The Application and Advancements of Transcranial Magnetic Stimulation in the Treatment of Phantom Limb Pain

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Abstract: Phantom limb pain (PLP) is a common and difficult-to-treat type of chronic neuropathic pain experienced after amputation, posing a considerable challenge in clinical management. Conventional treatments—reliant on opioids and invasive nerve blockade—often yield limited efficacy and are associated with drug dependency, tolerance, and surgical complications. In recent years, attention has shifted toward the neuroplasticity of the central nervous system, prompting the exploration of non-invasive neuromodulation strategies. Transcranial magnetic stimulation (TMS) is a non-invasive method that uses time-varying magnetic fields to generate electric currents in targeted areas of the cortex, enabling precise regulation of neuronal excitability. This approach opens a novel therapeutic avenue by targeting the pathological neuroplasticity underlying PLP. This review provides an in-depth synthesis of current research on the underlying mechanisms by which TMS contributes to the management of PLP. Through systematic literature analysis, we examine the effects of different TMS parameters, stimulation targets, and protocols in clinical trials. Furthermore, we critically analyze methodological characteristics and limitations in existing research and propose future directions under the framework of precision medicine, providing valuable theoretical insights for optimizing PLP treatment strategies.

Keywords: Phantom Limb Pain; Transcranial Magnetic Stimulation; Neuroplasticity; Neuromodulation

1. Introduction

Phantom limb pain (PLP), one of the most common types of chronic neuropathic pain following amputation, affects approximately 64% (95% CI: 60.0–68.05) of amputees worldwide[1]. The pathogenesis of PLP is complex and highly heterogeneous. Traditional pharmacological treatments, such as opioids, antidepressants, and anticonvulsants, are often accompanied by tolerance, dependence, and various side effects. While interventional treatments—such as nerve blocks or spinal cord stimulation—can provide rapid relief, they are invasive and often only produce short-term results[2]. Non-invasive approaches, such as mirror therapy, virtual reality training, and cognitive behavioral therapy, offer favorable safety profiles but are frequently limited by patient compliance and active engagement[3, 4].

Amid these challenges, transcranial magnetic stimulation (TMS) has garnered increasing interest as a non-invasive neuromodulatory method that modulates cortical excitability through electromagnetic induction. TMS employs high-frequency pulses to generate localized electric currents in targeted brain areas, effectively regulating neuronal excitability and synaptic plasticity. Its non-invasive nature, ease of operation, and relatively low incidence of side effects make it a promising alternative for PLP treatment[5].

This review presents a systematic overview of clinical evaluation approaches and prevalent treatment strategies for PLP, with a particular emphasis on recent clinical advances in the use of TMS. It explores the neuromodulatory effects of TMS and the neuroplastic mechanisms underlying its therapeutic potential. Additionally, we analyze key stimulation parameters—such as target location and frequency—with the goal of identifying the most effective protocols. Finally, we discuss the challenges and limitations currently faced in the clinical application of TMS, and explore future research directions, offering practical guidance and theoretical support for clinicians and researchers.

2. Clinical Assessment Methods and

Intervention Strategies for PLP

2.1 Definition and Clinical Characteristics of PLP

PLP is a complex neuropathic condition frequently experienced by amputees, marked by the sensation of pain in a limb that has been lost. Patients often describe the sensation as persistent burning, sharp stabbing, or cramping in the missing limb, with considerable variation in intensity and quality across individuals. PLP is not merely a sensory issue but a multidimensional condition that profoundly affects patients' psychological well-being and quality of life. Chronic pain may contribute to the development of comorbid psychological conditions, including depression, anxiety, and sleep disturbances[6]. PLP is frequently accompanied by phantom sensations, wherein patients feel the presence or movement of the absent limb. Clinical evidence indicates that negative emotions such as anxiety and depression significantly increase both the likelihood and severity of PLP. External stimuli—such as sudden temperature changes—can trigger abnormal nerve excitability at the amputation site, exacerbating pain. Furthermore, residual limb complications such as neuroma formation, nerve fiber hyperplasia, and scar tissue are also major contributors to ongoing pain.

