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Mechanisms and Clinical Effects of Neuromuscular Electrical Stimulation on Arthrogenic Muscle Inhibition Following Ankle Sprain

Jiayi Zhao

Golden Apple Jin Cheng No.1 Secondary School, Chengdu, China
johnnynb666@gmail.com

Abstract. Lateral ankle sprains (LAS) are acute musculoskeletal injuries with an extremely high incidence rate in competitive sports and daily physical activities worldwide; due to incomplete rehabilitation, up to 40% of patients ultimately develop chronic ankle instability (CAI). In exploring pathological models of CAI onset and progression, modern sports rehabilitation medicine clearly indicates that the core neurological barrier hindering patients from achieving dynamic joint rehabilitation is Articulo Muscular inhibition (AMI). AMI manifests as abnormal sensory input from the damaged joint capsule and torn ligaments, which continuously inhibits the surrounding, unaffected peroneal muscle groups, leading to a significant decrease in the excitability of the spinal motor neuron pool. Consequently, patients are unable to effectively mobilize motor units for tonic contraction through voluntary effort during the early stages of rehabilitation, trapping them in a vicious cycle of "injury—inhibition—atrophy—re-injury." Neuromuscular electrical stimulation (NMES)—a targeted electrotherapy modality—disrupts this neural inhibitory network by delivering specific pulsed currents through the skin to directly depolarize peripheral motor axons. This thereby elicits involuntary muscle contractions without the need for descending drive from the cerebral cortex. This article elaborates on the neuromodulation mechanisms of AMI following ankle injury, summarizing the latest evidence-based medical findings and clinical parameter designs for NMES in eliminating reflex inhibition, reshaping motor control, and enhancing joint stability, with the aim of providing a reference for the development of neurorehabilitation protocols for lower limb dysfunction.

Keywords: Neuromuscular Electrical Stimulation (NMES), Articulo-Muscular Inhibition (AMI), Ankle Sprain

1. Introduction

Lateral ankle sprains (LAS) are musculoskeletal injuries with an extremely high incidence rate in sports and daily physical activities; the mechanism of injury typically involves sudden, excessive inversion and internal rotation forces applied while the foot is in a plantar flexed position [1, 2]. The vast majority of acute injuries involve partial or complete structural tears of the lateral ankle ligament complex, with the anterior talofibular ligament (ATFL) and calcaneofibular ligament (CFL)

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being the most commonly affected [1]. Currently, conservative treatment protocols have been widely established in the medical community; however, clinical follow-up data indicate that if rehabilitation interventions are inadequate or unsystematic, up to 40% of patients with acute sprains will experience persistent residual symptoms that ultimately progress to chronic ankle instability (CAI) [2-4]. Clinically, CAI exhibits highly heterogeneous pathological characteristics, primarily manifested as a persistent sensation of joint "giving way" during weight-bearing walking or exercise, frequent recurrent inversion sprains, intractable chronic inflammatory pain, and severe impairment in the ability to perform dynamic balance tasks such as single-leg standing. This not only greatly limits patients' athletic performance but also significantly increases their long-term risk of secondary joint pathology [3, 4].

