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Editorial Commentary

Visual disturbances in migraine

In 1976, Barrett et al. (1) reported that the average evoked response to checkerboard stimulation of one-half of the visual field is recorded maximally from midline electrodes and electrodes placed over the hemisphere ipsilateral to the field of stimulation. To account for this surprising finding, they postulated that ipsilaterally sited electrodes would be best placed to detect activity in cortical generator areas on the medial and posteromedial surfaces of the contralateral visual cortex. In this issue of *Cephalalgia*, Shibata, Osawa and Iwata report that the amplitude of visual evoked potentials to a checkerboard pattern was greater in migraine with aura patients at the mid-occipital electrode than in migraine without aura patients or non-headache control subjects; furthermore, the amplitude of the visual evoked potential was greater contralateral to the visual aura than ipsilaterally in migraine with aura patients. Electroretinograms to the checkerboard pattern were normal in migraine patients, ruling out abnormalities at the retinal level. If these findings are interpreted in relation to the hypothetical location of cortical generator areas stimulated by the checkerboard pattern, they are consistent with the notion that residual neurological deficit persists between attacks of migraine with aura in the symptomatic part of the visual cortex. This deficit apparently induces comparative neuronal silence at the source of the visual aura but is surrounded by a zone of hyperactivity, perhaps resulting from damage to cortical inhibitory processes, that increases the amplitude of midline evoked potentials.

It seems unlikely that the same mechanism would contribute to photophobia, because this feature develops during attacks of migraine irrespective of the presence of a neurological prodrome. In this issue of *Cephalalgia*, Vanagaite et al. report that light-induced discomfort and pain intensified during attacks of migraine, but also persisted at moderate intensity during the headache-free interval. Neither the discomfort nor pain thresholds during the headache-free interval bore any relation to migraine features (i.e., pulsating pain, neurological prodrome, headache intensity or presence of gastrointestinal disturbances). More than one-third of patients thought that photophobia typically was greater on the painful side; nevertheless, sensitivity to light was similar on the symptomatic and non-symptomatic sides in 15 patients studied during an attack of unilateral migraine. This finding is at odds with a previous report that linked intensity of photophobia to trigeminal discharge during headache (2). To investigate the laterality of photophobia, Vanagaite et al. stimulated the right and left eyes sequentially, always starting with the right eye. Since sensitivity to light increased with repeated stimulation, the left eye was consistently more sensitive than the right; thus, an order effect may have masked pain-linked asymmetry of photophobia in some cases. The increase in sensitivity to light with repeated stimulation points to cumulative fatigue of an inhibitory subcortical process that normally suppresses sensations of glare and light-induced pain (3). Perhaps fatigue of this inhibitory mechanism increases sensations of photophobia in the presence of persistent trigeminal discharge during migraine headache, and heightens vulnerability to intense visual stimulation during the headache-free interval (4).

In sum, these two papers focus attention on cortical and subcortical processes which could contribute to visual disturbances in migraine. Finding out more about the source of these disturbances is important, because they might increase susceptibility to migraine and heighten discomfort during attacks.

References

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Power spectrum analysis on heart rate and diastolic blood pressure variability

Basic autonomic nervous system (ANS) abnormality in migraine during the inter-headache phase has been reported by several investigators. A decrease in the valsalva overshoot, orthostatic hypotension, as well as low plasma norepinephrine levels that did not increase in head-up tilting and a long recovery time after norepinephrine bolus injection were demonstrated in migraine. Other studies have shown iris muscle hypersensitivity, higher resting blood flow to the hand and a decreased sensitivity of beta-adrenergic receptors, all of which are compatible with sympathetic impairment.

In order to obtain quantitative information on sympathetic malfunction in migraine, spectral analysis of beat-to-beat fluctuations in heart rate (HR) as a non-invasive probe was utilized.

In a well-designed controlled study, Pierangeli et al. failed to show any abnormality regarding cardiovascular response to the tilt test or the Valsalva manoeuvre. Moreover, their study of power spectral analysis of heart rate and diastolic blood pressure in the supine position and during passive tilting was comparable to that of the control group. However, in previous studies using the power spectrum analysis of HR variability on 24-h ECG, enhanced low frequency HR fluctuations have been found, strongly suggesting sympathetic instability. Other HR studies have suggested sympathetic hypofunction. These conflicting data are hard to explain. On the one hand, the evidence for autonomic impairment in migraine is plenty and several controlled studies using different