

Abnormalities of Visual Evoked Potential in Migraine Patients

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Abstract

Background: Visual evoked potential (VEP) is regarded as a useful, reliable and non-invasive technique for the diagnosis of lesions in the optic pathway. This technique was used to investigate visual function in migraine.

Materials and Methods: 53 migraine patients (27 migraine cases with aura and 26 common migraine cases) and 55 controls were prospectively enrolled in this study. Visual evoked potential using the pattern reversal stimuli was performed in all patients and controls, and abnormalities of visual evoked potential were evaluated.

Results: In subjects with classic migraine mean latencies of the P100 wave was increased by 6.7% ($P < 0.05$) compared with controls. Common migraine subjects did not show significant difference with controls in regard to P100 latency ($P > 0.05$). Also no significant difference was observed in P100-N140 peak-to-peak amplitude between the patients and controls.

Conclusion: The results of this study demonstrate subtle neuronal damage within the visual system of migraine patients which may be due to repeated transient ischemia experienced during the aura or more likely as a constitutional change.

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Introduction

Visual evoked potential (VEP) has been used in clinical neurology for the diagnosis of lesions in the optic pathway, especially in the diagnosis of multiple sclerosis¹.

Although alterations in Evoked Potentials can be due to marked anatomical changes, more subtle abnormalities have been reported in conditions without signs of overt neurological damage such as migraine.^{2,3} Visual stimuli can precipitate migraine attacks and most migraine auras are visual, suggesting specific involvement of the visual system in the pathophysiology of migraine. Abnormalities of visual Evoked Potential in migraine patients were first shown by Kennard et al using pattern reversal method⁴ and found that the latency of the major positive wave was greater and the amplitude larger than in a group of age-matched controls. In this study, we investigated

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Table 1: Mean P100 latency in the three tested groups

Groups	Mean P100 latency of Right eye (mSec)	Mean P100 latency of left eye (mSec)
Migraine with aura (MA)	118.56	118.38
Migraine without aura (MO)	112.06	111.92
Controls	110.10	111.05

the nature of the VEP abnormalities in migraine patients.

Materials and Methods

Subjects

The control group consisted of 55 healthy volunteers (29 women and 26 men) aged 15 to 48 years. The patient group was comprised of 53 migraine cases (44 women and 9men) ranging in age from 15 to 57 years. All subjects had a visual acuity normal or corrected normal, and none of them had any visual disorder. The patient group fulfilled the International Headache Society (IHS)⁵ criteria for diagnosis of migraine with or without aura. The patient group was divided into two subgroups: migraine patients with aura (MA) (20 women and 7 men) and migraine patients without aura (MO) (24 women and 2 men). All patients with MA had a visual aura. Migraine frequency varied between one or more attacks per week to one attack per year. None of the patients had experienced an attack during the week prior to testing. All participants were assessed using a headache questionnaire and a clinical neurological examination including fundoscopy, measurement of visual acuity and external ocular movements. The study was performed at the electroneurodiagnostic clinic of Nemazi hospital and objectives of the test were explained to all subjects prior to the test session.

Techniques

Subjects were encouraged throughout the tests to

maintain their interest and concentration. VEPs were performed by checker board pattern reversal displayed on a TV monitor subtending 15°x 12° at a viewing distance of 100 cm.

The stimulus reversal rate was 2 per second and individual squares in the checker board pattern subtended a visual angle of 60°. The subjects were monitored while stimulation was done monocularly. Standard disk EEG electrodes were placed at the OZ position of the 10-20 international system (active electrode), the reference electrode was placed at FZ position, and ground electrode on the patient's hand. Electrode impedance was less than 5 kOhms and stimulation was done in whole field. Two hundred individual trials were averaged and a repeated trial to verify reproducibility of the results was performed. The latency of the N75, P100 and N140 and the P100-N140 peak-to-peak amplitude was measured. Statistical analysis was performed using software (SPSS version 10) and significance was defined as P<0.05.

Results

A summary of the mean of P100 latency, P100-N140 amplitude of the subjects are presented in the Tables 1 and 2. A comparison of the mentioned groups revealed a significant difference between P100 latency of MA with the other two groups (P=0.00) (Figure 1). However, no significant difference was observed with respect to

Table 2: Mean peak-to-peak amplitude of P100-N140 wave in the three tested groups

Groups	Mean amplitude of P ₁₀₀ -N ₁₄₀ of right eye (mV)	Mean amplitude of P ₁₀₀ -N ₁₄₀ of left eye (mV)
Migraine with aura (MA)	4.79	5.06
Migraine without aura (MO)	4.04	4.18
Controls	4.95	4.16

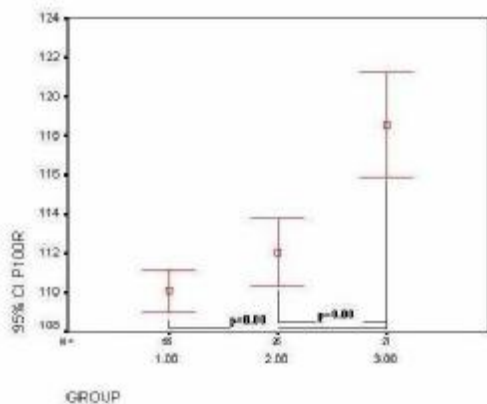


Figure 1: Relationship between mean P100 latency in the tested groups.

amplitude of P100-N140 ($P=0.284$).

P100 latency was 6.7% longer in subjects with classic migraine than controls ($P<0.05$) but the difference between patients with common migraine and controls in regard to P100 latency was 1.3% which was not statistically significant ($P>0.05$). There was no difference with respect to gender, in the P100 latency and amplitude of P100-N140. Also no statistically significant difference was observed between the right and left eye in each group.

Discussion

The most striking finding reported here, was longer P100 latency in migraine cases especially those with an aura. This is similar to the findings reported by Kennard et al⁴ who used checker board stimulation and found a prolonged P100 latency in migraine patients. Most previous studies of any size have confirmed prolongation of P100 latency^{4,6-8} but normal latencies have been reported in smaller surveys.⁹⁻¹¹ Also high P100 amplitudes were reported by some investigators which we were unable to confirm. The basis for the prolonged latencies in migraine is unclear. Kennard et al⁴ suggested that it may have a structural basis, due to ischaemic damage during repeated attacks. If this were so, a relation would have been expected between latency and duration of migraine which was not observed in the previous study.¹² Also, if relative cerebral ischaemia during a migraine aura can be considered as the cause, the abnormalities should be confined to MA, whereas our findings and those of a previous study¹² have shown some prolongation of P100 latency in MO. The possibility of the effect of ergotamine could still be considered, but if these were being exerted by recurrent is-

chaemia, one would expect that the change would be related to duration of migraine, in which case the results in MA and MO would be similar. Hyperexcitability of the brain in migraine might be the cause of the change in P100 latency and amplitude. Clinically, many patients with migraine are intolerant of noises and bright lights and some find these can even precipitate an attack. However, the neurophysiological correlate of the hyperexcitability could be the shorter latency and the higher amplitude of P100. Therefore, we suggest that the prolonged latencies are constitutional, perhaps due to synaptic delay.

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