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PHANTOM LYMB SYNDROME: ANATOMICAL MECHANISMS AND CLINICAL IMPLICATIONS

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Abstract

Phantom Limb Syndrome (PLS) is a complicated condition related to neurology in which individuals continue to perceive sensations, including pain, in a limb that has been amputated. Majority of amputees experience this phenomenon, and it reflects intricate neuroanatomical and neurophysiological adaptations across the peripheral and central nervous systems. It is important to differentiate it from residual limb pain or non-painful phantom phenomena. PLS is not only purely psychological experience, but it also causes various structural and functional changes within the nervous system. Understanding the anatomical mechanisms

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behind PLS provides insight into neuroplasticity and informs modern pain management strategies.

Keywords: phantom sensations; thalamocortical dysrhythmia; amputees; neuroma; adjacent somatotopic regions; prosthetic adaptation;

Introduction

Physicians and neuroscientists have been interested in Phantom Limb Syndrome for centuries as its clinical nature is not fully understood. It was firstly systematically described in the 19th century by American neurologist Silas Weir Mitchell observing conditions of American Civil War in detail. After that, PLS has been studied as complex neurophysiological phenomena rather than psychological one. The condition refers to the persistent perception of a missing limb even after amputation. According to studies between 80% and 95% of amputees experience phantom sensations, and a significant proportion develop phantom limb pain (PLP), which is one of the most prevalent post-amputation complications. Phantom sensations may include tingling, itching, pressure, movement, or severe burning pain localized to the absent limb. [6]

From an anatomical perspective, PLS challenges the assumption that sensation strictly depends on intact peripheral structures. Instead, it demonstrates that the central nervous system maintains a durable internal representation of the body, often referred to as the body schema. Even after the removal of peripheral input, cortical maps corresponding to the amputated limb persist and may become reorganized. Mostly, peripheral nerve injury at the amputation site results in the formation of neuroma and ectopic discharges. In the dorsal horn neurons of the spinal cord, increased excitability is observed due to loss of inhibitory control. [7]

Additionally, central sensitization, thalamocortical dysrhythmia, and changes in body schema contribute to permament phantom perceptions. According to a “neuromatrix” concept suggested by Ronald Melzack, pain is not merely a consequence of peripheral impulses, but rather an emergent phenomenon generated by widely distributed neuronal networks within the central nervous

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system(CNS). This theory explains why phantom sensations can remain present in the absence of sensory afferent signals.

From a clinical perspective, phantom limb syndrome represents both a neurological disorder and a significant challenge in rehabilitation medicine. Persistent phantom pain influences prosthetic adaptation, sleep quality, psychological status, and overall quality of life negatively. For this reason, understanding anatomical and neurophysiological basis of PLS is important to develop treatment strategies like mirror therapy, neuromodulation and pharmacological interventions.

Methods

At the amputation stump, neuromas form at the site of peripheral nerve injury and generate pathological ectopic impulses in damaged nerve fibers. These impulses are transmitted to spinal cord through dorsal root ganglia and are important to generate phantom sensations. Clinical neuropathalogic observations support this mechanism. [6]

Central sensitization mechanisms are analyzed based on studies that show increased excitability and inhibitory modulation in dorsal horn neurons of the spinal cord after deafferentation. Synaptic plasticity mediated by NMDA receptors is considered one of the main mechanisms underlying this sensitization. [2]

Functional MRI studies investigating changes in S1 and M1 cortical organization were identified and analyzed to evaluate reported patterns of cortical remapping. Additionally, studies addressing thalamocortical network alterations and central pain processing mechanisms were included to ensure comprehensive coverage of central nervous system involvement. [3,4]

Results

Peripheral Nervous System Changes

Severed peripheral nerves attempt regeneration after amputation. Neuromas-tangled masses of nerve fibers are often formed under the influence of this process. These tangled masses in nerve fibers can lead to spontaneous ectopic

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discharges due to abnormal sodium channel expression and heightened membrane excitability. These aberrant signals travel through the dorsal root ganglia into the spinal cord, where they may be interpreted as originating from the missing limb.

