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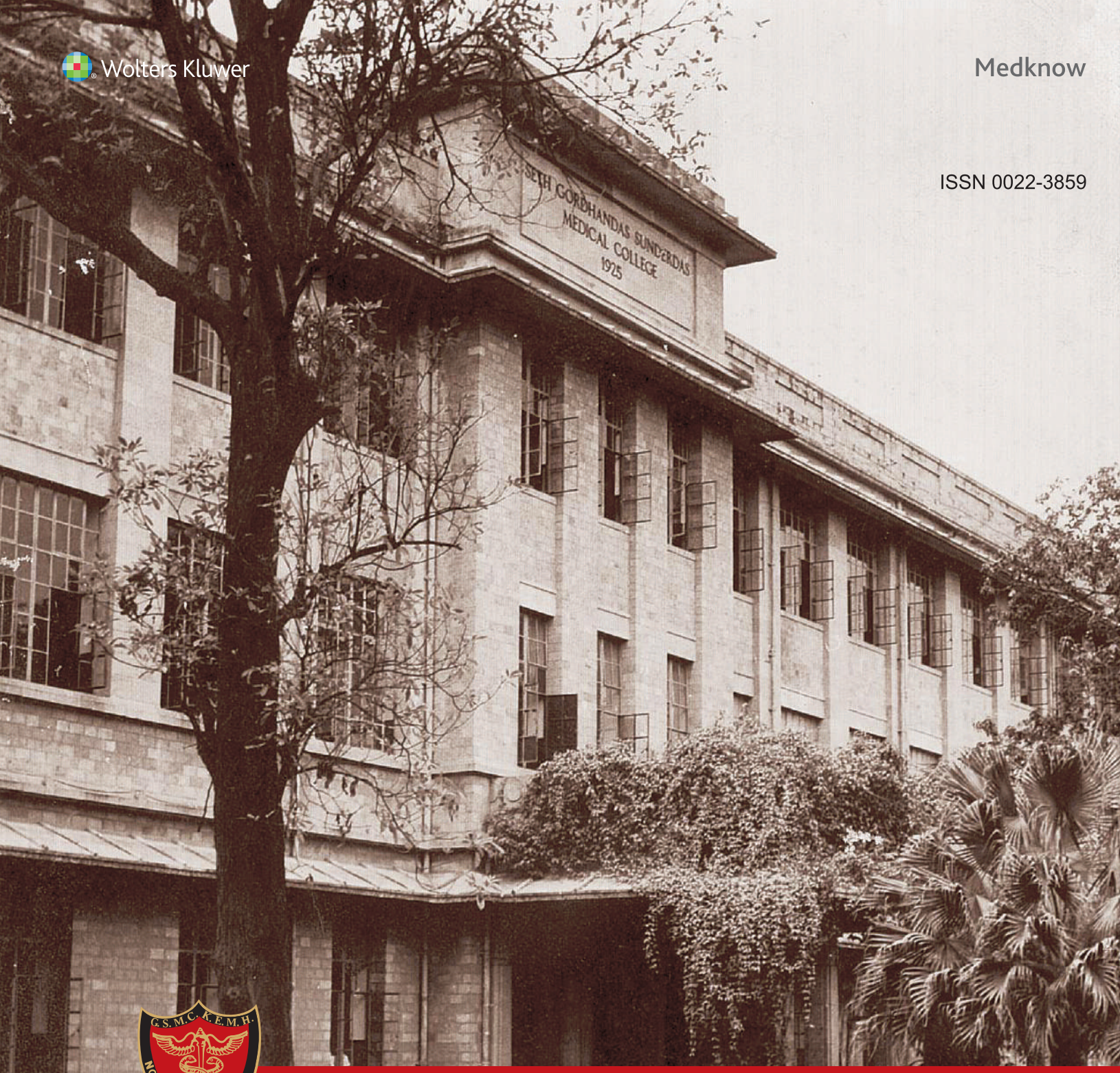

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Ultrasound guided dry needling: Relevance in chronic pain

Introduction

Dry needling (DN) is a specific treatment for myofascial pain syndromes (MPS) wherein a needle is inserted into a painful site in the muscle called myofascial trigger point (MTrP). An MTrP is defined as "a hyperirritable spot" within a painful taut band in a skeletal muscle associated with motor dysfunction, autonomic phenomena, and characteristic referral of pain.[1]

MTrPs exhibit a unique local twitch response (LTR) on needle insertion mediated by a spinal reflex. LTR can be seen/felt/recorded electromyographically and visualized with ultrasound. Active MTrPs are spontaneously painful and refer pain and paresthesia to distal sites. Latent MTrPs are painful only on palpation. Importantly, both active and latent MTrPs stimulate muscle nociceptors, disturb motor function by causing muscle stiffness and weakness, and restrict the range of motion (ROM).[2,3] DN causes immediate, complete analgesia by a physical "needle effect" without any injection.[4]

Ultrasound visualization of DN provides novel insights into muscle function and emphasizes that muscles seldom act in isolation. LTRs that are pathognomonic of MTrPs are routinely visualized in coworking muscles indicating that there is a much wider distribution of MTrPs than is currently assumed. For example, MTrPs in agonist muscles (flexors) apparently cause strain leading to reactive MTrPs in coworking muscles like antagonists (extensors), other agonists (synergists), and fixators.[5] Presumably, the MTrP numbers are insufficient to produce pain or tenderness above the perceptible threshold in these muscles. Routine confirmation of LTRs in coworking muscles has led to our hypothesis that pain and tenderness form just the tip of the iceberg, whereas the actual pain pathology lies in the whole muscle and its functional counterparts. Most of the LTRs seen during USGDN would neither be visible nor sought by the present DN practitioners who do not use ultrasound. Thus, ultrasound visualization revolutionizes DN into a more extensive as well as comprehensive version called USGDN, which addresses the interrelated functioning of the various structures in the body that work as parts of the whole: when one part suffers, it appears to involve all the others. Therefore, MPS becomes a neuromuscular problem and neuropathies become neuromyopathies manifesting with MPS. Both DN and USGDN use acupuncture needles because they are the thinnest available in the market (30–34 gauge), but the similarity ends there [see Table 1].

