The thalamus and cortex are more critical to migraine pathophysiology than the trigeminal nerve

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Migraine is central nervous system disorder and characterized by a severe lateralized pain in the ophthalmic branch of trigeminal nerve, occipital nerve and upper cervical root distribution. Headache attacks are preceded and or accompanied by alterations in sensory perception such as photophobia, phonophobia, osmophobia, and allodynia. A cerebral cortical phenomenon known as cortical spreading depression (CSD) was linked to lateralized headache and shown to be able to activate peripheral trigeminal fibers and second order trigeminal neurons in the brainstem nucleus (TNC) (Bolay et al, 2002). By activating trigeminovascular system, CSD is implicated in releasing CGRP and nitric oxide from trigeminal nerve endings and leading to neurogenic inflammation in the dura mater. CSD is a key to understand familial hemiplegic migraine phenotype, critical involvement of glutamatergic synapse, female hormonal influence and the efficacy of preventive anti-migraine drugs (Eikermann-Haerter et al, 2009; Ayata etal, 2006) .CSD is able activate thalamic reticular nucleus (TRN) (Tepe et al, 2015). TRN consists of GABAergic neurons that surround the thalamus and mainly functions as a gatekeeper of sensory outflow to the cortex, which is involved in selective attention, lateral inhibition, and discrimination of sensory stimuli. Sensorial perception is altered and prolonged during migraine headache attacks (Boran et al, 2016). Disruption of temporal discrimination of two consecutive sensorial stimuli seems specific to migraine headache attacks and proposed to be a neurophysiological marker for migraine (Vurallı et al, 2016, Vurallı et al, 2017). Research indicate a cortical alterations and dysfunctional thalamocortical oscillations take a role in activating ipsilateral brainstem pain structures and trigeminal pain nuclei. Migraine syndrome is not a trigeminal nerve mediated peripheral headache and the cerebral cortex and the thalamus have prime importance for migraine pathophysiology.