Ruprecht-Karls-Universität Heidelberg
Medizinische Fakultät Mannheim
Dissertations-Kurzfassung

The role of peripheral input and its contribution to phantom limb pain

Autor: Hongcai Liu
Institut / Klinik: Zentralinstitut für Seelische Gesundheit Mannheim (ZI)
Doktormutter: Prof. Dr. H. Flor

Following amputation, nearly all amputees report non-painful phantom phenomena and many of them suffer from chronic phantom limb pain (PLP) and residual limb pain (RLP). The etiology of PLP remains elusive and there is an ongoing debate on the role of peripheral and central mechanisms. Few studies have examined the entire somatosensory pathway from the truncated nerves to the cortex in amputees with PLP compared to those without PLP. The relationship between afferent input, somatosensory responses and the change in PLP remains unclear. The present thesis aimed at identifying whether peripheral afferent input can induce PLP and how it interacts with somatosensory processing and postamputation pain. Transcutaneous electrical nerve stimulation was applied on the truncated median nerve, the skin of the residual limb and the contralateral homologous nerve in twenty-two traumatic upper-limb amputees (12 with and 10 without PLP). Using somatosensory event-related potentials, the ascending volley was monitored through the brachial plexus, the spinal cord, the brainstem and the thalamus to the primary somatosensory cortex. There were no significant differences in the electrical potentials generated by the stimulation from the truncated nerve or the skin of the residual limb in amputees with and without PLP. Peripheral input could evoke PLP in amputees with chronic PLP (7/12), but not in amputees without a history of PLP (0/10). In addition, peripherally induced potentials through the spinal-subcortical segment were significantly positively associated with evoked RLP, but not PLP. Peripheral input can enhance PLP but seems insufficient to cause it. These findings indicate the multifactorial complexity of PLP and also suggest different mechanisms for PLP and RLP.