Furthermore, USGDN targets not only the pain but also the disability resulting from the structural impairment caused by a taut band that mechanically alters the function in all coworking muscles. This makes USGDN a specific treatment option for all chronic pains such as neuropathic pains, cancer pains, back pains, and arthritic pains, all of which manifest with MPS.[5]

This editorial discusses the latest findings on MTrPs and MPS and will put together the various jigsaw pieces scattered across pain literature to present a radically different understanding of chronic pain. We have assembled personal observations and the ultrasound evidence gathered over our experience of 16 years, to hypothesize that motor neuropathy is responsible for the ubiquitous prevalence of MPS seen in chronic pain conditions. As a specific treatment of MPS, USGDN has the potential to revolutionize the current practice of pain management.

Current Evidence: MTrPs

Electromyography of normal human neuromuscular endplate demonstrates discrete, random, positive miniature end-plate potentials (MEPPs) at approximately 6/s. The MTrP region is characterized by a barrage of potentials (110/s) called end-plate noise (EPN) due to the grossly (3×) increased release of acetylcholine from the nerve terminal.[1] This crescendo of EPN leads to muscle contracture associated with deep squeezing pain and autonomic manifestations of lightheadedness, diaphoresis, or nausea. Integrated hypothesis and its later modification propose that the relative lack of ATP (and perhaps oxygen), which is required to break the cross-bridges between actin and myosin filaments leads myosin filaments getting stuck at the Z band to form an MTrP.[1] Increased metabolic stress from sustained muscle contraction combined with a relative reduction of blood flow probably triggers increased release of inflammatory mediators (IM) and neurotransmitters, which ensure the generation and persistence of MTrPs in MPS. Abnormally contracted sarcomeres seen to be arranged unevenly on histopathology of human MTrPs corroborates this.[6]

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Table 1: Salient differences between acupuncture, ultrasound guided dry needling (USGDN), and conventional blindly performed dry needling (DN)

	Acupuncture	USGDN	Conventional DN
Diagnostic requirements	Solely based on Chinese philosophy	Both history and examination by a pain physician necessary for medical diagnosis of neuropathy and/or myofascial pain by physical demonstration of myofascial trigger points (MTrPs)	Demonstration of myofascial trigger points (MTrPs) necessary for diagnosis of myofascial pain
Process for detecting MTrPs and presence of MPS	There is no concept of muscles or MTrP in acupuncture	Application of just enough digital pressure to blanch the nail bed of examiner should provoke muscle pain. "Jump sign" is a jump in the muscle elicited when a taut band is strummed with the examiner's fingertips at 90^0 to the muscle. But the best confirmation is visualization of LTR pathognomonic of MTrP during USGDN.	Demonstration of MTrPs with digital pressure and "jump sign" on digital palpation or strumming with the examiner's fingertips
Needle insertion	Into specific acupoints on designated meridians. No anatomical references	Needles are inserted along both the length and breadth of all layers of muscles underlying the pain diagram drawn by patient, 2-3 cm apart, to address the multitudes of MTrPs, as confirmed by LTRs. This includes muscles exhibiting pain on digital pressure and jump sign and its coworking muscles (agonists, synergists, antagonists, fixators) as well as muscles in the kinetic chain of the original painful muscles.	Needles inserted into the most painful points (MTrPs) on palpable taut bands in muscles that have exhibited pain on digital pressure and jump sign on clinical examination.
Number of needles per session	6-10 (more used occasionally)	30-60 (more used occasionally) to address the multitudes of MTrPs as confirmed by LTRs in many muscles. This comprehensive needling might be the reason for the consistent efficacy of USGDN	6-10 (rarely more) This number of needles may leave many MTrPs in a muscle untreated.
Needle length	13-25 mm (rarely longer)	13-120 mm into the depths of muscle under ultrasound visualization. LTRs are most common in the deepest layers of muscle and deep-seated muscles like multifidus piriformis, Gemelli in back pain, serratus anterior just superficial to intercostal muscles in chest, etc.	Prudent and very real fear of complications dictates use of short 25-50 mm (rarely 75 mm) needles, which may not reach deep-seated MTrPs.
Duration of needle left *in situ*	Usually 20 mins	20-30 min. Needle is slowly and smoothly advanced in small increments, and when at maximum depth, left *in situ* till ultrasound shows muscle quiescence indicating deactivation of MTrPs (usually 20 min)	< 1 min. Needle is introduced, pumped, and then withdrawn all within a few seconds.
Sessions	Not specified	Usually, 8-10 treatment sessions but can go up to 20 sessions	Up to 6 treatment sessions
LTR visualization	Not anticipated nor looked for	LTRs are routinely visualized on ultrasound, hence no need to specially elicit them. LTRs seen in all coworking muscles, even where physical exam does not demonstrate MTrPs (might be below the pain threshold)	Efforts to see LTR through the skin by pumping the needle up and down multiple times
Practitioner expertise	Training in acupuncture	In-depth knowledge of muscle anatomy, sonoanatomy, and ability to steer needles under ultrasound essential	Knowledge of muscle anatomy is sufficient.
Influence of practitioner training	Acupuncturist trained to target acupoints	Pain physicians trained in various fluoroscopy and ultrasound guided interventions have the option to use USGDN as the sole treatment or in addition to neural interventions in neuropathic pains and MPS, targeting both pain and disability as two manifestations of the same pathology.	Physiotherapists trained to use DN as a part of physiotherapy in MPS. Pain relief is the main goal. Disability relief is not targeted.
Associated risks and complications	Neurovascular and visceral injuries reported	Bruising is the only complication because ultrasound visualization clearly demonstrates the moving pleura, peritoneum, and pulsating vessels thus avoiding the risk of visceral and neurovascular injuries.	Neurovascular and visceral injuries have been reported, including serious complications like pneumothorax.
Indications	Mainly for medical diseases and also for pain.	Indicated for pain, stiffness, and disability. Also useful in painless conditions such as vertigo and persistent hiccups, and spastic conditions like cerebral palsy, dystonias or deformities after stroke, postsurgical contractures, and keloids.	Only indicated for pain

