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Review Article

Repetitive Transcranial Magnetic Stimulation (rTMS) in Neurological Disorders: Current Evidence and Future Perspectives

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Abstract

This is a comprehensive review of the development, mechanisms of action, safety, and clinical applications of repetitive transcranial magnetic stimulation (rTMS) in various neurological conditions. It is particularly directed at clinical neurologists and aims to provide an in-depth analysis of both laboratory and clinical evidence, offering a clear overview of the current and potential future uses of this technique. The review also highlights the present limitations and challenges of rTMS in clinical practice. In summary, we state rTMS is a promising tool for clinical neurologists, especially in scenarios of therapeutic resistance. While its routine use still requires stronger evidence, the convergence of technological innovations and neuroscience positions this modality as a future cornerstone in neuromodulation.

Keywords: *Transcranial Magnetic Stimulation; Noninvasive Brain Stimulation; Neuromodulation; Neuroplasticity; Motor Dysfunction; Pain; Spasticity.*

Introduction

Repetitive transcranial magnetic stimulation (rTMS) is a non-invasive technique that modulates cortical excitability through the induction of focal electric currents (1).

Since its development in 1985 by Barker et al. (1), TMS has become a translational tool, enabling not only the exploration of brain connectivity (e.g., functional tractography) but also therapeutic applications in neuropsychiatric disorders.

Despite its growing clinical use, significant gaps remain in our understanding of the underlying mechanisms at the network level, particularly regarding the transsynaptic propagation of effects beyond the stimulated area (2).

TMS is currently considered the gold standard among non-invasive neuromodulation techniques, as a single stimulus can generate variable electric fields capable of depolarizing cortical neurons (3,4). It has a favorable safety profile and has received regulatory approval (FDA/EMA) for conditions such as treatment-resistant depression (5,6). Unlike invasive interventions (e.g., deep brain stimulation), rTMS carries lower risks of systemic effects or surgical complications.

The objectives of this review are to:

(1) Highlight the clinical relevance of rTMS in neurological practice,

(2) Review its historical development and neurophysiological foundations, and

(3) Provide an updated analysis of its mechanisms of action, safety profile, and indications in both neurology and psychiatry, with emphasis on recent evidence.

In the field of neurology, rTMS use has grown exponentially, with documented applications in post-stroke recovery (7,16), Parkinson's disease (7), neurological diagnosis (which will not be addressed in this article) (8), migraine and chronic pain (9), refractory epilepsy (10,11), multiple sclerosis (12), Alzheimer's disease (13), and movement disorders (14,15), supported by phase III clinical trials (see Table I).

Table 1

Diseases	Effects	Evidence
Parkinson's Disease (PD)	Improvement in motor symptoms (bradykinesia, rigidity) and non-motor symptoms (depression, apathy). High-frequency rTMS (≥5 Hz) over the motor cortex or dorsolateral prefrontal cortex (DLPFC) has shown moderate benefits.	Meta-analyses support its use as an adjunct therapy, although protocols are not yet standardized.
Chronic Pain and Migraine	In migraine, low-frequency rTMS (1 Hz) over the occipital cortex or DLPFC reduces attack frequency and intensity. In neuropathic pain (e.g., fibromyalgia, trigeminal neuralgia), it modulates pain circuits.	FDA-approved for migraine with aura (specific device). (rTMS)
Stroke	Promotes neuroplasticity and motor recovery post-stroke (rTMS inhibits the unaffected hemisphere or excites the affected one). Also improves aphasia and dysphagia in some cases.	Guidelines suggest its use in rehabilitation, but protocols vary. (rTMS)
Epilepsy	Low-frequency rTMS (1 Hz) may reduce cortical excitability and seizure frequency in focal epilepsies.	Promising results but limited to selected cases (e.g., accessible cortical foci). (rTMS)
Multiple Sclerosis (MS)	Improves fatigue, depression, and spasticity.	Preliminary studies show benefits, but more trials are needed. (rTMS)
Movement Disorders	Dystonia and Tremor: Some studies report improvement with rTMS, but evidence is limited.	(rTMS)
Alzheimer's Disease	Multisite rTMS combined with concurrent cognitive training may improve cognition, memory, apathy, and language in mild and early-stage AD (including mild cognitive impairment).	Clinical use is not recommended until long-term observational studies show multisite rTMS with cognitive training is more beneficial than single-site rTMS with cognitive training.

Development

Mechanism of Action

TMS is based on Faraday's law of electromagnetic induction, wherein an electric current discharged through a TMS coil generates a perpendicular magnetic field that crosses the skull and reaches the brain. This magnetic field induces an electric field and focal electric currents in the targeted brain tissue (3,4). When these induced electric currents are sufficiently strong, they depolarize neurons and elicit action potentials, known as TMS-induced neural discharges, which can be recorded using electroencephalography (EEG) (5), motor evoked potentials (MEPs) (2,6), or indirectly through functional MRI (fMRI) (7).

The effects of rTMS on cortical excitability depend on the specific stimulation parameters used in each protocol, as well as the geometry of the coil (8). As a general rule, low-frequency stimulation [LF ≤ 1 Hz] tends to reduce cortical excitability, whereas high-frequency stimulation [HF ≥ 5–20 Hz] tends to enhance it (6).

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The induced electric field is shaped by the tissue conductivity properties and reaches its maximum intensity in the superficial regions of the target cortical gyri and underlying white matter. TMS likely acts on axons of both excitatory and inhibitory neurons. The likelihood of individual axons generating an action potential in response to TMS depends on several factors:

- Axonal geometry,
- Degree of myelination,
- Spatial orientation relative to the applied field,
- Physiological state of the neuron (influenced by transsynaptic input, resting membrane potential, and firing rate) (7).

Computational models suggest that the main TMS targets are axonal terminals located at the crowns of the gyri and edges of the cortical folds (7).

Clinical Application of TMS in Stroke

Stroke remains one of the leading causes of neurological disability worldwide (17). Post-stroke complications frequently result in severe limitations in patients' daily functioning (19). Thus, the exploration of new technologies for stroke rehabilitation is essential to advance clinical experience in managing these patients and to promote a better understanding of the benefits, limitations, indications, contraindications, and precautions of such interventions.

In recent years, numerous studies have highlighted an irreplaceable role for TMS in the neurorehabilitation process following stroke. It has been applied to improve motor function in both upper and lower limbs, as well as language, swallowing, cognition, post-stroke depression, spasticity, and central post-stroke pain (see Table for further details). However, the precise mechanisms by which TMS exerts its therapeutic effects in stroke remain unclear, as do the optimal frequency, intensity, duration, and stimulation site.

Several studies suggest that rTMS, when combined with conventional rehabilitation strategies, can significantly reduce neurological impairment, improve functional independence in daily activities, and enhance overall quality of life in stroke survivors (20,21,22). These findings support rTMS as a potential complementary treatment in stroke rehabilitation.

Stimulation parameters such as site and timing may vary depending on the specific neurological deficits. Currently, the most commonly targeted regions in post-stroke rTMS protocols include:

- Primary motor cortex (M1)
- Left dorsolateral prefrontal cortex (DLPFC)
- Superior temporal gyrus
- Inferior frontal gyrus

Secondary somatosensory cortex (S2) (23).

Other cortical areas may also be targeted depending on their functional counterparts. Among these, M1 remains the most frequently used stimulation site, likely due to its central role in motor control and integration of functional commands (23). M1 is a multifunctional region, involved in motor execution, cognitive processing, speech, and swallowing. Given the multifactorial impairment seen in stroke, M1 is considered a key and irreplaceable target for TMS intervention.

TMS stimulation duration may vary, depending on the acute or chronic phase of stroke, disease severity, patient tolerance, and specific therapeutic goals.

Motor Dysfunction of the Upper Limb

Post-stroke motor dysfunction of the upper limb is primarily characterized by reduced movement, impaired coordination, and diminished dexterity. It affects approximately 55% to 75% of stroke survivors (24). Compared to the lower limb, upper limb recovery is typically slower and is most pronounced during the first six months post-stroke.

Unfortunately, due to limited access to early rehabilitation, many patients miss the "golden window" for upper limb recovery. Therefore, initiating early rehabilitation and identifying effective complementary therapies are critical priorities.

Several randomized controlled trials (RCTs) have shown that rTMS can modulate cortical excitability and re-establish interhemispheric inhibitory balance, thus improving motor function in stroke patients (25,26). For example, a study by Avenanti et al. demonstrated that high-frequency rTMS significantly enhanced cortical excitability in the lesioned hemisphere.

Promoting improved distal mobility, dexterity, and finger coordination in stroke patients (25). However, regarding motor function stimulation of the upper limbs, one study reported that bilateral TMS (high frequency on the lesioned hemisphere and low frequency on the unaffected hemisphere) was more beneficial than unilateral TMS for improving upper limb motor function (26). Nevertheless, a different meta-analysis revealed that this finding was only confirmed in the acute phase of stroke, while during the subacute and chronic phases, unilateral TMS applied to either the lesioned or unaffected hemisphere yielded better outcomes (27).

This suggests that enhancing cortical excitability in the lesioned hemisphere during the acute and subacute phases is the main strategy to improve neurological function of the upper limbs, whereas inhibiting cortical excitability in the unaffected hemisphere and reducing its inhibitory influence on the lesioned hemisphere during the chronic phase is the primary mechanism to enhance neurological function.

In conclusion, the different disease stages are important factors for determining the TMS treatment plan. A similar pattern applies to stimulation modalities; for example, Theta Burst Stimulation (TBS) is more beneficial for upper limb motor recovery during the acute phase, while rTMS is more effective during the subacute and chronic phases (27).

Furthermore, rTMS promotes fine motor recovery in the upper limbs post-stroke by indirectly modulating corticospinal tract excitability and directly enhancing cortical excitability. This process largely depends on the integrity of the corticospinal tract (CST) (28). Therefore, when applying rTMS to improve upper limb motor function post-stroke, CST integrity should be evaluated prior to designing the appropriate rTMS protocol. For example, low-frequency rTMS is more suitable for patients with high CST integrity, whereas high-frequency rTMS has shown more significant clinical effects in patients with low CST integrity (29). (Figure 1)

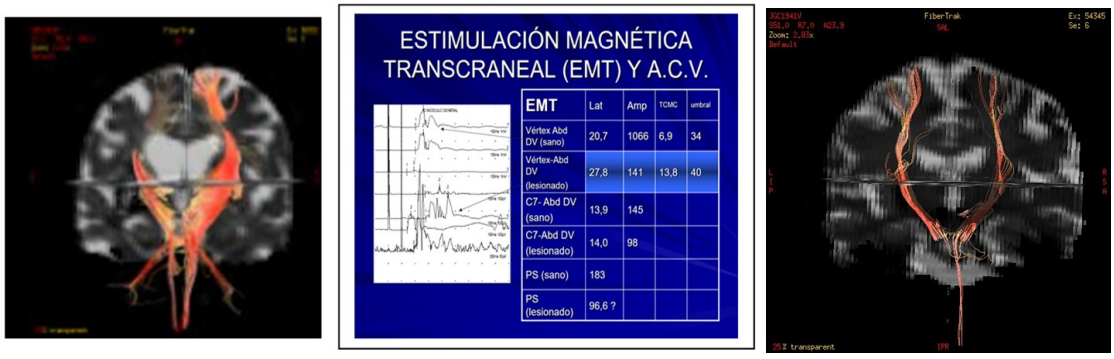

Figure 1. *Tractography of the pyramidal tract showing a marked reduction in its volume (left side) and only a few axons in the medulla due to axonal degeneration (arrow). Increased motor latency and reduced amplitude in the right hand. In the lower image, complete axonal degeneration of the pontine-medullary pyramidal tract, anticipating lack of response to rTMS.*

Lower Limb

Both lower limbs play a critical role in human motor function, as demonstrated by gait coordination and balance (30). After stroke, patients often suffer from lower limb motor dysfunction, gait abnormalities, and other sequelae that significantly impair their family and social participation.