Nociceptors have increased excitability mainly because of peripheral sensitization. Inflammatory mediators released after tissue injury enhance receptor responsiveness, lowering the threshold for action potential generation. Although the limb is absent, the residual nerves continue transmitting signals, creating the illusion of persistent presence.

Spinal Cord Mechanisms

At the level of the spinal cord, deafferentation leads to central sensitization within the dorsal horn. Reduced inhibitory interneuron activity and increased excitatory neurotransmission amplify nociceptive input. NMDA receptor activation plays a critical role in this process, strengthening synaptic transmission and contributing to long-term potentiation-like changes. This hyperexcitability may cause even minimal peripheral input or spontaneous neuronal firing to be perceived as intense pain. [2]

Cortical Reorganization

The most important part is that functional imaging studies have shown cortical reorganization within the primary somatosensory cortex(S1) and primary motor cortex(M1). Cortical remapping is a process in which the cortical representation of the body part that has been amputated is slowly taken over by adjacent somatotopic regions. For example, in people who have lost an arm, stimulating the face can cause activation of cortical areas that used to control the hand. This reorganization in the brain can lead to referred sensations that feel like they are coming from the missing limb. Functional imaging studies demonstrate that phantom limb pain has a strong correlation with this reorganization.

In addition to changes in the area of the brain that processes touch (the somatosensory cortex), there are also changes in the motor cortex and thalamus. When the normal connection between movement and sensory feedback is

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disrupted, it creates a mismatch between what a person intends to do and what they feel, which can make phantom pain worse. [8]

Neuromatrix Theory

The neuromatrix theory helps to understand how people feel their body and generate pain perception. It proposes that body perception is triggered by a distributed network of neurons which involve sensory, motor, and limbic regions. According to this model, the neural network encoding the limb remains intact despite its physical absence, therefore phantom limb sensations are generated. Emotional and cognitive factors may modulate this network, influencing pain perception. Meaning that stress, anxiety and depression might cause the pain to increase or vice versa. [3]

Discussion

The principle of neuroplasticity is demonstrated in Phantom Limb Syndrome, it is ability of the nervous system to reorganize structurally and functionally in response to injury. PLS is not caused by a single mechanism, various things generate it by interpreting together. For example, interaction of peripheral nerve changes, spinal sensitization, and cortical reorganization.

Clinically, this understanding informs treatment strategies that address the specific mechanisms involved in PLS. For instance, connection between motor intention and sensory perception is re-established by the visual feedback that can be provided in mirror therapy. Patients may relieve pain and decrease cortical mismatch when they observe the reflection of the intact limb. This approach emphasizes the significance of sensory integration in rehabilitation. [4]

Both peripheral and central mechanisms are targetted in pharmacological treatments, which involve NMDA receptor antagonists, antidepressants, and anticonvulsants. They are designed to adjust neural pathways that participate in pain processing. Apart form this, emerging interventions such as transcranial magnetic stimulation(TMS) and spinal cord stimulation(SCS) aim to modulate central excitability directly by representing cuttng-edge techniques. They

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contribute to enhance overall quality of life for individuals who suffer from PLS by offering new avenues for pain management and recovery.

Conclusion

Phantom Limb Syndrome (PLS) serves as a fascinating illustration of how anatomical structures influence conscious experience. Even when a limb is absent, the nervous system maintains and reorganizes its representation within both cortical and subcortical circuits. The formation of peripheral neuromas, spinal sensitization, and cortical remapping work together to elucidate the persistence of phantom sensations experienced by individuals. Ongoing research into these mechanisms not only deepens our understanding of brain plasticity but also paves the way for the development of more effective therapeutic interventions aimed at alleviating the discomfort associated with phantom limb sensations.

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