Central Nervous System Exhaustion

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Introduction

The Japanese Society of Fatigue Science provides the following definitions of fatigue and fatigue sensation: "Fatigue is defined as a loss in the ability and efficiency of mental and/or physical activities that is induced by excessive mental or physical activities or disease. The phrase "fatigue sensation" refers to the peculiar sense of discomfort, desire to rest, and decrease in motivation that frequently accompany exhaustion.

Some of the neural mechanisms underlying human acute physical fatigue, acute mental fatigue, chronic fatigue, and fatigue associated with human diseases and syndromes have recently been clarified by behavioural, electrophysiological, and neuroimaging studies using methods like Functional Magnetic Resonance Imaging (fMRI), Positron Emission Tomography (PET), and Magnetoencephalography (MEG).

Acute and Chronic Fatigue

Regarding acute physical fatigue, it is necessary to exert more voluntary effort in order to increase the motor output from the primary motor cortex (M1) in order to make up for the physical fatigue and maintain physical performance when performing a physical task and the active muscle fibres become fatigued. This keeps happening until the task demands the most volunteer effort. The facilitation system is a re-entrant neural circuit that connects the limbic system, basal ganglia, thalamus, orbitofrontal cortex, dorsolateral prefrontal cortex, anterior cingulate cortex, premotor area, supplementary motor area, and M1. It increases the motor output from M1 and is known to do so. Motivational input to this physical facilitation system increases SMA activity, which in turn increases M1 activity, increasing motor output to the peripheral system. However, during physical exhaustion, sensory information from the peripheral system is transmitted to M1, which reduces motor output. A system of inhibition increases the inhibitory input to M1 in order to restrict the recruitment of motor units and/or reduce the firing rate of the active motor units in M1. This neural network connects the spinal cord, TH, secondary somatosensory cortex, insular cortex (IC), posterior cingulate cortex (PCC), ACC, PM, SMA, and M1 to form the physical inhibition system. The harmony between these physical facilitation and inhibition systems primarily controls the motor output from M1.

Regarding acute mental exhaustion, a "dual regulation system" has been proposed as a conceptual model for cognitive task performance. According to this hypothesis, mental burden triggers the mental facilitation system, which helps people with mental weariness continue to execute cognitive tasks. The mental facilitation system is made up of the thalamic-frontal loop, which connects the limbic system, BG, TH, and FC. As motivational input to this system rises, so does its activation. The mental inhibition system is also activated by mental workload, which reduces the effectiveness of cognitive tasks. Involved in the mental inhibition system are the IC and PCC.Abrupt mental weariness is caused by acute mental workload, which activates the facilitation and inhibition systems in the brain. When the mental facilitation system is active, cognitive task performance is either maintained or improved, whereas when the mental inhibition system is active, cognitive task performance is compromised. Performance of the cognitive task is either degraded, maintained, or improved depending on how these two systems are activated in balance. Thus, through the dual regulation system, these two systems control how well we do cognitive tasks. Performance may be improved and acute weariness may be prevented by enhancing the physical or mental facilitation system through psychological, behavioural, or physical stimuli.

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However, if the facilitation system is overly improved at the expense of its functions, the system could become dysfunctional and produce more weariness. Driving challenges result from the facilitation system's malfunctions. The facilitation system is improved by motivation or voluntary effort, however more improvement of the system leads to further dysfunction of the system. Chronic or cumulative weariness could result from this.

Therefore, it has been suggested that chronic weariness results from a breakdown in the facilitation system. In fact, individuals with chronic fatigue display malfunction of the facilitation system during motor imagery task trials as well as during cognitive task trials, which may be caused by structural, functional, or metabolic impairment to any of the system's components. Because central sensitization and classical conditioning of the inhibition system have been successfully accomplished in humans, and an animal model of fatigue has been established using central sensitization and classical conditioning techniques, increased activation of the physical or mental inhibition system may be caused by repetitive and prolonged overwork and/or stress

Mechanism of Fatigue

However, if the facilitation system is overly improved at the expense of its functions, the system could become dysfunctional and produce more weariness. Driving challenges result from the facilitation system's malfunctions. The facilitation system is improved by motivation or voluntary effort, however more improvement of the system results in further dysfunction of the system. Numerous studies have revealed dysfunction and/or abnormal activation patterns in the brain regions involved in the facilitation and inhibition systems in human diseases or syndromes like multiple sclerosis and Chronic Fatigue Syndrome (CFS). Driving the facilitation system is difficult when the facilitation system is dysfunctional or when the inhibition system is strengthened. The facilitation and inhibition systems are improved by voluntary effort, but the improved inhibition system makes it harder to drive the facilitation system.

Conclusion

In these diseases and syndromes, this process results in severe exhaustion by causing dysfunction of the facilitation system and activation of the inhibition system through central sensitization or classical conditioning of the inhibition system.

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