TMS is an emerging therapy for stroke rehabilitation that can increase walking speed (31,32), correct gait asymmetry (32), reduce lower limb muscle spasticity (33,34), and improve balance and motor control (35,36), thereby enhancing lower limb function in stroke patients. A randomized controlled trial showed that rTMS significantly improved gait, motor control, and motor function in stroke patients, with sustained benefits observed during long-term follow-up (37).

This study was based on the interhemispheric inhibition theory, applying high-frequency rTMS to the affected hemisphere to promote cortical excitability reactivation, and low-frequency rTMS to the unaffected hemisphere to reduce cortical excitability and alleviate its over-inhibition, thus rebalancing bilateral interhemispheric inhibitory equilibrium. In this study, the bilateral rTMS regimen outperformed unilateral regimens, i.e., bilateral rTMS > high-frequency rTMS on the lesioned side > low-frequency rTMS on the unaffected side (27).

Indeed, most studies have shown that bilateral rTMS protocols outperform unilateral ones, akin to combined therapies generally outperforming monotherapies in certain diseases, which is not controversial.

It is believed that following brain injury, reduced excitability in the lesioned hemisphere is the primary cause of interhemispheric inhibitory imbalance, thus reactivation of cortical excitability in the lesioned hemisphere may be the main strategy to restore this balance, rather than reducing excitability in the unaffected hemisphere.

However, this predominance is not uniform; depending on etiology, lesion location, disease urgency, and individual patient differences, some patients respond better to reducing excitability in the unaffected hemisphere to promote interhemispheric inhibitory homeostasis.

A meta-analysis found that low-frequency rTMS in the unaffected hemisphere produced more clinically significant effects than high-frequency rTMS in the lesioned hemisphere during the chronic phase of stroke (23).

There is some controversy regarding whether TMS improves lower limb function post-stroke. For example, a meta-analysis showed that both high- and low-frequency rTMS positively affected walking speed in stroke patients (32). However, this was not supported by a study by Raffaella et al., who reported that while 20 Hz high-frequency rTMS significantly improved lower limb motor function in chronic stroke patients, it did not increase walking speed (38).

Additionally, Ying et al. reported that low-frequency (1 Hz) rTMS applied to the unaffected hemisphere did not significantly improve motor function or gait in stroke patients (39).

Contrastingly, another study found that low-frequency rTMS on the unaffected hemisphere improved lower limb muscle spasticity, thereby promoting better motor function (40). This idea was supported by studies from Soofia et al. and Liu et al. (41,42).

This variability in findings may be due to the complexity of walking, which involves multiple parameters such as gait speed and angle, step width and length. These parameters are regulated by multiple neural systems, including integrated cortical, subcortical, and spinal networks.

Similar to walking, multiple brain areas are activated, including the primary sensorimotor areas, primary motor areas, supplementary motor areas, basal ganglia, and cerebellum, all associated with increased cerebral blood flow (37).

This suggests that TMS improves lower limb motor function in stroke patients not only through specific TMS protocols but also via different stimulation sites and the interactions between brain regions.

Therefore, further studies are needed to generate more evidence on the diverse effects of TMS applications and to continue advancing its clinical implementation.

Cognitive Impairment

One study reported that one-third of stroke patients exhibit varying degrees of cognitive impairment, known as post-stroke cognitive impairment, which can rapidly progress to dementia and severely impact patients' quality of life. Therefore, active rehabilitation measures to prevent vascular dementia are critically important.

Studies have shown that TMS can improve cognitive function in patients with post-stroke cognitive impairment through anti-inflammatory effects and increased cerebral blood flow (43). For example, Takatoshi et al. reported that 10 Hz rTMS promotes improvements in memory, attention, and executive function in these patients by enhancing perfusion in ischemic brain regions (44). The dorsolateral prefrontal cortex (DLPFC) is often targeted for TMS to ameliorate post-stroke deficits, playing a key role in modulating higher cognitive functions such as memory, attention, and executive function (45,46). These studies demonstrated that high-frequency rTMS over the left DLPFC significantly improved executive function in patients.

Similarly, based on the theory of interhemispheric inhibitory balance, low-frequency rTMS applied to the right DLPFC also enhances cognitive and memory functions in these patients (47). Additionally, a meta-analysis showed that rTMS significantly improved cognitive impairment when applied for more than 4 weeks at stimulation intensities between 80% and 110% of the motor threshold (48).

Intermittent theta burst stimulation (iTBS) of the left DLPFC also significantly enhances cognitive functions such as executive function and semantic comprehension compared to sham stimulation in these patients (49).

Swallowing Disorders

More than 65% of new stroke patients annually present with dysphagia (50). Although dysphagia resolves spontaneously in most cases, between 11% and 50% of patients will develop persistent dysphagia without intervention (50). Therefore, preventing dysphagia from becoming a permanent sequela through external means is necessary.

Studies have demonstrated that TMS treatment applied to the M1 cortical region can promote improvement in swallowing function post-stroke (51). However, since the M1 area encompasses multiple neurological functions, precisely localizing the swallowing M1 functional area is a major challenge.

Li S. et al. (2020) used fMRI-guided TMS targeting the motor cortex to excite the orbicularis oris muscle, and surface electromyography detected motor evoked potentials (MEPs) in the submandibular complex (SMC) muscle as a method to localize stimulation targets within the M1 swallowing functional area. This study provides a more precise stimulation site for TMS aimed at improving post-stroke swallowing dysfunction. However, as this process is individualized, stimulation target locations within the SMC may differ slightly among patients, necessitating repositioning of the skeletal muscle membrane targets in different individuals for effective swallowing function improvement.

fMRI-guided rTMS can more accurately and effectively promote swallowing function recovery, especially high-frequency rTMS (51,52). Xiang et al. noted that high-frequency rTMS improved dysphagia in stroke patients more significantly and for a longer duration than low-frequency rTMS (52).

It is also worth noting that low-frequency rTMS (1 Hz) only improved appetite compared to conventional swallowing therapy but did not significantly impact swallowing function recovery.

Moreover, Cheng et al. (53) demonstrated significant swallowing function improvement in chronic stroke patients after applying 3000 pulses of 5 Hz rTMS over the tongue motor area of the affected hemisphere for two consecutive weeks. However, another study using the same stimulation parameters found no improvement in swallowing function at 2, 6, or even 12 months post-treatment (54). This contrasts with Cheng et al.'s findings.

These conflicting results suggest that the clinical efficacy of TMS may vary even within the same disease depending on severity, individual patient differences, or other external factors. Therefore, determining the optimal clinical efficacy of TMS still requires further investigation.

Aphasia

Post-stroke aphasia (PSA) is a common acquired language disorder in patients with acute or subacute stroke, which can cause varying degrees of impairment in four domains: auditory comprehension, reading, writing, and speech production (57). In the long term, this may lead to loss of self-confidence and can induce post-stroke depression in severe cases. Therefore, early treatment of patients with PSA is key to preventing post-stroke depression.

rTMS has been used since 2005 for the treatment of aphasia in patients with chronic stroke (58). The site of stimulation is an important factor in rTMS parameters affecting clinical efficacy. The superior temporal gyrus is the main cerebral region affected in sensory aphasia (also known as Wernicke's aphasia), and thus, it is preferentially chosen as the stimulation site in patients with Wernicke's aphasia (59). Similarly, another typical aphasia type, motor aphasia (also known as Broca's aphasia), involves lesions in the inferior frontal gyrus. Stimulation of this area significantly improves patients' spontaneous speech and repetition components (59). (Figure 2)

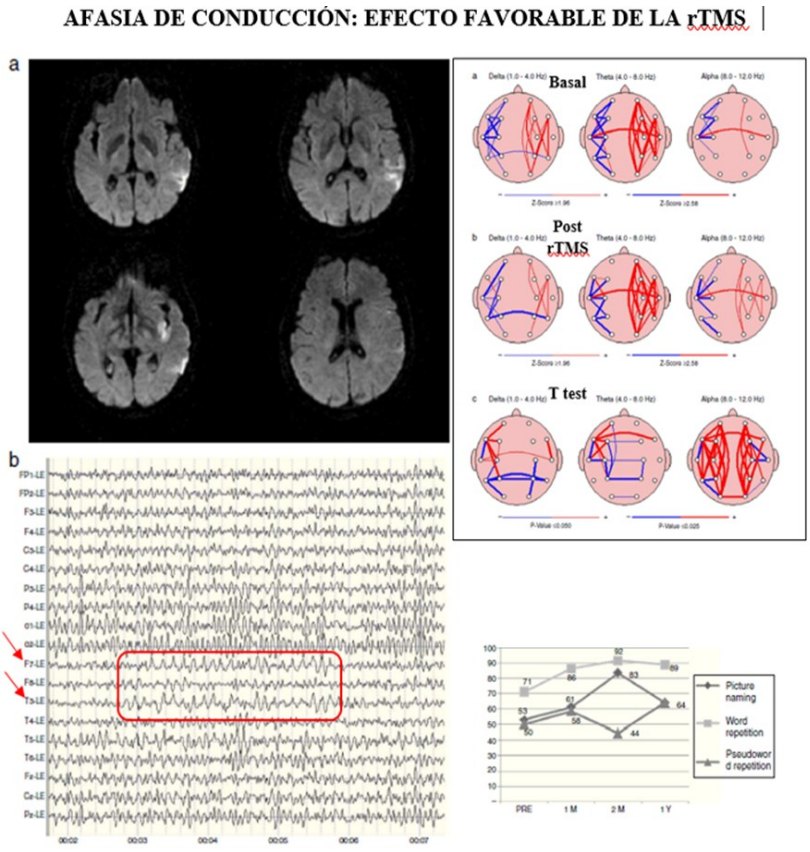

Figure 2. *Conduction aphasia due to a small infarct in Broca's area (a). EEG showing rhythmic theta activity over F7 and T3 (b). On the right, baseline reduction of coherence in the left hemisphere with compensatory increase in the right hemisphere. Following rTMS, a significant increase in left frontotemporal coherence is observed, associated with improved verbal output that persists up to one year.*

This evidence demonstrates the feasibility of selecting rTMS stimulation sites based on the lesioned brain areas corresponding to different aphasia types. However, although the lesioned brain area may be used as the stimulation site for patients with PSA, it remains unclear whether this site is the optimal stimulation target for rTMS, and no relevant studies currently confirm this.

