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Visual Evoked Potentials After Photostress in Patients With Primary Open-Angle Glaucoma and Ocular Hypertension

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Visual evoked potentials (VEPs) were assessed in basal condition and after photostress in normal subjects, in subjects with ocular hypertension (OHT), and in subjects with primary open-angle glaucoma (POAG). The VEPs recorded in basal condition showed that in patients with OHT and POAG a latency of the P100 peak was higher than in controls. The amplitudes were reduced in POAG patients but not in OHT patients. In all eyes, the VEPs recorded 20 s after photostress showed an increase in latency and a decrease in amplitude. In the control eyes, the functional recovery was complete after 80 s. In the eyes with OHP and in the eyes with POAG, the parameters of VEP after photostress underwent greater changes than in the control eyes. VEPs were superimposable on the basal condition (recovery time) at 73.2 s in OHT patients and at 113.2 s in POAG patients. The longer VEP recovery time after photostress observed in OHT and POAG patients could be attributed to the reduced functionality of the outer layers or the inner retinal layers of the central retina, or both. This test may be useful in the clinical evaluation for early diagnosis of glaucoma. Invest Ophthalmol Vis Sci 33:436–442, 1992

The macular photostress test (MPST[1]) assesses the recovery period of visual acuity after the central retina is dazzled with an ophthalmoscope.

A bleaching of a portion of the retina alters the adaptation process resulting in the formation of a scotoma. Return to a normal condition depends on the integrity of the complex pigmented epithelium-photoreceptors, which is functionally crucial for the resynthesis of retinal pigment.

The MPST was performed on normal subjects,[2–4] diabetic subjects,[5,6] and subjects suffering from glaucoma.[7]

Sherman and Henkind[8] studied the macular recovery period in subjects suffering from glaucoma. They found that macular recovery after photostress was altered. With their subjective method (MPST), they could not determine the relative contribution of the different functional layers of the retina to the delay in macular recovery caused by glaucoma.

An objective method for evaluating the visual function is to record the cortical potentials evoked by patterned stimuli—visual evoked potentials (VEPs)—or the electroretinographic signals (flash or pattern ERG). Flash ERG originates predominantly in the outer layers of the retina,[9] while the pattern ERG originates in the innermost retinal layers.[10–13]

Lovasik[14] and Franchi et al[15] studied the recovery of macular function using the VEP method after dazzling. Initially,[15] the VEP was evaluated in the control condition, then macular dazzling was produced, and the recovery time was evaluated. Recovery time is the time needed for the VEP to return to the control level. In all normal subjects, a total recovery of the VEP morphology was achieved after 60 s. In subjects suffering from maculopathy, the recovery time was noticeably longer. The length of the delay was correlated with the extent of the anatomo-functional changes in the photoreceptor-pigmented epithelium system.

In the light of these results we wanted to evaluate the following with an objective test: (1) the macular recovery time in normal subjects, analyzing the variation in VEP and its return to control condition after macular dazzling; (2) the macular recovery time in subjects with ocular hypertension (OHT) and in subjects with primary open-angle glaucoma (POAG), analyzing the variations of VEP and its recovery after macular dazzling; and (3) the possible differences between the recovery of the macular function after dazzling in normal subjects and dazzling in those with ocular hypertension or with primary open-angle glaucoma. Our goal was to evaluate whether a correlation existed between intraocular pressure and the recovery of vision after photostress.

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Subjects and Methods

Recording of VEPs was performed on 40 subjects. Only data from 29 subjects (42 eyes) were considered. In the other cases, the electrophysiological recordings presented too many artifacts per trial. Therefore, the single trials substantially exceeded the maximum duration acceptable for the present procedure.

We did not record pattern ERG or focal electroretinogram because the time required to obtain reliable readings is too long compared to the preestablished recording time. VEPs after photostress were recorded in 29 subjects—9 control subjects with normal intraocular pressure (IOP; 15 eyes), 9 patients with OHT (12 eyes), and 11 control patients with POAG (15 eyes).

