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Benefits from Theta Burst Stimulation in Post-stroke Rehabilitation: A Narrative Review

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Abstract

Stroke is an acute neurovascular central nervous system injury and often leads to neuronal death and permanent dysfunction. Its symptoms cover a variety of domains, such as motor dysfunction, unilateral neglect, aphasia, dysphagia, and cognitive impairment. As part of rehabilitation, an important task in post-stroke recovery is to relearn lost skills and regain as much independence as possible. Non-invasive brain stimulation, such as Theta burst stimulation (TBS), shows the potential to modify human brain plasticity for the rehabilitation of post-stroke patients. TBS is a patterned repetitive transcranial magnetic stimulation (rTMS) produced on conventional rTMS, which has been increasingly used in clinical rehabilitation in recent years, since its first application in 2005. The ability of TBS to affect brain function in post-stroke patients in terms of molecular biology and neurophysiology has been demonstrated in relevant basic research studies. This article provides a review of the therapeutic principles of TBS and its clinical application in post-stroke rehabilitation, offering clinical reference.

Keywords

Post-stroke, Theta burst stimulation, rehabilitation

1. Introduction

Stroke has become one of the leading causes of death and disability worldwide, according to statistics from the World Health Organization [1]. It causes various issues, including motor dysfunction, unilateral neglect, aphasia, dysphagia, and cognitive impairment. After 4 years of the onset of the disease, over 30% of patients still experienced difficulty participating autonomously or fulfilling social roles [2]. Continuously updated and iterative rehabilitation technologies such as TBS, offer more possibilities for optimal rehabilitation outcomes [3]. TBS is a type of conventional rTMS, characterized by transmitting a stimulation frequency of 50 Hz every 200ms, simulating a rhythm similar to that of θ. Previous studies have shown it to be a non-invasive brain stimulation (NIBS) with longer-lasting and more stable neural response effects [4]. Since its first application to modulate human motor cortical activity in 2005 [5], TBS has been gradually being used from the laboratory to the clinic to improve various post-stroke dysfunctions.

2. TBS

2.1 Types of TBS

TBS consists mainly of two types: intermittent TBS rapidly induces the excitation of neural functions and continuous TBS induces the inhibition of neural functions, increasing or decreasing the excitability of subcortical spinal

neurons at the site of stimulation, respectively [6]. TBS has a lower incidence of side effects compared to the traditional rTMS treatment paradigm and requires only a lower stimulus intensity and shorter stimulus duration to induce changes in cortical excitability equivalent to the traditional rTMS treatment paradigm and subsequent longer cortical excitatory effects.

2.2 Biochemistry of TBS

The structural integrity of the blood-brain barrier (BBB) is compromised after stroke [7, 8], leading to dysregulated solute transport and release of inflammatory cytokines driving peripheral immune cell infiltration [9]. This immune response may be beneficial in the early post-stroke phase, but the long-term destruction of the BBB and the subsequent neuroinflammatory response that occurs is detrimental to the repair process [10]. Studies show that TBS intervention can change gene expression pathways and improve neural remodeling and hemodialysis in post-stroke brain injury [11]. Aydin-Abidin et al. [12] demonstrated for the first time that acute rTMS specifically modulates the expression of zif268 and c-Fos in the rat cortex, depending on the mode of stimulation applied. Ljubisavljevic et al's [13] research found that long-term rTMS can positively affect the brain by promoting various changes in gene expression, including structural remodeling, neuroprotection, and neuronal plasticity. This can be a valuable tool in treating acute ischemia-reperfusion brain damage. Zong et al. [14] reported that TBS intervention significantly attenuated BBB permeability and protected the important BBB components ZO-1, claudin-5, occludin, and caveolin-1 from degradation, and ameliorated damage to vascular structure, morphology, and perfusion as well as local tissue oxygenation through alterations in gene expression products. This study reinforces the idea that TBS has a powerful protective effect against stroke.