In exploring the complex pathophysiological model underlying the onset and progression of CAI, modern sports rehabilitation medicine has increasingly recognized that joint-induced muscle inhibition (AMI) is the key neurological barrier that delays or even blocks the normal rehabilitation process [4-6]. Clinically and physiologically, AMI is clearly defined as a persistent, involuntary reflex inhibition occurring in muscle groups surrounding the injured joint that are structurally intact and unaffected [5, 7]. Specifically in the context of a lateral ankle sprain, abnormal sensory feedback from the damaged joint capsule and torn ligaments directly leads to a state of high inhibition in the spinal motor neuron pools innervating the peroneus longus and peroneus brevis muscles [5, 6]. This neurological blockade means that even if the muscle tissue of the patient's lateral muscle groups is anatomically completely healthy, the patient finds it difficult to effectively mobilize motor units for tonic contraction by issuing motor commands through voluntary will [4, 7]. Consequently, in the very early stages of rehabilitation, relying solely on traditional voluntary strength training often yields minimal results; not only does it fail to effectively induce muscle strength gains, but it also risks trapping patients in a vicious cycle of "joint injury—neural inhibition—muscle atrophy—dynamic instability—recurrent sprains" due to repeated frustration and pain [3, 4]. To completely break this pathological cycle, there is an urgent clinical need for targeted interventions capable of bypassing the impaired central nervous system inhibitory network [5, 8]. Neuromuscular electrical stimulation (NMES) is precisely such an intervention tool; by delivering pulsed currents with specific parameters through the skin, it acts directly on peripheral motor nerve axons to induce involuntary muscle contractions without requiring descending drive signals from the cerebral cortex, while simultaneously modulating spinal cord excitability through extensive proprioceptive afferent feedback [7, 8]. This literature review aims to thoroughly analyze the neurophysiological mechanisms underlying the development of AMI following a lateral ankle sprain and to systematically evaluate the latest clinical evidence regarding NMES in reversing muscle inhibition, improving dynamic muscle torque, and enhancing long-term ankle stability, with the goal of providing clinicians with a solid theoretical and evidence-based foundation for developing more scientific and effective ankle rehabilitation strategies [8, 9].

2. Pathophysiological mechanisms of AMI following ankle sprains

2.1. Abnormal changes in sensory input

The onset of AMI follows tissue trauma to the lateral ankle complex [1, 5]. Structural tears in the ATFL, a local inflammatory cascade, and intra-articular effusion alter the mechanical pressure within the joint capsule [5, 7]. These mechanical and chemical stimuli activate Class III and Class IV nociceptive afferent fibers while disrupting the normal firing patterns of low-threshold mechanoreceptors (such as Ruffini endings and Pacini corpuscles) [5, 7]. These abnormal sensory

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input signals are transmitted via Class Ia and Class II nerve fibers to the dorsal horn of the spinal cord, significantly altering the excitability of the spinal intermediate neuron network [5-7]. These fundamental changes in sensory input continuously transmit abnormal pathological impulses to the central nervous system, directly leading to functional disruption of the neural circuits innervating distal stabilizing muscle groups, and further exacerbating and consolidating the pathological progression of chronic ankle instability (CAI) [3, 4].

2.2. Decreased excitability of motor neurons in the spinal cord and supraspinal centers

The core neurophysiological feature of AMI is a significant decrease in the excitability of the α-motor neuron pool controlling the peroneal muscle group [5, 6]. Electrophysiological assessments show that the amplitude of the Hoffman reflex (H-reflex) is significantly reduced in patients with acute ankle sprains and CAI [5-7]. This attenuation reflects enhanced presynaptic and postsynaptic inhibition at the spinal cord level [6, 7]. Furthermore, abnormal sensory input is transmitted upward to the upper and middle spinal centers, leading to altered cortical motor excitability and weakened motor cortical drive [4, 5]. Consequently, the central nervous system is unable to generate sufficient descending commands to initiate dynamic muscle contraction [4, 7]. This inhibition occurs not only in the spinal cord but also affects subcortical centers. This prevents the peroneal muscle group from activating properly, resulting in a loss of dynamic joint protection, which ultimately directly triggers and exacerbates chronic ankle instability (CAI) [3, 4].

2.3. Functional Impairment of the peroneal muscle group

The peroneus longus and peroneus brevis muscles are the primary dynamic stabilizers against excessive foot inversion [4, 10]. When arthrogenic muscle inhibition (AMI) compromises these muscles, two major dynamic dysfunctions arise: a failure of selective activation and prolonged reflex response times [4, 5]. During sudden ankle inversion (e.g., stepping on uneven ground), inhibited peroneal muscles fail to generate timely eversion torque [4, 10]. Without prompt muscle activation, the joint easily inverts past its mechanical limits, leading to recurrent sprains and joint surface damage [1, 2]. This combined loss of strength and reaction speed impairs athletic performance [4, 10]. Biomechanically, it creates a harmful cycle that ultimately worsens chronic ankle instability (CAI) [2-4].