DN, dry needling; USGDN, ultrasound guided dry needling; MTrPs, myofascial trigger points; MPS, myofascial pain syndrome; LTR, local twitch response. Needles are left *in situ* for 20-30 min during USGDN because ultrasound videos have shown LTR activity to persist for about 15-20 min and rarely even 40 min (videos available), indicating that a longer needle sojourn in muscle is required to end the LTR and deactivate the MTrP. While the LTR is ongoing, the muscle appears to grip the needle, making withdrawal very painful and difficult. After the LTR subsides, the needle comes out smoothly and painlessly, indicating muscle relaxation

Microdialysis by Shah *et al.*[7] demonstrated significantly higher IM levels, comprising protons, bradykinin, calcitonin gene-related peptide, substance P, tumor necrosis factor, interleukin-1, serotonin, and norepinephrine in the local milieu of active MTrPs compared with latent MTrPs, which, in turn, is higher than that in normal muscle. Samples obtained before and after DN showed lower IM in the MTrP after DN, presumed to be due to increased local blood flow washing out the IM, which reduces pain and tenderness. These authors described MPS as a complex form of neuromuscular dysfunction: the neurogenic inflammation and IM in the tissue milieu of the MTrP stimulate muscle nociceptors and sensitize the afferent nociceptive nerves. This peripheral sensitization of muscle nociception progresses to central sensitization, and later limbic system dysfunction with ongoing initiation, sustenance, amplification, and perpetuation of MPS.[8]

Magnetic resonance elastography and 3D ultrasound can reliably assess and quantify the mechanical characteristics of the MTrP, such as localized areas of increased muscle stiffness at the taut

Table 2: Effectiveness of USGDN in 220 patients of complex regional pain syndrome (CRPS)

Anatomical location, age	Patient No, CRPS type	Budapest criteria	Budapest criteria, treatment given, disability of arm, shoulder and hand score (DASH), lower extremity function score (LEFS), ultrasound changes and post-treatment Budapest criteria, return to prior lifestyle
Upper extremity (UE)	168 patients 50 men 118 women 160 CRPS-1 patients and 8 CRPS-2 patients	+ve in all 168	The first 8 patients out of the 168 received only stellate (sympathetic ganglion) block (SGB) and continuous brachial plexus block (CBPB) and no USGDN. All 8 required 8-10 weeks of CBPB, which was difficult to maintain. One patient failed to improve. Later, 23 patients received SGB, CBPB, and USGDN. Addition of USGDN reduced the recovery time from 8-10 weeks to 3-6 weeks. Once the mechanism of cocontraction was understood we used USGDN as the sole treatment modality in the later 137 patients. We discontinued the blocks since all the complications were associated with the catheter used for CBPB and none with USGDN.
Post stroke CRPS	5 All had upper extremity CRPS	+ve in all 5	
Bilateral CRPS	5 Upper extremity CRPS	+ve	
Recurrent CRPS	2 Upper extremity CRPS	+ve	
Pediatric age group	2 Upper extremity CRPS	+ve	Budapest criteria: Resolution of all the symptoms and signs that form Budapest criteria like sensory, sudomotor, vasomotor, and motor manifestations was documented in 168 patients. Ultrasound demonstrated loss of outline, hyperechogenecity, reduction of muscle bulk in all patients. Seven patients with early CRPS with florid manifestations showed occasional muscle edema and intermuscular effusions. In some patients, the muscle disruption was limited to few muscles while patients with later CRPS showed marked changes in all the muscles. After 1 month of USGDN, all these muscles showed improvement with disappearance of muscle edema, intermuscular effusions, return of islands of hypoechoic muscle amid hyperechoic fibrous tissue, returning definition of muscle outlines, and increasing muscle bulk All patients showed >80% improvement in DASH scores, range of motion with restoration of normal hand functions. >98% patients returned to prior lifestyle.
Lower extremity (LE)	48 patients; 20 men 28 women 1 teenager. 44 CRPS-1 patients and 4 CRPS-2 patients	+ve in all 48	35 patients received neural interventions (NI) like continuous blocks of sciatic nerve(n16) and lumbar plexus,(n 8) lumbar sympathetic block,(n3) and PRF of composite nerve supply of knee along with USGDN and ankle (n 8). 13 patients received only USGDN. Lower extremity CRPS patients seemed to improve faster with continuous sciatic block which appeared to expedite and facilitate painless weight-bearing. The ultrasound changes with CRPS were similar to upper extremity CRPS and their response to USGDN was identical. Disability was assessed with lower extremity function score (LEFS). All patients showed resolution of all the symptoms and signs that form Budapest criteria like sensory, sudomotor, vasomotor, and motor manifestations. >80% improvement in LEFS with resumption of normal unaided walk in all the patients. >98% patients returned to prior lifestyle.
Chest wall	4 men CRPS-1	+ve in 4	All the 4 patients showed resolution of all the sensory, sudomotor, vasomotor, and motor symptoms and signs that form Budapest criteria with USGDN.

