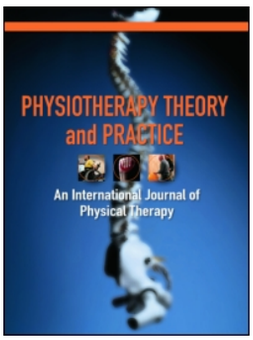

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Dry needling equilibration theory: A mechanistic explanation for enhancing sensorimotor function in individuals with chronic ankle instability

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ABSTRACT

Patients with chronic ankle instability (CAI) experience a dynamic interplay between impaired mechanical structures and sensorimotor deficiencies that contribute to recurrent sprains and sensations of instability. Concomitantly, muscular trigger points (MTrPs) are known to occur following trauma, maximal or submaximal concentric contractions, and unaccustomed eccentric loads. Additionally, MTrPs are theorized to be exacerbated in low-load and repetitive strain activities. MTrPs located within a muscle are associated with altered motor control, reaction delay, and decreased strength, deficits also found among those with CAI. Dry needling (DN) is reported to improve muscle range of motion, motor control, and pain in a myriad of neuromusculoskeletal conditions by decreasing spontaneous electrical activity and stiffness of taut muscle bands while improving filament overlap. Building on evidence supporting neuromechanical decoupling in chronic ligamentous injury with what is known about the development of MTrPs, this paper proposes a centrally mediated mechanism for improved sensorimotor function following DN for individuals with CAI. Dry needling equilibration theory (DNET) states that proprioception is improved following DN in the lower extremity by changing the muscle's length–tension relationship and leveraging minor acute discomfort to improve muscle spindle afferent information via the gamma motor system. The application of DNET for individuals with CAI may provide a mechanistic explanation for improved descending cortical output, resulting in enhanced sensorimotor function.

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Introduction

Ankle sprains are generally considered innocuous injuries; however, more than 40% of the ankle sprain patients will report at least one moderate-to-severe symptom in the 6–18 months following the initial injury. Further, as many as 74% of the patients develop chronic symptoms (Anandacoomarasamy and Barnsley, 2005; Braun, 1999; Konradsen, Bech, Ehrenbjerg, and Nickelsen, 2002). Chronic ankle instability (CAI) is defined by a history of ankle sprain accompanied by ongoing bouts of ankle instability, residual ankle sprain symptoms, and a decrease in patient-perceived function (Gribble et al., 2013). Aside from the disability experienced following acute ankle trauma, CAI has several long-term consequences including an increased risk of ankle osteoarthritis, limited physical activity, and diminished health-related quality of life (Al-Mahrouqi, MacDonald, Vicenzino, and Smith, 2018; Houston, Van Lunen, and Hoch, 2014; Saltzman et al., 2006; Wikstrom and Anderson, 2013; Wikstrom, Hubbard-Turner, and McKeon, 2013). Efforts to advance care for individuals with CAI have evolved from caring for simple anatomic laxity to the dynamic interplay between mechanical and sensorimotor impairments (Hiller, Kilbreath, and Refshauge, 2011).

Sensorimotor deficits, involving the wholesale integration of afferent information and efferent execution, following ligamentous ankle injuries were first described in the 1960s by Freeman, who described an "articular deafferentation" (Freeman, 1965; Freeman and Wyke, 1967) and further studied by Riemann and Lephart (2002a). Freeman (1965) asserted that peripheral mechanoreceptors were damaged following injury, thus impacting the cortex's ability to accurately detect the joint's location in space and enact a coordinated response. Based on Freeman's work, afferent feedback from articular structures also known as proprioception was believed to influence efferent responses creating a feedback loop. In chronic ligament injury, recent evidence reports evidence of neuroplastic alterations which may be primarily responsible for impaired physical performance and patient-reported dysfunction (Grooms, Appelbaum, and Onate, 2015).

