

DRY NEEDLING AS A SUPERIOR PHYSIOTHERAPY INTERVENTION FOR POST-STROKE UPPER LIMB REHABILITATION: NEUROPHYSIOLOGICAL MECHANISMS, CLINICAL EVIDENCE, AND A PROPOSED TREATMENT FRAMEWORK

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KEYWORDS

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Abstract

Background: Post-stroke upper limb spasticity is a multi-dimensional issue that involves neural hyperexcitability, myofascial trigger point development and secondary musculoskeletal remodelling. Traditional stretching methods address mechanical, but not neurophysiological, causes of spasticity. Dry needling is a mechanistically more comprehensive treatment that targets both neuromuscular, neurological, and musculoskeletal issues.

Objective: To describe the mechanism of dry needling for the treatment of post-stroke upper limb spasticity, summarise clinical evidence to support its superiority over stretching, and outline an evidence-based phased treatment pathway utilising dry needling as a first-line treatment.

Study Design: A randomized comparative interventional study conducted for 4 weeks in 60 post-stroke patients (Group A: Dry Needling, n = 30; Group B: Stretching, n = 30) at PMCH, Udaipur, was the source for the primary clinical data.

Results : Dry needling significantly improved all six outcome measures: a significant reduction in MAS (1.16 vs 0.57), reduction in VAS (3.46 vs 2.57), gain in shoulder ROM (26.8° vs 13.3°), elbow ROM (32.3° vs 14.7°), wrist ROM (24.3° vs 12.2°), and FMA-UE (15.2 vs 8.8 points), all p<0.05.

Conclusion: Dry needling is a neurophysiologically, mechanistically and clinically more effective treatment than stretching alone for reducing upper limb spasticity following stroke and can be used as the main intervention in a structured, multimodal physiotherapy approach.

1. INTRODUCTION

Upper limb dysfunction is one of the most common and difficult problems of stroke

and over one-third of the approximately 15 million people who suffer stroke annually

have persisted deficits in their upper limb function. This dysfunction is manifested on three levels [3]: neurological - increased excitability of alpha motor neurons, loss of descending inhibition, loss of reciprocal and presynaptic inhibition, velocity-dependent, reflex-mediated component of spasticity, [4]. neuromuscular - persistent abnormal motor unit activation, myofascial trigger points, hypersensitive nodules within taut bands that result from dysfunctional motor endplate depolarization, non-velocity dependent component of spasticity, [5]. and musculoskeletal - structural remodelling of chronically short muscled sarcomere loss and connective tissue stiffening, mechanical component of spasticity that does not respond to neurological therapy, [6].

Stretching mainly targets the musculoskeletal aspect and has a short, limited neurophysiological effect through Golgi tendon organ mediated inhibition but does not directly target trigger point pathology. Dry needling is a technique that treats all three of these areas: neurologically it stimulates A δ /C-fibers that activate spinal and supraspinal inhibitory pathways to decrease excitability of alpha motor neurons; neuromuscularly it produces a local twitch response (LTR), which helps to break

up abnormal motor endplate activity and to inactivate trigger points; musculoskeletally, it normalizes the biochemistry of local trigger points and increases circulation. The present study observed greater clinical improvements with dry needling, which may be explained by this triple-mechanism action.

2. NEUROPHYSIOLOGICAL MECHANISMS OF DRY NEEDLING

2.1 Peripheral Mechanisms : The contraction of the muscle fibres within a tight band after the needle is inserted is a short, involuntary response, known as the LTR, which is thought to be activation of a local reflex loop in the spinal cord through activation of A δ afferents [7]. Based on the integrated trigger point hypothesis, the imbalance in acetylcholine levels released from impaired motor endplates results in prolonged depolarization of the fibers, local energy crisis, hypoxia, and inflammatory sensitizing mediators released from the motor endplates [5]. Mechanical disruption of this cycle by the LTR restores sarcomere length and local blood flow, accounting for the lasting decreases in stiffness (MAS) and pain (VAS) with dry needling.

