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Visual evoked potentials in migraine

Abstract Migraine is a chronic disorder. Visual symptoms and hypersensitivity to light stimuli are common. The aim of this study is the analysis of visual system in migraineurs by visual evoked potentials (VEP). We studied 53 migraineurs (21 with prophylactic migraine treatment and 32 without preventive therapy) and 20 healthy control subjects. We found lower P100 latencies in migraineurs without therapy compared to controls. In treated patients, P100 latencies showed the same trend seen in the control group. We speculate a different responsiveness of the visual system in migraineurs probably due to a dysmodulation of sensor input leading to facilitation of visual processing.

Key words Migraine • Visual evoked potentials • Visual system

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Migraine is a common neurovascular disorder characterised by severe episodes of headache and autonomic-neurological symptoms. In clinical practice, we usually observe visual disturbances in migraineurs. The aura in migraine is usually visual (80–90% of cases). The majority of migraine attacks are accompanied by photophobia; approximately 60% of migraine attacks are caused by environmental light stimuli, like sunshine or restless and intermittent lights. There is also evidence that visual functions in migraineurs differ from those of normal subjects, even between attacks. Visual evoked potentials (VEP) are a functional option which facilitate assessment of the visual pathway. The presence of VEP changes was reported in previous studies but the results were often conflicting. In the headache-free interval, some authors showed difference of amplitude or latencies of P100 between migraineurs and healthy control subjects; others did not show any difference. With pattern reversal stimuli, Benna, Mariani, Drake, Tagliati and Polich were unable to demonstrate differences between migraineurs and controls [1–5]. In contrast, Kennard and Khalil showed a P100 latency increase in migraine patients between attacks, with a P100 amplitude increase in the patients studied by Khalil [6, 7]. Moreover, in an interictal study of sequential blocks of pattern-reversal VEP lasting 2 min, an amplitude increase over the time was shown [8]. This finding was explained as a potentiation of the response in patients as opposed to habituation in the control subject, in keeping with a similar study by Afra et al. where the stimulation lasted 15 min [9]. In 1999, Oelkers observed that habituation behaviour in migraine seemed to be affected in a complex way, depending on stimulating conditions, rather than being generally impaired [10].

The aims of the current study were to assess VEP changes in migraine and to evaluate the role of the preventive therapy in the balance of the visual functions.

We studied 73 subjects: 53 migraineurs recruited from a headache centre (48 females and 5 males, mean age 37.6 years) and 20 healthy control subjects (15 females and 5

males, mean age 34.6 years). Twenty-one migraineurs, 15 migraine without aura (MO) and 6 migraine with aura (MA), had a prophylactic migraine treatment and 32 (19 MO and 13 MA) had no preventive therapy. The drugs used were: beta blockers, antiepileptic drugs (sodium valproate), 5-HT receptor antagonists (pizotifen), calcium-channel blockers (flunarizine) and antidepressants (amitriptyline). The diagnosis of migraine was in accordance with the IHS classification. None of the patients had ocular disorders or other neurological diseases. The mean frequency of migraine attacks was 5 per month in MO and 1.5 per month in MA; they had a disease-history of about 15 years. Aura symptoms included visual (37% of patients), together with sensory (42%) and aphasic (21%) symptoms. The patients were studied at least 5 days after the last attack. All treated patients benefited from medication in terms of migraine frequency and pain intensity reduction. Recordings were performed by the same technician in standard conditions (in a quiet room with dimmed light, patients seated in an armchair, 1 m in front of a television monitor (mean luminance 240 cd/m^2)). Stimuli were presented as a checkerboard pattern of black and white squares subtending 4° 0.5' of arc (contrast 100%) at a reversal frequency of 3 Hz. With one eye patched, subjects were instructed to fixate on a point in the middle of the screen. The active electrode was inserted into the scalp in the midline over the occipital region, 2.5 cm above the inion. During uninterrupted stimulation, sequential blocks of 100 responses were averaged for a total duration of 9 min. We studied the amplitudes and latencies of the VEP components. We first compared VEP latencies and amplitudes between the right and the left eyes within each population. Because *t*-test revealed non-significant differences between the two eyes, we subsequently calculated mean values from both eyes for each subject. We estimated the variability of the responses in repeated stimulations with ANOVA for repeated measures test. We found that the P100 latencies did not change during the stimulation. Therefore, we used the 15 measurements of every group to obtain a much more reliable mean value; then, we used ANOVA one way test for group comparison. Migraineurs without preventive therapy presented lower P100 latencies compared with healthy control subjects; instead, the P100 latencies of treated migraineurs showed the same trend in the healthy control subjects (Fig. 1). In the group of migraineurs without prophylactic treatment, there was a trend toward a shorter latency in MA compared to MO (Fig. 2).