2.3 Electrophysiology of TBS

The recovery of motor evoked potential (MEP) amplitude to baseline levels is considered to be a valid neurophysiological indicator of TBS-guided cortical plasticity. The pharmacological profile of changes in MEP amplitude induced by LTP and LTD of synaptic plasticity is similar to that of LTD or LTP establishment at glutamatergic synapses in animal studies, and to that of drugs that modulate key receptors or channels involved in synaptic plasticity, such as those that modulate the N-methyl-D-aspartate (NMDA) receptor and Ca^{2+} channels [15]. The extent and rate of postsynaptic Ca^{2+} changes may determine whether glutamatergic synapses are enhanced, inhibited, or remain unchanged [16]. LDP is thought to occur through a rapid increase in postsynaptic Ca+ inward flow, followed by an increase in gene and protein expression, whereas a small amount of slow flow of Ca^{2+} induces LTD by decreasing postsynaptic neuronal activity. Pharmacological studies have shown that TBS induces γ-aminobutyric acidergic and NMDA-type glutamatergic synaptic plasticity [17, 18]. The normal balance of interhemispheric inhibition is disrupted after stroke, and enhanced inhibition from the contralateral hemisphere impairs activation in the ipsilateral region, with the healthy hemisphere exerting excessive inhibition on the affected hemisphere. TBS intervention induced changes in expression products and synaptic plasticity, which ultimately led to changes in cortical excitability [19].

3. Motor dysfunction

3.1 Clinical application

TBS stimulation of the damaged primary motor cortex (M1) combined with conventional rehabilitation training is a common form of TBS in the rehabilitation of motor dysfunction, and it has been demonstrated that this intervention can improve the recovery of motor function in post-stroke patients-especially upper limb motor function, improve motor relearning ability, and reduce spasticity of the affected limb [20, 21]. Saikaley et al. [22] conducted a net meta-analysis including 6,781 participants enrolled in six randomized controlled trials examining 20 non-conventional interventions and demonstrated that TBS was one of the eight non-conventional interventions that were significantly superior to conventional interventions for improving motor dysfunction post-stroke.

Currently, TBS is directed at the restoration of motor function and balance in the lower extremities, with the primary stimulation site being the cerebellum. Compared to physical therapy alone, iTBS combined with physical therapy resulted in significant improvements in balance function and gait [23]. Koch et al. [24] conducted a 3-week randomized, double-blind, controlled trial with 36 patients who had hemiparesis due to continuous ischemic chronic stroke in the contralateral middle cerebral artery region. It is concluded that cerebellar intermittent theta-burst stimulation promotes gait and balance recovery in stroke patients by acting on cerebellar cortical plasticity.

3.2 Newer paradigm explorations

The clinical application of newer TBS modalities is also being explored, and one such modality is priming TBS, in which iTBS or cTBS is applied for cortical priming activation before formal TBS stimulation. A study by Zhang et al. [25] included 42 chronic stroke patients in a 3-week randomized controlled trial, dividing the patients into a priming iTBS group, a non-priming iTBS group, and a sham stimulation group. After the treatment session, the priming iTBS group showed a more significant improvement in Fugl-Meyer upper extremity scores than the non-priming iTBS group and the sham stimulation group, and this improvement was relatively reduced at subsequent follow-up; At the same time, primed iTBS enhanced the effect of mirror visual feedback training higher β-sensorimotor event-related potentials in the patient's ipsilateral showing that it can promote post-stroke motor learning ability by enhancing the sensitivity of ipsilateral sensorimotor regions to the treatment.

3.3 Newer site exploration

Researchers are also exploring the use of other stimulation sites in TBS, such as the dorsal premotor cortex (PMd) and premotor area (PMA). Studies have shown that iTBS stimulation of PMd affects M1 excitability, but a threshold needs to be identified at which PMd stimulation is more effective versus patients who are more effectively stimulated by the standard stimulation method (M1) [26]; Functional imaging evidence suggests that the ipsilateral PMA, a higher cortex for motor control in the reticulospinal tract (RST), plays a key role in ipsilateral movement [27]. Another researcher conducted a rehabilitation assessment of elbow/wrist flexor/extensor spasticity and upper limb motor function with the Modified Ashworth Scale and Fugl-Meyer Upper Extremity Assessment Scale, which demonstrated that iTBS stimulation of the PMA improved spasticity and upper limb motor function promptly after stroke [28].