3. Neurophysiological mechanisms by which NMES overcomes AMI

3.1. Overcoming neural inhibition mechanisms

Unlike voluntary contraction, which relies entirely on the cerebral cortex to issue voluntary motor commands, NMES directly depolarizes motor axons at the peripheral level [5, 8]. Transcutaneous electrical stimulation delivers pulsed currents to the motor end-plates of the peroneal nerve; this artificial electrical stimulation alters the resting membrane potential of peripheral nerve fibers, generating action potentials that travel along the axons to the neuromuscular junction [7, 8]. By directly inducing muscle contraction at the peripheral level, NMES successfully bypasses the central autonomic drive blocked by AMI [5, 8]. This method of direct electrical stimulation, which bypasses central blockade, can activate inhibited muscle fibers without relying on descending commands from the brain, providing a reliable mechanostatic stimulus pathway for early rehabilitation [7, 8].

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3.2. Reducing presynaptic inhibition and resetting spinal reflex pathways

The therapeutic effects of NMES are not limited to peripheral muscle fibers but also extend to the central nervous system [5-7]. When NMES induces muscle contraction, it also generates a large amount of sensory afferent feedback that travels retrograde and anterograde along sensory nerve fibers to the spinal cord [7, 8]. This synchronized influx of sensory signals into the dorsal horn of the spinal cord modulates the activity of interneurons [6, 7]. Electrophysiological studies demonstrate that repeated NMES reduces presynaptic inhibition and restores H-reflex amplitude in α-motor neurons [5-7]. This process resets spinal reflex pathways and relieves neural inhibition on the peroneal muscles, which restores normal reflex feedback and supports long-term recovery of motor control [6, 7].

3.3. Restoration of autonomous activation and recruitment of the peroneal muscle group

Beyond spinal-level adaptations, NMES drives supraspinal neuroplasticity through mechanisms similar to long-term potentiation (LTP) [5, 8]. Sensory afferent signals generated by electrical stimulation project to the primary somatosensory and primary motor cortices [5, 7]. Combining NMES with voluntary effort (Superimposed NMES) synchronizes central motor intent with peripheral sensory feedback [5, 8]. This combined neural activation strengthens the corticospinal tract, reshapes motor cortex representation, and restores voluntary motor unit recruitment during functional movement [7, 8]. Through this central and peripheral remodeling, patients regain precise control over key stabilizing muscle groups in the lower limbs [4, 8].

4. Clinical efficacy and treatment parameters

4.1. Summary of clinical evidence and assessment measures

A growing body of clinical literature confirms that incorporating NMES into ankle rehabilitation programs can lead to measurable functional improvements across multiple key diagnostic indicators [5, 6]. In terms of surface electromyography (sEMG) amplitude, patients receiving NMES treatment exhibited a significant increase in the surface EMG amplitude of the peroneus longus muscle during maximum voluntary isometric contraction (MVIC), directly demonstrating that muscle activation failure had been reversed [5, 6]. In isokinetic and isometric muscle strength assessments, peak valgus torque increased significantly, successfully counteracting post-injury disuse atrophy and muscle weakness [5, 8]. In terms of dynamic postural control and functional scores, clinical trials have shown that dynamic single-leg balance, as assessed by the Y-Balance test, improved significantly, and patient-reported outcome measures such as the Cumberland Ankle Instability Test (CAIT) demonstrated a marked improvement in subjective stability scores [11, 12].

A double-blind, randomized controlled trial divided participants into an electrical stimulation group and a placebo group, with interventions conducted three times per week for 20 minutes each session. Ultimately, 38 participants completed the entire intervention and testing regimen. Dynamic data on pressure trajectory during static single-leg standing were collected using a force plate, and a two-factor repeated-measures analysis of variance was performed [12]. The results revealed significant interaction effects for the Cumberland Ankle Instability Scale score and balance parameters such as displacement X, displacement Y, 95% confidence ellipse area, root mean square error, and mean velocity of the center of pressure ($P < 0.05$, $0.103 \leq \eta^2 \leq 0.201$), between-group differences were observed in scale scores, X-axis displacement, root-mean-square displacement, and

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the area of the 95% confidence ellipse (P = 0.003–0.045, Cohen's d = 0.103–0.143) [12]. The results indicate that 6 weeks of neuromuscular electrical stimulation (NMES) of the peroneus longus muscle improves static balance control in young adults with chronic ankle instability, particularly enhancing frontal-plane ankle stability [12].

