Visual evoked response in transient monocular visual loss

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SUMMARY The pattern-reversal visual evoked response (VER) was recorded in 2 patients during transient monocular visual reduction. In both cases the VER was initially abolished. With recovery of vision there was gradual return of amplitude over a 3- to 8-minute period, while latencies remained unchanged from preattack values. These findings are discussed with regard to current understanding of the origins of the VER and relevant aspects of retinal electrophysiology.

Amaurosis fugax typically is the result of retinal micro-embolisation, classically from occlusive disease of the extracranial carotid artery,¹ or from other sources.²⁻⁶ Rarely, retinal vasospasm has been documented as a cause of transient visual loss.¹⁷⁻⁹ We had the opportunity to record the visual evoked response (VER) during monocular loss and subsequent return of vision in 2 patients. While other aspects of these cases were described elsewhere,^{8,9} the unique opportunity to record the VER prompted this report. To the best of our knowledge the VER has not been previously recorded during transient monocular visual reduction.

Materials and methods

During attacks of transient monocular visual loss the VER was obtained in different laboratories. Nevertheless the basic technique was similar, employing pattern-reversal stimulation with large checks (60' to 80' of arc) at low frequency (1-3 Hz), recording transient responses in each case.

Our first patient, wearing appropriate refractive error, was positioned 75 cm in front of a screen on to which was projected a high-contrast black-and-white checkerboard pattern. The entire pattern subtended 23° of central visual field with each individual square subtending 60' of arc. The average luminance at the screen was 270 cd/m². Reversal was produced by angular oscillation (1.2 Hz) of a mirror. Occipital

potentials were recorded with bipolar disc electrodes placed in the midline located 1 cm above theinion and at the vertex; an indifferent electrode was attached to the ear. The signal was passed through a preamplifier (Grass P-15) and a differential amplifier (Tektronix 3A9). Between 64 and 128 responses were averaged (Nicolet 1072 computer-averager) in at least 2 separate runs for each measurement. An observer monitored patient fixation throughout the course of VER testing. This technique produced a VER characterised by a small upward and larger downward deflection at approximately 100 ms. The negative component had the largest amplitude and was the most reliable complex. Latency was measured from the onset of the stimulus to the peak of this major negative component.

VER recordings for the second patient were obtained with a monopolar lead 1 cm above theinion and with a reference electrode at the mastoid process. The wave forms were recorded with a Nicolet CA-1000 system with television pattern generator. High-contrast black-and-white alternating check stimuli were presented at a reversal rate of 3 Hz, and 128 transient responses were averaged. At a distance of 4 feet (120 cm) each check subtended 80' of arc, with the entire screen subtending 10° 42' horizontally and 8° 30' vertically. Patient fixation was monitored continuously by an observer. With this technique the VER was characterised by a small negative deflection and larger positive component at approximately 100 ms. Latency was measured from the onset of the stimulus to the peak of this major upward deflection.

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Fig. 1 Case 1. Fluorescein angiography of right fundus, 55 seconds after onset of amaurotic fugax, demonstrates dye within vessels of the optic disc and segmental, incomplete filling of juxtapapillary retinal arterioles and veins. (Reprinted from Shaw et al.⁸).

Case reports and results

CASE 1

A 23-year-old black woman had recurrent amaurosis fugax in her right eye for 3 months. When she was initially examined, her visual acuity was 20/20 in each eye. Visual field examination showed a superior arcuate scotoma in the left eye. Ophthalmoscopy revealed dilated arterioles and veins in the right eye. The left fundus showed areas of vascular sheathing suggestive of previous vasculitis. The remainder of the examination was normal.

During examination the patient had repeated amaurotic attacks in the right eye. Right vision was instantaneously reduced to no light perception, with a nonreactive, amaurotic right pupil. Ophthalmoscopy revealed narrowing of retinal arterioles and veins, cessation of retinal blood flow, segmentation of the blood columns, and optic disc pallor. During an episode of transient visual loss fluorescein angiography showed early filling of the right optic disc and peripapillary capillaries, but fluorescein dye was not seen in the retinal arterioles until 30 seconds after injection (Fig. 1). The patient was found to have both SC haemoglobinopathy and systemic lupus erythematosus. After treatment with prednisone the amaurotic attacks abruptly subsided. The patient has remained asymptomatic.

VER was obtained at the onset of amaurosis fugax, and after 2, 5, 8, and 11 minutes (Fig. 2). At the onset of the attack no VER was recordable, and with return of retinal perfusion the VER amplitude gradually increased over a 5–8-minute period. In contrast, as VER amplitudes returned, latencies were

unchanged, measuring 94 ms (normal: 98 ± 6 ms).

