

Review Article**Dry Needling: Physiological Effects in Human Musculoskeletal Pain**Gargi Ray Chaudhuri¹, PhD¹Professor, Nopany Institute of Healthcare Studies*Corresponding Address:* Dr. Gargi Ray Chaudhuri, Nopany Institute of Healthcare Studies, 2C, Nando Mullick Lane, Kolkata- 700006; E-mail: raychaudhurig@yahoo.com

Abstract: Myofascial trigger points (MTrPs), characterized as local hypersensitive points, usually form a palpable taut band within the skeletal muscle fibre. Reduced blood flow causing local ischaemia and hypoxia forms the core of the MTrPs. Several mechanisms have been suggested to explain the pain relief of MTrPs by needle stimulation. The present review aims to understand the physiological basis of pain production and pain modulation and management of pain by investigating the general, neurological and biochemical mechanisms of action after Dry Needling (DN).

Keywords: MTrPs, Myofascial pain, Pain physiology, Dry Needling, Sensitization.

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Introduction

Myofascial pain is one of the most common examples of musculo-skeletal pain associated with active and latent myofascial trigger points defined as a hyperirritable spot in a taut band of skeletal muscle fibres.⁽¹⁾

During the past decades, clinical and scientific interest in DN has grown exponentially and various treatment effects are being credited to DN such as decreased pain, reduced muscle tension, improved range of motion, muscle strength and coordination in different musculoskeletal pain in addition to other neurological disorders like spasticity, muscle stiffness.⁽²⁻⁶⁾

DN uses a fine solid filiform needle and includes

an immediate reduction in local, referred and widespread pain though its action differs depending on locations, needle placement, depth of insertion and the needle force.⁽⁷⁻⁸⁾

The mechanism for relief of pain after DN application is based on potential physiological processes. The explanation of these processes is now needed to support the increased evidence of efficacy of DN.

Pain physiology and modulation

Pain is a complex phenomenon and includes both sensory discriminative and motivational affective components. It is a sensory experience that is accompanied by emotional responses and somatic as well as autonomic motor adjustments. Presumably the sensory discriminative component of pain depends on the Spinothalamic and Trigeminothalamic projection to the ventral posterior medial (VPM) nucleus of the contralateral thalamus.⁽⁹⁾ This nociceptive information is then transmitted to the Somatosensory regions (SI and SII) of the cerebral cortex. Sensory processing at these levels of the cortex results in (a) the perception of quality of pain (picking, burning aching); (b) the location of the painful stimulus (c) the intensity of the pain; (d) the duration of the pain.⁽¹⁰⁾

The motivational affective responses to painful stimuli include attention and arousal act, somatic and autonomic reflexes, endocrine responses and emotional changes. These account collectively for the unpleasant nature of painful stimuli. The motivational-affective responses depend

on activity transmitted in several ascending pathways including not only the Spinothalamic and Trigemino-thalamic tract but also the Spino-reticular and the Spino-mesencephalic tracts. Several other pathways also connect the spinal cord directly with limbic areas including amygdala but without a thalamic relay related to pain perception.⁽¹¹⁾

Eventually from the point of view of type of pain transmission there are dual pathways of pain signals into the central nervous system. Peripheral pain fiber that carries fast-sharp pain signals transmitted in the peripheral nerves to the spinal cord by small type A δ fibers at a velocity between 6 and 30 m/sec and conversely slow-chronic pain is transmitted by type-C fibers at velocities between 0.5 and 2 m/sec.⁽¹¹⁻¹²⁾ On entering the spinal cord, the pain signal takes two pathways to the brain through either the Neo-Spinothalamic tract (fast pain fibers and the Paleospinothalamic tract (slow pain fiber). These pain sensations can be categorized into nociceptive pain, hyperalgesia and allodynia, referred pain, neuropathic pain etc.⁽¹³⁾

Hyperalgesia and allodynia signify increased sensitivity of nociceptive afferent fibers. Injured cells produce pain by releasing chemicals such as K^+ that directly depolarizes nerve terminals and as a result of it, action potential formed making nociceptor more responsive. It also releases bradykinin and substance P, potent substances to sensitize nociceptive terminals. Moreover histamine from mast cells, prostaglandin from cell membrane, serotonin from platelets contribute to the inflammatory process eventually activating both A δ and C nerve ending.⁽¹⁴⁾

Substance P along with calcitonin gene-related peptide (CGRP), a neuropeptide from a nociceptor after an axon reflex, causes changes in the local environment producing vasodilatation and change in the capillary permeability resulting in plasma extravasations. Besides these, many regulatory and inflammatory cytokines like interleukin-1 β , tumor necrosis factor (TNF), neurotrophic factor

like nerve growth factor (NGF) can be released following local damage.⁽¹⁵⁾ Furthermore in chronic pain, enhanced pain facilitation may lead to “Central sensitization” which plays a synergistic role by augmenting nociceptive stimulation (hyperalgesia) followed by a higher pain feeling elicited by normally non-noxious stimuli (allodynia).⁽¹⁶⁻¹⁷⁾

