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Review Article

An Embodied Cognition Perspective on Neural Mechanism and Rehabilitation of Freezing of Gait

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Abstract

Freezing of gait (FoG) is a highly disabling and challenging symptom of dyskinesia. At present, the pathophysiological mechanism of frozen gait is not completely clear, and the specific mechanism of action observation and motor relearning rehabilitation training based on mirror visual feedback of embodied cognition is not clear. Cognitive, emotional and sensory signals are involved in each node of gait control motion loop, and multiple interactions among sensory, edge and cognitive systems regulate gait and posture control. This paper makes a brief review from the perspective of embodied cognition in order to provide new ideas for further improving the cognition of FoG, evaluating and developing more effective non-invasive intervention FoG nerve rehabilitation technology.

Keywords: Freezing of gait (FoG); Embodied cognition (EC); Mirror visual feedback (MVF); Nerve rehabilitation

Introduction

Gait is a complex process, involving the accompanying balance and movement process. Gait movement network involves spine central pattern generator, midbrain and cerebellum movement area, subthalamic nucleus movement area and various cortical areas, including frontal parietal lobe, auxiliary movement area and movement area [1]. Freezing of gait (FoG) is defined as the absence or significant reduction of short and occasional foot forward movement despite the intention of walking. FoG often occurs in many neurodegenerative diseases, such as Parkinson's disease (PD), multiple system atrophy (MSA), progressive supranuclear paralysis (PSP), normal stress hydrocephalus, etc. [2]. Meta-analysis showed that the prevalence of FoG was 50.6% in PD [3], 65.93% in MSA [2] and 45.6% in PSP [4], and the FoG gradually increased with the duration and severity of the disease. Among PD patients, the prevalence of FoG with a course of more than 10 years was the highest (70.8%), followed by a course of more than 5 years (53.3%). Hoehn-Yahr staging (H&Y) score ≤ 2.5 was 28.4%, and H&Y score ≥ 2.5 was 68.4% [5].

Pathophysiological Mechanism of FoG

FoG is a highly disabling and challenging symptom of dyskinesia, which often increases the risk of falling, the injury and fear related to falling, and the loss of independence. FoG attacks have obvious intermittent and unpredictable situational characteristics, and common triggering factors include emotional tension, environmen-

tal challenges (such as passing through the door, approaching the destination or turning), performing cognitive tasks while walking (that is, dual tasks), and reducing visual input (such as walking in the dark). According to the clinical phenotype of FoG trigger factors, it can be divided into sports type (appearing in turning), cognitive type (appearing in the process of dual tasks) and marginal type (appearing during emotional anxiety and tension) [6]. Priming effect refers to a psychological phenomenon that after being influenced by a certain stimulus, the perception and processing of the same stimulus will become more and more skilled, and it is a manifestation of implicit memory. It can be seen that because the specific triggering factors and priming effects of FoG vary from person to person, effective treatment may need to identify and locate these characteristic heterogeneity at the individual level. FOG has complex pathophysiology, and the specific mechanism is still unclear. At present, it is considered that the pathophysiology of FoG may involve the local primary sexual dysfunction and compensation network imbalance in the back motor circuit, and the cortical dysfunction and the projection of cerebellum to the basal ganglia and brain stem motor area may aggravate the performance of FoG nerve [7]. Some studies have found that gait festination and FoG in PD may be related to motor automaticity abnormality and sequence effect [8, 9].Gait festination and FoG can often exist at the same time or alone. Gait festination may occur before freezing gait, which shows that the initial pace becomes rapid and the stride length decreases, and then it becomes completely frozen. Patients with FoG often use very small steps to terminate the frozen gait [10]. Continuous effect can be seen in some patients before FoG, which shows that the stride length before gait freezing is gradually reduced and can be improved by external stimuli, suggesting that continuous effect and motor automation may have similar brain network mechanism. Gait disorder of PD can be divided into continuous gait disorder and paroxysmal gait disorder from the aspects of its characteristics, function, prognosis and mechanics, among which paroxysmal gait disorder mainly includes gait festination and FoG[11]. Classical pathophysiology in PD holds that the loss of dopaminergic neurons in substantia nigra and abnormal striatum function may lead to bradykinesia, which is difficult to explain the current neural mechanism of FoG abnormality. FoG can be divided into three types based on the potential neurobiological basis and related triggering events. One is characterized by movment disorder (such as difficulty in turning around in the same place), the second is characterized by increased anxiety (such as being more prone to appear in a hurry), and the third is characterized by attention deficit (such as having difficulty in performing cognitive dual tasks while walking) [12]. It can be seen that cognitive, emotional and sensory signals are involved in each node (cortex, basal ganglia, cerebellum and brain stem) that affects gait control, and the multiple interactions among sensory, marginal and cognitive systems regulate gait and posture control and other sports behaviors [13]. At present, although there are different treatment options for FoG, the combination of multi-modal methods including exercise training is usually needed in clinic, but the effective treatment method is still difficult to determine. Clinical dopa drugs and subthalamic nucleus or The globus pallidus interna-deep brain stimulation(GPi-DBS) therapy often have no obvious improvement on FoG phenomenon, which has become a difficult problem in clinical neurology, highlighting the complexity of FoG pathophysiological mechanism. At present, most studies focus on the symptomatic treatment of patients with confirmed FoG, but lack of methods to delay or prevent the onset of FoG. Therefore, clarifying the pathophysiological mechanism of FoG and exploring more effective evaluation and intervention methods are the key problems that need to be solved urgently in clinical neurology.

