Central Pain Conditions: From Mechanisms to Treatment

Troels S. Jensen

Dept. of Neurology and Danish Pain Research Center, Aarhus University Hospital, DK-8200 Aarhus N, Denmark.

Central pain (CP) represent different neuropathic pain conditions, where lesions of somatosensory pathways in the central nervous system (CNS) are assumed to be responsible for the pain. CP are - as their peripheral counterparts - characterised by pain in those body parts that corresponds to the brain or spinal cord territories, whose function has been disrupted by the CNS lesion. The distribution of pain in CP represents only a fraction of the area with reduced sensation, indicating that deafferentation is necessary for the pain. The occurrence of evoked pain, summation and aftersensations in the same body parts with reduced sensory discrimination indicates that hyperexcitability in addition to hyposensitivity also is a manifestation of CP. These findings have led to the proposal that partial or complete loss of afferent input result in hyperexcitability in certain neuronal pools and hence pain. The etiology of central neuropathic pain are multiple and includes pains caused by stroke, by spinal cord injury, by immunological disorders such as multiple sclerosis and transverse myelitis.

Treatment trials in patients with central pain may contribute to unravel mechanisms underlying these pain conditions. Tricyclic antidepressants has a pain relieving effect which may be related to a restoration of ascending monoaminergic input to the thalamus. Lidocaine an unspecific sodium channel blocker has in double-blind controlled trials shown efficacy on pain and evoked abnormality in patients with post stroke pain and in spinal cord injury pain. Lamotrigine a sodium channel blocker is efficacious in post stroke pain and possibly in incomplete spinal cord injury. Gabapentin and pregabalin both antihyperalgesics are likewise effective in relieving pain in spinal cord injury pain. Cannabinoids have a pain relieving effect in MS.