Post-Stroke Depression

Post-stroke depression (PSD) is the most common neuropsychiatric sequela in stroke patients, affecting approximately one-third of new cases each year. Currently, antidepressants are the most common treatment for PSD, but the clinical onset of pharmacological effects is delayed, and only a subset of patients experience significant improvement.

In 2008, it was demonstrated that rTMS treatment targeting the dorsolateral prefrontal cortex to improve PSD is safe and effective, with minimal side effects (60). rTMS is undoubtedly a safe and reliable alternative for PSD patients who do not respond to psychotherapy or who experience serious adverse effects from pharmacological treatment.

The traditional rTMS regimen 5 days per week for 4 to 6 weeks, has shown significant positive effects in patients with chronic major depression (60). However, this regimen is relatively lengthy.

Jessica et al. reported that accelerated rTMS (20 Hz, 110% motor threshold, 5 daily sessions over 4 days) significantly reduced post-stroke depression symptoms and maintained positive effects at 3 months follow-up (61). Furthermore, this study demonstrated that accelerated rTMS is safe and feasible in patients with subacute PSD, without risk of inducing seizures (61).

Based on these findings, we can provisionally conclude that accelerated rTMS is indeed more effective than conventional rTMS during the subacute and acute phases of PSD.

Spasticity

Early intervention with rehabilitation exercises is the cornerstone for the future social reintegration of stroke patients. However, post-stroke spasticity (PSS), a complication that can severely affect the recovery process, is a significant disorder that seriously impairs motor training and is characterized by velocity-dependent increased reflex tone. Not all stroke patients exhibit hypertonia clinically. Between 30% and 80% of stroke patients experience or are at risk of developing spasticity, which, if not treated promptly, can cause pain, contracture, deformity, or even joint stiffness and immobility (62).

Although numerous clinical studies have demonstrated that rTMS significantly reduces limb spasticity after stroke, a small number of studies have not shown similar results. This suggests that several factors—such as stimulation parameters, stimulation site, coil type, severity, and whether the patient is in the acute or chronic phase—may affect the efficacy of rTMS in reducing muscle spasticity (63). Different coil types exert varying degrees of influence on spasticity improvement; figure-of-eight coils, with greater focal stimulation, have a more significant effect in reducing spasticity symptoms compared to H-coils (63).

Secondly, the therapeutic effect on spasticity also varies according to the stimulation site. Besides the motor cortex, the cerebellum plays an important role in motor control and muscle tone stabilization. Theta Burst Stimulation applied to the cerebellum (cTBS) has been reported to reduce muscle spasticity in stroke patients by regulating corticospinal excitability through the cerebello-dentate-thalamo-cortical pathway (64). Therefore, cerebellar cTBS could be one of the most promising stimulation modalities for reducing muscle spasticity in the future development of transcranial magnetic stimulation (TMS).

Post-Stroke Central Pain

Post-stroke central pain (PSCP) is a common neuropathic pain sequela following ischemic brain injury, often manifested as sensory hypersensitivity or abnormalities in the region corresponding to the vascular lesion. It is frequently confused with stroke-induced shoulder subluxation.

Currently, PSCP is mainly treated with pharmacological agents such as anticonvulsants, which have shown good efficacy in symptom relief (66). However, in cases of intractable PSCP, medications may only serve as a placebo and fail to provide effective relief. A recent study revealed that rTMS targeting the motor cortex had a significant effect in alleviating refractory PSCP (67), although the duration of relief varied. This variability may relate to the ischemia location, disease progression stage, stimulation site, and individual patient differences. Therefore, further research into therapeutic parameters for PSCP improvement by rTMS is necessary.

Challenges and Perspectives

While rTMS is currently a common treatment for stroke, there are some safety risks associated with its use in humans. For example, in one study, 13 of 273 patients experienced adverse events such as headache, dizziness, rhinorrhea, syncope, and seizures (68). Among these, epilepsy is the most serious sequela and currently the most controversial issue. Safety guidelines for TMS indicate that epilepsy occurrence is influenced by various internal and external factors (69). External factors generally include equipment malfunction, operator error, and improper adjustment of rTMS parameters.

In general, it is necessary to inspect rTMS device hardware before initial use to prevent adverse reactions such as epilepsy caused by device inaccuracies (70). Furthermore, adverse reactions are also related to the type of rTMS, frequency, intensity, duration, and coil shape. Protocols with high frequencies, intensities above 120% of motor threshold, and short pulse intervals have a higher probability of inducing seizures (71,72).

In summary, triggers for adverse reactions to rTMS may also be influenced by other yet unidentified factors. Thus, deeper exploration of these triggers and the prevention of side effects such as seizures in patients remain urgent research lines. Moreover, although numerous animal experiments with rTMS have been conducted, few clinical trials have validated its use in humans, which undoubtedly constitutes a major obstacle for rTMS development.

Conclusion

To date, there is extensive clinical evidence supporting the value of rTMS for improving neurological deficits following stroke (motor function of upper and lower limbs, cognition, swallowing, language, mood, spasticity, and post-stroke neuropathic pain). While the exact mechanism by which rTMS improves post-stroke neurological deficits is not conclusive, the current status of rTMS is at a critical phase of translating its value into clinical application. At this stage, detailed documentation of the clinical efficacy, precautions, and adverse events of rTMS for stroke, as well as optimization and adjustment of stimulation parameters, is necessary.

TMS in Movement Disorders

Repetitive transcranial magnetic stimulation (rTMS) has demonstrated some degree of improvement in various movement disorders, such as Parkinson's disease (PD), dystonia, Huntington's disease, Tourette syndrome, and essential tremor (72)

Parkinson's Disease

Experimental studies suggest that rTMS induces changes in neurotransmitter release, synaptic efficiency, signaling pathways, and gene transcription (72,73). Additionally, rTMS stimulates neurogenesis, neuronal survival, and neurotransmitter release in patients with PD (72,73).

A possible mechanism of action involves the enhanced activity induced by high-frequency rTMS in the caudate nucleus, as well as the relief of dopamine deficiency within the nigrostriatal-thalamo-cortical circuit (74). Bradykinesia and tremor are two of the most disabling motor symptoms in PD and are believed to be associated with abnormal oscillations in the subthalamic nucleus (STN) (75). Bilateral stimulation of motor cortical regions has been shown to improve these symptoms (72–75).

The most favorable targets for high-frequency rTMS (rTMS-HF) include the primary motor cortex (M1), less focal motor cortex areas such as leg M1 or bilateral hand M1, and the dorsolateral prefrontal cortex (DLPFC). In contrast, the supplementary motor area (SMA) is the preferred target for low-frequency rTMS (rTMS-LF) (78–80).

Furthermore, rTMS targeting these areas has shown efficacy for levodopa-induced dyskinesia (LID) (77). Regarding depressive symptoms in PD, M1 stimulation with repeated rTMS-HF is recommended for treating motor symptoms, with potential benefits on mood (78–81).

A large double-cone coil applied to the leg M1 area may improve freezing of gait. rTMS has also been proposed as a treatment for cognitive impairment in PD, with additive effects to dopaminergic medications (82). Although the exact role of pathological neural oscillations in motor and cognitive function is complex, current evidence suggests these oscillations contribute to the observed deficits (83,84).

Currently, rTMS is considered an emerging strategy to improve certain motor symptoms and mood in PD, though optimal stimulation protocols remain to be established (see treatment protocols in Table IV).

Table II. Cerebrovascular Disease

Section	Protocol	Objective / Target	Parameters	Evidence / Outcome
1. Motor Recovery	**A. Inhibitory rTMS (1 Hz)** over Unaffected Hemisphere	Reduce contralateral hyperactivity interfering with recovery	- **Location:** M1 (unaffected hemisphere) - **Frequency:** 1 Hz - **Pulses:** 1200–1500/ session - **Intensity:** 90–110% MT -**Duration:** 10–15 sessions (1/day, 5 days/week)	- Significant motor improvement (Fugl-Meyer Assessment) - Synergistic with OT/PT*(Nowak et al., 2010)*
	B. Excitatory rTMS (5–10 Hz) over Affected Hemisphere	Stimulate neuroplasticity in the damaged hemisphere	- **Location:** M1 (affected hemisphere) - **Frequency:** 5–10 Hz - **Pulses:** 1000–2000/session - **Intensity:** 80–100% MT - **Duration:** 10–20 sessions	Greater benefit in subacute stroke (<6 months) *(Lefaucheur et al., 2014)*
	C. Dual Protocol (High & Low Frequency)	Combine excitatory and inhibitory protocols	- **Morning:** 10 Hz on affected M1 - **Afternoon:** 1 Hz on unaffected M1	Greater efficacy than monotherapy*(Wang et al., 2021)*
2. Aphasia (Left Hemisphere Stroke)	**A. Inhibitory rTMS (1 Hz)** over Right Hemisphere	Suppress overactivation in right homologous Broca's area	- **Target:** Pars triangularis (BA45) - **Frequency:** 1 Hz - **Pulses:** 1200 - **Intensity:** 90% MT - **Duration:** 10 sessions	Improvement in verbal fluency*(Naeser et al., 2012)*
	B. Excitatory rTMS (10 Hz) over Left Hemisphere	Enhance preserved perilesional language areas	- **Target:** Left perilesional area - **Frequency:** 10 Hz - **Pulses:** 1000 - **Intensity:** 80% MT - **Duration:** 15 sessions	Language function improvement (noted in studies)

Table III. Various protocols (cerebrovascular disease).

Category	Protocol	Mechanism / Target	Parameters	Evidence / Outcome
Dysphagia	**Excitatory rTMS (5 Hz)** over Pharyngeal Cortex	Stimulates pharyngeal cortex (M1 or inferior frontal region)	- **Frequency:** 5 Hz - **Pulses:** 500 • **Intensity:** 100% MT - **Ses-**	Reduces risk of aspiration *(Khedr et al., 2019)*
Spasticity	**Inhibitory rTMS (1 Hz)** over Contralateral M1	Suppresses cortical excitability linked to spasticity	- **Frequency:** 1 Hz	Reduces hypertonia in affected limbs *(Málly et al., 2018)*
Factors Influencing Response	—	—	- **Timing:** - Subacute (1–6 months): Greater plasticity - Chronic (>6 months): Modest benefits - **Stroke Location:** - Cortico-subcortical vs. brainstem - **Severity:** - Better response in mild-to-	Clinical outcomes vary based on timing, site, and severity

Table IV. RTMS Protocols in Parkinson's Disease.