The pathology of CRPS appears to be primarily motor; with formation of abundant MTrPs and taut bands in the agonist/antagonist muscles such as flexor/extensors, supinator/pronators, and adductor/abductors. The taut bands in these muscle groups impair reciprocal inhibition essential for smooth movements. The tautness in coworking muscles culminates in an abnormal cocontraction, which severely impedes all extremity and digital movements. Attempted movements of muscles tethered by constant cocontraction lead to friction at the digital tenosynovial sheaths giving rise to inflammation. Thus, the motor impairment due to cocontraction forms the primary pathology of CRPS giving rise to tenosynovial inflammation. Budapest criteria are, but manifestations of tenosynovial inflammation presenting with all its classical features; namely, rubor (redness, the vasomotor feature of CRPS), dolor (pain and other sensory features), calor (temperature asymmetry, another vasomotor feature), and tumor (swelling or sudomotor manifestation of CRPS). Relaxation of the cocontracted agonist/antagonist muscles of the CRPS-affected limb by USGDN automatically reduces the tenosynovial friction and resolves the inflammatory tendinosis in the hand, thereby reversing the pain, sensory features, warmth, and swelling (vasomotor and sudomotor) and allows a return of the normal coordination between the flexor (agonist) and extensor (antagonist) muscles with dramatic improvement of stiffness, weakness, and disability. Ultrasound documentation of structural disruption in CRPS-affected muscles, as well as their reversal after USGDN, supports this theory.[12-17] DASH, disability of arm shoulder hand score; LEFS, lower extremity function score; MTrPs-myofascial trigger points

band and the disordered recruitment of muscle fibers that results in weaker, painful, and incoordinate movements.[9,10]

DN Versus USGDN

The foundational work on MTrPs and DN was carried out by eminent physicians, yet the current practice and formal teaching of DN are carried out by physiotherapists. Needles are inserted by anatomical guesswork which can miss low-intensity LTRs, MTrPs in deeper muscles, and those in obese patients [Table 1]. There is no way to identify evolving, yet-to-become-painful MTrPs, nor the presence of taut bands, which are still too fine to be felt by gross clinical examination. MTrPs in coworking muscle groups are neither acknowledged nor elicited. These might well be the reasons for the findings of a 2017 systematic review and meta-analysis on DN effectiveness performed by physical therapists. The authors concluded that that the efficacy of DN in reducing pain in short-term follow-up had low-to-moderate quality evidence compared with no treatment. Evidence for the long-term benefit of DN is currently lacking.[11] These results

Table 3: A snapshot of the results in a few subsets of chronic pains treated from 2004 to 2019 out of 12,000 patients