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Articular and cutaneous receptors of the foot and ankle are some peripheral receptors that feed valuable proprioceptive information to the central nervous system (CNS) and have been found to be degraded in individuals with CAI (Freeman, 1965; Powell, Powden, Houston, and Hoch, 2014). While these receptors contribute valuable proprioceptive information, research has determined that disabling articular or cutaneous peripheral receptors near the ankle cannot reliably account for all the deficits in postural control seen in individuals with CAI (Feuerbach, Grabiner, Koh, and Weiker, 1994; Konradsen, Ravn, and Sorensen, 1993; McKeon et al., 2010). Additionally, research on inhibition of articular and ligamentous mechanoreceptors have been found to have no effect on muscle activation (Stone et al., 2013).

Proprioceptive information from muscles and tendons are an influential source of sensorimotor control (Konradsen, 2002). The muscle spindle is an extra-articular receptor that has been described as the "final common input" of proprioceptive information as it also incorporates feedback from cutaneous and articular mechanoreceptors (Johansson, Sjolander, and Sojka, 1991). Increased appreciation for the role of the muscle spindle has evolved the understanding of postural control to emphasize a feedforward mechanism that prepares joints for interaction with the environment along with the integration of feedback from the environment. This feedback-feedforward understanding places unique value on ascending and descending influences on the muscle spindle.

Muscle spindles can detect changes in static and dynamic muscle length through the gamma motor neuron. As a muscle shortens, the gamma motor neuron increases its firing rate to maintain an appropriate calibration with the extrafusal muscle fibers to ensure accurate information regarding muscle length. The need to increase input to the gamma motor neuron is conveyed to the spinal cord via Iα and type II (dynamic and static changes, respectively) afferent fibers from the muscle spindle. These fibers work in concert to create a monosynaptic reflex arc with the alpha motor neuron innervating the corresponding muscle as noted in the patellar tendon reflex. Additionally, this reciprocal relationship between the alpha and gamma motor neurons provides continuous feedback and normal and necessary tone to support joint stability in healthy systems (Riemann and Lephart, 2002b). A loss of calibration of the muscle spindle would result in misinformation to the spinal cord and supraspinal levels. Advances in neuroplasticity illustrate that motor output is directly influenced by sensory input. Faulty afferent information will ultimately result in dysfunctional motor output, creating or perpetuating chronic dysfunction (Needle, Lepley, and Grooms, 2017).

Muscular trigger points (MTrPs) are commonly found in both healthy and injured populations and are quantified by a tight band of skeletal muscle and a centrally sensitive nodule or locus that has reduced pain pressure thresholds (Lucas, Rich, and Polus, 2010). MTrPs may be characterized as active (ATrP) or latent (LTrP) depending on their unique characteristics. ATrPs spontaneously produce pain and are most associated with myofascial pain disorders, whereas LTrPs are pain-free unless compressed (Simons, Travell, and Simons, 1999). In the lower extremity alone, a recent study evaluating the presence of LTrPs 206 asymptomatic individuals, 77% had at least one trigger point with the average number of LTrPs being 7.5 ± 7.7, identified with excellent intra-rater reliability (Zuil-Escobar, Martinez-Cepa, Martin-Urrialde, and Gomez-Conesa, 2016). The presence of MTrPs suggests sensorimotor dysfunction as MTrPs are associated with many of the same symptoms of CAI and may be developed through the eccentric loading and repetitive strain that define CAI. MTrPs and CAI share characteristics such as altered spinal reflex excitability, central sensitization, altered muscular activation patterns, decreased strength, and increased reaction times (Ge, Monterde, Graven-Nielsen, and Arendt-Nielsen, 2014; Ge et al., 2009; Hoch and McKeon, 2014; Ibarra et al., 2011; Jaber et al., 2018; Kim, Ingersoll, and Hertel, 2012; Lucas, Rich, and Polus, 2010; Morihisa, Eskew, McNamara, and Young, 2016; Yassin et al., 2015). Therefore, MTrPs may contribute to the sensorimotor deficits found in individuals with CAI and be subsequently improved with their resolution.

