▪ Headache Medicine 2023, Supplement ▪ p-ISSN 2178-7468, e-ISSN 2763-6178

Headache Medicine

Theories About Migraine Aura During History: an Overview

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Categoria: Cefaleia na História e nas Artes

Introduction
The pathogenesis of migraine aura remains unclear, but is clinically dominated by one feature: the gradual spread of symptoms, usually over 5-20 min for each symptom. Many descriptions were made during time, from vasospastic theory and cortical ischaemia hypothesis to cortical spreading depression (CSD), described by Leão in 1944, which has been in focus since the 1980s, ally to new studies, like spreading oligaemia during migraine aura and subsequent discoveries of brainstem mechanisms.

Objective
To describe theories about migraine aura during history.

Methods
An integrative survey rose the data that was provided from the following scientific newspapers: PUBmed, BVsalud, EMBASE and CAPES. Once in these newspapers, the words 'migraine aura', 'theories', and 'history' were searched, combined with the boolean operator 'AND', providing the produced papers, without specified date, in english, spanish and portuguese. Furthermore, the original articles from the authors of the theories in this study were included. An overview was made.

Results
In 1941, Lashley published some observations of his own attacks of scintillating scotomas. He summarized his observations as follows: 'Maps of the scotomas of ophthalmic migraine sketched at brief intervals during an attack suggest that a wave of intense excitation is propagated at a rate of about 3 mm. per min. across the visual cortex. This wave is followed by complete inhibition of activity, with recovery progressing at the same rate'. In 1944, Leão described how EEG activity was successively depressed in different channels depending on their distance from an electric stimulus. Afterwards, he described that a wave of marked arterial dilation and increased blood flow in the pial vessels traveled simultaneously with the wave of CSD over the cerebral hemisphere. In 1945, Leão and Morrison proposed that CSD may be related to migraine with aura (MA) because of the slow development of scotoma and sensory symptoms. In 1969, Skinhøj and Paulson used the xenon-133 intracarotid technique with 16 detectors to investigate two patients. During the aura phase in one patient there was a reduction in regional cerebral blood flow (rCBF) of up to 66% compared with before the aura, a level known to be critical for normal oxygenation. The prerequisite for a more precise characterization of rCBF during migraine was the development by Lassen, with six patients with MA, the attacks were initiated by focal hyperaemia in three cases, and during the aura phase all patients developed rCBF reduction (oligaemia), which only in one case approached critical values. Oligaemia gradually spread anteriorly in the course of 15-45 min. In four cases severe headache was present concomitantly with oligaemia. Inspired by the rCBF results, Lauritzen investigated in 1982 CBF in rats during and in the wake of CSD by quantitative autoradiography. Cortical blood flow increased 218% during the CSD wave, but, more importantly, it decreased 15-27% after the hyperaemia and for more 1 hour after CSD. The changes in blood flow were largely limited to the cerebral cortex. After 1990, the CSD elicited by KCl significantly increased cell staining (c-fos protein-like immunoreactivity) within ipsilateral trigeminal nucleus caudalis (TNC). This was the first report demonstrating that neurophysiological events within the cerebral cortex can activate brainstem regions involved in pain processing via a trigeminovascular mechanism. In 2001, the study by Hadjikhani indicated that an electrophysiological event such as CSD generated the aura in the visual cortex.

Conclusion
Despite the theories are inconclusive about the migraine aura, the most relevant studies make an association between CSD and the rCBF. So many more studies are in process to elucidate this question on neuroscience.

Keywords: Migraine aura; Theories;History; Cortical spreading depression; Cerebral blood flow.