Conditions	Patients	Brief synopsis of improvements and the extent of benefit
Neuropathic pains[5,12-27]	1221	>90% pts achieved the endpoint of meaningful and lasting pain relief. Combination USGDN with PRF of local nerves or botulinum toxin A (Botox)/trigger point injections, showing that these were neuromyopathic pains
CRPS-1 & CRPS-2[5,12-17]	220	>95% of pts achieved end points of 100% pain relief, 90% disability relief, and return to work within USGDN (69/220 pts)
Postspine injury (formerly termed causalgia)	3	All pts had resolution of pains with lumbar sympathetic PRF + USGDN and discontinued opioids. One patient with a high-velocity rifle shot injury has no pain, can walk with calipers for exercise, and created a national record in pistol shooting. He returned to a desk job in the elite special forces of Indian army. He visits us once a year for maintenance USGDN. Two others after traffic accidents are largely pain-free and lead active lives.
Brachial plexus injuries (BPI)	11	10 pts had complete pain relief with only USGDN, indicating BPI pain was probably myofascial. 5 regained normal movements with USGDN suggesting that the motor deficit in BPI might be due to low grade co-contraction impeding movements
Poststroke pains	18	13 pts had >80% pain relief after 3 USGDN sessions and motor improvement after 10-12 sessions. 3 patients received ultrasound guided botox injection into muscles in addition to USGDN. Two patients did not improve
Deafferentation pains	2	One patient became pain-free with only USGDN and the other with intrathecal pump with baclofen for her painful spasms. USGDN led to motor improvement suggesting that motor deficit might be due to low-grade cocontraction impeding movements.
Phantom pains	3	One pediatric patient and 2 adults reported a distinct reduction of the frequency, duration as well as intensity of phantom pains with USGDN alone. One adult requires maintenance USGDN (2-3 sessions) once in 3-6 months.
Herpes and postherpetic neuralgia	35	30/35 pts received USGDN alone, local intercostal nerve PRF + USGDN in 5 pts. >90% achieved endpoint of reduction of pain hyperaesthesia and allodynia within 3 USGDN sessions and complete, lasting relief with 10-12 sessions with no later recurrences.
Trigeminal neuralgia (Article in review)	62	>80% pts achieved the endpoint of remission with complete pain relief with minimal or no medications with USGDN of masticatory and neck muscles alone (42/62 pts). Ultrasound demonstrates masticatory muscle twitches coinciding with neuromyalgic attacks. In 20/62 pts, mandibular nerve PRF preceded USGDN to reduce the frequent intense pains.
Postsurgical neuropathy[18,19,25]	108	>90% of patients achieved endpoint of pain relief and improved functionality, stop/reduce opioids with USGDN alone, or in combination with local blocks/PRF/botox, and physiotherapy.
Failed back surgery syndrome[18]	102	>70% of patients reached the endpoint of pain relief, stop/reduce opioids with combination of USGDN with neural interventions. Neural interventions improved pain but USGDN improved both pain and disability. Pts could resume active professional life after USGDN (One more Publication not included in references)
Back pain from various causes	1209	>85% of patients reached the endpoint of pain relief, could stop/reduce opioids, Neural interventions improved pain but USGDN relieved pain and allowed them to increase activity till they could resume active professional lives.
Knee pain from osteoarthritis[27]	396	>95% of patients achieved endpoints of pain relief, improved functionality graded by Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), stopped/reduced opioids. USGDN with or without neural interventions (PRF) led to consistent predictable increase in activity and resumption of active lifestyles.
Knee pain from rheumatoid arthritis (RA)	10	ALL patients achieved endpoint of pain relief, could stop/reduce opioids, neuromodulators but continued RA treatment. Combination of USGDN with PRF showed consistent predictable but dramatic pain reduction achieving hitherto impossible active lifestyles (SF16 and WOMAC) (Publication not included in references)
Frozen shoulder	110	>85% of patients achieved endpoint of pain relief, could stop/reduce analgesics. USGDN alone (15 sessions) or in combination with PRF of the composite nerve supply of all shoulder muscles allowed a sustained, painless return of all shoulder movements within 30-45 days.
Headaches and migraine	81	>95% of patients achieved endpoint of pain relief with USGDN and USG-guided Botox into all the neck muscles including those of suboccipital triangle. C1-C3 PRF was done in selected patients to address the composite nerve supply of neck muscles. The frequency and the severity of attacks and medications were reduced by >90%
Chronic pelvic pain[26]	20	>85% achieved the endpoint of meaningful pain relief, improved urinary/rectal function improved quality of life (SF16), could stop/reduce opioids after a combination of continuous caudal block, Botox, and USGDN of pelvic floor muscles.
Myofascial pains	647	>90% of patients achieved the endpoint of pain relief, could stop/reduce analgesics while increasing activities. Combination of USGDN with Botox/trigger point injections/PRF of nerves to local muscles allowed a sustained, painless return to higher levels of activity with physiotherapy for strengthening.
Writer's cramp	6	All 6 had complete pain relief of pain after 8-10 sessions of USGDN (publication not included in references)
Fibromyalgia	11	Endpoint of lasting pain relief was not possible but >60% had pain relief with a combination of USGDN with Botox/trigger point injections/PRF of nerves to local muscles allowed a better quality of life on SF16. But pains kept coming up elsewhere.
Cancer pain[19-24]	294	Neuropathic pains after cancer and its therapies are majorly neuromyopathic and respond to a combination of neural blocks, USGDN, and USGDN-guided Botox. >80% of patients achieved endpoint of a good quality of life with pains <1-2 with minimization of opioid doses.

CRPS, complex regional pain syndrome; PRF, pulsed radiofrequency; Pts, patients; USGDN, ultrasound guided dry needling; More references are available from our publications for the conditions treated but could not be included due to limited allowance for the number of references. Trigeminal neuralgia patients with frequent pains (VAS >6) were selected for PRF prior to USGDN.