Dry needling (DN) is considered a treatment for MTrPs that we propose may also affect local muscle spindles. DN is hypothesized to alter the excitability of the muscle spindle through improved muscle filament overlap, a known modulator of the gamma motor neuron (Knutson, 2000). The ability to improve muscle filament overlap and the quality of alpha–gamma co-activation would optimize proprioceptive control of the ankle, resulting in improved postural control. The authors propose dry needling equilibration theory (DNET) as a theoretical framework describing a centrally mediated mechanism responsible for improved sensorimotor function following DN treatment to MTrPs in the lower extremity. The purpose of this paper is to propose DN as a potential strategy for addressing sensorimotor deficits associated with CAI. It is hypothesized that patient outcomes for individuals with CAI may be improved through the clinical application of DN to create plastic cortical changes.

Literature review

Sensorimotor plasticity in CAI

Neuromechanical decoupling describes the loss in calibration between the nervous system and the mechanical properties of the surrounding muscle (Needle et al., 2014a). This occurs when the intrafusal muscle spindle and the extrafusal fibers in which it resides lose sensitivity to one another following a sudden stretch-type injury. As a result, accurate proprioceptive signaling that indicates the length and speed of muscle stretch no longer ascends to the CNS appropriately. This is believed to occur in individuals with CAI, ultimately resulting in decreased afferent traffic and which leads to impairments in motor control (Figure 1) (Needle et al., 2014a, 2013a). As the muscle spindle is known as the "final common input" of sensory information to the CNS, the loss of accurate sensory information will deprive higher centers from the ability to deliver appropriate feedback and feedforward motor control (Johansson, Sjolander, and Sojka, 1991; Needle, Lepley, and Grooms, 2017). A considerable body of research links increased chemonociceptors, gamma motor activity, and increased Ia and II muscle spindle output that have been proposed to create paraspinal muscular hypertonicity, sensitivity to pressure, and altered range of motion, all characteristics also associated with MTrPs (Knutson, 2000). Following injury, the nervous system undergoes a new Hebbian-type learning, based on degraded information from faulty receptors that ultimately induces neuroplastic change. Neuromechanical decoupling may help to explain why individuals respond in a myriad of ways following ligamentous injury, as the following experience depends more on the adaptations of the nervous system than it does on the mechanical properties supporting the joint.

Centrally mediated changes occur in conjunction with the development of CAI. Functionally, individuals with CAI exhibit altered motor control patterns which commonly manifest as deficits in postural control (Harkey et al., 2016; Hertel and Olmsted-Kramer, 2007; Jaber et al., 2018). Peripherally, individuals with CAI demonstrate increased nerve conduction latencies and decreased plantar foot cutaneous and vibrotactile sensation (Hoch and McKeon, 2014; Hoch, McKeon, and Andreatta, 2012; Powell, Powden, Houston, and

