

Brain circuitry underlying pain in response to imagined movement in people with spinal cord injury

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Introduction

Although there are many studies describing pain evoked by peripheral stimulation, we have recently reported that pain can be evoked in subjects with a cognitive task. In this study, we have used functional magnetic resonance imaging to explore the brain sites underlying the expression of this phenomenon.

Methods

Eleven clinically complete (ASIA A) thoracic SCI subjects with below-level neuropathic pain and 19 healthy controls were used in this study. The cerebral activation pattern during six 30 second periods of imagined right ankle plantarflexion and dorsiflexion was measured using fMRI (130 volumes, 57 axial slices, TR = 3s, TE = 30ms, flip angle = 90°, matrix size = 128*128, 3mm thick). During each movement imagery period, a recording of a car accelerator was used as an auditory guide. Each subject rated their ongoing pain prior to and during the movement imagery periods using a 10 cm VAS. All fMRI images were motion corrected, smoothed (6mm FWHM) and significant changes in signal intensity in all SCI and control subjects were determined using a repeated box-car model. Significant correlations between the percent change in pain intensity during the imagery periods and signal intensity increases were also assessed.

Results

In 9 out of 11 subjects with complete thoracic SCI and below-level neuropathic pain, imagined foot movements either evoked pain in a previously non-painful region or evoked a significant increase in pain within the region of on-going pain (3.2 ± 0.7 – 5.2 ± 0.8). In both controls and SCI subjects, movement imagery evoked signal increases in the supplementary motor area and cerebellar cortex. In SCI subjects, movement imagery also evoked increases in the left primary motor cortex (MI) and the right superior cerebellar cortex (corrected $p < 0.05$). In addition, in the SCI subjects, the magnitude of activation in the perigenual anterior cingulate cortex and right dorsolateral prefrontal cortex was significantly correlated with absolute increases in pain intensity ($r = 0.86$, $P < 0.002$; $r = 0.90$, $P = 0.001$, respectively).

Conclusion

This study demonstrates that in SCI subjects with neuropathic pain, a cognitive task is able to activate brain circuits involved in pain processing independent of peripheral inputs.