Section	Protocol Type	Target / Location	Parameters	Evidence / Outcome
1. Motor Symptoms	**High Frequency (Excitatory)**	M1 contralateral to symptoms or DLPFC	- **Frequency:** 5–20 Hz (commonly 10 Hz) - **Pulses:** 1000–2000/ session - **Intensity:** 80–120% MT - **Duration:** 10–20 sessions (5 days/ week)	Improves bradykinesia and rigidity *(Lefaucheur et al., 2019)*
	Low Frequency (Inhibitory)	SMA or unaffected hemisphere	- **Frequency:** 1 Hz - **Pulses:** 600–1200/ session - **Intensity:** 90–110% MT - **Duration:** 10–15 sessions	Reduces dyskinesias via cortical modulation *(Koch et al., 2020)*
2. Non-Motor Symptoms	**A. Depression / Apathy**	Left DLPFC (excitatory) or Right DLPFC (inhibitory)	- **Frequency:** 10 Hz (left) or 1 Hz (right) - **Pulses:** 3000/session - **Duration:** 15–30 sessions	Comparable efficacy to antidepressants *(Dobkin et al., 2016)*
	B. Cognitive Disorders	Bilateral DLPFC or parietal cortex	- **Frequency:** 5–10 Hz - **Duration:** 10–15 sessions	Mild executive function improvement *(Brys et al., 2016)*
3. Combined Protocols	**Example 1**	10 Hz over M1 (morning) + 1 Hz over SMA (afternoon)	—	Addresses both motor symptoms and dyskinesias
	Example 2	rTMS + Physical Rehabilitation	—	Enhances neuroplasticity *(Chang et al., 2021)*

TMS in Multiple Sclerosis

Multiple sclerosis (MS) is usually treated with disease-modifying therapies. However, despite increasingly effective treatments, patients develop relapsing-remitting MS (RRMS) and secondary progressive MS (SPMS). Since rTMS has no known interaction with MS drugs, it can be used as an adjunctive treatment to manage motor and sensory symptoms of MS (122).

Some MS symptoms are believed to be related to neuronal transmission in the brain. Low-frequency rTMS (LF-rTMS) on single neurons can cause prolonged inhibition of neuronal transmission, whereas high-frequency rTMS (HF-rTMS) can enhance neuronal transmission (122). Therefore, rTMS pulse trains modulate activity in the target brain region for minutes or even hours (123).

Thus, TMS may alleviate debilitating MS symptoms such as fatigue, spasticity, gait abnormalities, and manual dexterity impairments, which affect quality of life (QoL), especially in RRMS and SPMS patients (123–125).

Evidence on benefits for fatigue is contradictory. In 34 patients with secondary progressive MS, HF-rTMS (20 Hz) and intermittent theta burst stimulation (iTBS) an excitatory protocol with side effects similar to HF-rTMS—were used to manage spasticity. Both HF-rTMS and iTBS showed significant reduction in spasticity measured by the Modified Ashworth Scale compared to sham stimulation (127). iTBS produced a longer-lasting effect on the Subjective Evaluation of Spasticity Scale (SESS), and when administered after HF-rTMS, it also reduced pain and fatigue.

However, a systematic review and meta-analysis comparing transcranial direct current stimulation (tDCS), rTMS, and transcranial random noise stimulation (tRNS) did not find rTMS to be beneficial for fatigue (128).

No conclusive recommendation exists yet for therapeutic use of TMS in MS. Lefaucheur's evidence-based guidelines (81) suggest that iTBS targeting the leg motor cortex may be recommended for treating lower limb spasticity in MS. However, they do not recommend iTBS over the hand motor cortex to improve manual dexterity, nor high-frequency rTMS with an H-coil for fatigue. Given that iTBS and HF-rTMS with H-coil have shown some benefits, large-scale studies with established protocols are needed to determine how TMS can be effectively and routinely used therapeutically in MS (see treatment protocol in Table V).

Table V. rTMS Protocols in Alzheimer's Disease.

Category	Target / Mechanism	Protocol Parameters	Evidence / Outcome
1. Cognitive Symptoms	**A. Memory & Executive Functions**Stimulates frontal and temporoparietal networks	- **Frequency:** 10 Hz - **Location:** - Left DLPFC (executive function) - Left inferior parietal lobe (semantic memory) - **Pulses:** 2000/session (20 × 10s trains, 30s intervals) - **Intensity:** 80–100% MT - **Duration:** 5 days/week for 4–6 weeks	- Improved MMSE and ADAS-Cog - Effects lasting up to 3 months *(Rabey et al., 2013)*
	B. Language (Aphasia in Advanced AD) Enhances language output via Broca's area	- **Frequency:** 5 Hz - **Location:** Broca's area (dominant hemisphere) - **Pulses:** 1000/session - **Sessions:** 10	Experimental use for language deficits
2. Behavioral Symptoms	**C. Apathy & Depression**Modulates mood-related circuits	- **Frequency:** 10 Hz (left DLPFC) or 1 Hz (right DLPFC) - **Pulses:** 3000/session - **Sessions:** 15–20	Reductions in NPI depression/apathy scores *(Lee et al., 2016)*
	D. Agitation & Aggressiveness Experimental modulation of emotional centers	- **Frequency:** 1 Hz - **Target:** Medial prefrontal cortex	Preliminary evidence; under investigation
3. Combined Protocols	Cognition + Mood Regulation	- **Morning:** 10 Hz on left DLPFC (cognition) - **Afternoon:** 1 Hz on right DLPFC (depression) - **Duration:** 6 weeks	Integrated benefit for cognitive and behavioral symptoms
4. Clinical Considerations	Stage & Safety	- **Best response:** Mild-to-moderate AD - **Caution:** Limited efficacy in advanced AD - **Safety:** Generally well tolerated - **Avoid in:** Epilepsy or metallic implants - **Precision:** Neuronavigation recommended due to	Supports careful patient selection and targeting strategy

Cortical Dementia of the Alzheimer Type: Favorable Effect of RTMS On Certain Aspects of Cognitive Function

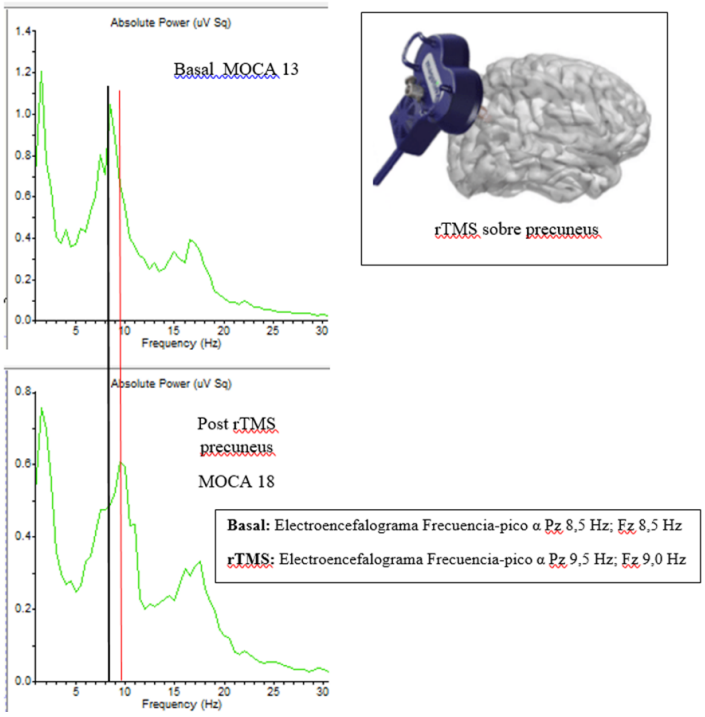

***Figure 3**. Clinical improvement (M.O.C.A) and EEG with acceleration of the parietal alpha-peak frequency of the Fast Fourier Transform after rTMS on precuneiform muscles (20 sessions)*

Table VI. rTMS Protocols for Specific Symptoms in Multiple Sclerosis (MS).

Symptom	Target / Mechanism	Suggested Protocol	Evidence / Outcome
A. Fatigue	Modulation of DLPFC and motor areas	- **Frequency:** 10 Hz (excitatory) - **Location:** Left DLPFC - **Pulses:** 1500–2000/session - **Intensity:** 80–100% MT - **Duration:** 10–15 sessions (5 days/week)	Significant fatigue reduction (MFIS) *(Tataroglu et al., 2020)*
B. Spasticity	Inhibition of cortical hyperexcitability	**Frequency:** 1 Hz (inhibitory) - **Location:** M1 contralateral to spastic limb - **Pulses:** 1200–1500/session - **Intensity:** 90–110% MT - **Duration:** 10 sessions	Moderate reduction in spasticity *(Ashworth Scale, Mori et al., 2021)*
C. Depression & Anxiety	Similar to major depression treatment protocols	- **Frequency:** 10 Hz (left DLPFC) or 1 Hz (right DLPFC) - **Pulses:** 3000/session - **Duration:** 20–30 sessions	Follows FDA-approved rTMS protocols for depression
D. Cognitive Dysfunction	Stimulation of frontoparietal networks	- **Frequency:** 5–10 Hz - **Location:** Bilateral DLPFC or parietal lobe - **Duration:** 10–15 sessions	Mild improvements in working memory and processing speed *(Sandrini et al., 2020)*

TMS in Epilepsy

Although antiepileptic drugs (AEDs) are the mainstay of epilepsy treatment, one-third of patients develop drug resistance. Many of these patients are not suitable candidates for surgical ablation. This group, at higher risk of morbidity, may respond to low-frequency TMS (LF-TMS) (78).

rTMS may reduce seizure likelihood in this population, probably due to its ability to induce prolonged inhibitory effects on synaptic potentials and focal cortical excitability (129).

No formal recommendation currently exists for therapeutic use of TMS in epilepsy. While the Cochrane review determined that rTMS is safe and effective in reducing epileptiform discharges, clear evidence for seizure frequency reduction is lacking (130). There is currently substantial variability in TMS techniques used in studies, reported outcomes, and the definition of drug-resistant epilepsy (130, 131).

Clinical Effects of rTMS in Cerebellar Ataxias

Low-frequency cerebellar TMS acts by reducing the inhibitory regulation of the cerebellar cortex over the dentate nucleus, thereby enhancing part of the impaired functionality of this nucleus. Additionally, a reduced inhibitory signal from Purkinje cells may enhance activation of the vestibular nuclei, resulting in improved balance in patients with cerebellar ataxias (132–136).

Spinocerebellar Ataxia

Dysfunction of the cerebellum and its connected neuronal networks causes a neurodegenerative disorder known as spinocerebellar ataxia (SCA). In a randomized, double-blind, placebo-controlled study, significant improvement was observed in clinical and kinematic outcomes of postural control in standing patients who completed a 4-week rTMS intervention with one month of follow-up, compared to patients who received sham intervention (136).

Hereditary Ataxias

The role of TMS in the diagnosis, pathophysiology, and treatment of genetically confirmed hereditary ataxias was examined in a critical review (135, 136). Hereditary ataxias are a heterogeneous group of neurodegenerative disorders affecting the motor cortex and corticospinal tract. In early involvement of the cortex and corticospinal tract, TMS has proven useful; similarly, repetitive cerebellar TMS (rTMS) has demonstrated efficacy as a therapeutic approach (136).