The control subjects had IOP <21 mmHg, normal visual acuity, normal visual field (Goldmann's perimetry), and no ocular or neurologic problems. The mean age of the control subjects was 52.6 ± 4.4 yr (mean ± SEM). They were age-matched to the OHT and POAG patients.

The OHT patients had intraocular pressure >21 mmHg, normal visual field, and normal cup/disc ratios (<0.4). The mean age was 53.2 ± 5.3 yr.

The POAG patients had intraocular pressure >21 mmHg, characteristic visual field losses, and cup/disc ratio of >0.5. The mean age was 52.1 ± 4.7 yr.

The subjects being examined were seated in a semidark room that was acoustically isolated. The display was surrounded by a uniform field of luminance 5 candela/m^2. The subjects were informed of the examination's nature and its diagnostic uses.

VEPs were recorded using the following method. The visual stimuli were checkerboard patterns (contrast 70%, mean luminance 110 cd/m^2) generated on a TV monitor and reversed in contrast at the rate of 2 reversals/s. At the viewing distance of 114 cm, the check side subtended 15 min of visual arc, and the screen of the monitor subtended 25°. The stimulation was monocular, after occlusion of the other eye.

Cup-shaped electrodes of silver-silver-chloride were fixed with collodion in the following positions: active electrode in Oz, reference electrode in Fpz, ground in left arm.

The interelectrode resistance was kept below 3 Kohm. The bioelectric signal was amplified (gain 20,000), filtered (band-pass 1–100 Hz), and averaged with artifact reject on a Cadwell 7400 (Polman, Bologna, Italy).

The recording session began with a preliminary experiment in which at least two VEPs were recorded, averaging over 100 stimulus periods, excluding the time of artifacts. The analysis time was 500 msec. The resulting waveforms were superimposed to check for the repeatability of the results.

The transient response is characterized by a certain number of waves with three peaks—of negative, positive, negative polarity, respectively. In normal subjects, these peaks have latencies of 75, 100, and 145 ms.

After this preliminary trial, a control VEP was recorded, reducing the averages to 40 events per trial (with no more than 2 sweeps discarded because of artifacts). This VEP record was defined as "basal," and it was kept on display on the computer screen.

Photostress then was induced for 30 s with a circular diffusing surface (the bulb of a 200 W lamp) that was centrally fixated by the subject from a distance of 20 cm and produced a central scotoma of 6° diameter.

Prior to the experiment, each subject was adapted to the ambient room light for 10 min; the pupil diameter was about 5 mm. During the photostress proce-

Table 1. Control patients: observed characteristics

Obs	*Eye*	*Sex*	*Age*	*Iop*	*C/D*	*VA*	*VF*	*P100b*	*P100 20s*	*RT*
TP	LE	F	53	15	0.20	10/10	N	89.58	102.08	72
TP	RE	F	53	14	0.20	10/10	N	89.58	102.08	76
LD	LE	M	56	12	0.35	10/10	N	87.50	102.08	78
LM	RE	M	48	15	0.30	10/10	N	97.91	106.25	70
LM	LE	M	48	13	0.30	10/10	N	95.83	110.41	76
AF	LE	F	47	13	0.25	10/10	N	91.66	108.33	72
PT	RE	M	56	12	0.30	10/10	N	95.83	106.16	70
PT	LE	M	56	14	0.30	10/10	N	91.66	110.41	74
NB	RE	F	58	16	0.30	10/10	N	95.83	103.87	76
LG	LE	F	57	15	0.20	10/10	N	95.83	104.16	76
LG	RE	F	57	14	0.25	10/10	N	93.75	106.25	72
MB	LE	M	47	16	0.35	10/10	N	91.66	102.08	72
MB	RE	M	47	14	0.35	10/10	N	91.66	102.08	70
EF	LE	F	52	13	0.30	10/10	N	97.91	108.33	68
EF	RE	F	52	14	0.30	10/10	N	91.66	102.08	76

Abbreviations: Iop = intraocular pressure in mmHg (mean of different measures); C/D = cup to disc ratio; VA = best corrected Snellen visual acuity; VF = Goldmann visual field; N = normal; P110b = latency time (msec) of peak P100 in basal recording; P100 20s = latency time (msec) of peak P100 at 20 sec after photostress; RT = recovery time of VEP morphology after photostress (sec).

