PAIN RESEARCH AND MANAGEMENT

Tinnitus: A pain syndrome in search for good therapy

Tinnitus is de ned as a phantom auditory perception, namely perception of sound without corresponding vibratory, mechanical activity in the cochlea. It is now evident that the pathology that causes most forms of tinnitus is in the central nervous system where some abnormal neural activity is generated and interpreted in a similar way as activity generated whe sound reaches the ear. Hearing loss is believed to trigger the perception of tinnitus in the central auditory system. However, there is no specilot chearing loss associated with the occurrence of tinnitus. Most forms of bilateral tinnitus are caused by abnormal neural activity in the central nervous system without damage of the ear. It is concept of somatic tinnitus is derived from observations that tinnitus can be evoked or modiled by somatic manoeuvres, and that tinnitus can develop acutely a er somatic insults to the face, head or neck. Extensive morphological and physiological evidence suggests that somatosensor auditory interactions in the nucleus cochlearis play a significant role in somatic tinnitus. It is derived to the spinal nerve at the cervical level and the trigeminal ganglion contain the primary somatosensory neurons that project to the nucleus cochlearis. Although neural plasticity is involved in the pathogenesis of tinnitus, there is also a group of patients with tinnitus who can be of vascular origin. Tinnitus patients with a hearing loss greater than 22 dB at 250 Hz were found to be good candidates for a reduction of tinnitus with sympathetic blockade. Sympathetic bres innervating the cochlea play a role