

Advances in the Application of rTMS in the Rehabilitation of Post-Stroke Swallowing Dysfunction

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Abstract

Stroke is currently one of the main causes of death and disability worldwide, with numerous reports indicating that it can lead to various functional impairments such as motor, sensory, swallowing, cognitive, emotional, and speech disorders following its occurrence. Among these, swallowing disorder is a relatively severe complication, which can easily lead to malnutrition, reduced quality of life, and aspiration pneumonia in patients, severely affecting their daily life and the outcome of the disease. Repetitive transcranial magnetic stimulation (rTMS) is a non-invasive brain stimulation technique, and many studies have shown that it can significantly improve swallowing disorders after stroke. This article summarizes the possible pathogenesis of swallowing disorders after stroke and the mechanism of action of rTMS on the therapeutic effects for patients.

Keywords

Repetitive Transcranial Magnetic Stimulation, Swallowing Dysfunction after Stroke, Rehabilitation Treatment

1. Introduction

Dysphagia after stroke (DAS) is a condition characterized by symptoms such as coughing when drinking water, difficulty swallowing, and repeated swallowing during the swallowing process [1]. It can easily lead to complications in patients, including aspiration, coughing, malnutrition, electrolyte imbalance, and even

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life-threatening aspiration pneumonia [2]. In patients with stroke, approximately 52% to 77.4% experience dysphagia [3] [4]. However, most patients will recover oral feeding within 30 days after onset [5]. This indicates that early effective swallowing rehabilitation treatment is crucial for DAS patients. Current treatment measures for DAS patients include conventional swallowing function training, feeding training, physical manipulation therapy, acupuncture, etc., in addition to which, a large body of research has demonstrated that repetitive transcranial magnetic stimulation (rTMS) can significantly improve the clinical symptoms of patients with dysphagia. In this article, we will discuss the pathogenesis of dysphagia, as well as the progress in research on the application of rTMS in DAS.

2. The Pathogenesis of Swallowing Dysfunction after Stroke

2.1. Damage to the Central Neural Network

Swallowing is a process of fine muscular activity that requires multiple sets of muscles to be activated in different sequences and with precise timing and force, such as opening the mouth on demand, pushing the tongue backward to deliver food, contracting of the cricopharyngeal muscle groups, airway closure, and opening of the esophageal sphincter. Errors in any of these stages can result in the patient developing dysphagia. This includes the control of swallowing movements by the brainstem swallowing center, the cerebral cortex, subcortical fibers, and the extrapyramidal system. The brainstem swallowing centers include not only the coordinated motor abilities and sensory inputs from multiple pairs of cerebral nerves to the swallowing muscles [6], but also the medullary swallowing centers that integrate and process the information from cerebral nerves and supramedullary inputs to elicit a swallowing response [7]; therefore, lesions of the brainstem are critical to the control of swallowing. The cortical swallowing centers of the brain play a key role in the initiation of swallowing [8], and a large number of studies now suggest that cortical swallowing centers are present in the frontal lobe, parietal lobe, temporal lobe, and anterior cingulate gyrus [9]. Dysfunction of many subcortical regions can also lead to dysphagia, such as the thalamus, the internal capsule, the insula, and the amygdala, which can also participate in the process of swallowing movements through the extrapyramidal system [10] [11]. Cerebellar and extrapyramidal damage may also cause swallowing dysfunction, which may be caused by dystonia in the muscles corresponding to swallowing movements [12].

2.2. Sensory System Damage

A complete sensory input-output link is essential during swallowing, and sensory decline in the oral cavity and pharynx may have difficulty in inducing the production of the swallowing reflex [13], and the input of sensory stimuli also causes an increase in the excitability of the corresponding areas of the cerebral cortex, which demonstrates the inextricable link between movement and sensation, and that sensory deficits have a significant impact on swallowing movements [14].

3. Hypothesis of the Principle of Repetitive Transcranial Magnetic Stimulation

rTMS is a non-invasive brain stimulation that consists of the passage of an electric current through a coil placed on the scalp, generating an electromagnetic field perpendicular to the coil, which can be independent of the soft tissues and bones of the scalp, and generating an induced current in the nerve cells of the corresponding cerebral cortex as a means of producing an activating or inhibiting effect on the cells in accordance with Faraday's Law of Electromagnetic Induction [15]. Based on the frequency of stimulation, it can be categorized into high-frequency stimulation (5 Hz HFrTMS) and above and low-frequency stimulation (≤ 1 Hz; LF-rTMS), where high-frequency stimulation increases cortical excitability and low-frequency stimulation decreases cortical excitability, and this effect persists for a period of time after stimulation is discontinued, and manifests itself mainly as long time period potentiation (LTP) and long time period depression (LTD) [16]. Based on this mechanism of action, there are many different hypotheses as to how it acts on the brain.

