The Thoracolumbar Fascia as a Source of Low Back Pain

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BACKGROUND: Low back pain (LBP) is still mainly ascribed to disorders of the spine (e.g. slipped disc), although unspecific LBP – which originates in the soft tissues of the low back – has a much higher prevalence. So far, the neuroanatomical and neurophysiological basis of this type of LBP is unknown.

METHODS: All results were obtained from anesthetized SD rats. Neuroanatomical experiments employing immunohistochemistry, neural tracers and activity markers (cFos) yielded data on the pathway of the nociceptive information in the spinal cord and brainstem. Electrophysiological recordings from dorsal horn neurons were performed to obtain information about the spinal processing of the nociceptive input from the soft tissues of the low back.

RESULTS: Neuroanatomy: Immunohistochemical investigation of the thoracolumbar fascia (TLF) revealed a dense innervation with afferent free nerve endings, including nociceptive ones. Neurons receiving nociceptive input from the low back and projecting to the brainstem (periaquaductal gray) were located mainly in laminae V of the dorsal horn. These cells were distributed over all lumbar and caudal segments of the lumbar spinal cord. In contrast, neurons receiving nociceptive input from a hindlimb muscle (gastrocnemius-soleus (GS)) showed a distribution restricted to the segments L5-L2. Electrophysiology: Neurons processing nociceptive input from the soft tissues of the low back at the vertebral level L4 and L5 were found predominantly in the segments L2-T13, i.e. several segments more cranially than the location of the receptive fields [1]. In animals with intact soft tissues, approximately 5% of all cells had input from the TLF. In rats in which a tonic-chronic inflammation of the multifidus muscle had been induced, the proportion rose significantly to 15%. In segment L3 - that does not normally receive input from the TLF - more than 10% of the cells responded to input from the TLF in myositis animals. Interestingly, after induction of the myositis the increase in the proportion of neurons responding to input from the TLF was higher than that of cells responding to input from the inflamed muscle.

CONCLUSIONS: Dorsal horn neurons receiving input from the soft tissues of the low back and projecting to higher nociceptive centres have a more extensive distribution in the spinal cord than has the GS muscle (cFos data). This could explain the diffuse nature of the LBP and, together with the high innervation density of the TLF, the higher prevalence of LBP compared to chronic pain in limb muscles. Under pathological conditions (myositis) the input to dorsal horn neurons from the TLF became particularly prominent. Collectively, our results suggest that the TLF is an important source of nociceptive input in chronic LBP patients.

REFERENCE