Table 2. OHT patients: observed characteristics

Obs	*Eye*	*Sex*	*Age*	*Iop*	*C/D*	*VA*	*VF*	*P100b*	*P100 20s*	*RT*
SG	LE	M	52	22	0.30	10/10	N	108.3	118.7	92
SG	RE	M	52	24	0.30	10/10	N	106.0	120.9	100
GU	LE	M	55	26	0.30	10/10	N	106.2	116.0	112
BR	RE	F	58	24	0.35	10/10	N	108.3	125.8	98
CN	RE	M	60	26	0.40	10/10	N	96.0	108.3	88
CO	LE	F	56	23	0.40	10/10	N	96.0	123.0	90
CO	RE	F	56	24	0.35	10/10	N	100.0	116.0	96
BO	LE	F	46	25	0.40	10/10	N	110.0	116.0	95
SP	LE	M	44	24	0.30	10/10	N	108.3	127.8	95
SP	RE	M	44	26	0.35	10/10	N	108.3	127.8	95
GR	RE	M	52	24	0.30	10/10	N	114.0	125.0	96
FD	LE	F	56	25	0.40	10/10	N	102.8	110.4	91

Abbreviations: Iop = intraocular pressure in mmHg (mean of different measures); C/D = cup to disc ratio: VA = best corrected Snellen visual acuity; VF = Goldmann visual field; N = normal; P100b = latency time (msec) of the peak P100 in basal recording; P100 20s = latency time (msec) of peak P100 at 20 sec after photostress; RT = recovery time of VEP morphology after photostress (sec).

dure, each of the subjects looked directly into the center of the light; the pupil diameter was about 2 mm.

Immediately after the photostress, fixation was shifted to the pattern stimulus and the recording of VEPs was started. Records were taken for successive periods of 20 s each (40 averaging every 20 s of recording and the corresponding record displayed on the screen) until the VEP waveform obtained was superimposable on the basal record. The corresponding time was considered "recovery time after photostress."

For all VEPs, the peak latency and peak amplitude of each wave were measured directly on the displayed records with a pair of cursors.

Results

We have considered the results obtained from basal VEPs in control eyes, in OHT eyes, and in POAG eyes; and VEPs after photostress in control eyes, in eyes with OHT, and in eyes with POAG.

In the analysis of VEP records, we evaluated the P100 latency, the temporal difference N75/N145, the N75-P100 amplitude, and the P100-N145 amplitude. The observed characteristics of all subjects are shown in Tables 1–3.

The differences between the control eyes and the eyes with OHT and POAG were statistically evaluated with a one-way analysis of variance for repeated measures.

Basal VEPs

In the control eyes, the parameters of VEPs (P100 latency, time difference N75/N145, N75-P100, and P100-N145 amplitudes) were within normal limits—ie, mean value ± 1 SD for N75-P100 and P100-N145 amplitudes, and mean value ± 3 SD for P100 latency

Table 3. POAG patients: observed characteristics

Obs	*Eye*	*Sex*	*Age*	*Iop*	*C/D*	*VA*	*VF*	*P100b*	*P100 20s*	*RT*
GI	LE	M	56	27	0.60	10/10	S	116.0	135.0	110
PA	LE	M	58	28	0.80	10/10	G	137.5	152.0	126
PA	RE	M	58	28	0.70	10/10	G	122.0	150.0	131
PL	LE	F	46	26	0.80	10/10	G	131.2	145.0	120
PL	RE	F	46	28	0.70	10/10	N	118.7	143.0	120
BR	RE	F	48	24	0.65	10/10	N	125.0	145.5	110
CN	RE	F	44	24	0.55	10/10	A	113.6	127.2	110
BO	LE	M	50	26	0.60	10/10	P	116.6	129.1	100
GR	LE	M	52	26	0.75	10/10	G	123.0	152.1	105
LC	LE	M	54	23	0.50	10/10	I	118.7	143.7	105
LC	RE	M	54	26	0.90	10/10	G	138.0	154.0	140
GU	LE	F	58	24	0.75	10/10	P	131.2	153.0	105
GU	RE	F	58	23	0.70	10/10	G	140.0	166.0	112
FA	LE	M	55	28	0.65	10/10	A	108.0	120.6	102
BU	LE	M	53	24	0.60	10/10	A	112.5	127.8	102

