BURST STIMULATION: BACKGROUND AND RESULTS
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The brain is a complex adaptive system, processing information from the internal and external environment. Information is transmitted within the nervous system in at least 2 separate firing patterns, tonic and burst firing. Burst firing consists of closely spaced spikes at high frequency (eg 500 Hz) followed by quiescent periods. It has been proposed that burst firing is a signal detector, a kind of wake up call, telling the brain new information has arrived, whereas tonic firing is a feature detector, it transmits the content of the new information. Thus burst can be seen as a start and stop signal at the beginning and end of quanta of information, which are transmitted in tonic mode. But burst firing also functions as a multiplexor, permitting selective routing of information via specific pathways.

The concept of burst stimulation is to mimic nature, and therapeutically using the advantages of burst firing.

Pain can be seen as a homeostatic emotion in which 2 ascending pathways transmit pain signals to the brain, increasing pain, and at least 1 descending pain pathway, suppressing pain. The medial spinothalamic ascending system, which ends in the dorsal anterior cingulate gyrus and anterior insula, fires in burst mode and encodes attention to pain, as well as the affective components of pain. The lateral spinothalamic system ends in the somatosensory cortex and encodes the discriminatory components of pain (where, intensity etc) and fires in tonic mode. The antinociceptive i.e. pain suppressing system also fires in burst mode so it can override the ascending pain transmission in the dorsal horn.

Lesions of the large myelinated fibers induce spontaneous bursting in adjacent C-fibers generating neuropathic pain. Spinal cord stimulation supposedly activates the remaining myelinated fibers to suppress this spontaneous C-fiber firing according to the pain gate theory. However Abeta fiber stimulation results in paresthesias. A preliminary non-placebo controlled study demonstrates that burst stimulation does not cause paresthesias and results in equally good to better pain suppression than tonic stimulation in neuropathic pain. This is confirmed by a placebo-controlled study as well as by multicentre European data. Furthermore attention to pain and pain changes is better suppressed by burst than tonic stimulation, as well as global pain.

EEG demonstrates that burst stimulation modulates the medial spinothalamic system more than tonic stimulation, explaining the different clinical features of burst and tonic stimulation. This is probably based on the multiplexing and routing function of bursts.

An animal study looking at nociceptive pain also demonstrates burst stimulation is better than tonic stimulation, suggesting that burst stimulation is beneficial for both neuropathic and nociceptive pain. This could explain its benefit in improving back pain in FBSS, which is often a combination of neuropathic and nociceptive pain.

More research is needed to optimize stimulation parameters (number of spikes, number of bursts, pulse widths, cycling etc).