Conclusions

The typical defects of myopia present topographical characteristics correlated to a certain extent with the refractive defect magnitude (pericaecal area) and also, in part, not ascribable to it (peripheral absolute defects). In order to discriminate defects of a myopic nature from those which are glaucomatous, it is not enough to look only at the perimetric indices, since GHT abnormalities (even if not too specific), asymmetric sensitivity between the two sides of the nasal hemimeridian according to the criteria we have proposed, and, of course, the presence of fascicular-type defects, according to Anderson's criteria, are all validly sensitive.

Key words: elevated myopia, glaucoma, computerized perimetry.

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Delayed postretinal neural conduction in glaucoma patients: Correlations between electrophysiological and computerized static perimetry parameters

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Purpose

In glaucoma patients, the clincal evaluation of the retina and optic nerve functions can be assess by Visual Field analysis or by recording Flash or Pattern Electroretinogram (ERG and PERG) and Visual Evoked Potentials (VEPs). By comparing the VEP peak latency and the PERG peak latency, it is possible to construct an index of postretinal neural conduction. Celesia et al. (1) suggested evaluating the difference between VEP P100 latency and PERG P50 latency, which measure he called the "RetinocorticalTime" (RCT). Marx et al. (2) proposed a different evaluation i.e. the difference between VEP N75 latency and PERG P50 latency and termed this measure the "Latency Window" (LW).

Since we recently found an impaired neural conduction in the postretinal visual pathways in glaucoma patients (3), our goal is to assess whether a correlation exists between electrophysiological parameters and static perimetry parameters, with a view to evaluating the possibility that the visual field defects observed in glaucoma patients might be ascribed to an impaired function of the retinal layers, or to an impaired neural conduction in the postretinal visual pathways, or to both.

Methods

Visual field by Humphrey Perimeter (Central 24-2 Threshold Test) and simultaneous recordings of PERG and VEP were assessed in 21 subjects with open angle glaucoma (POAG) and in 15 age-matched controls. The POAG patients were in pharmacological treatment with beta-blockers only; miotic or mydriatic drugs were not used. All subjects examined had best corrected visual acuity of 10/10 with refractive errors of ±2 spheres, and no general or neurologic pathologies. In the perimetric examination, the mean defect (MD) and the corrected pattern standard deviation (CPSD) were considered.

In the electrophysiological examination, the visual stimuli were checkerboard patterns (contrast 70%, mean luminance 100 cd/m² generated on a TV monitor and reversed in contrast at the rate of 2 reversals per sec.

The check edges subtended 15 minutes of visual angle. The stimulation was monocular.

PERG recordings

The bioelectrical signal was recorded by means of platinum hook electrodes inserted into the external corner of the inferior eyelid. Electroretinograms were derived bipolarly between the tested (active electrode) and the patched (reference electrode) eye. Local anaesthesia was provided by the application of novesine 0.4%. The ground electrode was in Fpz. The signal was amplified (gain 50000), filtered (band-pass 1-30 Hz) and averaged with automatic rejection of artifacts (200 events free of artifacts were averaged for every trial) by BM 6000 (Biomedica Mangoni, Pisa, Italy). The analysis time was 250 msecs.

VEP recordings

Cup-shaped electrodes were fixed in Oz (active electrode) and in Fpz (reference electrode); ground on left arm. The bioelectric signal was amplified (gain 20000), filtered (band-pass 1-100 Hz) and averaged (200 events). The analysis time was 250 msecs. We accepted PERG and VEP signals with signal-to-noise ratio >2.

Results

PERG: POAG eyes showed a significantly (ANOVA) delayed P50 peak latency against that of the control eyes (p<0.01) and correlated with MD (r = -0.643, p<0.002); the P50-N95 amplitudes were significantly lower in POAG eyes than in the control eyes (p<0.01) and correlated with MD (r=0.590, p=0.005).

VEP: in POAG eyes, we found a significantly delayed P100 peak latency against that of the control eyes (p<0.01) and correlated with MD (r=-0.822,

Table 1. POAG patients: observed characteristics.

Age		IOP	HUMPHREY MD		CPSD
54.05±2.75		21.33±1.20*	-3.16±1.0*		1.65±0.77*

VEP LAT	AMP	PERG LAT	AMP	RCT	LW
123±8.2*	5.9±2.5*	63.2±4.1*	0.6±0.1*	59.9±4.9*	34.52±4.8*

Humprey MD: mean deviation, CPSD: corrected pattern standard deviation; VEP LAT: P100 latency (msec), AMP: N75-P100 amplitude (microvolt); PERG LAT: P50 latency (msec), AMP: P50-N95 amplitude (microvolt); RCT = Retinocortical Time: difference between VEP P100 and PERG RG P50 latencies; LW = Latency Window: difference between VEP N75 and PERG P50 latencies; * = p<0.01 vs Controls.

p<0.01); the N75-P100 amplitudes were significantly lower in POAG eyes than in the control eyes (p<0.01) and correlated with MD (r=0.702, p<0.001).

RCT and LW were significantly greater in POAG eyes than in the control eyes (p<0.01); RCT and LW were correlated with MD (RCT: r=-0.844, p<0.001; LW: r=-0.810, p<0.001).

Conclusions

In patients with open angle glaucoma, the visual field defects could be ascribed to two sources of functional impairment, one retinal (impaired PERG) and one postretinal (impaired RCT and LW).

Key words. PERG, VEP, RCT, Visual Field, Glaucoma.

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