Truncal Ataxia

A placebo-controlled trial reported on the efficacy of cerebellar TMS for hereditary spinocerebellar degeneration (136, 137). Patients treated with active TMS showed significant reduction in truncal ataxia. Contraction of neck and shoulder muscles was evoked by active stimulation. Study findings revealed that disease type influenced rTMS efficacy (137).

Neurodegenerative Ataxia

A review by Benussi et al. (138) concluded that non-invasive brain stimulation has made substantial advances in developing specific stimulation protocols to regulate cerebellar excitability aiming to restore physiological cerebellar activity in patients with ataxia. Literature has shown that rTMS or transcranial direct current stimulation (tDCS) can be useful tools for patients with neurodegenerative ataxia

Placebo Effects

Substantial placebo effects exist in rTMS. This means its efficacy (response rates, remission rates, etc.) must be assessed not only in isolation but in comparison with placebo groups. Therefore, clinical studies lacking double-blind selection and placebo control provide limited information on whether clinical outcomes are attributable to direct neuromodulatory effects or indirect placebo effects. This is highly relevant not only in psychiatric applications but also in neurology, as is very evident, for example, in essential tremor. Most studies included in this review are placebo-controlled. However, concerns remain that some neurological disorder literature lacks adequate controls or proper selection.

Conclusions

Current Limitations and Need for Standardization

Although recent studies explore new TMS applications such as cerebellar stimulation in movement disorders (139)—methodological challenges remain. Optimization of this therapy requires greater standardization in:

- Study designs: Larger randomized controlled trials (RCTs).
- Stimulation parameters: Frequency, anatomical localization (e.g., individualized neuronavigation), coil type, and treatment duration.
- Outcome criteria: Objective measures (e.g., validated clinical scales) and biomarkers (neuroimaging, electrophysiology).

Current Evidence and Clinical Role

This review demonstrates that although TMS is not a first-line therapy, it offers significant benefits in:

- Drug-resistant patients: Symptomatic improvement in debilitating diseases (e.g., major depression, neuropathic pain).
- Neurorehabilitation: Post-stroke motor recovery (evidence level B) (138) and spasticity management in multiple sclerosis (128).

However, widespread clinical recommendation requires more phase III/IV evidence validating efficacy and cost-effectiveness.

Future Directions

Integration of emerging technologies may overcome current limitations:

- Artificial intelligence (AI): Optimization of protocols using predictive models (e.g., dose-response based on phenotypes).
- Advanced neuronavigation: Precise targeting of cortical sites with MRI tractography and functional MRI.
- Biomarkers: Identification of neurophysiological signatures (e.g., beta oscillation power) for personalized therapies.

Final Statement

TMS is a promising tool for the clinical neurologist, particularly in scenarios of therapeutic resistance. Although routine use requires further evidence, the convergence of technological and neuroscience innovations positions this modality as a future pillar in neuromodulation.

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References

1. Barker AT, Jalinous R, Freeston IL. Non-invasive magnetic stimulation of human motor cortex. The Lancet. Volumen: 1 Número: 8437: 1106-1107. 1985. DOI: 10.1016/S0140-6736(85)92413-4
2. Seewoo BJ, Etherington SJ, Rodger J. Transcranial magnetic stimulation. eLS.(2019) 2019:1-8. doi: 10.1002/9780470015902.a0028620.

3. Ali Jannati , Lindsay M. Oberman , Alexander Rotenberg , Alvaro Pascual-Leone. Assessing the mechanisms of brain plasticity by transcranial magnetic stimulation. Neuropsychopharmacology (2023) 48:191–208; https://doi.org/10.1038/s41386-022-01453-8

4. Xinlei Zhang, Lingling Zhu, Yuan Li , Hongna Yu, Tao Wang , Xiuli Chu. Therapeutic potential and mechanisms of repetitive transcranial magnetic stimulation in Alzheimer's disease: a literature review.European Journal of Medical Research (2025) 30:233 https://doi.org/10.1186/s40001-025-02493-8

5. Simone Rossia,, Andrea Antalb,, Sven Bestmannd, Marom Biksone, Carmen Brewerf , Jürgen Brockmöllerg , et al. Safety and recommendations for TMS use in healthy subjects and patient populations, with updates on training, ethical and regulatory issues: Expert Guidelines.Clin Neurophysiol. 2021 January ; 132(1): 269–306. doi:10.1016/j.clinph.2020.10.003.

6. Rossi S, Hallett M, Rossini PM, Pascual-Leone A. Safety, ethical considerations, and application guidelines for the use of transcranial magnetic stimulation in clinical practice and research. Clin Neurophysiol 2009;120:2008–39. 10.1016/j.clinph.2009.08.016. [PubMed: 19833552]

7. Vucic S, Kai-Hsiang Stanley Chen, Kiernan MC,Hallett M, Benninger D, Di Lazzaro V. et al. Clinical diagnostic utility of transcranial magnetic stimulation in neurological disorders. Updated report of an IFCN committee. Clin Neurophysiol. 2023 June ; 150: 131–175. doi:10.1016/j.clinph.2023.03.010.

8. Li S, Jiao R, Zhou X, Chen S. Motor recovery and antidepressant effects of repetitive transcranial magnetic stimulation on Parkinson disease: a PRISMA-compliant meta-analysis. Medicine 2020;99:18(e19642)

9. Iglesias AH. Transcranial magnetic stimulation as treatment in multiple neurologic conditions. Curr Neurol Neurosci Rep. (2020) 20:1-9.doi: 10.1007/s11910-020-1021-0

10. Tavakoli H, Heidarpanah A , A Literature Review of the Efficacy of Repetitive Transcranial Magnetic Stimulation on Epilepsy. Iran J Child Neurol. Spring 2022; 17 (1): 9-28

11. Walton D, Spencer DC, Nevitt SJ, Michael BD. Transcranial magnetic stimulation for the treatment of epilepsy. Cochrane Database of Systematic Reviews 2021, Issue 4. Art. No.: CD011025. DOI: 10.1002/14651858.CD011025.pub3.

12. Ruiz ML, Sospedra M, Arce SA, Tejeiro-Martínez J, Benito-León J. Current. Evidence on the potential therapeutic applications of transcranial magnetic stimulation in multiple sclerosis: a systematic review of the literature.Neurología. (2020) 37:199–215. doi: 10.1016/j.nrl.2018.03.023

13. Yi Ji1 , Chaoyi Yang , Xuerui Pang, Yibing Yan , Yue Wu et al. Repetitive transcranial magnetic stimulation in Alzheimer's disease: effects on neural and synaptic rehabilitation NEURAL REGENERATION RESEARCH | Vol 20 | No. 2 | February 2025

14. Frey J, Hess CW, Kugler L, Wajid M and Wagle Shukla A (2021) Transcranial Magnetic Stimulation in Tremor Syndromes: Pathophysiologic Insights and Therapeutic Role. Front. Neurol. 12:700026. doi: 10.3389/fneur.2021.700026

15. KimberleyT J, Borich,M R , Arora S, MD1, Siebner H R. Multiple sessions of low-frequency repetitive transcranial magnetic stimulation in focal hand dystonia: clinical and physiological effects. Restor Neurol Neurosci. 2013 ; 31(5): 533–542. doi:10.3233/RNN-120259.

16. Li Zhou , Yaju Jin , Danli Wu , Yongdan Cun , Chengcai Zhang , et al. Current evidence, clinical applications, and future directions of transcranial magnetic stimulation as a treatment for ischemic stroke. Sec. Translational Neuroscience .Volume 17 - 2023

17. Saini , V., Guada, L., and Yavagal, D. R. Global epidemiology of stroke and access to acute ischemic stroke interventions. Neurology 97, S6–s16. 2021. doi: 10.1212/WNL.0000000000012781.

18. Hallett M. Transcranial magnetic stimulation and the human brain. Nature.(2000) 406:147-50. doi: 10.1038/35018000

19. Chohan, S. A., Venkatesh, P. K., and How, C. H. Long-term complications of stroke and secondary prevention: an overview for primary care physicians. Singap. Med. J. 60, 616–620.2019. doi: 10.11622/smedj.2019158

20. Dionísio, A., Gouveia, R., Castelhano, J., Duarte, I. C., Santo, G. C., Sargento-Freitas, J.,et al. (2021). The role of continuous theta burst TMS in the neurorehabilitation of subacute stroke patients: a placebo-controlled study. Front. Neurol. 12:749798. doi:10.3389/fneur.2021.749798

21. Hoonhorst, M. H. J., Nijland, R. H. M., Emmelot, C. H., Kollen, B. J., and Kwakkel, G. TMS-induced central motor conduction time at the non-infarcted hemisphere is associated with spontaneous motor recovery of the paretic upper limb after severe stroke. Brain Sci. 11:648. 2021. doi: 10.3390/brainsci11050648

22. Zong, X., Gu, J., Zhou, S., Ding, D., Hu, Y., Tucker, L., et al. Continuous thetaburst stimulation enhances and sustains neurogenesis following ischemic stroke. Theranostics 12, 5710–5726.(2022) doi: 10.7150/thno.71832

23. Lee, C., Kim, Y., and Kaang, B. K. (2022). The primary motor cortex: The hub of motor learning in rodents. Neuroscience 485, 163–170. doi: 10.1016/j.neuroscience.2022.01.009

24. Wolf, S. L., Winstein, C. J., Miller, J. P., Taub, E., Uswatte, G., Morris, D., et al. Effect of constraint-induced movement therapy on upper extremity function 3 to 9 months after stroke: the EXCITE randomized clinical trial. JAMA 296, 2095–2104. (2006) doi: 10.1001/jama.296.17.2095

25. Kim, Y. H., You, S. H., Ko, M. H., Park, J. W., Lee, K. H., Jang, S. H., et al.Repetitive transcranial magnetic stimulation-induced corticomotor excitability and associated motor skill acquisition in chronic stroke. Stroke 37, 1471–1476. (2006) doi: 10.1161/01.STR.0000221233.55497.51

26. Long, H., Wang, H., Zhao, C., Duan, Q., Feng, F., Hui, N., et al. (2018). Effects of combining high- and low-frequency repetitive transcranial magnetic stimulation on upper limb hemiparesis in the early phase of stroke. Restor. Neurol. Neurosci. 36, 21–30.(2018)doi: 10.3233/RNN-170733

27. Chen, G., Lin, T., Wu, M., Cai, G., Ding, Q., Xu, J., et al. Effects of repetitive transcranial magnetic stimulation on upper-limb and finger function in stroke patients: a systematic review and meta-analysis of randomized controlled trials. Front. Neurol. 13:940467. (2022)doi: 10.3389/fneur.2022.940467

28. Wu, X., Wang, R., Wu, Q., Liao, C., Zhang, J., Jiao, H., et al. (2023). The effects of combined high-frequency repetitive transcranial magnetic stimulation and cervical nerve root magnetic stimulation on upper extremity motor recovery following stroke. Front. Neurosci. 17:1100464. (2023)doi: 10.3389/fnins.2023.1100464

