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Central sensitization in tension-type headache--possible pathophysiological mechanisms.

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Abstract

The aim of the present thesis was to investigate the pathophysiology of chronic tension-type headache with special reference to central mechanisms. Increased tenderness to palpation of pericranial myofascial tissues is the most apparent abnormality in patients with tension-type headache. A new piece of equipment, a so-called palpometer, that makes it possible to control the pressure intensity exerted during palpation, was developed. Thereafter, it was demonstrated that the measurement of tenderness could be compared between two observers if the palpation pressure was controlled, and that the Total Tenderness Scoring system was well suited for the scoring of tenderness during manual palpation. Subsequently, it was found that **pressure pain detection and tolerance thresholds were significantly decreased in the finger and tended to be decreased in the temporal region in chronic tension-type headache patients compared with controls. In addition, the electrical pain threshold in the cephalic region was significantly decreased in patients. It was concluded that the central pain sensitivity was increased in the patients probably due to sensitization of supraspinal neurones.** The stimulus-response function for palpation pressure vs. pain was found to be qualitatively altered in chronic tension-type headache patients compared with controls. The abnormality was related to the degree of tenderness and not to the diagnosis of tension-type headache. In support of this, the stimulus-response function was found to be qualitatively altered also in patients with fibromyalgia. It was concluded that the qualitatively altered nociception was probably due to central sensitization at the level of the spinal dorsal horn/trigeminal nucleus. Thereafter, the prophylactic effect of **amitriptyline, a non-selective serotonin (5-HT) reuptake inhibitor, and of citalopram, a highly selective 5-HT reuptake inhibitor, was examined in patients with chronic tension-type headache. Amitriptyline reduced headache significantly more than placebo, while citalopram had only a slight and insignificant effect. It was concluded that the blockade of 5-HT reuptake could only partly explain the efficacy of amitriptyline in tension-type headache, and that also other actions of amitriptyline, e.g. reduction of central sensitization, were involved.** Finally, the plasma 5-HT level, the platelet 5-HT level and the number of platelet 5-HT transporters were found to be normal in chronic tension-type headache. On the basis of the present and previous studies, a pathophysiological model for tension-type headache is presented. **According to the model, the main problem in chronic tension-type headache is central sensitization at the level of the spinal dorsal horn/trigeminal nucleus due to prolonged nociceptive inputs from pericranial myofascial tissues. The increased nociceptive input to supraspinal structures may in turn result in supraspinal sensitization. The central neuroplastic changes may affect the regulation of peripheral mechanisms and thereby lead to, for example, increased pericranial muscle activity or release of neurotransmitters in the myofascial tissues. By such mechanisms the central sensitization may be maintained even after the initial eliciting factors have been normalized, resulting in the conversion of episodic into chronic tension-type headache.** Future basic and clinical research should aim at identifying the source of peripheral nociception in order to prevent the development of central sensitization and at ways of reducing established sensitization.

This may lead to a much needed improvement in the treatment of chronic tension-type headache and other chronic myofascial pain conditions.

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