A systematic review and meta-analysis incorporating 23 randomized controlled trials comprehensively evaluated the effectiveness of NMES as an adjunct to ankle injury rehabilitation [9]. The results showed that the NMES group significantly outperformed the conventional control group on several key indicators: there was a more pronounced improvement in Cumberland Ankle Instability Test (CAIT) scores (MD = 2.82, 95% CI: 1.51–4.13, $P < 0.0001$), a more significant reduction in Visual Analog Scale (VAS) pain scores (SMD = −1.89, 95% CI: −2.73 to −1.04, $P < 0.0001$), and a higher clinical response rate (RR = 1.23, 95% CI: 1.14 to 1.33, $P < 0.00001$) [9]. Regarding gait parameters, the NMES group showed improvements in walking speed (MD = 0.06 m/s, 95% CI: 0.01–0.11), stride length (MD = 3.62 cm, 95% CI: 0.55–6.69), and ankle relative peak torque across all directions [9]. Although the overall stability index did not differ significantly between groups (SMD = −0.55, 95% CI: −1.919 to 0.10, P = 0.10), sensitivity analyses showed no publication bias [9]. These findings indicate that NMES increases valgus torque, optimizes gait, alleviates pain, and improves joint stability, with longer-term interventions yielding greater clinical benefit [9, 12].

4.2. Recommended intervention parameters and protocols

To optimize disinhibition while avoiding premature muscle fatigue or discomfort, electrical parameters should follow evidence-based guidelines [7, 8]. Specifically, a pulse frequency of 35–50 Hz is recommended to smoothly induce tonic muscle contractions [7, 8]. For pulse width, it is recommended to maintain a range of 200 μs to 400 μs, which allows for effective recruitment of peripheral motor nerves at an intensity level tolerable to the patient [7, 8]. Regarding duty cycle management, it is advisable to set the stimulation duration to 5 to 10 seconds, followed by a rest period of 10 to 20 seconds, maintaining a duty cycle ratio of 1:2 or 1:3 to effectively manage metabolic fatigue [7, 8]. In terms of clinical application, the use of Superimposed NMES is recommended, in which the clinician instructs the patient to actively contract the foot in an eversion direction while electrical stimulation pulses are being delivered, thereby maximizing the neuroplasticity effects on the central nervous system [7, 8].

5. Conclusion

In summary, arthrogenic muscle inhibition (AMI), as a core neurophysiological barrier in the pathological progression of lateral ankle sprains and chronic ankle instability (CAI), continuously suppresses the excitability of the spinal motor neuron pool through abnormal sensory input signals. This disrupts effective voluntary activation of the protective peroneal muscle group, thereby creating a cycle that perpetuates recurrent ankle sprains. Neuromuscular electrical stimulation (NMES), as a highly targeted modern electrophysiological intervention, can effectively bypass central nervous system inhibition through direct depolarization of peripheral motor nerve axons and, via extensive proprioceptive feedback, reset disrupted spinal reflex pathways, thereby promoting cortical motor reorganization. A wealth of modern, high-quality evidence-based medical data and meta-analysis results have fully demonstrated that NMES can significantly reverse failure of peroneal muscle activation, substantially increase peak dynamic eversion torque, optimize three-dimensional gait characteristics, significantly alleviate post-traumatic inflammatory pain, and comprehensively

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enhance patients' dynamic single-leg postural balance sensitivity and subjective joint stability. In the very early stages of conservative treatment for ankle trauma or during the golden window of postoperative rehabilitation, the proactive, early, and standardized implementation of a combined electrophysiological intervention protocol centered on superimposed NMES technology represents the most critical breakthrough for clinical rehabilitation physicians to break the pathological vicious cycle of AMI and prevent long-term functional instability. Future research should further focus on the precise, individualized adjustment of multimodal parameters and ultra-long-term follow-up cohorts to comprehensively advance the development of a precision digital rehabilitation system for the ankle joint.

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