Abbreviations: Iop = intraocular pressure in mmHg (mean of different measures); C/D = cup to disc ratio; VA = best corrected Snellen visual acuity; VF = Goldmann visual field; A = arcuate scotoma; I = inferior nasal arcuate scotoma; G = general constriction; N = nasal step; P = paracentral step; S = superior nasal step; P100b = latency time (msec) of peak P100 in basal recording; P100 20s = latency time (msec) of the peak P100 at 20 sec after photostress; RT = recovery time of VEP morphology after photostress (sec).

Table 4. Mean increase in P100 latency and mean percentage decrease in amplitude N75-P100 at 20, 40, and 60 sec after photostress

	Latency P100		*Amplitude N75-P100*	
	M	*SD*	*M*	*SD*
20 sec				
C	11.92	3.12 msec	−0.16	0.10%
OHT	13.68	6.15 msec	−0.27	0.10%
POAG	19.47	5.88 msec	−0.33	0.12%
40 sec				
C	8.47	2.77 msec	−0.14	0.08%
OHT	7.31	3.92 msec	−0.22	0.06%
POAG	13.54	4.64 msec	−0.25	0.11%
60 sec				
C	4.54	2.71 msec	−0.12	0.15%
OHT	5.55	3.42 msec	−0.14	0.06%
POAG	10.78	4.91 msec	−0.19	0.12%

M = mean; SD = one standard deviation of the mean values.

and time difference N75/N145. In OHT and POAG patients, the P100 latency was greater than in the control eyes (OHT: $F(1,25) = 49.86$, $P < 0.01$; POAG: $F(1,18) = 123.88$, $P < 0.01$).

VEP amplitudes of the OHT patients were comparable to the control ones ($F(1,25) = 0.27$, $P > 0.05$), but POAG patients showed reduction in amplitude ($F(1,28) = 8.84$, $P < 0.01$). The time difference N75/145 was comparable in the three groups.

VEPs After Photostress: Control Eyes

Examples of records of one normal subject (FA) are shown in Figure 1. The mean data are presented in Tables 4 and in Figures 2 and 3.

At 20 s after photostress, we observed an increase in P100 latency and a decrease in amplitudes N75-P100 and P100-N145. At 40 and 60 s after photostress, the P100 latencies were shorter with respect to the values observed at 20 s, but they were still longer than in the basal VEP. The amplitudes increased with respect to the values observed at 20 s, but they were still lower than the basal ones. At 80 s after photostress, the record was superimposable on the basal one, indicating perfect recovery. The time difference N75/N145 did not change after photostress.

VEPs After Photostress in Eyes With OHT and POAG

Examples of records of one subject with OHT (SP) and records of one subject with POAG (PA) are shown in Figure 1. The mean data are presented in Tables 4 and in Figures 2 and 3. At 20, 40, and 60 s after photostress, we observed in these patients the same changes found in the control eyes.

The VEPs recorded at 20 s after photostress showed P100 peaks of longer latency and smaller amplitude compared to the values observed in basal records. At 40 s and 60 s after photostress, the P100 latency progressively diminished, although it was longer than in the basal VEP. The amplitude increased progressively but remained lower than in the VEP recorded before the photostress. The time difference N75/N145 did not change after photostress. The mean increments in P100 latency observed at 20, 40, and 60 s after photostress in OHT patients were comparable to the increment observed in the control eyes ($F(1,79) = 0.25$, $P > 0.05$). In POAG patients, the mean increments were higher than in the controls ($F(1,88) = 30.94$, $P < 0.01$).

The mean reductions in amplitude observed at 20, 40, and 60 s after photostress in the OHT patients were comparable to the reduction observed in the control eyes ($F(1,79) = 0.25$, $P > 0.05$). In POAG patients, the mean reductions were lower than in the controls ($F(1,88) = 30.94$, $P < 0.01$).