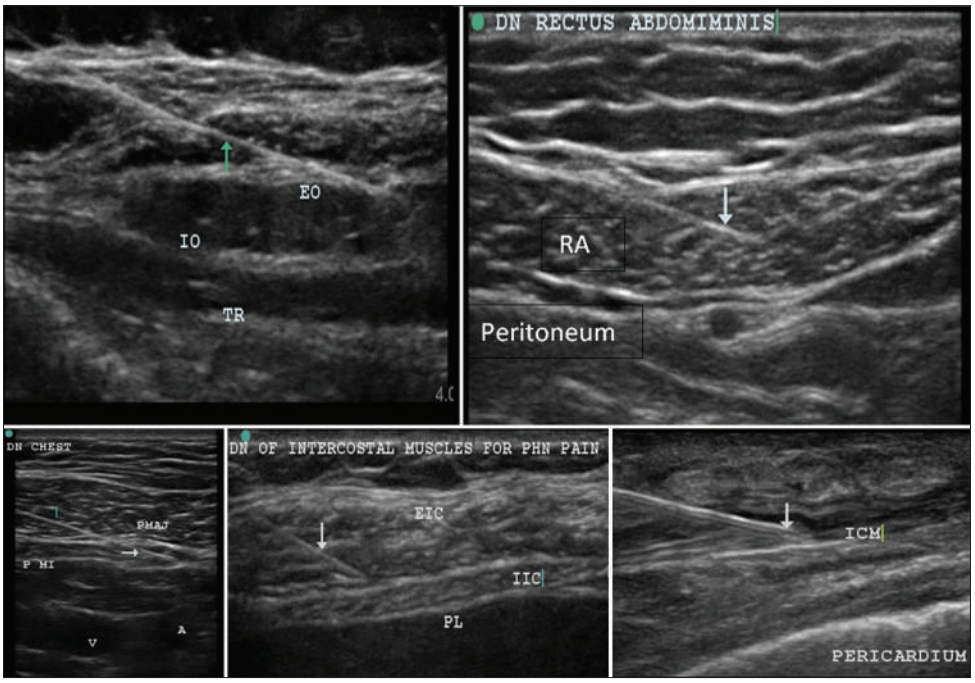

Figure 1: Advantages of ultrasound visualization and the necessity for ultrasound visualization during dry needling.Ultrasound images showing needling of abdominal wall muscles (*top left* and *right*), chest wall muscles (*bottom left*), and intercostal muscles (*bottom middle* and *right*). Ultrasonography allows direct visualization of pleura, peritoneum, pericardium, and neurovascular structures so that needles can be steered into muscles while safeguarding these vital structures. EO, external oblique; IO, internal oblique; TR, transversus abdominis; DN, Dry needling, RA-rectus abdominis; PMAJ, Pectoralis major; P MI, Pectoralis minor; V, subclavian vein; A, subclavian artery; PHN, post-herpetic neuralgia; EIC, external intercostal muscle and the muscle superficial to it is the serratus anterior; IIC, internal intercostal muscles; PL, pleura, ICM-intercostal muscle

are very markedly different from the effectiveness of USGDN at our center, in a variety of pain conditions [Tables 2 and 3].[5,12-27]

USGDN requires a thorough knowledge of sonoanatomy and the ability to steer needles under ultrasound visualization, which ensures accurate needle placement [Figure 1] while avoiding visceral, pleural, and neurovascular injury, all of which have been reported with blindly performed DN.[1] But we believe that by far the most significant lacuna of blind DN is that it confines the practitioner's understanding of MPS to a few detectable MTrPs. In contrast, USGDN emphasizes the actual enormity of the MPS problem by demonstrating LTRs in coworking muscles as the needle passes through various muscle layers, particularly in the deepest muscles closest to ribs, pleura, and peritoneum. Furthermore, observation of structural disruption of muscle in complex regional pain syndrome (CRPS) and its reversal by USGDN has led to a novel understanding of CRPS pathophysiology [Figure 2, Table 2].[12-17]

Ultrasound Observation of the Sequence of Events in Muscles and Their Clinical Correlation during USGDN (Videos 1-14)

1. The sudden sharp LTR visualized on needle introduction is associated with a sense of heaviness/sharp pain. Patients with moderate-to-severe pain may show additionally sustained twitches that follow LTR. After needle removal, the original pain and stiffness show perceptible reduction.
2. Physicians down the ages could never objectively confirm the presence of pain. However, severe pain can be seen as constant shimmers (twitches) in painful muscles at rest, whereas its normal counterpart has none. Shocks in trigeminal neuralgia coincide with sudden muscle twitches, which subside 5–15 min after LTR. The resolution of twitches corresponds to perceptible pain relief after needle removal. This makes ultrasound the first investigative modality to objectively "demonstrate" pain and its relief by USGDN.
3. The post-USGDN pain relief is reflected as muscle quiescence in the next session. Patients report stiffness reduction as well. Cumulative reduction of pain, stiffness, and weakness over successive USGDN sessions translates into a complete reversal of pain and disability in a variety of neuropathic conditions [Figure 3].[5,12-27]

Potential of USGDN to Revolutionize Neuropathic Pain Treatment

Neuropathic pains that form the majority of chronic pains are defined as "pains arising as a direct consequence of a lesion or disease affecting the somatosensory system."[28] Hence, current treatments target the different components of the somatosensory pain pathway, including:

1. Central nervous system (oral medications, intravenous lignocaine/ketamine infusions)
2. Spinal cord (epidural injections, neurostimulation, intrathecal drug delivery)

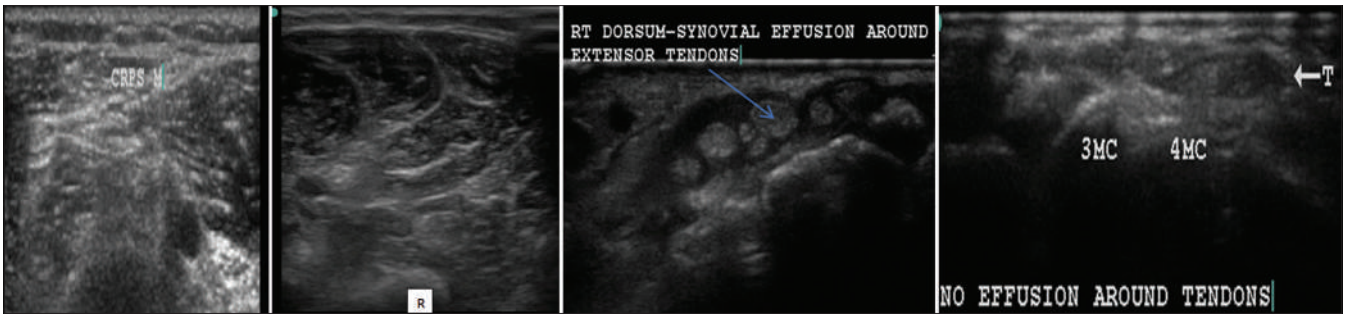

Figure 2: Muscle ultrasonography as a diagnostic and prognostic tool in CRPS. Ultrasound images before and after USGDN of the CRPS-affected hand. Ultrasound images of the flexor muscles of forearm just below the elbow before USGDN showing a complete loss of normal outlines and individual muscles cannot be distinguished 1st image. Loss of normal muscle structure is a consistent diagnostic feature of CRPS. The 2nd image after USGDN shows the return of normal outlines as well as return of hypoechoic muscle fibers in the muscles. The bony outlines of radius and ulna obscured by the hyperechoic echoes pre-USGDN become clearer after treatment. There is also an increase in muscle bulk, compared with 1st image. 3rd image shows the tenosynovial effusion around the extensor tendons presumably due to tenosynovial friction from the pull on the tendons by cocontracted digital extensor and the flexor muscles. 4th image shows no effusion post-USGDN. USGDN routinely relaxes agonist/antagonist muscle groups, relieves the effusion, resolves the hallmark stiffness and immobilization of CRPS, and restores the normal reciprocal inhibition between agonist/antagonist muscle groups essential for coordination of movements. MC, metacarpal bone; T, tendons