29. Wang, Y., Xu, N., Wang, R., and Zai, W. Systematic review and network meta-analysis of effects of noninvasive brain stimulation on post-stroke cognitive impairment. Front. Neurosci. 16:1082383.(2022) doi: 10.3389/fnins.2022.1082383

30. Paul, S. L., Srikanth, V. K., and Thrift, A. G. The large and growing burden of stroke. Curr. Drug Targets 8, 786–793. (2007)doi: 10.2174/138945007781077418 Li, Y., Fan, J., Yang, J., He, C., and Li, S. Effects of repetitive transcranial-magnetic stimulation on walking and balance function after stroke: a systematic review and meta-analysis. Am. J. Phys. Med. Rehabil. 97, 773–781.(2018) doi: 10.1097/PHM.0000000000000948

31. Vaz, P. G., Salazar, A., Stein, C., Marchese, R. R., Lukrafka, J. L., Plentz, R. D. M., et al. Noninvasive brain stimulation combined with other therapies improves gait speed after stroke: a systematic review and meta-analysis. Top. Stroke Rehabil. 26,201–213. (2019)doi: 10.1080/10749357.2019.1565696

32. Naghdi, S., Ansari, N. N., Rastgoo, M., Forogh, B., Jalaie, S., and Olyaei, G. A pilot study on the effects of low frequency repetitive transcranial magnetic stimulation on lower extremity spasticity and motor neuron excitability in patients after stroke. J. Bodyw. Mov. Ther. 19, 616–623. (2015)doi: 10.1016/j.jbmt.2014.10.001

33. Rastgoo, M., Naghdi, S., Nakhostin Ansari, N., Olyaei, G., Jalaei, S., Forogh, B., et al. Effects of repetitive transcranial magnetic stimulation on lower extremity spasticity and motor function in stroke patients. Disabil. Rehabil. 38, 1918 –1926. (2016)doi:10.3109/09638288.2015.1107780

34. Wang, R. Y., Tseng, H. Y., Liao, K. K., Wang, C. J., Lai, K. L., and Yang, Y. R. rTMS combined with task-oriented training to improve symmetry of interhemispheric corticomotor excitability and gait performance after stroke: a randomized trial.

35. Neurorehabil. Neural Repair 26, 222–230. (2012)doi: 10.1177/1545968311423265

36. Kang, N., Lee, R. D., Lee, J. H., and Hwang, M. H. Functional balance and postural control improvements in patients with stroke after noninvasive brain stimulation: a meta-analysis. Arch. Phys. Med. Rehabil. 101, 141–153.(2020) doi: 10.1016/j.apmr.2019.09.003

37. Veldema, J., and Gharabaghi, A. Non-invasive brain stimulation for improving gait, balance, and lower limbs motor function in stroke. J. Neuroeng. Rehabil. 19:84. (2022)doi:10.1186/s12984-022-01062-y

38. Chieffo, R., de Prezzo, S., Houdayer, E., Nuara, A., di Maggio, G., Coppi, E., et al. (2014). Deep repetitive transcranial magnetic stimulation with H-coil on lower limb motor function in chronic stroke: a pilot study. Arch. Phys. Med. Rehabil. 95, 1141–1147.(2014)doi: 10.1016/j.apmr.2014.02.019

39. Huang, Y. Z., Lin, L. F., Chang, K. H., Hu, C. J., Liou, T. H., and Lin, Y. N. (2018). Priming with 1-Hz repetitive transcranial magnetic stimulation over Contralesional leg motor cortex does not increase the rate of regaining ambulation within 3 months of stroke: a randomized controlled trial. Am. J. Phys. Med. Rehabil. 97, 339–345.(2018) doi: 10.1097/PHM.0000000000000850

40. Rastgoo, M., Naghdi, S., Nakhostin Ansari, N., Olyaei, G., Jalaei, S., Forogh, B., et al. Effects of repetitive transcranial magnetic stimulation on lower extremity spasticity and motor function in stroke patients. Disabil. Rehabil. 38, 1918 –1926. (2016)doi:10.3109/09638288.2015.1107780

41. Naghdi, S., Ansari, N. N., Rastgoo, M., Forogh, B., Jalaie, S., and Olyaei, G. A pilot study on the effects of low frequency repetitive transcranial magnetic stimulation on lower extremity spasticity and motor neuron excitability in patients after stroke. J. Bodyw. Mov. Ther. 19, 616–623.(2015) doi: 10.1016/j.jbmt.2014.10.001

42. Liu, Y., Li, H., Zhang, J., Zhao, Q. Q., Mei, H. N., and Ma, J. A meta-analysis: whether repetitive transcranial magnetic stimulation improves dysfunction caused by stroke with lower limb spasticity. Evid. Based Complement. Alternat. Med. 2021, 1–10. doi: 10.1155/2021/721929

43. Cha, B., Kim, J., Kim, J. M., Yin, L., Li, K., Li, Y., et al. (2022). Therapeutic effect of repetitive transcranial magnetic stimulation for Post-stroke vascular cognitive impairment: a prospective pilot study. Front. Neurol. 13:813597. doi: 10.3389/fneur.2022.813597

44. Hara, T., Shanmugalingam, A., McIntyre, A., and Burhan, A. M. (2021). The effect of non-invasive brain stimulation (NIBS) on attention and memory function in stroke rehabilitation patients: a systematic review and meta-analysis. Diagnostics 11:227. (2021)doi:10.3390/diagnostics11020227

45. Bressler, S. L., and Menon, V. Large-scale brain networks in cognition: emerging methods and principles. Trends Cogn. Sci. 14, 277–290. (2010)doi: 10.1016/j.tics.2010.04.004

46. Yin, M., Liu, Y., Zhang, L., Zheng, H., Peng, L., Ai, Y., et al. Effects of rTMS treatment on cognitive impairment and resting-state brain activity in stroke patients: a randomized clinical trial. Front. Neural. Circuits 14:563777.(2020) doi: 10.3389/fncir.2020.563777

47. Yingli, B., Zunke, G., Wei, C., and Shiyan, W. Cerebral activity manipulation of low-frequency repetitive transcranial magnetic stimulation in post-stroke patients with cognitive impairment. Front. Neurol. 13:951209. (2022)doi: 10.3389/fneur.2022.951209

48. Wang, L., Zhu, Q. X., Zhong, M. H., Zhou, R.-Z., Liu, X.-Q., Tang, N.-S., et al. (2022). Effects of corticospinal tract integrity on upper limb motor function recovery in stroke patients treated with repetitive transcranial magnetic stimulation. J. Integr. Neurosci. 21:50.(2022) doi: 10.31083/j.jin2102050

49. Li, W., Wen, Q., Xie, Y. H., Hu, A. L., Wu, Q., and Wang, Y. X. Improvement of poststroke cognitive impairment by intermittent theta bursts: a double-blind randomized controlled trial. Brain Behav. 12:e2569. (2022)doi: 10.1002/brb3.2569

50. Kumar, S., Selim, M. H., and Caplan, L. R. Medical complications after stroke. Lancet Neurol. 9, 105–118.(2010) doi: 10.1016/S1474-4422(09)70266-2

51. Du, J., Yang, F., Liu, L., Hu, J., Cai, B., Liu, W., et al. Repetitive transcranial magnetic stimulation for rehabilitation of poststroke dysphagia: a randomized, double-blind clinical trial. Clin. Neurophysiol. 127, 1907–1913.(2016) doi:10.1016/j.clinph.2015.11.04

52. Liao, X., Xing, G., Guo, Z., Jin, Y., Tang, Q., He, B., et al. Repetitive transcranial magnetic stimulation as an alternative therapy for dysphagia after stroke: a systematic review and meta-analysis. Clin. Rehabil. 31, 289–298. (2017). doi:10.1177/0269215516644771

53. Cheng, I. K., Chan, K. M., Wong, C. S., and Cheung, R. T. F. Preliminary evidence of the effects of high-frequency repetitive transcranial magnetic stimulation (rTMS) on swallowing functions in post-stroke individuals with chronic dysphagia. Int. J. Lang. Commun. Disord. 50, 389–396.(2015) doi: 10.1111/1460-6984.12144

54. Cheng, I. K. Y., Chan, K. M. K., Wong, C. S., Li, L., Chiu, K., Cheung, R., et al. Neuronavigated high-frequency repetitive transcranial magnetic stimulation for chronic post-stroke dysphagia: a randomized controlled study. J. Rehabil. Med. 49, 475–481.(2017) doi: 10.2340/16501977-2235

55. Park, E., Kim, M. S., Chang, W. H., Oh, S. M., Kim, Y. K., Lee, A., et al. Effects of bilateral repetitive transcranial magnetic stimulation on post-stroke dysphagia. Brain Stimul. 10, 75–82. (2017)doi: 10.1016/j.brs.2016.08.005

56. Wen, X., Liu, Z., Zhong, L., Peng, Y., Wang, J., Liu, H., et al. The effectiveness of repetitive transcranial magnetic stimulation for Post-stroke dysphagia: a systematic review and meta-analysis. Front. Hum. Neurosci. 16:841781. (2022) doi: 10.3389/ fnhum.2022.841781

57. Hara, T., and Abo, M. New treatment strategy using repetitive transcranial magnetic stimulation for Post-stroke aphasia. Diagnostics (Basel) 11:1853. (2021)doi: 10.3390/diagnostics11101853

58. Gholami, M., Pourbaghi, N., and Taghvatalab, S. Evaluation of rTMS in patients with poststroke aphasia: a systematic review and focused meta-analysis. Neurol. Sci. 43, 4685–4694. (2022). doi: 10.1007/s10072-022-06092-x

59. Ren, C., Zhang, G., Xu, X., Hao, J., Fang, H., Chen, P., et al. The effect of rTMS over the different targets on language recovery in stroke patients with global aphasia: a randomized sham-controlled study. Biomed. Res. Int. 2019, 1–7. (2019). doi: 10.1155/2019/4589056

60. Duan, H., Yan, X., Meng, S., Qiu, L., Zhang, J., Yang, C., et al.Effectiveness evaluation of repetitive transcranial magnetic stimulation therapy combined with mindfulness-based stress reduction for people with Post-stroke depression: a randomized controlled trial. Int. J. Environ. Res. Public Health 20:930.(2023) doi: 10.3390/ijerph20020930

61. Frey, J., Najib, U., Lilly, C., and Adcock, A. (2020). Novel TMS for stroke and depression (NoTSAD): accelerated repetitive transcranial magnetic stimulation as a safe and effective treatment for Post-stroke depression. Front. Neurol. 11:788.(2020) doi: 10.3389/fneur.2020.00788

62. Wei, X., Xia, N., Li, Y. A., Gu, M., Zhang, T., Gao, W., et al. Immediate and short-term effects of continuous theta burst transcranial magnetic stimulation over contralesional premotor area on post-stroke spasticity in patients with severe hemiplegia: study protocol for a randomized controlled trial. Front. Neurol. 13:895580. (2022)doi: 10.3389/ fneur.2022.895580

