GUESTEDITORIAL

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To MD To neuroscientist To patient To therapist

Thalamic Sensory Syndromes

The role of the thalamus in sensation, as suggested by Luys in 1865 (Cambier, 1982), was finally confirmed by the classic paper of Dejerine & Roussy (1906), in which the appearance of sensory disturbances in the course of lesions of the ventral posterior nucleus of the thalamus was described. The ventral posterior nucleus seems to be organized into different functional part. It has been shown that its somatic afferents are somatotopically organized, with tongue, face, and fingers having a large representation in the medio-ventral portion of the nucleus (VPM), whereas trunk and legs are represented more dorsolaterally (VPL). The varying contributions of single areas of the thalamus to the elaboration of single modalities of sensation coming from different body regions explains the variety of sensory stroke syndromes reported. Depending on the extents of ventrolateral thalamic stroke, sensory disturbances can be isolated or can be associated with ataxia and hemiparesis, alone or in combination (Schmahmann, 2003).

The most typical clinical presentation of sensory disturbances due to thalamic stroke is represented by the thalamic syndrome of Dejerine and Roussay. Patients often report paraesthesias, numbness, heaviness of the involved area, or pain, but occasionally

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can be unaware of a profound sensory loss. The hemi-hypoesthesia typically involves the entire hemibody, including the trunk, although often accentuated periorally and distally on the upper limb. Sensory loss is more or less pronounced for spinothalamic modalities of sensation and usually is severe for lemniscal modalities.

A pure sensory stroke should be diagnosed only in the presence of isolated sensory disturbances involving at least two body areas and it is suggestive of, but not specific for, thalamic stroke. Besides the involvement of three body areas together, partial sensory syndromes have been described. All modalities of sensation can be affected, but a dissociated loss is rather common (Carrera *et al.*, 2004). Cytoarchitectural studies have demonstrated that anatomical separation of the spinothalamic and dorsal column/medial lemniscus sensory modalities in the thalamus. In fact, patients with pure hemisensory loss consisting of impaired dorsal column modalities (proprioception, stereognosis, graphaesthesia, two-point discrimination) in the absence of lateral spinothalamic dysfunction (pain, temperature, light touch) upon clinical examination were described (Paciaroni & Bogousslavsky, 1998).

It is described that, when thalamic infarct is limited to the posterior portion of the ventral nuclei, patients report numbness and paraesthesia, and when infarcts are larger and affect the fibers passing from the medial and lateral portions of the thalamus to the cortical sensory zones, sensory loss is more severe and that “thalamic pain” often develops later (Caplan *et al.*, 1988). Central pain is often described in terms both of thermal stimuli, such as burning, and of pain evoked by cold or tactile stimuli that are not normally painful (allodynia). It usually appears after a latency of weeks or months (rarely years) and can be felt in the upper or lower limb, the face, the tongue, hand, and foot together, or the entire

hemibody. The identification of a thalamic nucleus specific for pain sensation that is cytoarchitectonically located in the posterior thalamus, could also explain this thalamic pain syndrome (Craig *et al.*, 1994, Kim *et al.*, 2007). The mechanisms behind thalamic pain are not known. Partial cortical deafferentation due to impairment of spinothalamic afferents seems to be crucial for its development (Willis & Westlund, 1997). Another hypothesis proposes that pain is due to a lesion that removes an inhibition exerted by the medial lemniscal pathways on neurons in the thalamus and cortex involved in the perception of pain (Boivie *et al.*, 1989).

Thalamic pain is a poorly recognized entity that can interfere with rehabilitation, reduce the quality of life, and interfere with the activities of daily living and recreational activities. Treatment options for thalamic pain are limited; at present, amitriptyline is the drug of first choice. Other drugs including antidepressants, anticonvulsants, antiarrhythmics, opioids and N-methyl-d-aspartate antagonists may provide relief for some patients who do not respond to amitriptyline (Hansson, 2004). Progress has also occurred in thalamic pain treatment with motor cortex stimulation (MCS), which probably opens a period of neuro-modulation of the cortical areas controlling pain. A better understanding of the MCS mechanism of action will probably make it possible to adjust better the stimulation parameters. The conclusions of multicentered randomised studies, now in progress, will be very useful and are likely to promote further research and clinical applications in this field.

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