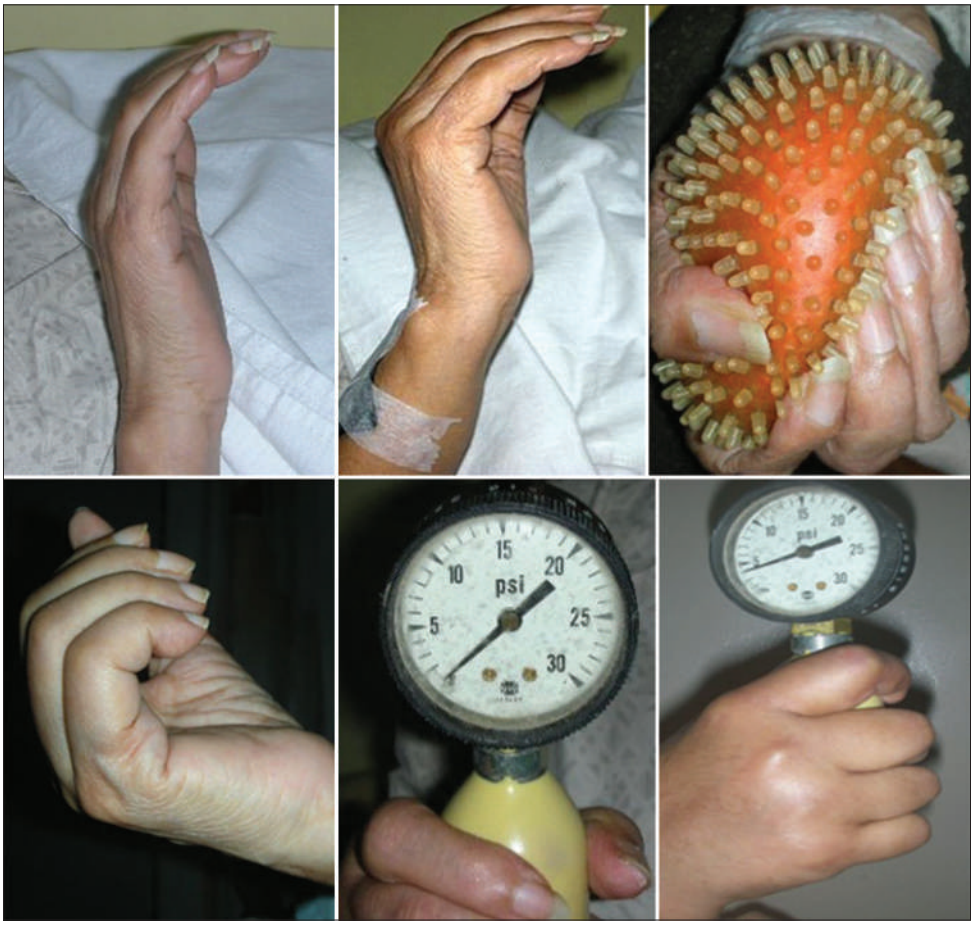

Figure 3: Cumulative result of serial ultrasound guided dry needling (USGDN) of agonist and antagonist muscles (digital flexors and extensors) with sequential improvement of pain stiffness and weakness in patients of complex regional pain syndrome. *Top row*: Appearance of the hand on *day 3* (*left*) with the fingers fixed in minimal flexion with inability to further flex the fingers, *day 7* (*middle*) where she is able to flex the fingers. She is squeezing the ball on *day 10* (*right*) after USGDN was initiated on *day 1* carried out thrice weekly. *Bottom row*: Appearance of the hand on *day 12* (*left*) showing a gradual increase in the flexion at the metacarpophalangeal and interphalangeal joints to enable the formation of a fist to hold a dynamometer on *day 17* (*middle*) and with gradual increase in grip strength from 0 pounds per square inch to 4 pounds per square inch by *day 22* (*right*)

3. Somatic nervous system (peripheral plexus and nerve blocks, neuropreservative/ablative radiofrequency procedures)
4. Sympathetic ganglia (stellate/lumbar sympathetic/ganglion impar/coeliac/hypogastric plexus blocks)

However, the above interventions leave behind significant residual pains for which opioids are routinely prescribed, in both cancer and noncancer pains. Unfortunately, neuropathic pains are notoriously resistant to opioids leading to drug-seeking behavior, a major contributor to the present opioid crisis in the west.

