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STUDY OF CEREBRAL HEMODYNAMICS IN PATIENTS WITH MIGRAINE

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Abstact: Headache is one of the most common ailments; migraine is one of the most prevalent and disabling neurological disorders and cluster headache presents as one of the most excruciating pain disorders. Both are complex disorder characterized by recurrent episodes of headache. A key feature is that various triggers can set off an attack providing the opportunity to explore disease mechanisms by experimentally inducing attacks. This review summarizes neuroimaging and hemodynamic studies in human in provoked and spontaneous attacks of migraine and cluster headache.

Keywords: Brain imaging, cerebral hemodynamics, cluster headache, headache provocation, migraine.

Cerebral hemodynamics during different phases of the migraine attack demonstrate alterations in cerebral blood flow and perfusion, vessel caliber, cortical and sub-cortical function, underscoring that migraine pathophysiology is highly complex. Migraine attacks might begin in diencephalic and brainstem areas, whereas migraine aura is a cortical phenomenon. In cluster headache pathophysiology, the hypothalamus might also play a pivotal role, whereas the pattern of cerebral blood flood differs from migraine. For both disorders, alterations of arterial blood vessel diameter might be more an epiphenomenon of the attack than a causative trigger.Studying cerebral hemodynamics in provocation models are important in the search for specific biomarkers in the hope to discover future targets for more specific and effective mechanism-based anti-headache treatment.[1]

CH is characterized by (almost) strict unilateral headache with prominent cranio-autonomic features, such as pupillomotor dysfunction, ptosis, lacrimation, conjunctival injection, rhinorhea, nasal congestion, ear fullness and others. The main dynamic feature of CH is that it occurs in “clusters,” i.e. not equally distributed over time. Typically, in the beginning, patients with CH have periods of recurring, often daily attacks over several weeks (“bouts”) followed by months or years of freedom from attacks (“out of bout”). Moreover, during bouts, patients suffer from recurring attacks, which often occur on a daily basis, and at the same time during the day. From a clinical perspective, thus, an internal clock might be sought turning on single CH attacks and/or switching between in-bout and out of bout. Migraine and CH are characterized by recurrent headache attacks of essentially different, but overlapping pathophysiology.[3] To study the individual, in general unpredictable, attack, provocation studies assessing cerebral hemodynamics and neurovascular coupling are necessary and will be reviewed here in detail. Most such studies were on migraine, for which reason migraine will be covered most, but CH will be included as well.Premonitory symptoms thus appear to be the first clinical trace of the migraine attack,[6] some of them persisting into the headache or even postdromal phase, others disappearing with the onset of head pain. Understanding the generation of the premonitory phase

therefore might offer insights on how and where migraine attacks may begin.[2] Functional brain imaging would be the method of choice, but the unpredictability of the individual attack in episodic migraine in the context of substantial logistic expense would make such approach in particular difficult. At least three different approaches are conceivable and have been followed: triggering of premonitory symptoms (and its subsequent migraine attacks), testing of several patients at random time points with prospective diary-based identification of the time point within the migraine cycle, and sequential examination of individual patients on a regular basis with catching the premonitory phase "by accident."[4]

In summary, the earliest clinically comprehensible phase of the migraine attack, the premonitory phase, might be a key to our understanding of migraine pathophysiology. Being able to provoke attacks using GTN as well as scanning during various time points of the migraine cycle has proven useful in identifying the hypothalamus as a likely structure driving the individual attack. Further studies investigating what in turn might regulate the hypothalamus, be it hormonal, vascular, more complex events such as CSD, would be a next step to deepen our understanding of migraine pathophysiology.

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