MODULATION OF PHANTOM LIMB PAIN USING EPIDURAL STIMULATION OF THE CERVICAL DORSAL SPINAL CORD

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INTRODUCTION

Pain is a common comorbidity of conditions such as peripheral nerve injury, substance-induced neuropathy, and trauma. Nearly 1.5 billion people worldwide suffer from chronic pain with the estimated cost of health care nearly \$275 billion. The mechanisms of neuropathic pain are poorly understood and its evaluation in humans is complex because most stimuli required to induce neuropathic pain produce irreversible damage. Recent evidence suggests that the incidence of chronic phantom limb pain can be regulated by delivering sensory feedback that is relevant to the amputated limb. This study aims to determine whether cervical spinal root stimulation to elicit sensations localized to the amputated arm can also result in concomitant changes in PLP

METHODS

All procedures were approved by the University of Pittsburgh Institutional Review Board and the US Army Human Research Protection Office. Two study participants were implanted with three 8 or 16 contact spinal cord stimulation leads (Boston Scientific) in the lateral epidural space of the cervical spinal cord. Stimulation electrode, amplitude, frequency and pulse width were varied across trials. The location, intensity and modality of the evoked percepts was recorded. The intensity of PLP was recorded on a visual analog scale (VAS) after every stimulation trial. Additionally, the McGill Pain Questionnaire (MPQ) was administered on a weekly basis, and again one month following explantation. The leads were explanted after 2-4 weeks.

RESULTS

A total of 1,493 trials evoked localized sensations, of which 580 PLP episodes were reported (38.9%) at a mean intensity of 2.5 ± 1.9 on the VAS. For the 115 electrodes that evoked a sensation, stimulation amplitude and pulse width were related to the intensity and incidence of PLP respectively. Furthermore, a clinically significant (>5 points) reduction in PLP was observed on the MPQ in subject 1 (9 points) and subject 2 (8 points) at 1-month follow-up. Additionally, the effect of stimulation electrode location on

PLP modulation as well as the correlation between the modality of stimulation evoked non-PLP sensation and the incidence of PLP is being explored.

CONCLUSION

This study suggests that stimulation amplitude and pulse width may modulate the intensity and frequency of a PLP episode. We further observed time-dependent PLP modulation such that the immediate post-stimulation phase was associated with increased PLP that may be coupled to a long-term reduction in PLP.