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 [illegible] amputation due to a sick state presenting with an unsalvageable limb.

Stump neuroma discomfort, prosthesis, brosis, and residual local tissue in ammation can all cause comparable postoperative pain symptoms. PLP patients experience scorching, stinging, agonizing, and piercing pain, as well as a shi ing 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. e 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 rst degree pseudounipolar neurons in the dorsal root ganglion, where it decussates and ascends to third degree neurons in the thalamus [3]. is 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? is 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 a er 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. ey linked it to the loss of GABAergic inhibition as well as glutamate-induced long-term potentiation modi cations 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 bres 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,

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

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