach the MTPs, and elicit a local trigger response (Zhou, 2015). Usually, weekly treatments for three to four weeks are needed (Zhou, 2015).

**Adverse reactions and contraindications to DN.**

Overall, DN has been found to be a safe therapeutic option for MP (Boyles et al., 2015; Zhou, 2015). One study found the most common adverse effects of DN are bruising, bleeding and pain (Boyles et al., 2015). These adverse reactions are considered mild as they are short term, do not require any treatment and only occur approximately 20% of the time (Boyles et al., 2015). Moderate to severe adverse events are fainting, headache and nausea which occur less than 0.04% of the time (Boyles et al., 2015). Other additional associated risk are hematoma, infection, pneumothorax and nerve lesions (Boyles et al., 2015). Contraindications to DN are
bleeding disorders, anticoagulation therapy, local or systemic infections, and the inability to rest the treated region after the procedure (Yap, 2007).

**Physiology of DN.**

There is emerging research on the physiologic effects of DN, but the exact mechanism of deactivation of MTPs is unknown (Cagnie et al., 2013). Many sources speak to disrupting the endplate, however there is no basic research demonstrating this (Cagnie et al., 2013). There have been many proposed hypotheses as to how DN affects MTPs and MP (Borg-Stein & Simons, Boyles et al., 2015; 2002). One proposed hypothesis is that DN hyper-stimulates the pain generating area and thereby normalizes the local sensory inputs (Boyles et al., 2015). Another hypothesis is that DN stimulates local alpha-delta reserve fibers which cause natural opioid mediating pain suppression (Boyles et al., 2015). A third hypothesis is that DN causes inhibitory interneurons to be stimulated which prevents normal pain transmission to the sensory cortex (Boyles et al., 2015). Finally, there is evidence that DN may correct the altered chemical milieu found at MTPs (Boyles et al., 2015).

One of the most common theories in the literature is that when the needle enters the muscle tissue, it disrupts the cell membrane of individual muscle fibres and causes a discharging, which is called ‘insertional activity” (Cagnie et al., 2013; Resteghini, 2006). This would cause a decrease in spontaneous electrical activity and then a relaxation from the direct local electrical stimulus or a reflex mechanism (Cagnie et al., 2013; Resteghini, 2006). The result is that the shortened muscle fasciculate and relaxes (Cagnie et al., 2013; Resteghini, 2006). It has been suggested that the needle insertion in the endplate zone may lead to increased discharge and decrease the amount of available acetylcholine which chases a decrease in the spontaneous electrical activity (Cagnie et al., 2013). Or, another theory is that the discharge may elicit a local
twitch response which could cause a change in the length and tension of the muscles, stimulating mechanical receptors (Cagnie et al., 2013). When the needle is rotated within the muscle, it can produce an intense stimulation, and the insertion is converted to a linear motion which shortens the muscle fibres locally which causes activation of the muscle spindles and Golgi tendon organs, leading to more muscle relaxation via local spinal reflexes (Resteghini, 2006).

It has also been suggested that DN may increase the muscle blood flow and oxygenation, possibly by the release of vasoactive substances which cause an axon reflex and increase vasodilation (Cagnie et al., 2013). One study found that angiogenesis, vasodilation and altered glucose metabolism in hypoxic tissues is treated (Cagnie et al., 2013).

**Clinical Evidence for DN in the Trapezius Muscle**

In recent years, there has been an increased interest in DN as a therapy for MP and MTPs. A literature review was completed on the current research for use of DN in the trapezius muscle for MP and MTPs. Databases used to search for literature were CNAHL, PubMED, MEDLINE and Web of Science. Search terms used were “dry needling”, “dry needling therapy”, “trigger point needling”, “intramuscular stimulation therapy”, “trapezius”, “trapezius muscle” and “trapezius muscle pain”. From the search results, studies were eliminated and selected based on title, abstract, publication date and appropriateness to topic. The studies that were examined, and to be discussed were largely published between 2013 and 2017, with some dating back to 2007.

Of the research examined, there was a variety of different types of studies such as single and double blinded randomised control trials, interventional studies with and without control groups, case studies and prospective studies. DN was examined in combination with and against
many other different management strategies or interventions such as stretching, cryotherapy, and compression; at and outside trigger points, and with needle tail heating.

DN was examined without any comparison group in several studies (Abbaszadeh-Amirdehi, Ansari, Naghdí, Olyaei & Nourbakhsh, 2017; Gerber et al., 2015; Gerber et al., 2017; Mejuto-Vazquez, Salom-Moreno, Ortega-Santiago, Truyols-Domínguez & Fernández-de-Las-Penas, 2014). All of these studies found that DN had a significant effect on outcomes which included pain intensity, pressure pain threshold and range of movement. Two of the studies also examined the effect of DN on MTP status and found that DN had a positive effect on the status of latent or active MTPs (Gerber et al., 2015, Gerber et al., 2017).