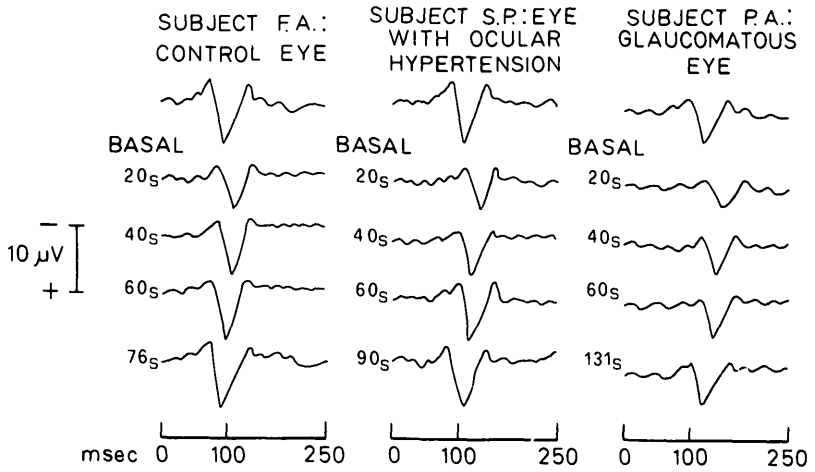

Fig. 1. VEP layout of subjects F.A. (control eye), S.P. (OHP eye) and P.A. (POAG eye) in normal condition (basal) and 20, 40, and 60 sec after photostress. In comparison with the control eye records, in the eye with OHT and the eye with POAG, VEPs recorded at 20, 40, and 60 sec after photostress show a longer P100 latency and a reduced amplitude. The VEPs are superimposable on the basal waveform at 76 sec in the control eye, in the OHT eye at 90 sec, and at 131 sec in the POAG eye.

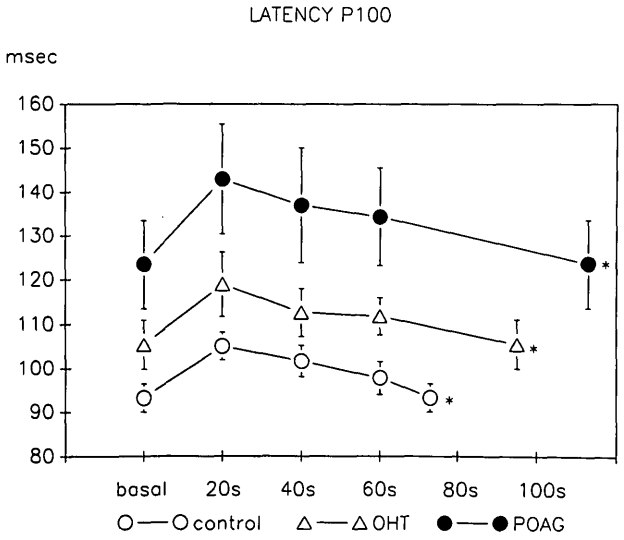

Fig. 2. Graphic representation of mean values of Latency P100 in basal condition and 20, 40, 60, 80, 100, and 120 sec after photostress. Error bars represent one standard error of the mean. Recovery time after photostress (*) is 73.3 sec in the control eyes, 95.1 sec for the OHT eyes, and 113.2 sec for the POAG eyes.

The VEPs were superimposable on the basal VEP (recovery time after photostress) at 95.1 ± 6.5 s in the eyes with OHT and at 113.2 ± 11.8 s in the eyes with POAG. This time is much longer than in the control subjects(OHT: $F(1,25) = 131.76$, $P < 0.01$; POAG: $F(1,28) = 160.79$, $P < 0.01$).

Discussion

The intent of our research was to evaluate the VEP recovery time after photostress in OHP patients and in POAG patients.

In OHT and POAG patients, the VEPs recorded before photostress showed P100 latency longer than in controls; the amplitudes were reduced in POAG patients but not in OHT patients. These changes of VEPs in OHT and POAG patients have been attributed to a selective reduced functionality of the inner retinal layers.[16–28] This is supported by a histological study[29] that showed a loss of large ganglion cell fibers in POAG patients.