The endogenous opioid system helps to modulate pain from their ability to inhibit directly the ascending transmission of nociceptive information from the spinal cord dorsal horn cells. This system works through the activation of μ , δ and κ receptors that are widely distributed in both peripheral and central nervous system related to nociception.⁽¹⁴⁾ They are also able to activate pain control circuit from the level of midbrain to spinal cord. It can also block many of the local reflexes that results from pain signals, especially the withdrawal reflex.⁽¹⁸⁾

Melzack and Wall in 1965 published “Gate theory of pain control” which has been modified as segmental inhibition where A β fiber can lead to an inhibition in the spinal cord by blocking the synaptic transmission between A δ and C fibers for their comparative slower information transmission.⁽¹⁹⁾

Functional aspect of Dry Needling (DN)

The myofascial trigger point function consists of development of taut band which results in a formation of abnormal endplate potential due to excessive acetylcholine (Ach) release in the neuromuscular junction. This excessive Ach release produces a sustained sarcomere contraction that could lead to local ischaemia or hypoxia in the muscle.⁽²⁰⁻²¹⁾

Several researchers tried to postulate the theory of deactivation of trigger points by Dry Needling. As per the explanation a ‘Local Twitch response (LTR)’ makes localized contraction of the affected muscle fibre after Dry Needling where deep Dry Needling gives analgesic effect compared to superficial.⁽²²⁾

It has been accepted that DN may disrupt the integrity of dysfunctional end plate. It can influence the spontaneous electrical activity (SEA) by eliciting a local twitch response (LTR) or can effectively suppress SEA when LTR are elicited. As a result, DN can immediately reduce available SEA store leading to lesser SEA or LTR which may alter the length and tension of the muscle fiber thus stimulating mechanoreceptors.⁽²³⁻²⁴⁾

Changes in muscle fiber architecture

Dry Needling creates a local stretch in contracting cytoskeletal structure which causes sarcomere to return to their relaxation length by reducing the amount of 'overlap' between actin and myosin fiber muscle.⁽²⁵⁻²⁶⁾

Biochemical changes

Local ischaemia or hypoxia has been proved to be a reason for taut band formation. Needling may stimulate release of vasoactive substances like substance P or CGRP which upon activation of A δ and C fiber via flare response lead to local vasodilatation.⁽²⁷⁾ Pain associated with myofascial trigger point is related to stimulation of a unique cascade of cytokines that are integral to the inflammatory response.⁽²⁸⁾ Low Oxygen level lead to a significant drop in pH that excite muscle nociceptors.⁽²⁹⁾ At the molecular level, nociceptors express in ion channels for stimulus transduction and action potential generation and as a result of it a large number of receptors for inflammatory and other mediators can be expressed. These receptors are coupled to ion channels and often activate second messenger system that influence ion channels signal transduction.⁽³⁰⁾ Transient receptor protein (TRP) is the first cloned nociceptive ion channel where transient receptor potential cation channel subfamily V member like TRPV1, TRPV2 or TRPV3 mediates the viscous sensation after their transcription.⁽³¹⁾

Most voltage-gated Na⁺ channels are blocked by tetrodotoxin (TTX), many small DRG cells express TTX resistant (TTX-R), Na⁺ channels,

in addition to the TTX- Sensitive (TTX-S) Na⁺ channels. This TTX-R-Na⁺ currents are influenced by inflammatory mediators.⁽³²⁾ Some acid sensing ion channels are Na⁺ channels opened by low pH which is an important aspect of inflammation and formation of action potential.⁽³³⁾

Several proteins like hypoxia- inducible factor – 1 α (HIF-1 α) COX-2, vascular endothelial growth factor (VEGF) found to be active by Dry Needling may promote angiogenesis, vasodilatation and alter glucose metabolism in hypoxic tissue.⁽²⁹⁾

Neurophysiological effect

Peripheral activation of A δ and C fibre nociceptors is modified by a number of sensitising and analgesic agents. Local twitch response(LTR) is defined as an involuntary localized and temporary contraction in one part of the taut muscle during trigger point needling. Low twitch response produced by DN may cause significant lower concentration of substance P and CGRP. It has been found that DN in biceps muscles enhanced the β -endorphin levels in biceps muscle.⁽³⁴⁻³⁵⁾ It has been hypothesized that site- specific dry needling may be mediated by segmental inhibitory effects, evoked by selective stimulation of large myelinated fibre in the myofascial trigger point.⁽³⁶⁾