63. Xia, Y., Xu, Y., Li, Y., Lu, Y., and Wang, Z. Comparative efficacy of different repetitive transcranial magnetic stimulation protocols for stroke: a network meta-analysis. Front. Neurol. 13:918786. doi: 10.3389/fneur.2022.918786

64. Li, D., Cheng, A., Zhang, Z., Sun, Y., and Liu, Y. (2021). Effects of low-frequency repetitive transcranial magnetic stimulation combined with cerebellar continuous theta burst stimulation on spasticity and limb dyskinesia in patients with stroke. BMC Neurol. 21:369.(2021) doi: 10.1186/s12883-021-02406-2

65. Qin, Y., Liu, X., Zhang, Y., Wu, J., and Wang, X. (2023). Effects of transcranial combined with peripheral repetitive magnetic stimulation on limb spasticity and resting-state brain activity in stroke patients. Front. Hum. Neurosci. 17:992424.(2023) doi: 10.3389/fnhum.2023.992424

66. Bo, Z., Jian, Y., Yan, L., Gangfeng, G., Xiaojing, L., Xiaolan, L., et al. Pharmacotherapies for central Post-stroke pain: a systematic review and networkmeta-analysis. Oxidative Med. Cell. Longev. 2022, 1–10. doi: 10.1155/2022/3511385

67. Malfitano, C., Rossetti, A., Scarano, S., Malloggi, C., and Tesio, L. Efficacy of repetitive transcranial magnetic stimulation for acute central Post-stroke pain: a case study. Front. Neurol. 12:742567. (2021). doi: 10.3389/ fneur.2021.742567

68. Qiao, J., Ye, Q. P., Wu, Z. M., Dai, Y., and Dou, Z. L. (2022). The effect and optimal parameters of repetitive transcranial magnetic stimulation on poststroke dysphagia: a meta-analysis of randomized controlled trials. Front. Neurosci. 16:845737. doi: 10.3389/fnins.2022.845737

69. Walton, D., Spencer, D. C., Nevitt, S. J., and Michael, B. D. Transcranial magnetic stimulation for the treatment of epilepsy. Cochrane Database Syst. Rev. 4:Cd011025. (2021). doi: 10.1002/14651858.CD011025.pub3

70. Bae, E. H., Schrader, L. M., Machii, K., Alonso-Alonso, M., Riviello, J. J. Jr.,Pascual-Leone, A., et al. Safety and tolerability of repetitive transcranial magnetic stimulation in patients with epilepsy: a review of the literature. Epilepsy Behav. 10, 521–528. (2007). doi: 10.1016/j.yebeh.2007.03.004

71. Tendler, A., Roth, Y., and Zangen, A. Rate of inadvertently induced seizures with deep repetitive transcranial magnetic stimulation. Brain Stimul. 11, 1410–1414. (2018). doi:10.1016/j.brs.2018.09.001

72. Arias-Carrión O. Basic mechanisms of rTMS: implications in Parkinson's disease. Int Arch Med. (2008) 1:1-8. doi: 10.1186/1755-7682-1-2

73. Yang, M. J. (2023). Clinical effect of high-frequency repetitive transcranial magnetic stimulation in the treatment of cognitive dysfunction in Parkinson's disease. Chin. Mod. Med. 12, 19–24. doi: 10.3969/j.issn.1674-4721.2023.12.006

74. Yang Y , Yan Z, Chang W , Ding J, Xu H. Effect of different modalities of transcranial magnetic stimulation on Parkinson's patients cognitive impairment and long-term effectiveness: a systematic review and network meta-analysis. Front. Neurosci. 18:1354864.(2024) doi: 10.3389/fnins.2024.1354864

75. González-García N, Armony JL, Soto J, Trejo D, Alegría MA, Drucker-Colín R. Effects of rTMS on Parkinson's disease: a longitudinal fMRI study. J Neurol. (2011) 258:1268-80. doi: 10.1007/s00415-011-5923-2

76. Alemam AI, Eltantawi MA. Repetitive transcranial magnetic stimulation in treatment of levodopa-induced dyskinesia in Parkinson's Disease. J Neurol Res. (2019) 9:28-34. doi: 10.14740/jnr512

77. Weinberger M, Hutchison WD, Dostrovsky JO. Pathological subthalamic nucleus oscillations in PD: can they be the cause of bradykinesia and akinesia? Exp Neurol. (2009) 219:58–61.doi: 10.1016/j.expneurol.2009.05.014

78. Habib S, Hamid U, Jamil A, Zainab AZ, Yousuf T, Habib S, et al. Transcranial magnetic stimulation as a therapeutic option for neurologic and psychiatric illnesses. Cureus. (2018) 10:e3456. doi: 10.7759/cureus.3456

79. Latorre A, Rocchi L, Berardelli A, Bhatia KP, Rothwell JC. The use of transcranial magnetic stimulation as a treatment for movement disorders: a critical review. Movement Disord. (2019) 34:769-82. doi: 10.1002/mds.27705

80. Machado S, Arias-Carrión O, Paes F, Vieira RT, Caixeta L, Novaes F, et al. Repetitive transcranial magnetic stimulation for clinical applications in neurological and psychiatric disorders: an overview. Eurasian J Med. (2013) 45:191. doi: 10.5152/eajm.2013.39

81. Lefaucheur J-P. Evidence-based guidelines on the therapeutic use of repetitive transcranial magnetic stimulation (rTMS): an update (2014-2018).Clin Neurophysiol. (2020) 131:474-528. doi: 10.1016/j.clinph.2020.02.003

82. Khedr EM, Mohamed KO, Ali AM, Hasan AM. The effect of repetitive transcranial magnetic stimulation on cognitive impairment in Parkinson's disease with dementia: pilot study. Restor Neurol Neurosci. (2020) 38:55-66.doi: 10.3233/RNN-190956

83. Teo W-P, Hendy AM, Goodwill AM, Loftus AM. Transcranial alternating current stimulation: a potential modulator for pathological oscillations in Parkinson's disease? Front Neurol. (2017) 8:185.doi: 10.3389/fneur.2017.00185

84. Zhang Q, Aldridge GM, Narayanan NS, Anderson SW, Uc EY. Approach to cognitive impairment in Parkinson's disease. Neurotherapeutics. (2020)17:1495-510. doi: 10.1007/s13311-020-00963-x

85. Tyvaert L, Houdayer E, Devanne H, Monaca C, Cassim F, Derambure P.The effect of repetitive transcranial magnetic stimulation on dystonia: a clinical and pathophysiological approach. Neurophysiologie Clinique/Clin Neurophysiol. (2006) 36:135-43. doi: 10.1016/j.neucli.2006.08.007

86. Quartarone A, Rizzo V, Terranova C, Cacciola A, Milardi D, Calamuneri A,et al. Therapeutic use of non-invasive brain stimulation in dystonia. Front Neurosci. (2017) 11:423. doi: 10.3389/fnins.2017.00423

87. Sharma K, Cucca A, Lee A, Agarwal S, Frucht SJ, Biagioni MC. Transcranial magnetic stimulation therapy for focal leg dystonia: a case report. J Clin Movement Disord. (2019) 6:1-4. doi: 10.1186/s40734-019-0076-z

88. Huang YZ, Edwards MJ, Bhatia KP, Rothwell JC. One-Hz repetitive transcranial magnetic stimulation of the premotor cortex alters reciprocal inhibition in DYT1 dystonia. Movement Disord. (2004) 19:54-9.doi: 10.1002/mds.10627

89. Lozeron P, Poujois A, Richard A, Masmoudi S, Meppiel E, Woimant F, et al.Contribution of TMS and rTMS in the understanding of the pathophysiology and in the treatment of dystonia. Front Neural Circuits. (2016) 10:90.doi: 10.3389/fncir.2016.00090

90. Erro R, Tinazzi M, Morgante F, Bhatia KP. Non-invasive brain stimulation for dystonia: therapeutic implications. Eur J Neurol. (2017) 24:1228-e64.doi: 10.1111/ene.13363

91. Siebner HR, Filipovic SR, Rowe JB, Cordivari C, Gerschlager W, Rothwell JC, et al. Patients with focal arm dystonia have increased sensitivity to slow-frequency repetitive TMS of the dorsal premotor cortex. Brain. (2003) 126:2710-25. doi:10.1093/brain/awg282

92. Kimberley TJ, Borich MR, Arora S, Siebner HR. Multiple sessions of low-frequency repetitive transcranial magnetic stimulation in focal hand dystonia: clinical and physiological effects. Restor Neurol Neurosci. (2013)31:533-42. doi: 10.3233/RNN-120259

93. Medina F, Tunez I. Huntington's disease: the value of transcranial meganetic stimulation. Curr Med Chem. (2010) 17:2482-91.doi: 10.2174/092986710791556078

94. Brown KE, Neva JL, Ledwell NM, Boyd LA. Use of transcranial magnetic stimulation in the treatment of selected movement disorders. Degener Neurol Neuromusc Dis. (2014) 4:133. doi: 10.2147/DNND.S70079

95. Taber MT, Fibiger HC. Electrical stimulation of the prefrontal cortex increases dopamine release in the nucleus accumbens of the rat: modulation by metabotropic glutamate receptors. J Neurosci. (1995) 15:3896-904. doi: 10.1523/JNEUROSCI.15-05-03896.1995

96. You Z-B, Tzschentke TM, Brodin E, Wise RA. Electrical stimulation of the prefrontal cortex increases cholecystokinin, glutamate, and dopamine release in the nucleus accumbens: an in vivo microdialysis study in freely moving rats. J Neurosci. (1998) 18:6492-500.doi: 10.1523/JNEUROSCI.18-16-06492.1998

97. Klöppel S, Draganski B, Siebner HR, Tabrizi SJ, Weiller C, Frackowiak RS. Functional compensation of motor function in pre-symptomatic Huntington's disease. Brain. (2009) 132:1624-32. doi: 10.1093/brain/awp081

98. Brusa L, Versace V, Koch G, Bernardi G, Iani C, Stanzione P, et al.Improvement of choreic movements by 1Hz repetitive transcranial magnetic stimulation in Huntington's disease patients. Annals Neurol. (2005) 58:655-6.doi: 10.1002/ana.20613

99. Shukla A, Jayarajan RN, Muralidharan K, Jain S. Repetitive transcranial magnetic stimulation not beneficial in severe choreiform movements of Huntington disease. J ECT. (2013) 29:e16-7.doi: 10.1097/YCT.0b013e3182711dfc

100. Groiss SJ, Netz J, Lange HW, Buetefisch CM. Frequency dependent effects of rTMS on motor and cognitive functions in Huntington's disease. Basal Ganglia. (2012) 2:41-8. doi: 10.1016/j.baga.2011.12.001