Another management strategy that DN was compared against was stretching and cryotherapy. In one study, DN and stretch was compared to stretch and cryotherapy (Priyanki & Tilak Francis, 2017). Another study compared DN and passive stretching against just passive stretch (Cerezo-Tellez et al., 2016). Both studies found that DN and stretch was superior and more effective in meeting or addressing study outcomes compared to just passive stretch or cryotherapy and stretch, and in addition had higher levels of patient satisfaction. Both studies examined pain intensity using the verbal scale analogue (VAS) and range of motion as their outcomes, and Cerezo-Tellez et al. (2016) also examined muscle strength. Cerezo-Tellez et al. (2016) considered the effect over three weeks, while Priyanki and Tilak Francis (2017) examined a longer period over 10 treatments.

A couple of studies compared DN and compression of MTPs (Ziaeifar et al., 2014; Ziaeifar, Arab & Nourbakhsh, 2016). Both found that DN had a greater effect on measured outcomes than compression. The outcomes that were measured by both were pain intensity and pressure pain threshold. The study by Ziaeifar et al. (2014) evaluated over three treatments while
the study done by Ziaeifar, Arab and Nourbakhsh (2016) measured immediately after each treatment and 48 hours later. One study by Martin-Pintado-Zugasti et al. (2015) examined if compression after DN helped with intensity of post needleling soreness. The studies found that compression did help with duration and intensity of post needleling soreness, and that DN and compression also seem to improve range of motion. These outcomes were measured periodically up to 72 hours after a treatment.

Several other studies examined DN in unique ways. One study by Pecos-Martin et al., (2015) compared effectiveness of DN in MTPs to DN in the same muscle but not at the MTP. They measured VAS, pressure pain threshold and degree of disability for three treatments. Overall, they found DN in MTPs to have greatest effect on measured outcomes compared to DN not at MTPs. Another study compared DN to intramuscular stimulation (IMS) which is the addition of needling in the paraspinal muscle (Ga, Choi, Park & Yoon, 2007). This study found that IMS had a more continued effect on pain and decreased rates of depression. A study by Wang, Gao, Hou and Li (2014) examined DN with needle tail heating and just DN. They measured VAS for pain and pressure pain threshold, and found that DN was effective overall, while DN with needle tail heating had even better effects on pain and in the long term. They assessed outcomes at baseline, 7 days, 1 month and 3 months post treatment.

Another study examined DN in the trapezius muscle not just for pain but also to analyze the effect of DN on oxygenation (Jimbo, Atsuta, Kobayashi & Matsuno, 2008). They found that DN decreased pain on the day of treatment and the day after. It did not improve oxygenation immediately but there was improved muscular aerobic capacity the next day. However, it is important to note that they used ‘tender points’ which could be MTPs but were not specified as such.
Finally, one case study of DN in the trapezius muscle found that DN in the trapezius muscle decreased pain and disability for the individual (Pavkovich, 2015). For this individual, it did not improve range of motion but this outcome was expected as the patient had had previous spinal fusions which affected her range of motion.

Most of these studies evaluated the effect on their measured outcome immediately or shortly after the treatment. A couple considered the outcomes a few weeks to a month after treatment or followed the patient over multiple treatments (Cerezo-Tellez et al., 2016; Gerber et al., 2015; Gerber et al., 2017; Priyanki & Tilak Francis, 2017). Only one study followed the patient for longer periods of time and that was the study by Wang et al. (2014) which assessed 3 months after treatment.

To summarize, DN was found to be a superior option as a therapeutic treatment in addressing MP and MPTs. In all of the studies examined, some combination of pain intensity, pressure pain threshold and range of motion was used as primary outcomes to evaluate the therapeutic effectiveness of DN on MP and MPTs. DN was compared individually against no treatment, individually against other non-pharmacologic management strategies, or in combination with non-pharmacologic management strategies against just the individual non-pharmacologic management strategy. In each scenario, DN was found to have positive effect pain intensity and pressure pain threshold, and improved range of motion which indicates that DN is a good therapeutic treatment option for DN and MTPs.