3.1. Interhemispheric Inhibition Model

The interhemispheric inhibition (IHI) model refers to the fact that the two cerebral hemispheres are connected via the corpus callosum [17] which not only transmits information directly to each other between the two hemispheres, but also dynamically regulates their reciprocal inhibitory or excitatory effects to a dynamic equilibrium, and after a stroke, this inhibitory equilibrium is broken, which will cause the healthy hemisphere's inhibition of the affected hemisphere to be enhanced while the affected hemisphere's inhibition of the healthy hemisphere to be weakened, leading to dysphagia [18]. Based on this theory, a large number of studies have demonstrated that the balance can be maintained by inhibiting the excitability of the healthy hemisphere or increasing the excitability of the affected hemisphere, thus improving the symptoms of post-stroke dysphagia.

3.2. Healthy Side Compensation Model

After stroke, the brain will organize the nerves around the damaged area to re-establish new circuits, which is mainly manifested in the re-awakening of the nerves in the uninjured area, connection, and the occurrence of new dendrites and spines [19]. When the swallowing center on the affected side is destroyed, the remodeling ability of the remaining neurons cannot meet the functional recovery needs, the healthy side of the swallowing center will compensate for the increase in the neural pathways of the region, in order to improve the recovery of swallowing function. When the affected side is the healthy side of the swallowing center, the healthy side will increase the neural pathways of the region, so as to improve the symptoms of dysphagia and function recovery, and when the affected side is the dominant hemisphere of the swallowing center, it will also promote the transfer of the dominant hemisphere to the healthy side [20].

3.3. Bimodal Equilibrium-Restoration of Equilibrium Model

Di Pino G [18] argued that the interhemispheric connection as inhibition or compensation after stroke is related to the neural pathways that survived the stroke and the reserve of neural remodeling in the region, and that the healthy-side compensatory model may be more helpful for functional recovery if the surviving tissues and reserve of the lesion area are low, and the interhemispheric inhibitory model may be more helpful for functional recovery if the lesion area is sufficiently well endowed with the neural pathways and reserves, which has implications for this will help us to develop individualized plans for rTMS, but this model needs more imaging and neurological examinations to monitor and judge before treatment, so as to develop individual treatment plans, and there are fewer studies in this direction now.

4. Repetitive Transcranial Magnetic Stimulation Mechanism

4.1. Regulation of Neuroplasticity

After stroke, neuroplasticity is the basis for the recovery of brain function, and brain plasticity will follow a fixed time line and also increase in a limited period of time [21], and rTMS is one of the factors that regulate the enhancement process, and high-frequency repetitive transcranial magnetic stimulation (HF-rTMS) will elevate the excitability of the cerebral cortex, and when the cerebral cortex excitability is elevated, it will be more favorable for this process to take place [22], therefore, in the recovery period of stroke, rTMS can be used to enhance neuroplasticity, promote the compensation of the cerebral cortex, improve central nerve conduction, and help the recovery of swallowing function [23].

4.2. Regulation of Neurotransmitters

After stroke, the release of neurotransmitters will be affected [24], which will cause a series of clinical symptoms, including cognition, memory, motor, swallowing dysfunction, etc. Therefore, regulating the release of neurotransmitters and the expression of the corresponding genes can improve the swallowing dysfunction after stroke, and the study of Ikeda T [25] showed that rTMS can regulate the mRNA expression level of some neurotransmitter genes in the mouse brain, thereby regulating the neurotransmitter expression level, thereby regulating neurotransmitter secretion levels and affecting neuronal activity. At the same time, studies have shown that rTMS can also affect the release level of cholinergic neurons [26], which has a more obvious effect on improving the excitability of neurons. In addition, rTMS also has a more significant effect on some other neurotransmitters, such as brain neurotrophic factor [27], which can improve post-stroke dysfunction.