Central sensitization represents an enhancement in the function of neurons and circuits in nociceptive pathways caused by increases in membrane excitability and synaptic efficacy as well as by reduced inhibition and is the manifestation of the somatosensory nervous system in response to activity, inflammation and neural injury.⁽³⁷⁾ In case of central sensitization, DN may stimulate both large myelinated fibers (A β and δ) and C fibers indirectly via release of inflammatory mediators and send positive stimulation to the spinal cord and can activate higher mediators centers involved in pain processing.⁽³⁸⁾

Effects of Endogenous opioids

Periaquiductalgray matter (PAG) is a central part of

the opioid circuitry. DN may act through release of endogenous opioid that via stimulating nociceptive fibers may activate the enkephalinergic inhibitory dorsal horn interneurons. Besides this, serotonergic and noradrenergic descending inhibitory system work through stimulation of A δ fibers.⁽³⁹⁾

Conditioned Pain Modulation (CPM)

Conditioned pain modulation is a paradigm that has been increasingly used over the past few years to assess endogenous analgesia capacity in healthy individuals and pain patients.⁽⁴⁰⁾ DN may act through this endogenous pain inhibiting system.⁽⁴¹⁾

Considering all mechanisms and study of dry needling, it appears that DN acts mostly on highly complex network of all physiological processes along with biochemical and cellular evidences. Central and peripheral network should be taken principally under consideration to establish its way of functioning. Researchers should concentrate and explore to what the other possible physiological basis could be attributed to Dry Needling so that the future goal could be aimed to fill in the current voids.

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References

- 1) Simons DG. Review of enigmatic MTrPs as a common cause of enigmatic musculoskeletal pain and dysfunction. *J ElectromyogrKinesiol.* 2004;14:95-107.
- 2) Rainy CE. The use of trigger point dry needling and intramuscular electrical stimulation for a subject with chronic low back pain: a case report. *Int J sports phys Ther.* 2013; 8(2):145-161.
- 3) Pablo H, Orlando M. A case study looking at the effectiveness of Deep Dry needling for the management of hypertonia. *J musculoskelet pain.* 2010;15(2):55-60.
- 4) Calvo S, Quintero I, Pablo H. Effects of dry needling (DNHS techniques) on the contractile properties of spastic muscle in a patient: a case report. *IntJrehabil res.* Post author corrections: June 2016 [In press].
- 5) Srbely JZ, Dickey JP, Lee DC, et al. Dry needle stimulation of myofascial trigger points evokes segmental anti-nociceptive effects. *J Rehabil Med.* 2010; 42:463-468.
- 6) Cagnie B, Dewitte V, Barbe T, et al. Physiologic effects of dry needling. *Curr Pain Headache Rep.* 2013; 17:348-355.
- 7) Vulfsons S, Ratmansky M, Kalichman L. Trigger point needling: techniques and outcome. *Curr pain Headache Rep.* 2012;16(5):407-412.
- 8) Hong CZ. Lidocaine injection versus dry needling to myofascial trigger point: The importance of the local twitch response. *Am. J. Rehabil.* 1994; 73(4): 256-263.
- 9) Berne RM, Levy MM, Koeppen B, et al. The somatosensory system. In: *Physiology 5th Eds.* Mosby Publication, 2004:110-116.
- 10) Constanzo RM, Gardner EP. A quantitative analysis of responses of direction-sensitive neurons in somatosensory cortex of awake monkeys. *J Neurophysiol.* 1980; 43:1319-1341.
- 11) Guyton AC, Hall JE. *Textbook of Medical Physiology.* 10th Ed. Elsevier 2004: 553-555.
- 12) Hopkins K. Show me where it hurts: Tracing the pathways of pain. *J NIH Res.* 1997; 9:37-43.
- 13) Barrett KE, Barman SM, Boitano S, et al. *Ganong's Review of Medical Physiology.* 25th ed. Tata Mc grow Hill publication 2015.
- 14) Patel N. *Physiology of Pain.* In: Patel N, Kopf A, eds. *Guide to pain management in low resource setting.* 2010:13-18.
- 15) Chou LW, Kao MJ, Lin JG. Probable mechanism of needling therapies for myofascial pain control. *Evidence based complementary and alternative medicine.* 2012; vol 2012:1-11.
- 16) Ossipov MH. The perception and endogenous modulation of pain. *Scientificia.* 2012; vol 2012:1-25.
- 17) Nijs J, Van HB, Oostendrop RA. Recognition of central sensitization in patients with musculoskeletal pain: Application of pain neurophysiology in manual therapy practice. *Man.Ther.* 2010; 15(2):135-141.
- 18) Bach FW. Beta -endorphin in the brain: a role in nociception. *ActaAnaesthesiol scan.* 1997; 41:133-140.
- 19) Melzac R, Wall PD. Pain Mechanism: A new theory. A gate control system modulates sensory inputs from the skin before it evokes pain perception and response. *Science.* 1965; 150:971-997.
- 20) Simons DG. Review of enigmatic MTrPs as a common cause of enigmatic musculoskeletal pain and dysfunction. *J ElectromyogrKinesiol.* 2004; 14:95-107.