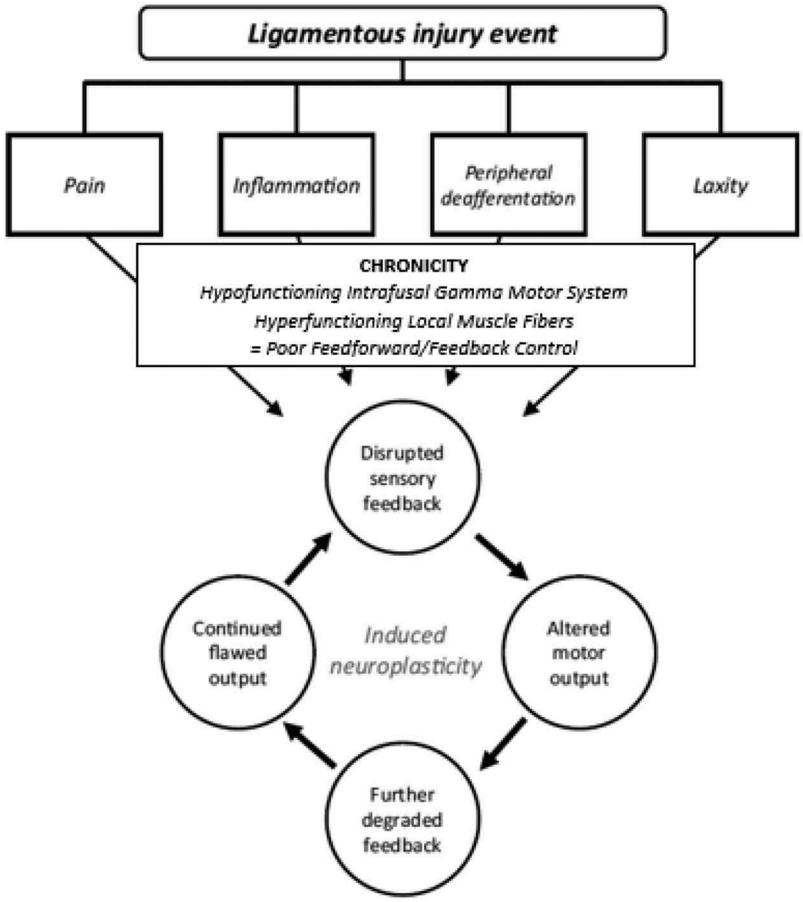

Figure 1. Theorized paradigm of the role of ligamentous injury for inducing neuroplasticity and its effects on sensorimotor function (Adapted from Needle, Lepley, and Grooms 2017).

Hoch, 2014). Centrally, individuals with CAI demonstrate blunted spinal reflex excitability and somatosensory cortex activity with smaller motor somatotopic representations (Harkey et al., 2016; Kim, Hart, Saliba, and Hertel, 2016; Kosik et al., 2017; Needle et al., 2014b). Additionally, individuals with CAI have been found to have increased cortical resting motor thresholds and dampened corticospinal excitability (Hoch and McKeon, 2014; Palmieri-Smith, Hopkins, and Brown, 2009; Pietrosimone and Gribble, 2012). Individuals with CAI experience neuroplasticity throughout proprioceptive systems with few interventions that seek to directly affect these neuromechanical aberrations.

Neurology of a trigger point

Theories describing the probable mechanisms underlying the physiology of MTrP genesis have been developing since 1981 (Hubbard and Berkoff, 1993; Simons, 1981). Evidence using both electrodiagnostic and histopathologic methods have been used to illustrate the presence of damaged motor endplates near MTrPs that release excessive ACh. These result in a cascade of negative effects on local musculature that include local sarcomere contracture, ischemia, increased metabolism, energy crisis and nociception sensitization that have been detected by these methods (Shah and Gilliams, 2008; Simons, Travell, and Simons, 1999). In 2004, the Expanded MTrP Hypothesis built on previous MTrP genesis theories and utilized additional evidence to include damaging effects of hypoxia, the presence of miniature endplate potentials, along with peripheral and central sensitization found in active MTrPs (Gerwin, Dommerholt, and Shah, 2004). Additionally, most recent evidence suggests that the abnormal endplate noise found to be associated with MTrPs is ultimately the result of dysfunctional muscle contraction or injury that increases sympathetic nervous system activity and ACh release (Liu et al., 2017). Chemical mediators at the motor endplate are known to increase ACh effectiveness through the inhibition of acetylcholinesterase and ACh receptor upregulation. These result in miniature end plate potentials that induce sarcomere contraction and the taut muscular bands that are characteristic of MTrPs (Gerwin, Dommerholt, and Shah, 2004). While a causal relationship has yet to be secured, recent research has demonstrated the ability to create an MTrP in rat and mouse models by increasing neurotransmission through the administration of an anticholinesterasic agent (Margalef et al., 2019). These experimentally generated MTrPs exhibited local glycosaminoglycans, palpable taut bands, contraction knots, and local twitch responses when treated with needle stimulation, established hallmarks for MTrPs.

