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A Short Note on Phantom Limb

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Short Communication

Limb salvage is a major part of podiatry, with the goal of preserving the function and length of the lower extremity while also addressing any co-morbidities or infections. Amputation of the lower extremities anatomy may be necessary in the course of treatment, as determined by the clinician and the patient. Vascular disease, trauma, infection, and malignancy are all causes of lower limb amputation. Indeed, vascular disease is a common cause of lower extremity amputations, with higher rates among individuals aged 65 and up. Every year, 1.7 million people in the United States lose a limb, with 185,000 new lower extremity amputations accounting for roughly 86 percent of all amputations. Infection, trauma, and stump pain are all possible causes of pain. In the aftermath of surgery Patients with painful post-amputation sensations were first documented in the 16th century, and such symptoms were later labelled as phantom limb pain in the 19th century [1].

More recently, phantom limb experiences have been reported in 60 to 80 percent of amputees. The prevalence of phantom limb pain has risen from 2% in the past to higher percentages today. Patients were less prone to mention pain symptoms in the past than they are now, which could explain the disparity in incidence rates. Sherman, on the other hand, claims that just 17 percent of phantom limb concerns are handled by doctors. As a result, determining what defines phantom pain is critical in order to give effective care [2]. Phantom pain occurs following amputation and/or nerve injury to a limb, organ, or other tissue. Phantom limb pain is most common in podiatry after limb amputation due to a sick state presenting with an unsalvageable limb.

Stump neuroma discomfort, prosthesis, fibrosis, and residual local tissue inflammation can all cause comparable postoperative pain symptoms. PLP patients experience scorching, stinging, agonizing, and piercing pain, as well as a shifting warmth and cold sensation to the severed area that waxes and wanes. Environmental, emotional, or physical factors may trigger the onset of symptoms. Various neurologic processes in the human body allow for the reception, transfer, recognition, and reaction to a variety of inputs. The anterolateral system transports pain, temperature, crude touch, and pressure sensory information to the central nervous system, with pain and temperature information passing through lateral spinothalamic pathways to the parietal cortex. Pain from the lower extremities is conveyed from a peripheral receptor to first degree pseudounipolar neurons in the dorsal root ganglion, where it decussates and ascends to third degree neurons in the thalamus [3]. This sensory data will eventually reach the primary sensory cortex in the parietal lobe's post central gyrus, which houses the sensory homunculus. It's unsurprising that such a complex information highway would be disrupted by an amputation and from the periphery may have the potential to cause brain problems. How does pain, which serves as a defence mechanism for the human body, become chronic and unremitting following limb loss? This is an issue that researchers are still debating today, with no clear answer.

Phantom limb pain is more common in people who have had longer periods of stump pain, and it is more likely to go away as the stump pain goes away. Researchers discovered that after a nerve is entirely severed, the dorsal root ganglion cells change. With the capacity for plasticity development at the dorsal horn and other regions, the dorsal root ganglion cells become more active and sensitive to chemical and mechanical changes. Higher glutamate and NMDA concentrations correspond with increased sensitivity, which contributes to allodynia and hyperalgesia at the molecular level. Further explained the importance of maladaptive behaviour for pain and phantom limb pain, plasticity and the development of memory are important. They linked it to the loss of GABAergic inhibition as well as glutamate-induced long-term potentiation modifications and structural changes such as myelination and axonal sprouting. In addition to the aforementioned neurotransmitters, norepinephrine, a key ligand, may give light on the sympathetic nervous system's role in pain sensitivity regulation. Animals' postganglionic sympathetic nerve fibres become stimulated and hence more sensitive as norepinephrine levels rise, and this enhanced sensitivity can contribute to higher pain awareness. Patients may show evidence of localized alterations in addition to molecular abnormalities. Up regulation of sodium channels is linked to more frequent bouts of pain on a local level. A neuroma can grow where a nerve is severed at the local amputation site. Local chemical, physical, and mechanical stimulation can activate neuroma activity, resulting in pain feeling [4].

The notion of neural plasticity asserts that the adult brain is capable of dynamic regulation and is an integral aspect of the nervous system's adaptable structure throughout an individual's lifespan. It has been proposed that short- and long-term retraining consists of modulations to brain regions involving grey matter and white matter. Gray matter alterations are thought to include glycogenesis, vascularization, and synaptogenesis, while white matter modifications include axonal sprouting and myelination. The relationship between neuronal plasticity in the cortex and the development of extremities lesions or sensory pathologic alterations in monkeys has also been studied. Jiang looked into how brain grey matter and white matter plasticity developed following a lower leg amputation. Using tract-based spatial statistics and tractography analysis, researchers compared 18 patients with right lower limb amputation to 18 healthy controls [5]. They looked for changes in white matter cortical thickness and fractional anisotropy using T1 MRI. The fractional anisotropy decreased in white matter sections of the right superior corona radiate in the right temporal lobe, left PMC, and right inferior fronto-occipital fasciculus in amputation patients. Furthermore, the left premotor cortex thinning was seen, with smaller clusters in the visual-to-motor areas. They found no significant alterations in the aforementioned locations in

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healthy control individuals, indicating that the changes in cortical areas reflecting an amputated leg are real [6].

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