2.2 Assessment Methods for PLP

Due to the multifactorial and heterogeneous nature of PLP, clinical evaluation must be comprehensive and systematic. Clinicians should gather detailed medical histories, conduct thorough physical examinations, and assess psychological status using standardized instruments to understand the patient's condition holistically and develop targeted treatment plans. Pain rating scales are essential tools in clinical and research settings. The Visual Analogue Scale (VAS) allows patients to mark their pain intensity on a linear scale, offering an intuitive reflection of their subjective experience. The Numerical Rating Scale (NRS) uses a 0–10 range for more precise quantification. The McGill Pain Questionnaire captures not only intensity but also sensory, affective, and evaluative dimensions of the pain experience.

Given the high prevalence of psychological disturbances among amputees—due to altered body image, functional loss, and social adaptation challenges—psychological evaluation is equally critical. Instruments such as the Hamilton Anxiety Rating Scale and the Hamilton Depression Rating Scale are widely used to quantify emotional states and provide a baseline for subsequent individualized interventions[7].

Despite the usefulness of these subjective scales, their inherent limitations—particularly in capturing the spatiotemporal dynamics of pain—have driven the search for more objective and quantifiable assessment tools. Progress in neuroscience has facilitated the incorporation of neuroimaging modalities, such as functional magnetic resonance imaging (fMRI) and electroencephalography (EEG), into both clinical assessments and research applications[8]. These tools offer deeper insights into the neuropathophysiology of PLP and help monitor treatment effects. For instance, fMRI can detect changes in blood-oxygen-level-dependent signals, illustrating cortical reorganization and alterations in pain-related networks after amputation. Foell et al. (2014)[9] showed that mirror therapy significantly reduced somatosensory cortex displacement—from an average of 15.4 mm to 2.9 mm—which strongly correlated with pain relief. Duarte et al. (2020)[10] demonstrated that mirror therapy activates bilateral primary motor cortices, somatosensory cortex (S1), and visual cortex, with visual activation negatively correlating with pain intensity—highlighting the role of visual pathways in pain modulation. Andoh et al. (2020)[11] further found that primary motor cortex (M1) activation intensity was positively correlated with pain ratings in PLP patients, reinforcing the value of fMRI in mapping the link between cortical plasticity and pain.

In parallel, EEG—due to its high temporal resolution—can capture dynamic neural oscillations in various frequency bands (alpha, beta, theta) and evaluate neurophysiological responses to interventions such as peripheral nerve or transcutaneous electrical nerve stimulation. Kleeva et al. [12] reported that EEG could effectively track PLP fluctuations and responses to different neurostimulation methods, offering a physiological biomarker for PLP. The integration of neuroimaging with conventional scales thus provides a more comprehensive foundation for accurate

diagnosis and outcome evaluation in PLP, with significant clinical implications.

2.3 Common Treatment Strategies for PLP

To date, no standardized treatment protocol has been established for PLP. Based on current clinical practices and research, treatment strategies can be broadly categorized into four groups: pharmacological therapy, interventional procedures, physical/cognitive therapies, and neuromodulation techniques (**Table 1**).

Table 1. Common Treatment Strategies for PLP

Category	Main Methods	Advantages	Limitations / Disadvantages
Pharmacological Therapy	Opioids, antidepressants, anticonvulsants, topical agents	Widely applicable; some agents also improve mood	Long-term use may cause tolerance, dependence, and side effects
Interventional Therapy	Nerve blocks, spinal cord stimulation	Direct interruption of pain transmission; effective relief	Invasive, higher surgical risks, costly, often short-term effect
Physical and Cognitive Therapy	Mirror therapy, virtual reality, physical rehabilitation, CBT	Non-invasive with fewer side effects; improves neuroplasticity and emotional status	Requires high patient compliance; variable efficacy
Neuromodulation Techniques	rTMS, transcranial direct current stimulation (tDCS)	Non-invasive modulation of cortical plasticity; effective in some patients	Long-term efficacy unclear; relatively high equipment and operation costs

Due to significant individual variability in the neuropathology of PLP, amputation history, and psychological state, single-modality treatments often fall short. As a result, personalized and multimodal treatment approaches are crucial, designed to align with each patient's unique clinical characteristics.