Ultimately, MTrPs represent a local upregulation of neuromechanical activity resulting from damaged muscle. In addition to ligamentous damage, sudden ankle inversion traumatizes local musculature and very likely results in the formation or activation of an MTrP. A self-perpetuating, hyper-functioning, and dysfunctional cycle is developed, and the characteristic taut bands and abnormal miniature endplate potentials are sustained. The documented consequences of MTrP include chaotic muscle activation patterns, decreased efficiency of reciprocal inhibition and increased muscular activity (Ge, Monterde, Graven-Nielsen, and Arendt-Nielsen, 2014; Ibarra et al., 2011; Lucas, Polus, and Rich, 2004; Lucas, Rich, and Polus, 2010). These may all contribute to the early-onset of muscle fatigue that is found to be four times faster at MTrPs than at non-MTrPs that may lead to overload of nearby motor units that may contribute and perpetuate overuse (Ge, Arendt-Nielsen, and Madeleine, 2012). Additionally, MTrPs also have increased sarcomere overlap, resulting in local stiffness within a muscle (Cagnie et al., 2013; Chen et al., 2007; Maher, Hayes, and Shinohara, 2013). Non-uniformity within the muscle may affect its length–tension relationship, ultimately compromising the fidelity of nuclear bag and chain afferent signaling and subsequently altering sensory processing (Ge et al., 2009). Finally, a gross decrease in strength is noted in individuals with MTrPs when compared to those without in a double-blind study of upper extremity strength (Celik and Yeldan, 2011). Ultimately the combination of neurologic and muscular consequences of an MTrP creates a local neuromechanical dysregulation.

Dry needling in rehabilitation

DN is a treatment tool that directly affects the neurophysiology of muscle, thereby likely affecting nearby muscle spindles. The change in treated structures is hypothesized to alter the excitability of the mechanoreceptors by altering the chemical environment and muscle filament overlap, two known modulators of the gamma motor neuron (Knutson, 2000). DN is a treatment that reduces trigger points and is associated with locally decreased nociceptive mediators, monofilament overlap, spontaneous electrical activity (SEA), and patient-reported pain (Baldry, 2002; Boyles, Fowler, Ramsey, and Burrows, 2015; Chen et al., 2001; Dommerholt, 2011; Hsieh, Chou, Joe, and Hong, 2011; Melzack, 1981; Shah and Gilliams, 2008).

Table 1. Alterations in key structures and improvements with ON.

Depressed sensorimotor activity	Dry needling-induced equilibration	Excited neuromuscular activity
Muscle spindle Cutaneous receptors Iα afferent signals	**Increased gamma motor neuron**	Spontaneous electrical activity (SEA)
Spinal reflex excitability	**Decreased SEA = recalibration of intra- and extrafusal muscle fibers**	Local muscle metabolism
Spinocerebellar tracts		Intramuscular length–tension relationship increased
Somatosensory and somatomotor cortex activity	***Resulting in improved feedforward and feedback mechanisms.***	Altered motor control strategies
Accuracy of motor output		

DN can be an effective treatment for MTrPs in a broad variety of muscle groups (Boyles, Fowler, Ramsey, and Burrows, 2015). A systematic review of randomized controlled trials identified DN treatment as having positive effects on pain, range of motion, and at least one patient-reported measure of function or quality of life in multiple body regions. Strong evidence supports the use of DN to treat upper-quarter myofascial pain both immediately following and 4 weeks after treatment (Kietrys et al., 2013). Furthermore, DN was more effective for reducing pain and improving pain pressure threshold in those with musculoskeletal pain compared to no treatment, sham needling, or other physical therapy interventions (Gattie, Cleland, and Snodgrass, 2017). Additionally, the authors determined that DN was found to have superior functional outcomes in a myriad of musculoskeletal conditions when compared to no treatment or sham needling with low-quality evidence (Gattie, Cleland, and Snodgrass, 2017).