There are several limitations within the individual studies and with the collection of information that can be concluded from them. While several of the studies were randomised controlled trials with placebo and blinded conditions there were several that were not. As double blinded, randomized controlled trials are considered to be the strongest level of evidence, the
conclusions of the studies using other designs are not considered as strong. Overall, the long-term efficacy and effects of DN on MP and MTPs were not explored as the majority of the studies only evaluated the effects and outcomes of DN for an immediate or short-term effect. In general, there is no standardized procedural application of DN which means that each study likely used different methods, procedures and approaches to DN which could have an effect on the results. Some of the sample populations from the different studies affect the conclusions’ generalizability and transferability, and therefore the external validity of those results. For example, Gerber et al. (2017) used only university students and many of the studies had quite small sample sizes (Jimbo et al., 2008; Maher, Hayes & Shinohara, 2013). Finally, there were many variables between the different studies, for example, some examined DN with no control or comparison group, some combined or compared it with stretch, cryotherapy, pressure, etc.

**Description of Project**

To address NPs lack of knowledge regarding MP and DN, an article for publication will be submitted to The Journal of Nurse Practitioners. The article will be written to fit either as a “Case Challenge” or “Clinical Features” article. Through this article, knowledge on MP and MTPs, clinical signs and symptoms, the diagnostic reasoning process, and evaluation and management strategies with a focus on DN will be disseminated. This publication was selected because the target population for this project is NPs and this journal is a key publication for the Nurse Practitioner profession. The mode of using an article to disseminate knowledge was selected as it has the potential to reach a large number of relevant people. One weakness of a manuscript publication is that it will be difficult to assess if the goals of this project, to address a knowledge gap, will be met.
Conclusion

To conclude, MP is a common complaint yet is often underdiagnosed and under treated (Shah et al., 2008; Borg-Stein, 2004; Yap, 2007). It is caused by the development of active or latent MTPs which are hyperirritable nodules within a taut band of skeletal muscle (Borg-Stein & Simons, 2002; Shah et al., 2008). MTPs are associated with pain at rest, tenderness with palpation and can affect motor function causing decreased range of motion and increased stiffness (Borg-Stein & Simons, 2002; Shah et al., 2008; Shah, 2008). They can be identified with gentle palpation, and a thorough medical, neurological and musculoskeletal assessment should be completed (Borg-Stein & Simons, 2002). Imaging will not show MP or MTPs but should be conducted, along with other investigations, to exclude other disease processes and causes (Borg-Stein, 2004).

There are several different pharmacological approaches to MP. The most commonly used approach is NSAID therapy, although there is minimal amount of literature addressing their effectiveness in treating MP (Borg-Stein & Simons, 2002). Use of Trizanidine and Botulinum toxin are promising therapies for MP but more evidence is needed their use and efficacy (Borg-Stein & Simons, 2002). Finally, antidepressants and Tramadol do not have literature exploring their use for MP, but may address complaints that commonly exist simultaneously with MP such as osteoarthritis, chronic low back pain, sleep, and chronic tension headaches (Borg-Stein & Simons, 2002).

There are several non-pharmacological approaches to MTPs such as postural, mechanical and ergonomic modifications, stress reduction, massage, TENS, US, heat, stretch, exercise and DN. Majority of the approaches have little or weak evidence to support their use for MP (Borg-Stein & Simons, 2002). Massage has a few studies which support its use as a solo therapy or in
combination with stretch (Borg-Stein & Simons, 2002; Borg-Stein, 2004; Yap, 2007). The evidence regarding TENS has shown mixed results in its use for MP (Yap, 2007). Stretch is the basis of MP treatment and works to address muscle shortening (Borg-Stein & Simons, 2002). Based on the literature reviewed, DN, which is the insertion of a needle into the MTP to elicit a local twitch response, appears to be the most effective treatment for MP. With regards to MTPs in the trapezius muscle, literature is showing that DN is an effective treatment option. This indicates that for patients with MP and MTPs in the trapezius muscle, DN is an effective treatment option that can be offered.

The goal of this project is to provide NPs with a better theoretical and clinical understanding of MP and MTPs, how to diagnose MP, what the different treatment options are, and specifics about the use and efficacy of DN as a treatment option for MTPs in the trapezius muscle. The increased knowledge and understanding will address the problem of under diagnosis of MP and lead to effective management strategies for it.

Limitations of this project and the current evidence means that further research needs to be completed on DN therapy to add to strengthen level of evidence with further replication of current evidence. Additionally, to support some of the studies comparing DN to other MP treatment options or considering DN in combination with other treatment options, further research on the use of DN in combination therapy or as a solo therapy over other options is also needed. Finally, the long-term effects of DN on pain and range of motion outcomes can be further researched and explored. However, due to the nature of DN as an intervention, it could be challenging to design a double blinded randomized controlled trial study which would provide the highest level of evidence strength.
References


