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BRIEF COMMUNICATION

Reversal of phantom pain and hand-to-face remapping after brachial plexus avulsion

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Abstract

Following left brachial plexus avulsion, a 20-year-old man had phantom limb pain and remapping of sensation from his paralyzed hand onto his face. Mirror therapy (15 min daily, 5 days/week) led immediately to good movement of the phantom limb with decreased pain. Within 2 weeks following nerve graft surgery, remapping of hand sensation onto the face disappeared along with resolution of phantom limb pain. Mirror therapy coupled with nerve grafting may relieve phantom limb pain due to brachial plexus avulsion and reverse hand-to-face remapping, suggesting that both peripheral and central mechanisms mediate development of phantom limb pain and cortical reorganization/neuroplasticity after brachial plexus avulsion.

Individuals suffering severe deafferentation injuries, such as brachial plexus avulsion (BPA), can experience phantom limb pain even with an intact limb.[1,2] Pain in the setting of BPA is often severe, difficult to treat, and can be disabling.[3] Routine medications for neuropathic-type pain such as anticonvulsants, tricyclic antidepressants, and selective serotonine and norepinephrine reuptake inhibitors are often ineffective. Cannabinoid-based analgesics have also demonstrated unclear benefits.[4] More invasive procedures such as spinal cord stimulation or dorsal root entry zone (DREZ) lesioning are available, but not all patients are ideal candidates. For instance, while DREZ lesioning is a neurosurgical procedure that may lead to lasting relief in up to two thirds of patients, in the setting of MRI evidence of damage to the dorsal horn or DREZ, no patients demonstrated resolution of pain with this procedure.[5] Transcranial magnetic stimulation (TMS) is a noninvasive treatment alternative which has been shown to trigger neuronal plasticity to produce long-lasting therapeutic benefit.[6] Therapeutic effects of motor cortex stimulation on deafferentation pain suggest that the core mechanisms underlying deafferentation pain also interact with the motor system.[7]

There are many theories as to why the pain from BPA is so severe. Deafferentation pain may result from reorganization of the nervous system after nerve injury via processes that interact with the pain matrix in the brain.[7] Loss of normal afferent input to the central nervous system along with spontaneous firing of the dorsal horn neurons can lead to changes at the level of the spinal cord and the brain. In chronic pain, at the level of the spinal cord the receptive field of wide-dynamic range neurons at the substantia gelatinosa can increase, leading to non-painful stimuli being interpreted as painful.[8] Regarding changes in the brain, fMRI has demonstrated differing patterns of activity when comparing persons with limb loss with and without phantom limb pain.[9]

The manner in which cortical remapping occurs may be linked to the development of severe pain. In upper extremity amputees, reorganization of the sensory maps in a topographical manner has been reported, with sensation of the hand transferred to the face.[10] Mirror therapy

may induce cortical and fMRI changes that lead to improvements in deafferentation pain.

A 20-year-old man had left BPA of C6-T1 due to a motorcycle accident. Although the limb was anatomically intact, he complained of constant, unremitting moderate to severe (3.5–9/10) left hand/arm phantom pain, experienced as a frozen immobile limb, with throbbing, electrical shocks, stabbing, cramping, aching, and tenderness, which started immediately upon awakening after injury. Neurological examination at 6 months after injury revealed that he had the capability to shrug the shoulders fully, flaccid paralysis of the left upper extremity, and complete anesthesia to light touch, pain, and temperature from the elbow distally and decreased light touch over the lateral and posterior upper arm as well as the anterior shoulder and upper chest. Light touch and cold applied to the left buccal and jaw region resulted in the patient reporting the same sensations in his left hand, a phenomenon known as remapping.[11]

He began mirror therapy (15 min daily, 5 days/week), placing a mirror in the mid-sagittal plane between his intact and paralyzed upper limbs and attempting to move his left hand/arm while viewing the reflection of his right hand/arm moving (finger, wrist and elbow extension/flexion, and rotation of the wrist). He immediately reported good movement of the phantom with resolution of pain (7–0/10). After completing his first treatment session, the limb resumed its immobile state and pain returned to pretreatment level. Following 1 month of treatment, his pain had decreased to 4/10. At month 8 after injury, he underwent brachial plexus exploration and nerve grafting of the spinal accessory to the musculocutaneous nerve. Referred sensations to the left hand elicited by touching the left face continued to be reported immediately following the surgery. However, within 2 weeks the referred sensations disappeared concomitant with resolution of phantom pain. After a further 2 weeks, minimal shoulder rotation was noted on examination.

Although nerve grafting alone may have led to resolution of both the phantom pain and the referred sensations, the response of our patient to mirror therapy prior to surgical intervention demonstrates that this treatment is effective in providing relief from phantom pain in the setting of BPA. As our patient experienced both resolution of phantom pain and reversal of hand-to-face remapping, this suggests that both peripheral and central mechanisms mediate development of phantom pain and cortical reorganization/neuroplasticity after BPA. Cortical reorganization/neuroplasticity in phantom pain secondary to major limb amputation has long been thought to be due to neurons forming new connections with neighboring cortical regions.[12] In this case, the rapidity of phantom pain onset/relief and changes seen in hand-to-face remapping would be most easily explained by unmasking of existing, but previously quiescent, excitatory or inhibitory synaptic connections instead of formation of de novo (new) connections. This reinforces the view of brain function as a continuously shifting equilibrium, with constant formation and reformation of synaptic connections in response to changes in the surrounding environment, allowing us to partially reset the equilibrium using the noninvasive procedure of mirror therapy.

Conflict of Interest

All authors have no conflicts of interest to report.

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