CASE 2

Four days prior to examination a 48-year-old man reported the onset of repeated episodes of 'almost

PRE-ATTACK

ONSET OF ATTACK

2 MIN. AFTER ONSET

5 MIN. AFTER ONSET

8 MIN. AFTER ONSET

11 MIN AFTER ONSET

5µv
50msec

Fig. 2 Case 1. Serial recordings of pattern-reversal VER during an attack of amaurosis fugax on the right due to spasm of the central retinal artery.

Fig. 3 Case 2. Fluorescein angiography of the left eye 8 seconds after the onset of ocular migraine reveals filling of retinal vessels only inferotemporally. The remainder of the vessels filled approximately 30 seconds later. (Reprinted from Kline and Kelly⁹).

total blindness' of the left eye lasting about 2 minutes. During these episodes he experienced progressive loss of peripheral visual field but maintained a small portion of central visual field, which he described as 'shaped like a leaf.' The patient had experienced classic cluster headaches for the past 25 years but none in the previous 6 months. There was no family history of migraine. While he was asymptomatic,

neuro-ophthalmological testing was entirely normal, including funduscopy examination.

While he was in hospital the monocular attacks of visual reduction, each lasting approximately 2 minutes, were studied in detail. The patient maintained visual acuity of 20/30 on the left, and a small central island of visual field. During the attacks the retinal veins narrowed. There was delay in the appearance of fluorescein dye in branches of the central retinal artery yet prompt filling of 2 cilioretinal vessels (Fig. 3). Haematological and cardiac investigations, including echocardiography, gave normal results, and no abnormalities were found on carotid angiography. Diagnosed as having ocular migraine, the patient was placed on propranolol, with prompt cessation of visual symptoms.

As seen in Fig. 4, at the onset of the attack of ocular migraine the VER was abolished. While amplitude returned over a 2–3-minute period, the latencies (102 ms) were essentially unchanged from preattack values (normal: $99 \text{ ms} \pm 7$).

Fig. 4 Case 2. Serial recordings of pattern-reversal VER during an episode of transient visual reduction of the left eye due to ocular migraine.

Discussion

The series of electrical and electrochemical events that leads to the elaboration of the VER begins in the photoreceptors of the retina and culminates in the occipital cortex. Pattern-reversal VER has been found to show a more consistent waveform and to be more sensitive in detecting lesions of the visual pathways than has flash response.¹⁰ In the assessment of afferent input to the visual cortex pattern-reversal VER has been studied in a variety of diseases of the optic nerve, including optic neuritis,¹¹ ischaemic optic

neuropathy,^{12,13} toxic amblyopia,¹⁴ glaucoma,¹⁵ and compressive optic neuropathy.¹⁶ Comparison of amplitude and latency abnormalities has at times distinguished among such optic nerve diseases. For example, in the acute phase of demyelinating optic neuritis the amplitude of the pattern-reversal VER is reduced, and it returns to normal with clinical recovery, while characteristically prolonged latency remains.¹¹ However, in many instances it is difficult to separate the relative contribution of axonal degeneration and demyelination when VER abnormalities occur.

The 2 cases presented here provide an opportunity to examine selective impairment of axonal transmission within the optic nerve without disturbance of myelin sheaths. The central retinal artery is the sole vascular supply of the inner retinal layers (ganglion cells, inner plexiform layer, inner nuclear layer).¹⁷ With temporary interruption of blood flow, inner retinal activity ceases and no VER is generated. With resumption of blood flow there is progressive increases in retinal neuronal function, and VER amplitude gradually returns (Figs. 2 and 4). But with gradual return of inner retinal function there is nevertheless a constant value for VER latency.

Investigations into the genesis of the VER have dealt with contributions from striate (area 17) and extrastriate (areas 18 and 19) cortex.^{18–20} An understanding of the relationship between the VER and underlying neural events is far from complete. The only study dealing with the origin of the transient pattern reversal VER was done by Halliday and Michael,¹⁸ who found that the largest amplitude responses were recorded at electrodes located 5 to 7.5 cm in front of theinion, a site anterior to the striate cortex. They concluded that the major deflection occurring at approximately 100 ms is generated in extrastriate cortex.

Our studies demonstrate that return of inner retinal function, with no damage to optic nerve myelin, causes no alteration in VER latency. This phenomenon suggests that the inner retina responds in an all-or-none fashion. Current understanding of retinal electrophysiology supports this interpretation. With visual excitation the majority of retinal neural cells respond in a slowly graded manner, with the exception of retinal ganglion cells and some amacrine cells, where depolarisation leads to an all-or-none action potential.^{21,22} With interruption and subsequent return of inner retinal activity there is a gradual and progressive return of VER amplitude, presumably

reflecting the number of functioning ganglion cells. yet an immediate restitution of latency occurs, possibly due to the all-or-none action potentials of the retinal ganglion cells.

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