Central pain in spinal cord injury

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ABSTRACT

The studies have been carried out at the Danish Pain Research Center, University of Aarhus. Central neuropathic pain is an important but also neglected problem following spinal cord injury (SCI) and it is often chronic, disabling, and resistant to treatment. The major aim of the present dissertation was to evaluate the underlying mechanisms of central pain in SCI.

I a postal survey we found that chronic pain is present in about 70% of patients with SCI and chronic central neuropathic pain in 30-50%. To clarify mechanisms underlying central pain following SCI, we have carried out clinical studies using quantitative sensory testing, MRI, and pharmacological trials. Based on distinctions between: 1) at-level pain and below-level pain, 2) spontaneous and evoked types of pain, and 3) painful and non-painful SCI, it has been possible to clarify distinct mechanisms of SCI pain.

The main findings were:

– Evoked types of pain are more common in SCI patients with central pain.
– Lesions in central grey matter are larger in SCI patients with central pain.
– Spinothalamic tract lesions are equally common in SCI patients with and without central pain.

Thus, the development of central pain in SCI does not seem to depend on interruption of the spinothalamic tract but may depend on grey matter lesions and the generation of a “spinal pain generator” of hyperexcitable cells at the rostral part of the spinal lesion. It is hypothesized that central pain results from excitatory input from the spinal generator ascending in multisynaptic pathways to deafferented higher order neurons with receptive fields below injury level causing pain sensation in these areas.

An intravenous drug trial using the sodium channel blocker lidocaine supported the role of neuronal hyperexcitability in SCI pain. However, treatment of SCI neuropathic pain remains challenging. Long-term trials are generally disappointing. Lamotrigine failed to relieve neuropathic pain following SCI, although subgroup analysis suggested efficacy in a subgroup of patients that had evoked pain.

Although we have come far in the understanding of the mechanisms of central SCI pain, there are still unsettled issues that should be addressed in future clinical studies. We need to further understand the interaction between spinal and supraspinal changes following a spinal injury and to identify new treatment targets.