3. Mechanisms and Clinical Progress of TMS in the Treatment of PLP

3.1 Basic Principles of TMS

TMS is a non-invasive neuromodulation technique that employs electromagnetic induction to modulate brain activity. By placing a specialized coil on the scalp, brief high-intensity magnetic pulses are delivered, penetrating the skull to generate electric currents in targeted cortical areas, thereby facilitating or suppressing neuronal activity in specific brain regions[13]. Compared with invasive procedures, TMS allows for targeted modulation of brain activity without surgical intervention or implants, significantly reducing the risk of infection or complications. Furthermore, its stimulation parameters—such as intensity, frequency, and pulse count—can be flexibly adjusted for precise control.

TMS operates in several modes depending on the stimulation waveform and temporal pattern, including single-pulse, paired-pulse, repetitive TMS (rTMS), and patterned burst protocols [14]. Among them, rTMS delivers repeated pulses to modulate cortical excitability and synaptic plasticity, reshaping neural networks. The U.S. Food and Drug Administration has approved rTMS as a treatment for individuals with depression unresponsive to conventional therapies, and research continues into its application for conditions such as anxiety disorders, Parkinson's disease, epilepsy, post-stroke recovery, and chronic pain management[5, 15].

3.2 Proposed Mechanisms of TMS in PLP Treatment

Several neurophysiological mechanisms have been proposed to explain how TMS alleviates PLP, reflecting the complexity of its therapeutic effects.

3.2.1 Cortical reorganization and pain network modulation

Neuroimaging research indicates that PLP is strongly associated with maladaptive reorganization within the sensorimotor cortex after amputation[16]. The loss of sensory input from the amputated limb leads to invasion of adjacent cortical regions (e.g., face or trunk), resulting in abnormal cortical remapping[17]. At the same time, increased activity in pain-related areas—such as the anterior cingulate cortex, insula, and thalamus—can enhance nociceptive processing along the spinothalamic-cortical pathway[18]. TMS is

thought to reverse these maladaptive changes by reshaping the cortical somatotopic map and modulating functional connectivity within pain networks, thereby disrupting aberrant central pain processing loops[19].

3.2.2 Induction of synaptic plasticity and restoration of excitatory–inhibitory balance

rTMS can evoke long-term potentiation- or long-term depression-like effects, thereby modulating synaptic plasticity within pain-related neural circuits[20]. Animal studies have shown that amputation leads to enhanced excitatory transmission mediated by NMDA receptors and reduced GABAergic inhibition, contributing to cortical hyperexcitability and pathological remodeling[21]. TMS can produce bidirectional regulatory effects: low-frequency rTMS (≤1 Hz) enhances cortical inhibition, thereby dampening overactive pain pathways [22, 23], while high-frequency rTMS (≥5 Hz) elevates cortical excitability, potentially activating glutamatergic neurons and facilitating the release of endogenous opioids such as β-endorphins, which increase the pain threshold[21]. These regulatory effects help restore the disrupted cortical excitability balance post-amputation, reducing pain amplification.

3.2.3 Peripheral–central signal integration and reversal of central sensitization

PLP arises from the interplay between peripheral nerve injury and central pain processing dysfunction[20]. Long-term amputation can result in central sensitization of the spinal cord and cerebral cortex, leading to persistent activation of nociceptive pathways. TMS may help reverse this sensitization by inhibiting abnormal peripheral input and restoring the brain's ability to filter and integrate sensory signals appropriately, thereby alleviating chronic pain[24].

3.3 Clinical Progress of TMS in PLP Treatment

Clinical studies indicate that the analgesic effects of rTMS depend heavily on key stimulation parameters, such as the targeted brain region, frequency, intensity, pulse count, and duration of treatment [25]. A comprehensive review of existing studies allows us to summarize the current clinical progress of TMS in the treatment of PLP, with particular focus on stimulation frequency and target site selection.

