proportion of patients discontinuing treatment for this reason indicate that in future studies, more attention should be given to these problems. Substances with a longer life-time, or those affecting central mechanisms, do not necessarily offer a better solution, as potential side effects could be more pronounced or persist longer.

References

3. Dahlöf CGH. Unpublished data (with permission)

V Pfaffenrath

Photic driving in migraine

In this issue of Cephalalgia, the study by Puca et al. on steady state visual evoked potentials provides additional data confirming that hyperexcitability to light is a hallmark of the cerebral cortex in migraineurs between attacks. Since the seminal observation by Golla & Winter (1) of an increased photic drive of the EEG in migraineurs, cortical hyperreactivity to visual stimuli has been demonstrated with various methods, e.g. epidemiological surveys (2) functional tests (3, 4), steady state (5), flash (6) or pattern reversal (7, 8) evoked potentials. It is not clear whether the cortical hyperexcitability is due to lack of inhibition by intrinsic GABAergic neurons (4) or to an abnormal modulation of the cortex by subcortical (monoaminergic) pathways (9). It was hypothesized that the former, which postulates a loss of cortical interneurons, might be an acquired consequence of repeated insults (hypoxia/spreading depression) to the visual cortex, while the latter could be a genetic abnormality. One major interest of Puca et al.’s study is that they find a positive correlation between visual reactivity and family history of migraine or autonomic symptoms during the attack, which favors the hypothesis of an inherited, subcortical dysfunction. Unfortunately, the reliability of information concerning severity of attack-associated symptoms and affected family members can be questioned in the absence of diary card data and of family interviews. Because of their importance for migraine pathogenesis, the correlations found here need to be confirmed using these more stringent methods.

References


J Schoenen

Temporalis exteroceptive and tension-type headache

In this issue of Cephalalgia, Bendtsen et al. demonstrate that the temporalis exteroceptive silent period is not different in patients with chronic tension-type headache versus controls. While these results conflict with most previous reports (1–3), they are in accord with a recent study (4). Bendtsen et al. conducted a well-designed experiment using a blinded observer and a large sample of subjects, unlike previously reported studies. However, the methods used to measure ES1 and ES2 were different from those used by previous investigators. In particular, the duration used for most patients was 0.5 compared to the standard 0.2 (5). The authors also found a small, but significant decrease in ES2 in 10 patients when the stimulus duration was reduced to 0.2. This further strengthens the notion that a standardized method should be used by all laboratories for future studies in ES2. Also, the results should be corroborated by other investigators using blinded measures for observation. With the advent of magnetic stimulation, research in neurophysiology of headache may be corroborated by measuring both the interoceptive and exteroceptive silent period in patients with tension-type headache.

References


KMA Welch