Neural Mechanism of Embodied Cognition Participating in FoG

FoG is aggravated in excitement, tension, narrow space or approaching the target and turning, suggesting that it may have potential cognitive and sensory network involvement. Among psychological factors, nervous anxiety or great psychological pressure will induce and aggravate FoG[14]. Embodied cognition (EC) is based on the influence of sports system on situational cognition, and its two core elements are body (including brain) and environment. The process of embodied cognition is based on sensory, motor and emotional systems. EC theory mainly includes action-perception interaction, Mirror Neuron System (MNS) and embodied semantics. Because gait requires active cognitive and motor control systems, theta and β-band oscillations in the frontal lobe in the cortical-basal ganglia loop may be very important for starting and executing gait, and may be related to FoG. It is found that the mirror visual feedback (MVF) may affect the subthreshold activities of bilateral sensorimotor cortex during the preparation and execution of exercise observed in the EEG of healthy participants and stroke patients [15]. MRCP is an EEG pattern with frequency of 0~5Hz and amplitude of 5~30V, which starts about 1.5~2 seconds before the start of autonomic movement. Considering the significance of the projection of basal ganglia-thalamus-cortex nerve loop on the anterior auxiliary motor area, it is considered that the changes of different components of MRCP may be related to the characteristics of FoG [16]. Studies have shown that verbs expressing movement will activate brain regions related to body information processing, and the generation of verbs acting in unison promotes the execution of actions, which proves that body patterns are also implicitly activated in the process of vocabulary information processing. The strong expression of all movements and the combination of neural codes in the motor cortex connecting all limbs may help to transfer skills across limbs and provide a useful framework for thinking about how the motor system constructs movement and brain-computer interface [17].

The latest research found that there are EC abnormalities in Alzheimer's disease (AD), PD and Amyotrophic Lateral Sclerosis(ALS) in neurodegenerative diseases, even without other cognitive defects, which have a negative impact on the prognosis of the disease. The correlation mechanism of EC between emotion and motor control is a research hotspot at present. It is found that PD patients' response to emotional cues that cause disgust is impaired, and their flexibility of motor resonance to disgusting situations is reduced. PD patients may have defects in "transforming" aversion motivation state into physiological response, and emotional state is involved in the underlying pathological mechanism of FoG in PD pa-

tients [18, 19]. EC is rooted in the "here and now" situation and the self-body in motion control. Self-ownership and self-initiative are considered as the basis of EC self, and many experimental studies are also being carried out around this theme. The development of innovative technology for neurological rehabilitation based on EC mechanism may be a promising treatment method to improve the movement and cognition affected by diseases [20].