3.3.1 Stimulation frequency

High-frequency stimulation is generally associated with increased cortical excitability, whereas low-frequency stimulation tends to exert inhibitory effects[13]. For instance, a randomized controlled trial by Ahmed et al. (2011) [26] involved administering 20 Hz rTMS over the contralateral M1 in 27 patients with PLP over a five-day course. The results demonstrated an approximate 55% reduction in VAS scores following the fifth session in the active stimulation group, with a sustained 39% pain reduction observed at the two-month follow-up—significantly outperforming the sham group. In another recent study, low-frequency (1 Hz) rTMS was delivered to the dorsolateral prefrontal cortex (DLPFC) contralateral to the amputation site over ten sessions. The median VAS score decreased markedly from a baseline of 6.5 to nearly zero, and remained significantly lower than baseline at the 60-day follow-up [23]. This effect remained significant at the 60-day follow-up. Overall, while both high- and low-frequency rTMS have shown efficacy in treating PLP, there is no consensus on which frequency is superior. Although current parameter selection often reflects the pathophysiology of the targeted area, robust evidence from large randomized controlled trials is still needed to determine optimal frequency settings.

3.3.2 Stimulation targets

The M1 plays a central role in pain and motor regulation, with extensive connections to the thalamus, cingulate cortex, and insula[27]. M1 is the most commonly targeted site in PLP studies. Malavera et al. (2016)[28] demonstrated significant VAS score reductions using 10 Hz rTMS over contralateral M1 in 54 lower-limb amputees. Similarly, Ahmed et al. [26] confirmed significant pain relief using 20 Hz rTMS. Low-frequency stimulation of M1 has also shown benefit: Wang et al. (2022)[29] used 1 Hz rTMS over contralateral M1 in lower-limb amputees, resulting in immediate and 3-month pain relief.

The DLPFC is involved in the cognitive-emotional aspects of pain and modulates pain-related brain networks via cortico-limbic pathways[23]. Recent trials indicate that low-frequency rTMS over DLPFC can also alleviate PLP. Vats et al. (2024)[23] reported a near-complete elimination of pain in the real stimulation group, with effects persisting

through 60-day follow-up. Case reports have combined contralateral S1 and DLPFC stimulation, yielding a reduction in VAS from 5 to 1[30].

The S1, due to its role in pain perception and cortical reorganization, is also a potential target. Lee et al. (2015)[31] applied 1 Hz rTMS over S1 in lower-limb amputees for six treatment cycles, reducing VAS from 9 to 2 and maintaining relief for 3 months. Scibilia et al. (2018)[32] used navigated rTMS to stimulate M1, S1, and DLPFC and observed enhanced connectivity in the postcentral gyrus associated with pain reduction.

Target localization strategies vary. M1 hotspots are usually identified via motor evoked potentials, while DLPFC is commonly located 5 cm anterior to M1[23]. Some researchers have used fMRI-guided neuronavigation to tailor TMS targeting[32]. Although no standardized protocol exists for individualized targeting in PLP, future studies may integrate anatomical and functional imaging with electrophysiological data to optimize precision therapy.

To assist in clinical decision-making, several studies have summarized TMS parameters and efficacy data, as shown in Table 2 of this review. While there is still no universally accepted stimulation protocol, the Chinese expert consensus recommends high-frequency rTMS over the contralateral M1 as a first-line non-pharmacological treatment for PLP[14]. Most effective protocols involve 10 Hz stimulation at 80–90% of Resting Motor Threshold (RMT), delivering 1500–3000 pulses per session over 5–10 sessions. This regimen appears to produce meaningful analgesic effects, possibly by modulating maladaptive plasticity and restoring cortical network balance [13, 33].

