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Can the symptoms associated with migraine provide information on brain structures triggering the migraine attacks?

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Introduction

The structures that trigger the migraine attack have been enigmatic for decades. The concepts contending that a migraine attack begins in the brainstem have been described by Burstein et al. (2012) and others stating that it starts in the cortex have been described by Goadsby et al. (2009).

Keywords

Migraine, associated symptoms, cortex, brainstem.

Purpose

To determine whether the above concepts are still valid today and if migraine associated symptoms: photophobia, phonophobia, nausea and vomiting can provide us with information about brain structures triggering the migraine.

Material and methods

The material was synthesized proceon the basis of international articles – randomized studies, cohorts, clinical cases and others over the past 20 years and some older articles to clarify the onset of a migraine attack and concrete mechanisms „starting” this process of pain and suffering. The PubMed database was used in order to select the data from the literature.

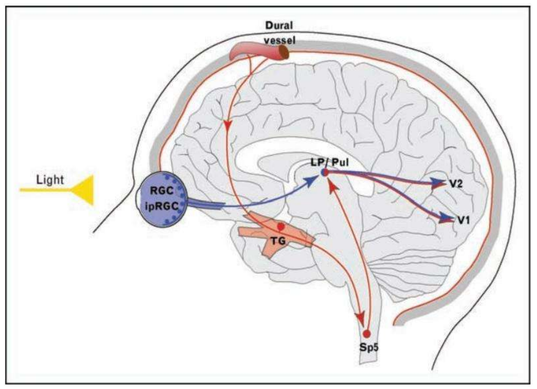
Results

A possible perception of light due to intrinsically photosensitive retinal ganglion cells (ipRGCs) containing melanopsin photopigment has been identified. Noseda et al. determined that ipRGCs project directly to the thalamic neurons processing and transferring both visual and nociceptive signals to the somatosensory association cortex. The studies on phonophobia using magnetic resonance imaging (MRI) showed a significant activation of the temporal lobe and the cuneus. In the study conducted by Maniyar et al. on the pathogenesis of nausea and vomiting associated with migraine the results demonstrated the involvement of the nucleus tractus solitarius located in the brainstem.

Conclusions

This analysis confirmed the hypothesis that the brainstem remains to be the anatomical structure responsible for triggering the migraine in patients with a prevalence of nausea and vomiting but the cortex remains to be responsible for it's triggering in patients having photophobia and phonophobia.

Fig. 1. Proposed mechanism for enhanced sensitivity to light during migraine through the convergence of nociceptive signals from the meninges on thalamic neurons that project to the visual cortices. (Noseda, R., & Burstein, R. (2011)).



Red depicts the trigeminovascular pathway. Blue depicts the visual pathway from the retina to the visual cortex. ipRGCs, intrinsically photosensitive retinal ganglion cells; LP, lateral posterior nucleus; Pul, pulvinar; RGCs, retinal ganglion cells; V1, primary visual cortex; V2 secondary visual cortex; Sp5, spinal trigeminal nucleus; TG, trigeminal ganglion.

Fig. 2. Potential relationships between pain, nausea and other associated features. (Kelman, L., & Tanis, D. (2006)).

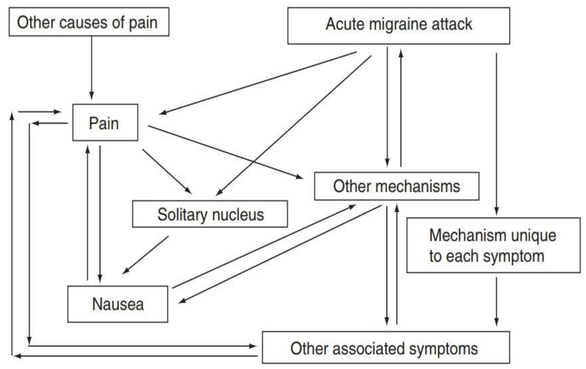
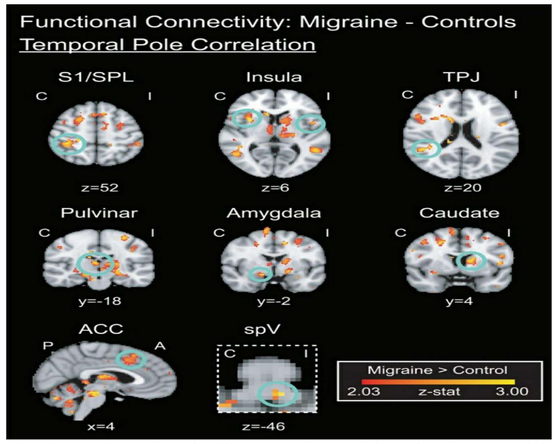


Fig. 3. Functional connectivity contrast of the anterior Temporal Pole during intermittent heat stimuli (pain threshold þ1 C) in interictal migraine patients—controls. (Moulton, E. A., Becerra, L., Maleki, N., Pendse, G., Tully, S., Hargreaves, R., ... Borsook, D. (2010)).



The TP in interictal migraine patients has significantly enhanced functional connectivity within areas commonly activated by experimental pain, as well as in multimodal sensory processing areas. A, anterior; ACC, C, contralateral; I, ipsilateral: P, posterior, S1, primary somatosensory cortex; SPL, superior parietal lobe; spV, and TPJ, temporoparietal junction.