The addition of DN to the fibularis longus muscle within a traditional rehabilitation plan demonstrated superior self-reported outcomes in individuals with CAI (Salom-Moreno et al., 2015). In this study, subjects who received DN treatment in addition to traditional balance exercises reported significantly greater improvements 8 weeks after their last DN treatment and four weeks after their last exercise treatment over traditional exercise alone. Furthermore, DN MTrPs of the fibularis longus muscle in individuals with CAI improved ankle strength, balance, and hop testing outcomes (Rossi et al., 2017). These subjects demonstrated immediate improvement following a single treatment without additional rehabilitative care. These studies together demonstrate that DN treatment has already demonstrated the ability to have enhanced or stand-alone improvements in both clinician- and patient-oriented outcomes in individuals with CAI.

Dry needling equilibration theory

DNET proposes a mechanism for DN's positive effects on balance that links the depressed sensorimotor system with the excited local mechanistic changes within the muscle (Table 1). DNET hypothesizes that intramuscular dynamics are normalized, resulting in conditions effective for the transference of accurate and effective afferent and efferent information. DNET describes the process of neuromechanical calibration that occurs in neuromechanically decoupled and neuromechanically dysregulated structures (Figure 2).

DNET describes the probable mechanisms initiated by DN that create a cascade of responses (Figure 3) that help to explain the positive effects of DN and its ability to harmonize the dysfunctional neuromechanical environment. DN is accomplished through the insertion of a thin, solid, filiform needle into an identified MTrP in a skeletal muscle belly (Figure 3, Step 1). The insertion of the flexible needle into an MTrP can cause "twitch responses" which are a sudden and focal contraction of the taut band (Dommerholt, Bron, and Franssen, 2006). Once a twitch response is elicited, a "fast in, fast out" technique that moves the needle up and down for approximately 30–45 seconds is used to elicit any additional twitch responses. The silencing of the twitch response indicates a normalization of muscle length that is followed by a decreased tone and taut bands in the treated muscle (Chen et al., 2001). Following a twitch response, SEA activity is reduced ensuring that the local cycle of muscle contracture will not resume (Ge, Fernandez-de-Las-Penas, and Yue, 2011; Hsieh, Chou, Joe, and Hong, 2011; Hsieh et al., 2014).

As a result of improved intramuscular length–tension relationships, the muscle spindle may send optimized sensory information to the CNS. As muscle spindles remain the "final common input" to the CNS, improving spindle proprioceptive accuracy is valuable to resultant postural control (Figure 3, Step 2). Higher muscle spindle activity is associated with