Table 2. Summary of TMS Stimulation Parameters and Analgesic Efficacy in PLP Treatment

Study (Year)	Sample Size (Treatment / Control)	Target Site	Frequency & Intensity	Pulses per Session	Treatment Course	Analgesic Effect
Ahmed et al., (2011) [26]	27 (17/10)	Contralateral M1	20 Hz, 80% RMT	~2000	5 sessions / 1 week	Significant reduction in VAS and LANSS; analgesia maintained for 1–2 months; serum β-endorphin increased post-treatment
Di Rollo et al. (2011)[34]	1	Contralateral M1	1 Hz, 80% RMT	600	15 sessions / 3 weeks	VAS reduced from 6 to 4; effect lasted 3 weeks
Lee et al., (2015)[31]	1	Contralateral M1 + S1	1 Hz, 85% RMT	800	6 cycles: 10 sessions each for M1 and S1	S1 stimulation reduced VAS from 9 to 2; effect lasted ~3 months
Malavera et al., (2016) [28]	54 (27/27)	Contralateral M1	10 Hz, 90% RMT	1200	10 sessions / 2 weeks	Real stimulation group showed 53.4% VAS reduction vs. 22.9% in sham (p=0.03); no significant difference at 30 days
Grammer et al. (2015)[30]	1	Contralateral S1 + DLPFC	S1: 1 Hz, 100% RMT; DLPFC: 10 Hz, 120% RMT	S1: 2000; DLPFC: 3000	28 sessions / 6 weeks (5 S1, 23 DLPFC)	VAS decreased from 5 to 1 after full course
Xu et al., (2019) [35]	24 (12/12)	M1 (control) vs. DLPFC (trial)	10 Hz, 90% RMT	1000	20 sessions / 4 weeks	Significant SF-MPQ reduction in DLPFC group
Wanget al., (2022)[29]	26 (12/14)	Contralateral M1	1 Hz, 80% RMT	900	10 sessions / 2 weeks	Immediate and 3-month VAS reduction sustained
Vats et al., (2024)[23]	19 (10/9)	Contralateral DLPFC	1 Hz, 90% RMT	1200	10 sessions / 2 weeks	VAS dropped from 6.5 to 0; remained ~0.5 at 60-day follow-up

Note: RMT = Resting Motor Threshold; VAS = Visual Analogue Scale; LANSS = Leeds Assessment of Neuropathic Symptoms and Signs; SF-MPQ = Short-Form McGill Pain Questionnaire.

3.4 Safety and Common Side Effects of TMS

Current clinical evidence indicates that rTMS is generally safe and well tolerated in the treatment of PLP, with no reports of serious adverse events[15, 29]. For example, in the study by Malavera et al. (2016)[28], no major

complications were observed among 54 participants undergoing 10 Hz stimulation. Similarly, Wang et al. (2022)[29] reported no dizziness, headaches, or other discomfort in their cohort of 26 patients.

The most frequently observed side effects are mild and short-lived, such as temporary headaches, fatigue, dizziness, or neck discomfort. In rare cases, transient blurred vision has been reported, which typically resolves shortly after treatment cessation [31]. Overall, rTMS is considered a well-tolerated option, provided it is not used in patients with contraindications such as epilepsy or implanted metallic cranial devices.

The 2024 Chinese Expert Consensus on Phantom Limb Pain[36] recommends rTMS as a second-line treatment, particularly for patients who are unresponsive to pharmacological therapy or unsuitable for invasive procedures. However, it is important to note that therapeutic outcomes vary among individuals, and symptom recurrence may occur after cessation of treatment in some cases.

4. Limitations and Challenges of TMS Therapy

Although TMS has demonstrated considerable clinical potential as a novel non-invasive neuromodulation technique for alleviating symptoms of various neurological and psychiatric disorders, its application in the treatment of PLP still faces numerous technical, clinical, and ethical challenges. These include the precise control of stimulation parameters, development of personalized treatment protocols, evaluation of long-term efficacy, management of side effects, and concerns about treatment cost and accessibility—all of which warrant further exploration.

