Assessment of spinal cord stimulation-based modulation in the spontaneous hyperexcitability model of neuropathic pain

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Background

Spinal cord stimulation (SCS) is a clinical therapy for intractable neuropathic pain in the lower limbs and low back. Its mechanism of action remains unclear despite its clinical use for over 50 years. The gate control theory is a theoretical framework by which electrical stimulation of the dorsal column can block transmission of peripheral pain signals from being perceived in the brain, but this theory has not been able to explain why SCS seems to preferentially block chronic neuropathic and not nociceptive pain. Few studies have explored how recruitment of Ab afferents can inhibit transmission of spontaneous nociceptive activity. Here, I use 4-aminopyridine to pharmacologically induce spontaneous activity and to investigate if recruitment of Ab afferents from the segment of interest is required for modulation of spontaneous nociceptive activity in the dorsal spinal cord.

Methods

Adult mouse spinal cord- dorsal root ganglia preparation

4-aminopyridine (4-AP): K+ channel blocker

Step 1: Characterize spontaneous nociceptive and non-nociceptive activity generated with 4-aminopyridine (10-20 mM)

Step 2: Investigate SCS modulation of spontaneous nociceptive activity at subthreshold and threshold amplitudes for dorsal root Ab recruitment

Histological evidence of superficial dorsal horn activation in 4-AP spontaneous hyperexcitability model

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References


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