4.3. Regulation of Glial Cells

Astrocytes are the most abundant glial cells in the body, and they are activated in two different phenotypes after stroke, type A1 and type A2. Type A1 astrocytes

secrete inflammatory cytokines and neurotoxic mediators, which trigger neuroinflammation and further aggravate craniocerebral injuries after stroke, while type A2 astrocytes secrete anti-inflammatory cytokines and nerve growth factors to promote nerve regeneration, in order to protect neurological function. Studies have shown that rTMS inhibits the production of type A1 astrocytes [28], promotes the transformation of type A1 astrocytes to type A2 and increases the secretion of trophic factors by type A2 astrocytes [29]. Therefore, rTMS can alleviate the neuroinflammatory response, minimize the exacerbation of brain injury, and promote the regeneration of the nerves to assist in post-stroke rehabilitation.

4.4. Regulation of Inflammatory Cytokines

The secretion of inflammatory cytokines will be rapidly activated after stroke, which includes two types: pro-inflammatory cytokines such as IL-1, IL-6, IL-11, IL-17, IL-23 and interferon- γ (IFN- γ), tumor necrosis factor- α (TNF- α), etc., and anti-inflammatory cytokines, including IL-4, IL-6, IL-10 and TGF- β , etc. [30]. Among them, IL-6 has both anti-inflammatory and pro-inflammatory properties [31]. rTMS can simultaneously inhibit the secretion of pro-inflammatory cytokines and promote the production of anti-inflammatory cytokines after stroke [32]-[35], thus reducing the damage of inflammation and promoting tissue repair after stroke. At the same time, pro-inflammatory cytokines can also affect synaptic plasticity, thereby influencing neurological recovery after stroke [36].

5. Differences and Advantages Over Traditional Treatments

Conventional swallowing treatments such as routine swallowing exercises, neuromuscular electrical stimulation, and transcranial direct current stimulation can, to some extent, promote the recovery of various dysfunctions after stroke. However, these rehabilitation programs also have some drawbacks, such as slow onset of action, time-consuming, and poor patient compliance [37]. rTMS, on the other hand, is superior to conventional therapies in terms of onset time, efficacy, long-term effect, and relief of complications. Clinical data show that 10 Hz high-frequency rTMS can increase the amplitude of motor evoked potentials (MEP) in the swallowing cortex by 30% - 50%, with a sustained effect of 3 - 6 months, which cannot be achieved by peripheral electrical stimulation [38]. It has also been demonstrated that rTMS synchronizes the activation of the insula-basal ganglia-cerebellar loop (a key swallowing neural network), whereas conventional methods only improve a single link [39]. In terms of breadth of applicability, in brainstem stroke patients, rTMS can still produce efficacy through cortical remodeling when NMES fails due to laryngeal return nerve injury [40]. Studies have shown that rTMS has a positive effect in terms of time to effect, rate of reduction of osmotic malabsorption, improvement in FOIS score, and 6-month recurrence rate [41] [42].

6. Contraindications and Limitations

Many studies have proved that rTMS is a safe and effective treatment, but there

are some side effects, such as pins-and-needles pain at the site of stimulation or headache, a small number of patients experience mild sleep disturbances and mood changes after the treatment, and a part of the case studies also suggest that rTMS may induce epilepsy, but this can be avoided as much as possible by following the safety guidelines to avoid the development of such side effects [43]. The clinical outcomes of rTMS are currently being evaluated. The clinical efficacy of rTMS has been confirmed by many case studies and feasibility studies, but there is still a lack of studies such as sham-controlled experiments and proof-of-principle studies of clinical efficacy with the same types of stimuli [44].

7. Summary and Outlook

rTMS is a highly precise and safe treatment that allows good control of the frequency, intensity, and location of stimulation [45]. rTMS should probably be applied early in the history of the disease for more relevant therapeutic effects, since post-stroke swallowing dysfunction usually recovers rapidly. This paper describes the mechanism of generation of post-stroke dysphagia and the principle of action of rTMS, which may help in the selection of treatment options for patients in the clinic. Although the current study proves that rTMS can help in post-stroke rehabilitation, the specific stimulation parameters and sites are not standardized and the positioning of stimulation in the treatment varies from person to person. Future studies should focus on identifying the optimal rTMS protocol and incorporating multicenter, randomized controlled trials to verify its efficacy. In addition, further studies are needed to investigate the impact of rTMS on long-term prognosis, such as the 6-month recurrence rate. Overall, rTMS, as a potential treatment for post-stroke dysphagia, has shown some advantages in improving swallowing function and related indicators, but more high-quality studies are needed to support its clinical application [46]. Meanwhile, some studies have suggested the use of imaging and 3D localization with neuronavigation technology as a way to precisely stimulate the desired site, but this option is cumbersome, and large-sample, multicenter studies should be conducted in the future to determine and provide more authoritative treatment options to choose from.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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