The observed decrease of P100 latency in patients with migraine is not easy to explain. Morphological changes in the optic nerves and the central pathways can be excluded because the three groups were well matched with regard to sex and age characteristics. This finding suggests a different responsiveness of the visual system in migraineurs due to a dysmodulation of sensor input, leading to facilitation of visual processing. With preventive migraine therapy, this difference disappears. Further study is necessary to understand this interesting topic and to make clear the differences between MA and MO visual responsiveness.

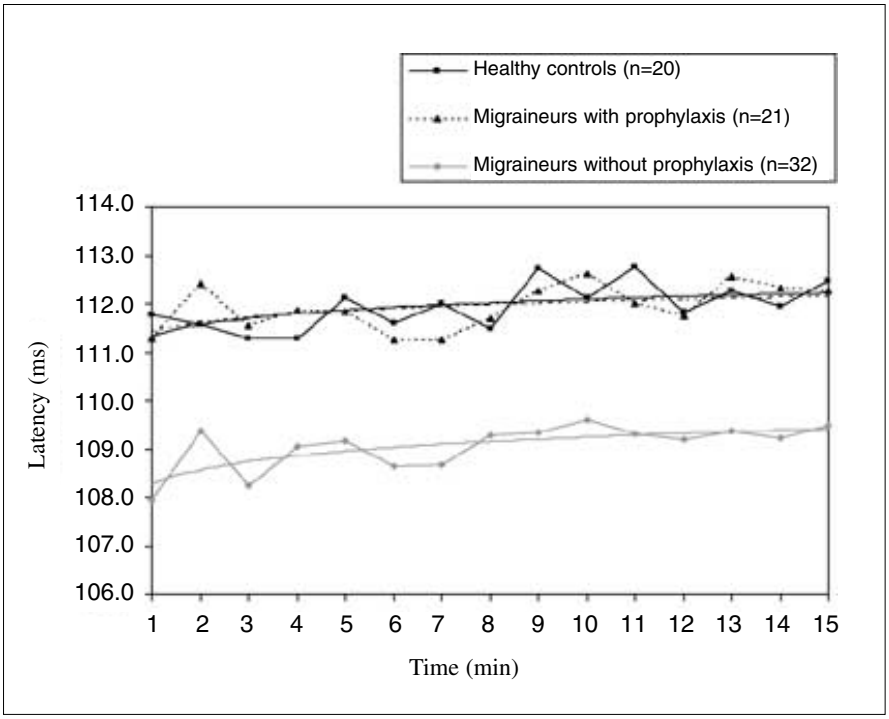

Fig. 1 P100 latencies in healthy control subjects, migraine patients without and with prophylaxis

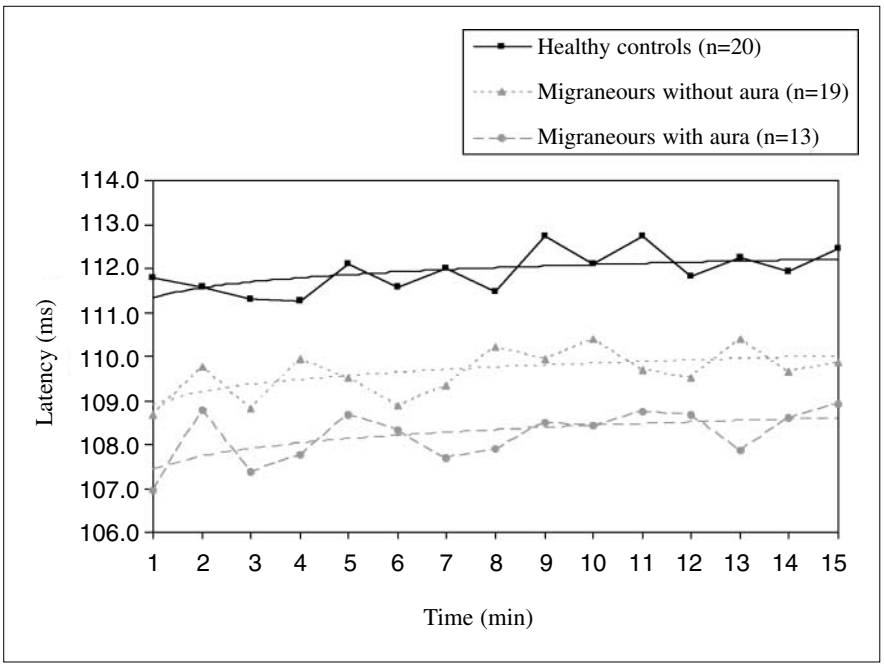

Fig. 2 P100 latencies in healthy control subjects, migraine patients without and with aura without prophylaxis

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