Firstly, TMS is limited in its ability to regulate deep brain regions such as the thalamus and cingulate cortex. Due to physical constraints on magnetic field penetration and spatial resolution, it is difficult for TMS to accurately target neural circuits located in the deeper cortical or subcortical layers. Moreover, the molecular and cellular mechanisms underlying TMS-induced pain relief in PLP remain incompletely understood. More in-depth and systematic basic research is needed to elucidate how TMS affects neuroplasticity, neurotransmitter systems, and pain-related network reorganization at a mechanistic level.

Secondly, the therapeutic efficacy of TMS in PLP is highly dependent on accurate configuration of stimulation parameters. Currently, there is no standardized protocol for clinical use. Differences in frequency, intensity, stimulation target, and treatment duration among existing studies make direct comparison difficult and hinder clinical translation. Moreover, considerable individual variability—including factors like amputation level and cause, time elapsed since amputation, and accompanying symptoms—can substantially influence a patient's response to TMS. These inter-individual variations underscore the need for more flexible, personalized treatment strategies.

Another major challenge lies in the durability of therapeutic effects. While many studies report short-term analgesic benefits, there is a lack of systematic investigation into long-term efficacy. Some patients experience a recurrence of symptoms soon after the treatment course ends, indicating that a single course of rTMS may not provide lasting pain relief and that repeated or maintenance sessions may be required. Furthermore, the absence of reliable prognostic biomarkers hinders the development of individualized protocols. Currently, clinicians cannot accurately predict which patients are likely to respond favorably to rTMS, limiting the efficiency and cost-effectiveness of the intervention.

Future advances in neuroscience are expected to address these challenges by integrating multimodal neuroimaging and neurophysiological tools—such as fMRI, EEG, and fNIRS—to enable real-time monitoring of brain activity and individualized adjustment of stimulation parameters. Combining TMS with emerging therapies like virtual reality, mirror therapy, and cognitive behavioral therapy may produce synergistic effects and improve clinical outcomes. The development of closed-loop TMS systems, which adapt stimulation in response to ongoing brain signals, will be crucial for optimizing both efficacy and safety. Additionally, there is a pressing need for large-scale, multicenter randomized controlled trials to standardize TMS protocols, identify optimal stimulation parameters, and assess long-term effects including pain relief, functional gains, and enhanced quality of life.

5. Conclusion and Future Perspectives

As an innovative neuromodulation technique, TMS offers a promising non-invasive therapeutic option for alleviating PLP by modulating cortical neuroplasticity and reshaping dysfunctional neural network activity. High-frequency rTMS targeting the contralateral M1 has demonstrated significant short-term analgesic effects. At the same time, emerging evidence suggests that low-frequency stimulation of the DLPFC or S1 may also hold potential for pain relief in PLP patients.

Despite its encouraging results, current research on TMS in PLP remains constrained by several limitations. Most existing studies are characterized by small sample sizes, lack of long-term follow-up, and significant heterogeneity in stimulation parameters—including frequency, intensity, target site, and treatment duration. Additionally, few studies have employed multimodal neuroimaging or individualized targeting approaches to optimize treatment efficacy. These factors restrict the generalizability of the findings and pose significant challenges for clinical implementation. Future research efforts should aim to address the following key challenges: First, developing optimized multimodal intervention strategies that combine TMS with mirror therapy, virtual reality, and cognitive-behavioral therapy could enhance therapeutic outcomes through synergistic mechanisms. Second, the advancement of real-time neurofeedback-guided TMS systems—integrating EEG, fMRI, or fNIRS data—may enable the dynamic adjustment of stimulation parameters based on patients' neural activity patterns, thereby improving treatment precision and safety. Lastly, well-designed large-scale, multicenter randomized controlled trials are essential to establish standardized stimulation protocols, confirm long-term efficacy, and comprehensively assess functional outcomes such as pain reduction, mental health status, and overall quality of life.

In summary, with ongoing advancements in neuroscience and neuromodulation, TMS is anticipated to become a vital component in the integrated treatment of PLP. Offering a non-invasive, individualized approach to neuromodulation, TMS shows significant promise in enhancing rehabilitation effectiveness and improving the quality of life for amputees experiencing PLP.

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