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REVIEW ARTICLE

Application of noninvasive brain stimulation for post-stroke dysphagia rehabilitation

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Abstract Noninvasive brain stimulation (NIBS), commonly consisting of transcranial magnetic stimulation (TMS), transcranial direct-current stimulation (tDCS), as well as paired associative stimulation (PAS), has attracted increased interest and been applied experimentally in the treatment of post-stroke dysphagia (PSD). This review presented a synopsis of the current research for the application of NIBS on PSD. The intention here was to understand the current research progress and limitations in this field and to stimulate potential research questions not yet investigated for the application of NIBS on patients with PSD. Here we successively reviewed advances of repetitive TMS (rTMS), tDCS, and PAS techniques on both healthy participants and PSD patients in three aspects, including scientific researches about dysphagia mechanism, applied studies about stimulation parameters, and clinical trials about their therapeutic effects. The techniques of NIBS, especially rTMS, have been used by the researchers to explore the different mechanisms between swallowing recovery and extremity rehabilitation. The key findings included the important role of intact hemisphere reorganization for PSD recovery, and the use of NIBS on the contra-lesional side as a therapeutic potential for dysphagia rehabilitation. Though significant results were achieved in most studies by using NIBS on swallowing rehabilitation, it is still difficult to draw conclusions for the efficacy of these neurostimulation techniques, considering the great disparities between studies.

Conflicts of interest: All authors declare no conflicts of interest.

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Introduction

Dysphagia, a swallowing disorder, can be divided into oropharyngeal dysphagia and esophageal dysphagia based on the different stages of deglutition. Oropharyngeal dysphagia, resulting from either oropharyngeal swallowing dysfunction or perceived difficulty in the process of swallowing, is usually a manifestation of a systemic disease rather than a disease specific to the oropharynx [1]. Stroke is a representative cause of oropharyngeal dysphagia, and in acute stroke, the prevalence of dysphagia has been reported as being between 37% and 78% [2]. Post-stroke dysphagia (PSD), which is a common medical complication that affects many patients in the first few hours and days after ictus, is associated with increased mortality and morbidity, partially due to aspiration, pneumonia, and malnutrition [3]. In most patients, PSD can improve spontaneously; however, in approximately 11—50% of patients, it is a long-term disability [2,3].

The primary goal of treatment for dysphagia after stroke is to improve the amount and variety of food and liquid which are swallowed orally while minimizing the risk of aspiration and related complications. The currently used treatment methods for PSD include posture training, dietary modifications, swallowing exercises, drug therapy, oromotor stimulation, neuromuscular electrical stimulation, botulinum toxin injection, and noninvasive brain stimulation (NIBS) [4].

As a powerful method to modulate human brain function, NIBS commonly consists of transcranial magnetic stimulation (TMS), transcranial direct-current stimulation (tDCS), and paired associative stimulation (PAS). PAS is derived by combining peripheral stimulation to the targeted muscle with TMS or tDCS over the representational area of that muscle in the motor cortex [5,6]. The reasons why NIBS can be utilized for the PSD rehabilitation mainly include the following: first, PSD has been believed to be associated with damage to the cortex and subcortical structures, including, but not limited to, the lower motor neurons of the swallowing center in the brainstem; second, cortical reorganization, known as neuroplasticity [7], which could be purposefully modulated by NIBS, as described below, leads to swallowing recovery. Although the application of NIBS exhibited synergistic effects over time [4], it is still unreliable for proposing any specific recruitment criteria due to the limited number of well-designed, long-term follow-up studies. Basically, the successful implementation of these techniques as interventional strategies will rely on an improved understanding of the underlying neuronal correlates of functional recovery [6].

In this paper, we sequentially review the progress made by utilizing TMS, tDCS, and PAS on healthy participants and PSD patients, with the goals of investigating whether NIBS has brought light to the mechanism research of PSD, verifying whether the usage of NIBS on PSD rehabilitation has shown satisfactory results, and determining whether some methodological limitations remain in need of further investigation.

Utilization of TMS on PSD

Approximately three decades ago, Barker et al. [8] demonstrated that it was possible to stimulate both nerves and the brain using external magnetic stimulation. TMS then started to be used in clinical neurology to study the central motor conduction time. Depending on the stimulation parameters, TMS can excite or inhibit the brain, thus allowing the functional mapping of cortical regions and the creation of transient functional lesions [9]. Compared with single-pulse TMS, which can depolarize neurons and evoke measurable effects, trains of stimuli (repetitive TMS, rTMS) can provide novel insights into the pathophysiology of the neural circuitry, which have been widely utilized in the areas of motor and speech recovery [10]. The application of TMS on dysphagia stroke can be summarized in the following three aspects.

The mechanistic research of PSD using TMS

When discussing research on the mechanisms of dysphagia, it is necessary to mention Dr. Hamdy et al. [11] who were the first to use TMS on this subject. They used TMS in 20 healthy participants, two decades ago, to describe the physiological characteristics of the corticofugal pathways to swallowing muscles [11]. They found, for the first time, that the muscles involved in swallowing appeared to be represented bilaterally on the precentral cortex, which displayed interhemispheric asymmetry, independent of handedness. These findings proved that the cortex plays an important role in regulating the brainstem swallowing program.

A year later, they published additional experimental results on this subject [12]. To acquire cortical stimulations, TMS was used on 20 post-stroke patients with or without dysphagia for the first time. The authors found that the PSD patients had smaller pharyngeal responses on the unaffected hemisphere than did patients who retained normal swallowing. This result was consistent with their prior finding of the presence of interhemispheric asymmetry with the swallowing motor function and suggested that dysphagia after unilateral hemispheric stroke was related to the magnitude of pharyngeal motor representation in the unaffected hemisphere.

Based on these results, Hamdy et al. [13] speculated that the recovery of swallowing in PSD patients could be explained by the compensatory reorganization of swallowing function in the intact hemisphere rather than the restoration of swallowing function in the damaged hemisphere, and they performed another clinical study to verify this speculation. After 3 months of follow-up for 28 patients who had a unilateral hemispheric stroke, the researchers demonstrated that the cortical map representation of the pharyngeal musculature in the undamaged hemisphere increased markedly in size in the PSD patients who recovered swallowing, but that there was no change in patients who had persistent dysphagia or who did not have dysphagia throughout. These observations verified the speculation that the recovery of PSD may be dependent on compensatory strategies of cortical reorganization, through neuroplastic changes, which can mainly be observed in the undamaged hemisphere. By using the TMS technique, these findings on the mechanism of PSD and its recovery have laid the theoretical foundations for PSD rehabilitation and have facilitated further research on the neuroplasticity of the pharyngeal motor cortex in association with its functional outcome [7,14].