101. Klöppel S, Draganski B, Siebner HR, Tabrizi SJ, Weiller C, Frackowiak RS. Functional compensation of motor function in pre-symptomatic Huntington's disease. Brain. (2009) 132:1624-32. doi: 10.1093/brain/awp081

102. Khalifa N, Edebol Eeg-Olofsson K. Low-frequent Repetitive Transcranial Magnetic Stimulation (rTMS) in adolescents with Tourette syndrome. J Neonatal Pediatr Med. (2017) 3:S1013. doi: 10.4172/2572-4983.1000S1013

103. Kleimaker M, Kleimaker A, Weissbach A, Colzato LS, Beste C, Bäumer T,et al. Non-invasive brain stimulation for the treatment of gilles de la tourette syndrome. Front Neurol. (2020) 11:1539. doi: 10.3389/fneur.2020.592258

104. Grados M, Huselid R, Duque-Serrano L. Transcranial magnetic stimulation in Tourette syndrome: a historical perspective, its current use and the influence of comorbidities in treatment response. Brain Sci. (2018) 8:129.doi: 10.3390/brainsci8070129

105. Chae J-H, Nahas Z, Wassermann E, Li X, Sethuraman G, Gilbert D,et al. A pilot safety study of repetitive transcranial magnetic stimulation (rTMS) in Tourette's syndrome. Cogn Behav Neurol. (2004) 17:109-17.doi 10.1097/01.wnn.0000116253.78804.3ª

106. Frey J, Hess CW, Kugler L,WajidM,Wagle Shukla A. Transcranial magnetic stimulation in tremor syndromes: pathophysiologic insights and therapeutic role. Front Neurol. (2021) 2021:1482. doi: 10.3389/fneur.2021.700026

107. Shih LC, Pascual-Leone A. Non-invasive brain stimulation for essential tremor. Tremor Other Hyperkin Movements. (2017) 7:458. doi: 10.5334/tohm.377

108. Reis J, Swayne O, Vandermeeren Y, Camus M, Dimyan M, Harris-Love M,et al. Contribution of transcranial magnetic stimulation to the understanding of cortical mechanisms involved in motor control. J Physiol. (2008) 586:325-51. doi: 10.1113/jphysiol.2007.144824

109. Kang N, Cauraugh JH. Does non-invasive brain stimulation reduce essential tremor? A systematic review and meta-analysis. PLoS ONE. (2017)12:e0185462. doi: 10.1371/journal.pone.0185462

110. Bologna M, Rocchi L, Leodori G, Paparella G, Conte A, Kahn N, et al. Cerebellar continuous theta burst stimulation in essential tremor. Cerebellum. (2015) 14:133-41. doi: 10.1007/s12311-014-0621-0

111. Bologna M, Di Biasio F, Conte A, Iezzi E, Modugno N, Berardelli A. Effects of cerebellar continuous theta burst stimulation on resting tremor in Parkinson's disease. Parkinson Relat Disord. (2015) 21:1061-6.doi: 10.1016/j.parkreldis.2015.06.015

112. Weiler M, Stieger KC, Long JM, Rapp PR. Transcranial magnetic stimulation in Alzheimer's disease: are we ready? Eneuro. (2020) 7.doi: 10.1523/ENEURO.0235-19.2019

113. Guerra A, Assenza F, Bressi F, Scrascia F, Del Duca M, Ursini F, et al.Transcranial magnetic stimulation studies in Alzheimer's disease. Int J Alzheimers Dis. (2011) 2011:263817. doi: 10.4061/2011/263817

114. Lee J, Lee AY. Transcranial magnetic stimulation parameters as neurophysiological biomarkers in Alzheimer's disease. Annals Clin Neurophysiol. (2021) 23:7-16. doi: 10.14253/acn.2021.23.1.7

115. Makowiecki K, Harvey AR, Sherrard RM, Rodger J. Low-intensity repetitive transcranial magnetic stimulation improves abnormal visual cortical circuit topography and upregulates BDNF in mice. J Neurosci. (2014) 34:10780-92.doi: 10.1523/JNEUROSCI.0723-14.2014

116. Tokay T, Holl N, Kirschstein T, Zschorlich V, Köhling R. High-frequency magnetic stimulation induces long-term potentiation in rat hippocampal slices. Neurosci Lett. (2009) 461:150-4. doi: 10.1016/j.neulet.2009.06.032

117. Kumar S, Zomorrodi R, Ghazala Z, Goodman MS, Blumberger DM, Cheam A, et al. Extent of dorsolateral prefrontal cortex plasticity and its association with working memory in patients with Alzheimer disease. JAMA Psychiatry. (2017) 74:1266-74. doi: 10.1001/jamapsychiatry.2017.3292\

118. LenzM, Galanis C,Müller-Dahlhaus F, Opitz A,Wierenga CJ, Szabó G, et al. Repetitive magnetic stimulation induces plasticity of inhibitory synapses. Nat Commun. (2016) 7:1-13. doi: 10.1038/ncomms10020

119. Holczer A, Németh VL, Vékony T, Vécsei L, Klivényi P, Must A. Noninvasive brain stimulation in Alzheimer's disease and mild cognitive impairment—a state-of-the-art review on methodological characteristics and stimulation parameters. Front Hum Neurosci. (2020) 14:179.doi: 10.3389/fnhum.2020.00179

120. Wang X, Mao Z, Ling Z, Yu X. Repetitive transcranial magnetic stimulation for cognitive impairment in Alzheimer's disease: a meta-analysis of randomized controlled trials. J Neurol. (2020) 267:791-801.doi: 10.1007/s00415-019-09644-y

121. Lin Y, JiangW-J, Shan P-Y, LuM,Wang T, Li R-H, et al. The role of repetitive transcranial magnetic stimulation (rTMS) in the treatment of cognitive impairment in patients with Alzheimer's disease: a systematic review and meta-analysis. J Neurol Sci. (2019) 398:184-91. doi: 10.1016/j.jns.2019.01.038

122. Centonze D, Petta F, Versace V, Rossi S, Torelli F, Prosperetti C, et al. Effects of motor cortex rTMS on lower urinary tract dysfunction in multiple sclerosis. Multiple Scler J. (2007) 13:269-71. doi: 10.1177/1352458506070729

123. Elzamarany E, Afifi L, El-Fayoumy NM, Salah H, Nada M. Motor cortex rTMS improves dexterity in relapsing-remitting and secondary progressive multiple sclerosis. Acta Neurologica Belgica. (2016) 116:145-50.doi: 10.1007/s13760-015-0540-y

124. Koch G, Rossi S, Prosperetti C, Codecà C, Monteleone F, Petrosini L, et al.Improvement of hand dexterity following motor cortex rTMS in multiple sclerosis patients with cerebellar impairment.Multiple Scler J. (2008) 14:995-8. doi: 10.1177/1352458508088710

125. Mori F, Codecà C, Kusayanagi H, Monteleone F, Boffa L, Rimano A, et al. Effects of intermittent theta burst stimulation on spasticity in patients with multiple sclerosis. Eur J Neurol. (2010) 17:295-300.doi: 10.1111/j.1468-1331.2009.02806.x

126. Liu M, Fan S, Xu Y, Cui L. Non-invasive brain stimulation for fatigue in multiple sclerosis patients: a systematic review and meta-analysis. Multiple Scler Relat Disord. (2019) 36:101375. doi: 10.1016/j.msard.2019.08.017

127. Boutière C, Rey C, Zaaraoui W, Le Troter A, Rico A, Crespy L, et al.Improvement of spasticity following intermittent theta burst stimulation in multiple sclerosis is associated with modulation of resting-state functional connectivity of the primary motor cortices.Multiple Scler J. (2017) 23:855-63.doi:10.1177/1352458516661640

128. Korzhova J, Bakulin I, Sinitsyn D, Poydasheva A, Suponeva N, ZakharovaM, et al. High-frequency repetitive transcranial magnetic stimulationand intermittent theta-burst stimulation for spasticity management in secondary progressive multiple sclerosis. Eur J Neurol. (2019) 26:680-e44.doi: 10.1111/ene.13877

129. Chen R, Spencer DC, Weston J, Nolan SJ. Transcranial magnetic stimulation for the treatment of epilepsy. Cochr Database Syst Rev. (2016)2016:CD011025. doi: 10.1002/14651858.CD011025.pub2

130. HsuW-Y, Cheng C-H, LinM-W, Shih Y-H, Liao K-K, Lin Y-Y. Antiepileptic effects of low frequency repetitive transcranial magnetic stimulation: a metaanalysis.Epilep Res. (2011) 96:231-40. doi: 10.1016/j.eplepsyres.2011.06.002

131. Mahajan UV, Parker JJ, Williams NR, Bhati MT, Ku S, Grant G, et al.Adjunctive repetitive transcranial magnetic stimulation delivers superior quality of life for focal epilepsy compared to anti-epileptic drugs: a meta-analytic utility prediction study. Brain Stimul. (2020) 13:430-2.doi: 10.1016/j.brs.2019.12.006

132. Farzan F,Wu Y,Manor B, Anastasio EM, LoughM, Novak V, et al. Cerebellar TMS in treatment of a patient with cerebellar ataxia: evidence from clinical, biomechanics and neurophysiological assessments. Cerebellum.(2013) 12:707-12. doi: 10.1007/s12311-013-0485-8

133. Manor B, Greenstein PE, Davila-Perez P, Wakefield S, Zhou J, Pascual-Leone A. Repetitive transcranial magnetic stimulation in spinocerebellar ataxia: a pilot randomized controlled trial. Front Neurol. (2019) 10:73.doi: 10.3389/fneur.2019.00073

134. Pauly MG, Steinmeier A, Bolte C, Hamami F, Tzvi E, Münchau A, et al.Cerebellar rTMS and PAS effectively induce cerebellar plasticity. Sci Rep.(2021) 11:1-13. doi: 10.1038/s41598-021-82496-7

135. Schwenkreis P, Tegenthoff M, Witscher K, Börnke C, Przuntek H, Malin JP, et al. Motor cortex activation by transcranial magnetic stimulation in ataxia patients depends on the genetic defect. Brain. (2002) 125:301-9.doi: 10.1093/brain/awf023

136. Groiss SJ, Ugawa Y. Cerebellar stimulation in ataxia. Cerebellum. (2012)11:440-2. doi: 10.1007/s12311-011-0329-3

137. Shiga Y, Tsuda T, Itoyama Y, Shimizu H, Miyazawa K, Jin K,et al. Transcranial magnetic stimulation alleviates truncal ataxia in spinocerebellar degeneration. J Neurol Neurosurg Psychiatry. (2002)72:124-6. doi: 10.1136/jnnp.72.1.124

138. Benussi A, Pascual-Leone A, Borroni B. Non-invasive cerebellar stimulation in neurodegenerative ataxia: a literature review. Int J Mol Sci. (2020) 21:1948. doi: 10.3390/ijms21061948

139. Ugawa Y, Shimo Y, Terao Y. Future of tanscranial magnetic stimulation in movement disorders: introduction of novel methods. J Movement Disord. (2020) 13:115. doi: 10.14802/jmd.19083