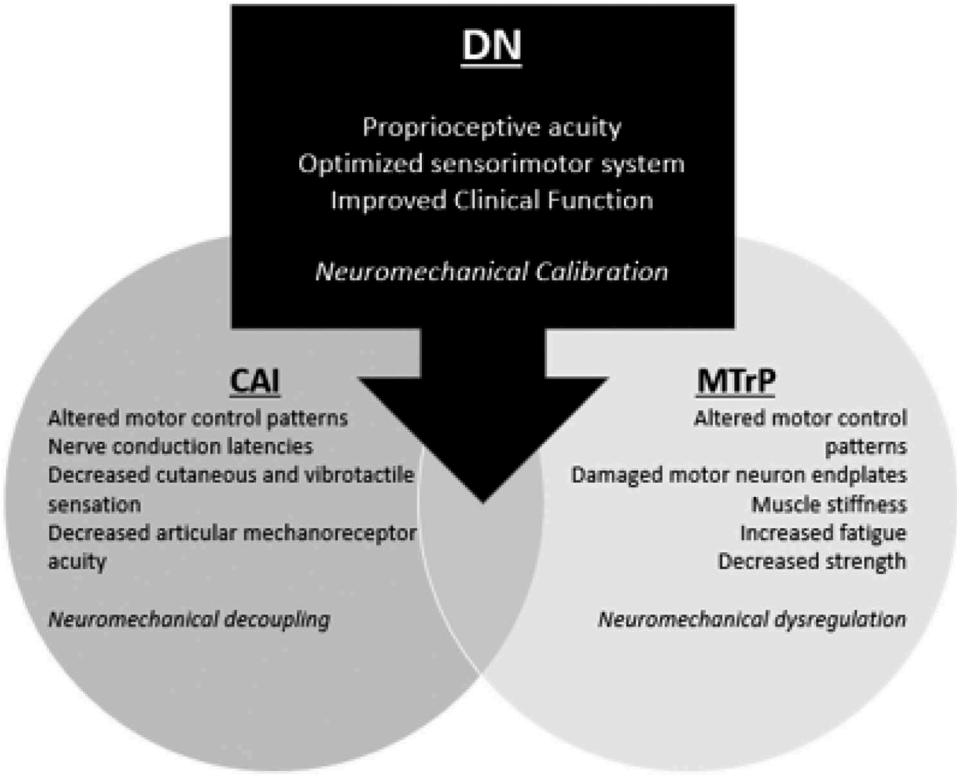

Figure 2. Hypothesized relationship of DN treatment and symptomology of CAI and MtrP as neuromuscular pathologies.

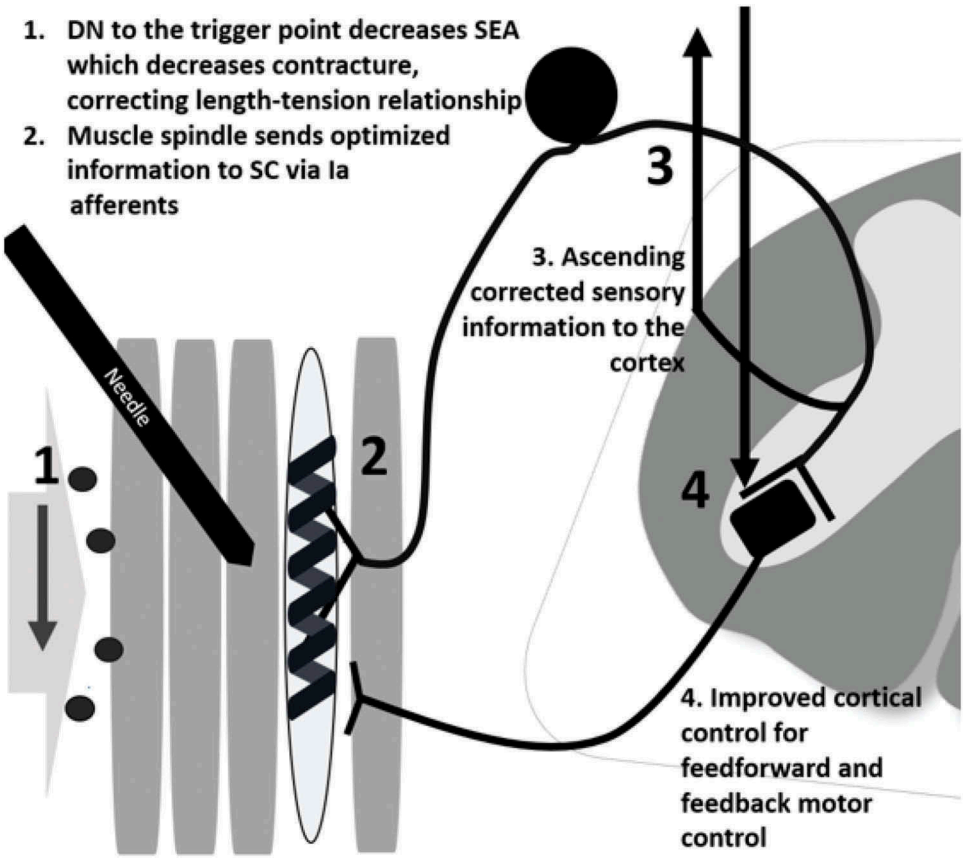

Figure 3. Proposed mechanism for dry needling equilibration theory.