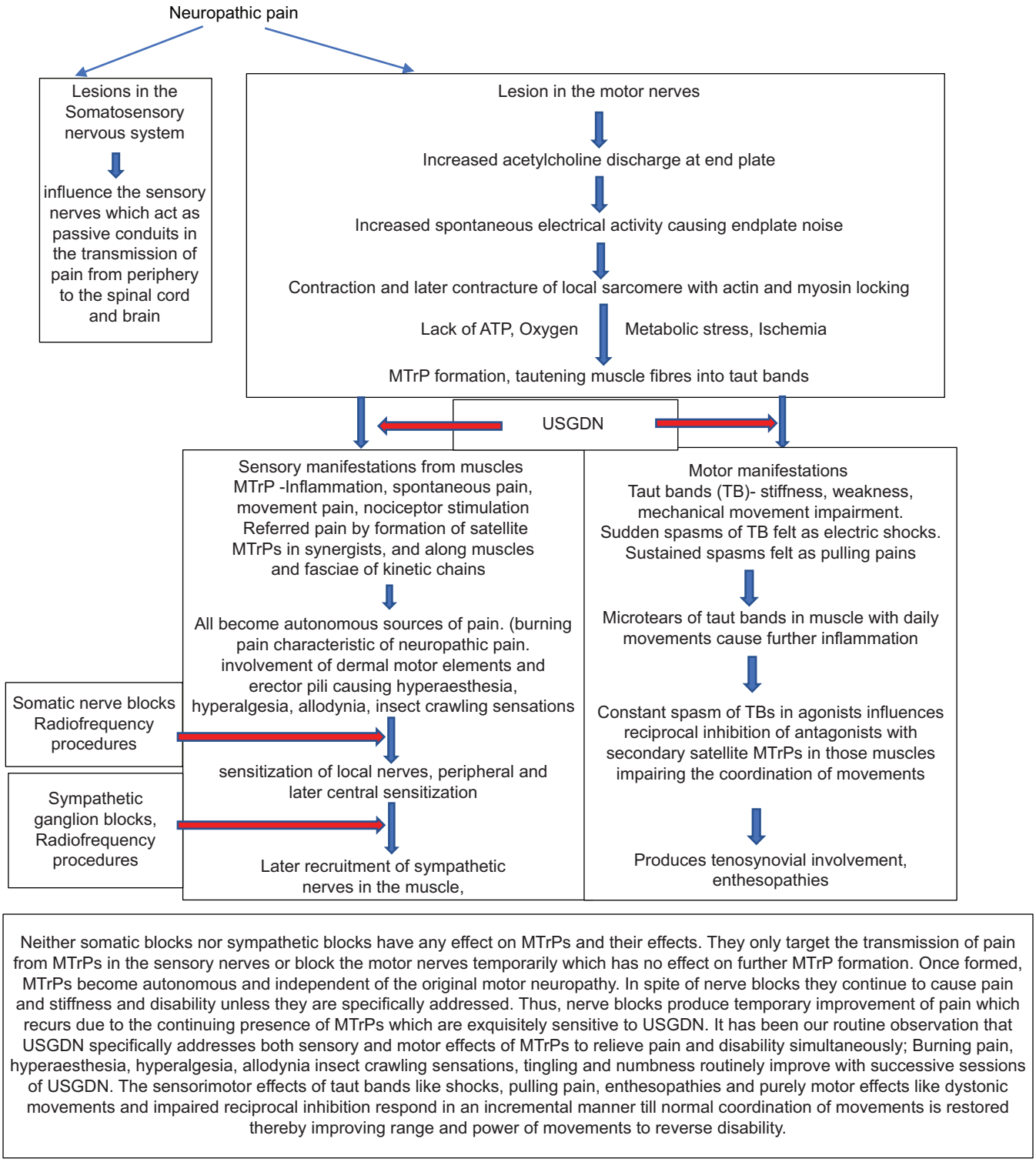

Figure 4: A diagrammatic representation of a working hypothesis to explain neuromyopathy; sequence of events leading to MTrP generation, production of inflammatory mediators, consequences of MTrP, and taut bands. The relevance of neural blocks and USGDN in this sequence of events and the significance of USGDN in neuropathic pains are shown

Figure 4 shows the diagrammatic representation of a working hypothesis, which links the robust evidence for MTrP genesis to motor neuropathy,[1-10] to explain the staggering prevalence of MPS (70%–95%) in chronic pains.[29-32] Instead of dismissing this MPS as "secondary" to pain and disuse,[28] this hypothesis emphasizes that muscles are not just passive expressors of neuropathy/neuromyopathy but are its dynamic perpetrators, facilitators, sustainers, and amplifiers. The sheer interdependent complexity of muscle function ensures the production of myriad bizarre symptoms, which are the hallmark of neuropathic pain syndromes. These pains from MPS persist despite surgery, opioid administration, and current pain management interventions, including spinal cord stimulation.

These recalcitrant pains from MPS are comprehensively addressed by systematic USGDN and USGDN-guided botulinum toxin injection, with a dramatic and lasting reversal of many chronic pain conditions [Tables 2 and 3], including the residual pains that persist after current neural interventions. USGDN is unique in providing not just pain relief but lasting disability relief, which all the other current interventions fail to achieve. Routine and predictable relief of "sensory neuropathic symptoms" such as burning, allodynia, hyperalgesia, and hyperaesthesia (in postherpetic neuralgia, postsurgical neuropathy, postlaminectomy syndrome, cancer pains, brachial plexus injuries, CRPS, etc.) by USGDN suggests an interesting possibility that these "sensory" symptoms might actually be manifestations of intense spasm of underlying muscles and/or dermal motor elements and erector pili muscles.[16]

Conclusion

A paradigm change of the current understanding of neuropathy that includes the motor as much as the somatosensory system not only explains the ubiquitous presence of MPS in chronic pains but also opens up new exciting possibilities in pain management. Effective treatments such as USGDN alone or in combination with neural interventions could significantly reduce the biological contribution to the biopsychosocial model of chronic pain. Further research is required to unequivocally prove the concept of neuromyopathy and the efficacy of USGDN over opioids through well-designed clinical trials.

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