After photostress, in the control eyes, the VEP recorded 20 s after dazzling presented a latency increase and an amplitude decrease. After 80 s, a normal condition was reached, and therefore the functional recovery was complete. The changes induced by photostress on the VEPs are generally attributable to the diminished capacity of macular photoreceptors to produce a sufficient electrotonic potential after dazzling.[14,15]

In the eyes with OHT or POAG, the parameters of VEP after photostress showed larger changes and longer recovery times (OHT: 95.1 ± 6.5 s; POAG: 113.2 ± 11.8 s) than in the control eyes. This may be related to stress caused by the pressure on the photoreceptors or on the inner retinal layer of the macular region.

Previous reports[23,30–36] indicate that the flash ERG is not modified after ocular hypertension or in glaucoma. This leads us to believe that the sensory layer of the retina is not functionally sensitive to the amount of intraocular pressure.

The evidence that ocular hypertension mainly affects the proximal retinal layers came from a recent study on humans that showed experimentally induced ocular hypertension produced modifications of VEP and pattern ERG but not of flash ERG.[37,38]

These observations are supported by an experimental model of retinal ischemia in cats.[39] Here, the intraocular pressure was increased and the average arterial pressure was reduced until a total block of the choroid-retina circulation was obtained. Ten minutes after release from ischemia, the flash ERG had already recovered, while the VEP and pattern ERG were still depressed, even 2 h later. This reveals that the neurons of the proximal layers are more vulnerable to pressure damage than those of the distal layers.

That the flash ERG is not affected by ocular hypertension does not rule out a possible impairment of functionality of the sensory layers of the macula. Indeed, the flash ERG reflects the activity of the outer layers of the whole retina, and the contribution of the macular region to the flash ERG is negligible.

A more sensitive way to test the functionality of the macular region is offered by the focal ERG in response to the light modulated within a small central area.

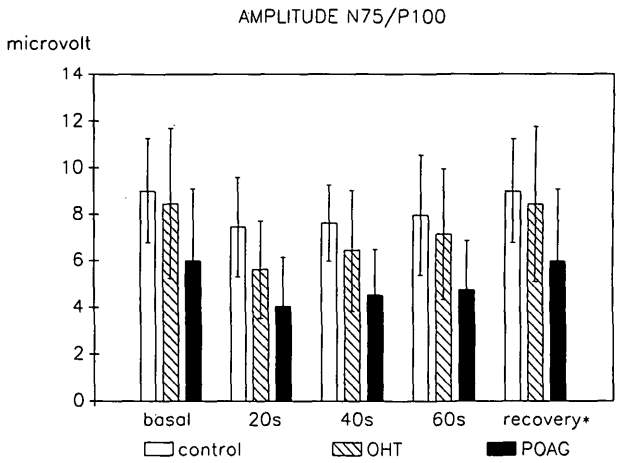

Fig. 3. Histograms of mean values of VEPs amplitude in basal condition and 20, 40, and 60 sec after photostress. The vertical lines represent one standard deviation. Recovery time after photostress (*) is 73.3 sec for the control eyes, 95.1 sec for the OHT eyes, and 113.2 sec for the POAG eyes.

According to a recent report,[40] glaucoma may cause a reduction of amplitude of the focal ERG in response to a central 9° stimulus. This may imply suffering of the outer retinal layers and even of the photoreceptors secondary to the pressure damage. These changes are accompanied by a reduction of VEP amplitudes and psychophysical flicker sensitivity. These findings, however, are limited to stimuli modulated at temporal frequencies higher than 10 Hz and are noticeable especially at 30–50 Hz. The changes in VEP responses may reveal selective damage of thick nerve fibers, which are thought to mediate the responses to high temporal frequencies.

The longer VEP recovery time after photostress observed in OHT and POAG patients therefore could be attributed to the reduced functionality of the outer retinal layers[40] or of the inner retinal layers of the central retina, or both.

This electrophysiological test may be applicable to the early diagnosis of pathologies such as glaucoma, in which the retinal structure undergoes anatomical and pathological changes that produce a functional deficit.

Key words: VEPs, photostress, IOP, OHT, POAG

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