4. Unilateral neglect

Unilateral neglect can be defined as deficits in perception, attention, and action on the contralateral side of the brain injury [29]. The model of reciprocal inhibition between the cerebral hemispheres and the plasticity of the central nervous system is the theoretical basis for noninvasive neuromodulation to improve unilateral neglect after stroke [30]. In recent years, a large number of randomized controlled trials have assessed the efficacy of TBS for unilateral neglect in multi-session design trials, and overall, unilateral neglect-related outcome indicators have improved [31]. Cazzoli et al. [32] applied cTBS to the left posterior parietal cortex of patients with subacute left-sided neglect for two consecutive days, in contrast to a sham stimulation group. The results showed a 37% improvement in the spontaneous daily behavior of neglected patients after repeated applications of cTBS, and this improvement persisted for at least 3 weeks after stimulation.

Most unilateral neglect programs for TBS interventions after stroke target the left parietal cortex. Cao et al. [33] applied iTBS intervention on DLPFC for 10 days. 80% of the resting motor threshold stimulation group showed a greater reduction in resting state functional connectivity strength compared to 40% of the resting motor threshold applied stimulation group, and the improvement in the line segmentation test and star elimination test was more significant. This study applied iTBS on the undamaged hemisphere, which seems to go against the interhemispheric competition model. However, the stimulation was performed on the DLPFC, a locus that is considered a key brain region involved in top-down attentional control. Excitatory stimulation of this region may enhance compensatory functions for visuospatial neglect independent of the interhemispheric competition model.

5. Aphasia

Aphasia is a common syndrome in which the dominant hemisphere is damaged post-stroke. Neuroimaging studies have shown a relevant basis for demonstrating that decreasing the excitability of the mirror language area in the nondominant hemisphere is one of the possible mechanisms of action in the rehabilitation of aphasia [34]. The posterior part of the inferior frontal gyrus (pIFG) in humans is recognized as part of the human mirror neuron system and plays a key role in mapping sensation to motor action. Most aphasia studies apply TBS to the right BA45 [35]. To assess whether cTBS improves naming performance in patients with aphasia at different stages after stroke, a randomized, sham-controlled, crossover trial of 18 patients with right-handed aphasia was tested on a picture-naming task and verbal-independent vigilance before and after the implementation of cTBS in the right Brodmann area 45 localized in the electroencephalogram system. Outcomes compared with the sham control group after the intervention, naming scores improved significantly, naming latency was significantly shorter, and patients in the subacute

post-stroke phase showed the greatest improvement.

Other investigators have explored the effect of iTBS stimulation of the left hemisphere pIFG region on post-stroke aphasia. Restle et al. [36] divided healthy subjects into three different intervention groups under the guidance of fMRI, the left pIFG for the iTBS group, the cTBS group, and the intermediate TBS (imTBS) group, to compare the speech repetition accuracy of sentences before and after the TBS intervention. The results showed that the accuracy of utterance repetition improved after iTBS, and to a lesser extent after imTBS, but remained unchanged after cTBS. Chouet al. [37] applied 10 sessions of iTBS to the left BA45, and the results showed significant improvement in the iTBS group in terms of conversational, descriptive, and expressive scores, as well as auditory comprehension, reading comprehension, and matching scores compared to the sham-stimulation group. Conversational, descriptive, and expressive scores characterize speech production, and matching scores characterize speech perception. The facilitation of left pIFG stimulation by iTBS in the above studies suggests that the human left hemisphere pIFG plays a relevant role in the repetition of speech perception to articulatory output and that the combination of repetitive speech training with iTBS of the left pIFG could be used to explore the treatment of post-stroke aphasia.