MNS plays a leading role in EC, updating and creating its own Body Schema by using the behavior simulation of itself and others. The main brain regions related to EC involve human MNS and its anatomical correlation. The visual input of this system mainly comes from the posterior part of the middle temporal gyrus and the superior temporal sulcus, and the motor output is sent to the inferior parietal lobule of the primary motor cortex, and then to the anterior motor cortex and the posterior inferior frontal gyrus [21]. In addition, the left and right judgment of limbs is often impaired in patients with hemiplegia after stroke, which proves that the body schema of patients has changed [22]. Studies have shown that the structure and connectivity of the striated body area (EBA) are involved in coding body representations and body perception disorders [23]. Self-illusion is not only an illusion caused by the integration of multiple senses and/or sensory movements, but also an self-representation that incorporates external objects into the brain. MNS is regarded as the neural basis for EC to recognize situations, and the neural basis for self-recognition is regarded as essentially the same as MNS [24].

Mechanism of Mirror Visual Feedback in FoG Rehabilitation

MNS-based MVF sports training strategies, such as motion observation and motion imagination, are helpful to promote sports relearning. Research shows that action observation and motor imagination training can effectively improve the disease severity, quality of life, balance ability and gait of PD patients [25]. MVF can also increase the excitability of primary motor cortex during asynchronous hand movements, which can effectively promote the recovery of hemiplegic limbs after stroke [26]. At present, motion observation has been recommended as an effective rehabilitation tool for PD patients, especially for FoG[27]. In the rehabilitation practice based on MVF, it is found that the mirror illusion of non-paralytic limb voluntary movement often has a regulating effect on the activation of visual motor and sensory motor brain. Based on EEG β -band (15-28 Hz) analysis, it is found that MVF combined with target directional movement is related to the reduction of hemispheric asymmetry in the preparation and execution stages of exercise, emphasizing the potential importance of incorporating visual moving targets into tasks based on MVF to maximize the rehabilitation results of training activities [28].

Although modern medicine has been able to effectively diagnose, treat and prevent many diseases, the understanding of behavior control of involuntary movement in dyskinesia diseases such as tremor and FoG is still very limited. Modern medicine has become accustomed to looking at the body from the perspective of natural science, which is one-sided. Many medical phenomena, such as the most common clinical pain, do not fully conform to the characteristics of biomedical research. Virtual reality (VR) technology has the advantage of immersive visual experience, and the functional integration of MNS and sensorimotor cortex under the visual perception of action is one of the theoretical foundations for the application of action observation to neural rehabilitation. Virtual Mirror Visual Feedback (VR-MVF) Adding MVF to VR situation is an effective means to improve interactive performance. Compared with traditional action observation, the visual representation of self-action in VR scene further stimulates the activity of MNS core cortex, which can adjust the excitability of bilateral cortex and promote its functional integration with sensorimotor cortex [13, 29]. EEG research shows that the use of VR may affect several brain regions (possibly including MNS) involved in sports planning and sports relearning, thus leading to the enhancement of sports performance [30]. It can be seen that VR-MVF technology is a rehabilitation technology to improve sensorimotor maladjustment through the advanced cognitive center of cerebral cortex. Self-modeling with immersive VR head-mounted display video and physical practice of personalized motion strategy, such as virtual doorway and corridor environment, are feasible and acceptable methods to solve FoG patients [31, 32].

Future Research and Conclusion

At present, the pathophysiological mechanism of FoG is not clear, and the specific mechanism of MVF rehabilitation training is not clear. Reconstructing the priming effect model of FoG patients based on EC theory may promote and enhance motor relearning, which needs more detailed research to clarify the target of EC in neural rehabilitation and sufficient evidence to clarify the relationship between EC body language, emotion and motor control. It can be used to further strengthen the motor control function of FoG patients by presenting specially designed embodied cognitive tasks on the computer screen or having a positive impact on obtaining new priming effects. In the future, EEG, functional magnetic resonance imaging (fMRI), far infrared thermal imaging and other inspection methods, combined with the flexibility of emerging VR technology, will help to further improve the cognition of FoG, evaluate and develop more effective non-invasive intervention FoG neural rehabilitation technology through MVF video self-modeling.

Author Contributions

Wang GQ, Jin P, Ma XF, Fu XM,Wen X, Bai X and Han YZ contributed to the conceptualization and wrote and edited the manuscript. All authors contributed to the article and approved the submitted version.

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