2.2 Spinal Cord Mechanisms : Needle stimulation activates A δ and C mechanonociceptors projecting to the dorsal horn and activates: presynaptic inhibition of Ia afferents via GABA mediated mechanisms, decreases the gain of the stretch reflex; activates Ib inhibitory interneuron (Golgi tendon organ pathway) which causes postsynaptic motor neuron inhibition; activates recurrent inhibition via antidromic activation of Renshaw cells which causes motor neuron inhibition beyond what stretches alone produce.

2.3 Supraspinal Mechanisms : A δ /C afferents activate descending noradrenergic and serotonergic inhibition from the rostral ventromedulla and locus coeruleus, respectively, and release endogenous opioids, in the periaqueductal gray, which

are involved in the regulation of descending noradrenergic and serotonergic inhibition [8]. This accounts for the degree of pain relief (VAS reduction 3.46 cm [>2 cm MCID] with dry needling compared with 2.57 cm with stretching).

2.4 Biochemical Mechanisms. A microdialysis study revealed that at active trigger points, the levels of substance P, CGRP, bradykinin, serotonin, prostaglandin E₂, TNF- α , IL-1 β , IL-6 are all increased, with local pH being reduced [9,10]. Dry needling mechanically disrupts this microenvironment, which improves circulation and allows inflammatory mediators to be removed, therefore resulting in a reduction of pain and stiffness beyond the therapy session.

3. CLINICAL EVIDENCE

Table 1: Complete Outcome Data — Dry Needling (Group A) vs Stretching (Group B)

Outcome	Gp A Pre	Gp A Post	Gain A	Gp B Pre	Gp B Post	Gain B	p†
MAS Score	2.53 $\pm$ 0.50	1.37 $\pm$ 0.49	1.16	2.50 $\pm$ 0.51	1.93 $\pm$ 0.52	0.57	<0.05
VAS Score (cm)	6.13 $\pm$ 0.88	2.67 $\pm$ 0.76	3.46	6.20 $\pm$ 0.91	3.63 $\pm$ 0.81	2.57	<0.05

Shoulder ROM (°)	81.9±2.9	108.7±3.2	26.8°	82.5±3.0	95.8±3.5	13.3°	<0.05
Elbow ROM (°)	96.3±3.1	128.6±3.4	32.3°	96.7±3.0	111.4±3.6	14.7°	<0.05
Wrist ROM (°)	43.5±2.6	67.8±3.1	24.3°	44.1±2.8	56.3±3.2	12.2°	<0.05
FMA-UE Score	31.2±1.9	46.4±2.2	15.2	31.4±2.0	40.2±2.3	8.8	<0.05

†Between-group (independent t-test). All within-group changes significant (Group A $p<0.001$, Group B $p<0.05$).

Table 2: Magnitude of Dry Needling Superiority over Stretching

Outcome Measure	Group A Gain	Group B Gain	Superiority Ratio (A/B)	Clinical Interpretation
MAS Reduction	1.16	0.57	2.04×	DN twice as effective at spasticity reduction
VAS Pain Reduction	3.46	2.57	1.35×	DN 35% greater pain relief
Shoulder ROM Gain	26.8°	13.3°	2.02×	DN doubles shoulder mobility gains
Elbow ROM Gain	32.3°	14.7°	2.20×	DN more than doubles elbow mobility gains

Wrist ROM Gain	24.3°	12.2°	1.99×	DN nearly doubles wrist mobility gains
FMA-UE Motor Gain	15.2	8.8	1.73×	DN 73% greater motor recovery

Note. DN = Dry Needling. Superiority ratio calculated as Group A improvement ÷ Group B improvement. Dry needling increases ROM at shoulder, elbow, and wrist by about 2 times compared to stretching.