- 21) Hong CZ, Simons DG, Pathophysiologic mechanisms of myofascial trigger points. *Arch Phys Med Rehabil.* 1998; 78: 863-872.
- 22) Ceccherelli F, Rigoni MT, Gangliardi G, et al. Comparison of superficial and deep acupuncture in the treatment of lumbar myofascial pain : a double-blind randomized controlled study. *Clin J Pain.* 2002; 18(3):149-150.
- 23) Hsich YL, Chou LW, Soc YS, et al. Spinal cord mechanism involving the remote effects of Dry Needling on the trigger spots in rabbit skeletal muscle. *Arch Phys Med Rehabil.* 2011; 92(7): 1098-1105.
- 24) Baldry P. *Accupuncture, trigger points and musculoskeletal pain.* 3rd Ed. Churchill Livingstone : 2005.
- 25) Dommerholt J. Dry Needling in orthopedic physiological therapy Practice. *Ortho Phys TherPract.* 2004; 16(3):15-20.
- 26) Dommerholt J, Mayoral D M O, Grobli C, Trigger point Dry Needling. *Myofascial trigger points: pathophysiology and evidence- informed diagnosis and management.* 2009: 159-190.
- 27) Sato A, Sato Y, Shimena M et al. Calcitonin gene-related peptide produces skeletal muscle vasodilation following antidromic stimulation of unmyelinated afferents in the dorsal root in rats. *Neurosci Lett.* 2000; 283(2):137-140.
- 28) Gerwin RD, Dommerholt J, Shah JP. An expression of Simon's integrated hypothesis of trigger point formation. *Current pain and headache reports.* 2004; 8(6):468-475.
- 29) Gautam M, Benson CJ, Shukla KA. Increased response of muscle sensory neurons to decreases in pH after muscle inflammation. *Neurosciences.* 2010; 170:893-900.
- 30) Caterina MJ, Leffler A, Malmberg AB et al. Impaired nociception and pain in mice lacking the capsaicin receptor. *Science.* 2000; 288:306-313.
- 31) Simon T, Andem PP and Clifford JW. Transient receptor channels: targeting point at the source. *Nature Reviews. Drug Discovery.*
- 32) Akopian AN, Soulova V, England S, et al. The tetrodotoxin – resistant sodium channel SNS has a specialized function in pain pathways. *Nat. Neurosci.* 1999; 2(6):541-548.
- 33) Nicolas V. Acid sensing ion channels (ASICs): new targets for the analgesic effects of non steroidal anti-inflammatory drugs (NSAIDs). *Current Drug Targets-inflammation and allergy* 2004; 3(1):71-79.
- 34) Shah JP, Gilliams EA. Uncovering the biochemical milieu of myofascial trigger points using in vivo micro dialysis : an application of muscle pain concepts to myofascial pain syndrome. *J Bodyw Mov Ther.* 2008;12:371-384.
- 35) Hsieh YL, Yang SA, Yang CC et al. Dry needling at myofascial trigger spots of rabbit skeletal muscles modulates the biochemicals associated with pain inflammation and hypoxia. *Evid Based complement. Altern Med.* 2012; vol 2012.
- 36) Karst M, Rollnik JD, Fink M, et al. Pressure pain threshold and needle acupuncture in chronic tension-type headache – a double-blind placebo-controlled study. *Pain* 2000; 88: 199-203.
- 37) Kumar SP, Saha S. Mechanism based classification of pain for physical therapy management in palliative care: A clinical commentary. *Indian J Palliat Care.* 2011; 17(1):80-86.
- 38) Niddam DM, Chan RC, Lee SH. Central modulation of pain evoked from myofascial trigger points. *Clin J Pain.* 2007; 23(5):440-448.
- 39) Millan MJ. Descending control of pain. *Prog. Neurobiol.* 2002; 66(6):355-474.
- 40) Nir RR, Yarnitsky D. Conditioned pain modulation. Current opinion in supportive and palliative care. 2015; 9(2):131-137.
- 41) Leung L. Neurophysiological basis of acupuncture induced analgesia – an updated review. *J Accupunct Meridian Stud.* 2012; 5(6):261-270.

