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Editorial Commentary

Sensitivity to light and noise in tension-type and cervicogenic headache

Attempts to identify discrete sets of symptoms which define clinically recognized headache syndromes in the migraine-tension headache spectrum have largely met with failure, primarily because many patients describe features of both types of headache (1). The substantial overlap of clinical symptomatology suggests that similar mechanisms are active during different types of headache, perhaps developing secondarily in response to a primary pathophysiological process.

In this issue of *Cephalalgia*, Vanagaite Vingen and Stovner report that patients with tension-type or cervicogenic headache were more sensitive to light and sound than headache-free controls, even when patients were tested during the headache-free interval. In a series of related studies, Vanagaite Vingen and colleagues reported similar findings for patients with migraine (2, 3). In addition, patients with cluster headaches were unusually sensitive to light and sound during bouts but not during the remission period between bouts (4). Painful stimulation of the head intensifies visual discomfort in migraine sufferers but not controls (5, 6), suggesting that mechanisms that normally suppress photophobia are disrupted during the headache-free interval as well as during migraine. The persistence of phonophobia in various forms of headache implies that a similar process modifies sensitivity to sound; however, experimental evidence to support this view is lacking (5).

The message to come from the series of papers by Vanagaite Vingen and colleagues is that symptoms such as photophobia and phonophobia are not specific enough to be useful for differential diagnosis of headache syndromes. This point is particularly important for tightening the diagnostic criteria for cervicogenic headache, where many of the symptoms may arise because of convergence of pain impulses on a "final common pathway" that is active during other forms of headache (7).

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PD Drummond

QEEG in migraine without aura

De Tommaso and co-workers provide interesting data on spontaneous and visually evoked EEG in migraine without aura (this issue). Most importantly, they recorded EEG both during headache and in a pain-free phase. Using blinded data analysis, the authors found consistent changes in brain electrical activity during headache, i.e., slowing and asymmetry of the posterior alpha peak frequency. In addition, they observed increased left temporal theta power during pain (the majority had left-sided symptoms). A large proportion (14 of 16 patients) had slowing outside the control group range, thus EEG spectral analysis (QEEG) during headache can possibly be used as a marker of migraine. It remains for this to be confirmed in studies with larger control and patient groups.

The authors found reduction (normalization) of photic driving amplitude during headache. Both background EEG frequency slowing and visually evoked EEG power reduction can reflect cortical inhibition in the late headache phase, while increased theta power tentatively suggests that a different pattern of reactivity is found in the temporal lobe. Whether this presumed cortical inhibition was triggered by a wave of depolarization, as suggested by the authors, or by another (e.g., vascular) process cannot definitely be answered by EEG studies alone. It should be noted that these results do not necessarily imply that synaptic inhibition is excessive.

QEEG is certainly more objective than conventional EEG interpretation. Still, a number of possible pitfalls have to be avoided, e.g., epoch selection bias, state fluctuations, non-cerebral electrical generators, and electrode artifacts. The method is mainly useful in research, and it is generally recommended to use QEEG only in conjunction with visual EEG interpretation performed by a skilled physician (1).

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T Sand

Endothelin in migraine patients

In 1991, Edmeads suggested the involvement of the endothelial derived constricting factor, endothelin, and its endothelial counterpart, nitric oxide (NO), respectively, in the pathophysiology of vascular changes (vasoconstric tion and subsequent vasodilation) during migraine attacks (1).

The first demonstration of increased levels of ET-1 during migraine attacks by Färkkilä et al. (2) was confirmed successively by our group (3) both in migraine patients with and without aura (3). In our study the significant rise of this vasoconstrictive peptide in plasma persisted also between 4 and 6 h from the onset of attacks. In the present research, a rise of endothelin in plasma was also found ictally, with the highest values occurring in the first 2 h after the start of the migraine attack. Unlike our study, the values of ET-1 fell 3–4 h after the beginning of attacks, even to values lower than those measured interictally.

Several factors putatively involved in the early events of the migraine attack increase the production of endothelin, such as serotonin, norepinephrine, hypoxia, arachidonic acid and its by-products or by-products of cyclooxy-genase activity. On the other hand, the higher levels of endothelin might be the consequence of hemodynamic shear stress in the vasodilatory phase. This may represent a physiological vasoconstricting mechanism counterbalancing the abnormal vasodilation due to the production of sensory neuropeptides by trigeminovascular activation, or to the local release of prostacyclin and NO. However, endothelin itself can stimulate the NO.

A role for ET-1 in mediating neurogenic inflammation has been proposed (4). However, the endothelin antagonist bosentan that blocks expression of neurogenic inflammation was not effective in aborting migraine attacks (5). The mode of action of the 5HT-agonists might suggest that they could also exercise their vasoconstrictive action by blocking endothelin production and release. In light of this, ET-1 could be considered a "natural migraine compound".

This is the first attempt to study the effect of 5HT abortive migraine drugs on ET-1, although results are not conclusive. A definite answer regarding the source of the ET-1 rise during migraine attacks could be obtained if blood samples were drawn from the jugular blood. Further research is also needed to investigate the sequential production of vasoactive substances with vasoconstricting effects (primarily endothelin) and vasodilating effects (trigeminovascular neuropeptides, NO, prostacyclin) and the relationships between their peripheral and jugular venous blood levels to hemodynamic changes and any migraine attacks.

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C Sarchielli

Migraine in childhood and adolescence

In this issue of *Cephalalgia*, Gherpelli and co-workers report a study on headache in children and adolescents <15 years of age. The study focuses on migraine and reports two major findings; on the one hand the relationship between age and migraine type, headache characteristics, and associated symptoms of the International Headache Society (IHS) classification, on the other the sensitivity and specificity of the IHS criteria of migraine without aura. Similar to earlier reports, the prevalence of aura, pulsating quality, and unilateral location was higher in older children. Sensitivity was >70% for duration, pain intensity, aggravation with physical activity, photophobia, and phonophobia. Specificity was >70% for aggravation with physical activity and all concomitant symptoms. Even though duration of 2–48 h 5HT had a good sensitivity, almost 60% of children with a migrainous disorder (IHS 1.7) had a headache duration of <2 h. Accordingly, Gherpelli and co-workers propose to withdraw the duration criterion of migraine without aura completely.

Since 1988, the IHS criteria have been applied in a series of population- and clinic-based studies on headache in childhood. The majority of these studies have focused on migraine and only a few have included tension-type or other headaches. In total, the studies comprise more than 1,800 (range 25–395) children suffering from migraine. The studies were performed in several European countries, in Canada and in the United States. The work by Gherpelli et al. adds important data from a South American country, Brazil.

Including the authors of the current paper, eight research groups (1–8) have made specific proposals regarding a forthcoming revision of the current IHS criteria for migraine in childhood. Seven of the eight groups have proposed a reduction in the required minimum duration of childhood migraine from 2 to 1 h. Four groups have proposed changes to the unilaterality criterion, reflecting the observation that many children suffering from migraine give a bilateral fronto/temporal or median frontal location. Another four groups have proposed revisions of the photophobia/phonophobia criterion. Single proposals include revising the minimum number of attacks, intensity, aggravation with physical activity, and nausea. None of the groups have suggested revisions of quality, vomiting, or aura.

The literature on the IHS criteria for childhood migraine is enriched by this well-written paper by Gherpelli and co-workers. The international childhood headache community would appreciate hearing more from our Brazilian colleagues, and other researchers throughout the world.

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C WÖBER-BINGÖL