6. Dysphagia

From 37 to 78% of patients after stroke suffer from various degrees of dysphagia, which can induce serious complications such as dehydration, malnutrition, aspiration pneumonia, and electrolyte disorders. TBS is effective in improving post-stroke dysphagia, and in the current study, the TBS stimulation site was mostly chosen to be the supraglottic muscle group corresponding to the motor cortical area of the brain. Zhang et al. [38] investigated changes in the temporal dynamics of interregional connectivity induced by iTBS after cTBS in the contralateral supraspinatus cortex. The results showed that iTBS reversed the inhibitory effect of cTBS on the contralateral supraspinatus cortex.

Researchers have also explored the effectiveness and safety of cerebellar TBS for the treatment of post-stroke dysphagia. Rao et al. [39] randomized 70 patients with post-stroke dysphagia into a bilateral cerebellar iTBS group and a sham stimulation group, and swallowing function was assessed at baseline, 2 weeks post-intervention and 4 weeks of follow-up using fiberoptic endoscopic dysphagia severity scale (FEDSS); penetration aspiration scale (PAS); Standardized Swallowing Assessment (SSA); and Functional Oral Intake Scale (FOIS). It indicates that iTBS, as a more effective TMS stimulation modality, can effectively improve swallowing function by stimulating bilateral cerebellar hemispheres, and also reflects that 100% resting motor threshold bilateral cerebellar iTBS is a relatively safe treatment.

7. Cognitive impairment

Post-stroke cognitive impairment (PSCI) is a common post-stroke complication characterized by cognitive impairment, mainly in the form of impaired executive function [40]. It results in the loss of self-care, social participation, and work skills of the patients, placing a heavy burden on their families and society. At present, there is still a lack of effective treatments for PSCI. In theorized that pharmacological treatments such as acetylcholinesterase inhibitors may improve PSCI, however, studies have shown that such pharmacological treatments have only short-term benefits and are associated with varying degrees of side effects [41]. Cognitive training is a common clinical treatment for cognitive impairment, which can delay or improve cognitive decline through repeated training. Its efficacy varies from person to person, depending to some extent on the patient's level of commitment and cooperation. Researchers have explored the use of iTBS for the rehabilitation of cognitive deficits after stroke; however, so far there have been fewer studies on the rationale for the role of iTBS in improving cognitive function in terms of neuroplasticity, and the mechanisms are still unclear [42].

A study by Chu et al. [43] that randomly assigned 60 PSCI patients with post-stroke cognitive impairment to receive iTBS, tDCS, or cognitive training alone demonstrated that iTBS plus cognitive training improved cognitive functioning and quality of life in patients with PSCI compared to cognitive training alone. Li et al. [44] found that iTBS effectively and safely improved overall cognitive dysfunction, including semantic comprehension and executive function, and positively affected memory function. Tsai et al. [45] systematically examined the effectiveness of iTBS on post-stroke cognitive deficits through 2 protocols. The results showed significant improvements in overall cognitive, attentional, and memory functions in the iTBS group with post-treatment cognitive dysfunction compared with the sham stimulation group.

8. Prospects and shortcomings

As mentioned earlier TBS has the conventional functional characteristics of rTMS, as well as the unique advantages of shorter interventions and longer duration of action that it does not have, as well as the disadvantages of a relatively unclear mechanism of action and a higher rate of seizure induction. Individual variability in TBS-induced synaptic plasticity is large, and the relevant factors that cause individual variability include age, genetic polymorphisms, menstrual cycle, blood glucose level, and sleep quality [46]. Although a series of basic and clinical applications of TBS in the field of post-stroke rehabilitation have been explored, the number of large-scale and rigorous clinical trials in this field is small. The sample sizes included in the trials are small, and several small, less rigorously designed trials provide the only available data on the design of the TBS parameters and the selection of the intervention sites, so there are still many shortcomings in the evidence for TBS in post-stroke rehabilitation. The efficacy of the TBS intervention needs to be replicated by including a large number of patients in randomized clinical trials to further determine the potential of TBS to restore all functions of post-stroke.

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