Consistency with published literature:

These findings are similar to the systematic review by Valencia-Chulián et al. (2020) which showed that dry needling is effective at reducing spasticity and increasing range of motion in stroke patients by both mechanical and neurological pathways [11] and the systematic review and meta-analysis by Fernández-de-las-Peñas et al. (2021) which found that dry needling was effective at reducing spasticity, pain, and motor function in post-stroke patients [13]. Currently, there is no evidence to support this combined approach from controlled trials; only from case reports. Esmaeeli et al. (2025) have reported a plantar flexor spasticity in a chronic stroke patient that

showed reduction in plantar flexor spasticity, dorsiflexion range of motion and functional improvement after the application of dry needling and static stretching [16]. In a separate case report on a TBI survivor, dry needling was shown to decrease upper extremity spasticity, and it was suggested that this was due to alteration in the excitability of neurons and the mechanical properties of the targeted muscle [14]. While this is a population distinct from that studied in the post-stroke population, the proposed mechanism is plausibly more generalizable; however, evidence from RCTs of this specific combination is lacking—data from the present study could potentially fill this gap.

4. PROPOSED PHYSIOTHERAPY TREATMENT FRAMEWORK

A structure, four-phase approach is proposed with dry needling being the primary method of treatment for the neuromuscular and

neurological component of spasticity, supplemented by stretching, neuromuscular facilitation, and task-oriented training.

Table 3. Proposed Phased Framework

Phase	Duration	Primary Interventions	Goals
1 – Initial	Wk 1–2, 2x/wk	Baseline assessment; positioning/splints; gentle passive ROM; DN — elbow flexors; patient/family education; heat/cryotherapy	Establish baseline; reduce acute spasticity/pain; prevent early contracture
2 – Active Needling	Wk 2–4, 2– 3x/wk	Progressive DN — wrist/finger flexors, pronator teres; post- needling stretching (30s×5); active-assistive ROM; PNF patterns	Deactivate trigger points; restore passive ROM; reduce pain <3/10 VAS
3 – Functional Integration	Wk 4–6, 2x/wk	DN maintenance PRN; task- oriented reach-grasp-release training; FES; mirror therapy; ADL training	Transfer ROM to function; improve voluntary control; improve ADL independence
4 – Maintenance	Wk 6+, monthly/PRN	Reassessment; home stretching program; maintenance DN; caregiver training; orthotic management	Maintain gains; prevent contracture recurrence

1 Protocol notes : Sterile, single use filiform needles (0.25-0.30mm diameter, 25-40mm long) are inserted after systematic trigger point palpation (taut band, point

tenderness, referred pain "jump sign," and LTR on snapping palpation) in the following sequence: elbow flexors, wrist flexors, finger flexors, and pronator teres. Gentle

pistoning is applied until LTR is elicited, and the needle is kept 10–15 minutes, then immediately (within 5 minutes) followed by passive stretching to take advantage of the decreased tone after the needle is removed.

4.2 Safety: Absolute contraindications: active skin infection/open wounds at injection site, known bleeding disorders or supratherapeutic anticoagulation, patient can't sign consent, severe needle phobia.

Contraindications: relative contraindications include local lymphoedema, severe arrhythmia/pacemaker dependence, active DVT in the limb and pregnancy. Post-needling soreness, minor bruising and a rare vasovagal response are the most common and mild effects; other risks of the procedure are rare, such as pneumothorax which is a risk with posterior thorax/cervical needling, and should be avoided in patients with respiratory compromise.