improved cortical excitability (Needle et al., 2018). Ultimately, this results in increased accuracy of ascending information to supraspinal structures, resulting in improved descending output (Figure 3, Step 4). Continued training under these conditions would likely result in neuroplastic changes that would combat maladaptive sensorimotor changes that have likely occurred in individuals with chronic ligament injury.

Applying DNET to CAI rehabilitation

The purpose of this paper was to propose a mechanism to describe how DN may improve sensorimotor precision in individuals with CAI through the DNET. DNET describes how DN improves proprioceptive input by improving intramuscular length–tension relationships and utilizing nociception to re-calibrate local muscle spindles. Postural control strategies are thus advanced secondary to improved input, resulting in precision motor output.

Ankle injuries are far from innocuous, and the effects throughout the nervous system are far reaching. CAI results in neuroplastic changes at all levels of the nervous system, resulting in negative effects of both the afferent and efferent pathways. It is likely that the genesis of CAI merely represents an inability to address the neurologic components of the acute ligament injury. Research continues to affirm a failure of the CNS to appropriately regulate muscle tone in individuals with CAI, resulting in altered motor patterns, recurrent sprains, and chronic conditions like osteoarthritis (Al-Mahrouqi, MacDonald, Vicenzino, and Smith, 2018; Hiller, Kilbreath, and Refshauge, 2011; Jaber et al., 2018; Needle et al., 2013b). DN is suggested to improve the precision of muscle spindles, resulting in improved cortical functioning. Training under these conditions may result in improved proprioceptive ability, thereby combatting the maladaptive effects of chronic joint injury. Additionally, DN is the only direct treatment for MTrPs and has an established ability to reduce SEA and tissue stiffness (Chen et al., 2001; Maher, Hayes, and Shinohara, 2013). It follows that decreasing SEA abolishes the mechanical effects of increased myofilament overlap and altered length–tension relationships, resulting in a decrease of tissue stiffness that may inappropriately alter muscle spindle activity (Baldry, Yunus, and Inanici, 2001; Dommerholt, 2004).

While other therapies have shown improvement in cortical excitability by affecting mechanoreceptors (vibration, electrical stimulation, manipulation, and massage), DN is the only one that directly treats and reduces the activity of MTrPs affecting the muscle spindle. This treatment is advantageous in comparison to other therapies that purpose to improve sensorimotor control through both feedback and feedforward mechanisms because it affects the "final common input" of the sensorimotor system, the muscle spindle.

Future research

While DNET bridges maladaptive neuroplastic changes in individuals with CAI and treatment of MTrPs, additional research is necessary to support and broaden its application. Future researchers should deepen our mechanistic understanding of why DN induces positive changes in the MTrP. Additionally, prospectively addressing the development of an MTrP would enable researchers to establish clarified relationships on the initiation and propagation of MTrP factors. Finally, DNET would be best supported with research that included long-term follow-ups on its effect on negative outcomes associated with CAI.

Conclusion

DNET describes the ability to improve clinical function using DN in individuals with chronic ligament injury through improved sensorimotor input to the CNS, resulting in improved functional output. This is accomplished through a recalibration of the muscle spindle to the extrafusal fibers via the treatment of the MTrP with DN decreasing SEA and correcting muscle length–tension relationships. Training under improved conditions will likely result in positive neuroplastic changes that will combat the maladaptive sensorimotor plasticity resulting from chronic injury.

Declaration of interest

The authors declare no conflict of interest.

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