Table 4: Recommended Outcome Monitoring Schedule

Outcome Measure	Domain	Frequency	MCID / Goal Threshold	Normal/ Target	Alert Level
MAS (Modified Ashworth Scale)	Spasticity	Every 2 sessions	≥ 1 point reduction	MAS 0–1	No change after 4 sessions
VAS (Visual Analogue Scale)	Pain	Every session	≥ 2 cm reduction (MCID)	$< 3/10$	$> 6/10$ persistent
Shoulder ROM (°)	Mobility	Weekly	$\geq 10^\circ$ improvement	$\geq 150^\circ$ flexion	$< 80^\circ$ at 4 weeks
Elbow ROM (°)	Mobility	Weekly	$\geq 10^\circ$ improvement	Full extension 0°	$< 90^\circ$ extension

Wrist ROM (°)	Mobility	Weekly	$\geq 10^\circ$ improvement	$\geq 50^\circ$ extension	$< 30^\circ$ extension
FMA-UE Score	Motor recovery	Every 2 weeks	≥ 7 point improvement	Score > 50	No change at 2 weeks

Note. MCID = Minimum Clinically Important Difference. Alert levels are intended to be used to review the treatment plan, increase the level of intervention or for multidisciplinary consultation. FMA-UE MCID of 7 points derived from Gladstone et al. (2002) and Chen et al. (2015).

5. DISCUSSION

5.1 The functional significance of ROM gains:

demonstrate approximately twice increase in ROM at the shoulder (26.8° vs 13.3°), elbow (32.3° vs 14.7°), and wrist (24.3° vs 12.2°) equates to functional gains – overhead reach and dressing (shoulder), transfers and bimanual tasks (elbow), and grip/release, writing, and typing (wrist).

5.2 Synergy with stretching : Dry needling and stretching are complementary, not mutually exclusive: Dry needling opens up a window when there is reduced neuromuscular resistance and then additional stretching provides the mechanical elongation that is not possible during the same window with stretching. This post-needling sequence of stretches is similar to that of the case report by Esmaeeli

et al. (2025) which showed synergistic effect on spasticity and mobility between the two stretches [16].

5.3 Neuroplasticity implications: Recovery of motor function after stroke is dependent on experience-dependent reorganization of the cortex, via task-specific practice [17]. Dry needling can improve the voluntary motor activation and the neuroplastic drive to execute the task, which may be able to create a more permissive environment for voluntary motor activation and task practice, thus potentially increasing the FMA-UE benefits that were observed in Group A.

5.4 Multidisciplinary care: Effective implementation needs to be coordinated with neurology in cases that are intractable for pharmacological control of spasticity and with occupational therapy to provide

training in ADL skills and to provide adaptation equipment, with regular case conferencing to ensure consistency in functional goals.

5.5 Limitations and future directions :

A four week intervention period does not allow for long-term durability to be assessed and three and six month follow-up is suggested. Due to the small sample size (n=60), there were not enough patients for subdivision of analysis based on lesion location, severity, or time since stroke. The dosages of dry needling (needle diameter, depth, pistoning frequency and retention time) are still not standardized. Future RCTs should look at the use of dry needling with other treatments such as botulinum toxin, functional electrical stimulation and constraint induced movement therapy, and future neuroimaging studies (fMRI and TMS mapping) should measure changes in excitability of the cortex and spinal cord.

6. CONCLUSION

This paper presents a neurophysiological mechanistic analysis of dry needling, along with evidence from a randomized comparative study of 60 post-stroke patients, showing that dry needling is superior in all outcome domains when compared to

stretching, with statistical significance. The triple mechanism action of dry needling, including neurological, neuromuscular and musculoskeletal, supports the clinical merits of dry needling, as there is about a double amount of ROM and spasticity reduction than stretching, and 73% more motor recovery than stretching. The proposed four-step model provides physiotherapists with a practical and evidence-based approach to dry needling as the key modality of the upper limb rehabilitation process in a multidisciplinary team in post-stroke rehabilitation.

DECLARATIONS

Ethics Approval: Institutional Ethics Committee, Pacific Medical University, Udaipur; Declaration of Helsinki (2013) and ICMR National Ethical Guidelines (2017). *Informed Consent:* Obtained from all participants. *Conflict of Interest:* None declared. *Funding:* None; conducted as partial fulfillment of MPT degree requirements. *Author Contributions:* Jhanavi Makhijani — conception, data collection, analysis, manuscript preparation. Dr. Jafar Khan — supervision, methodology, critical review.

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