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Pattern-Evoked Potentials and Optic Nerve Fiber Loss in Monocular Laser-Induced Glaucoma

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ERG and VEP responses to counterphase checkerboard stimuli were obtained from cynomolgus monkeys with monocular glaucoma induced by laser photocoagulation of the trabecular meshwork. The glaucomatous eyes showed reductions of PERG amplitude that were directly related to the histologically defined nerve damage. VEP amplitudes were also reduced in the glaucomatous eyes, but were more variable and less affected by damage than the PERG responses. An acute increase in eye pressure to 40 mm Hg in eyes without damage had no detectable effect on PERG amplitudes. Invest Ophthalmol Vis Sci 30:897-907, 1989

The flash-evoked electroretinogram (ERG) has most often been reported to be unaffected by glaucoma. Whereas the ERG is thought to reflect only the activity of radial retinal elements, that is, photoreceptors, bipolar cells and Müllner cells, glaucoma damages only retinal ganglion cells. Recent studies have suggested that the "pattern-evoked" ERG (PERG), produced with counterphase grating or checkerboard stimuli, may reflect inner retinal activity. This conclusion is not unanimous. Sherman and van den Berg et al have reported that optic nerve disease has little effect on PERG amplitude. Spekreijse and coworkers proposed that the PERG is a nonlinear component of the luminance response, having little to do with patterned stimuli per se. However, other studies have concluded that the PERG is a complex potential that depends on both luminance and pattern parameters.

The origin of the PERG is a matter of intense interest for glaucomatologists, who are searching for a reliable clinical test of early glaucomatous optic nerve damage. Quigley and coworkers have shown that the visual field, long considered the definitive indicator of glaucomatous damage, can appear normal when a substantial minority of optic nerve axons have been destroyed. Any test that could reliably detect early glaucomatous damage would thus be of great diagnostic value.

The PERG is very small (<10 μV) and variable, even in normal subjects, and it is also vulnerable to blink and eye movement artifacts. Even though there is much evidence that PERG amplitudes are significantly reduced in glaucoma, it is still not clear whether these reductions are detectable in individual patients, who may have: (1) both eyes similarly affected but by unknown amounts; (2) preretinal opacities with unknown effects on PERG amplitudes; (3) large normal differences between left- and right-eye PERG amplitudes; or (4) normal PERG amplitudes that are much larger or smaller than mean values.

The current study is the first to examine the effect of graded optic nerve damage on PERG responses with histopathological confirmation of the degree of damage. We recorded PERGs from monkeys with unilateral laser-induced glaucoma, and confirmed that PERG amplitudes are reduced in the glaucomatous eyes when compared to the normal eyes of these monkeys. In addition, we found that the PERG reductions are highly correlated with optic nerve axon loss. We tested the degree of variability in the PERG responses in each animal and between animals to assess the PERG's potential clinical usefulness. For comparison purposes, we recorded pattern visual evoked cortical potentials (VEPs), flash ERGs and oscillatory potentials in addition to the PERGs.

Materials and Methods

Animal Preparation

All procedures used in this study were performed in accordance with the ARVO Resolution on the Use
of Animals in Research, and were approved by our Animal Care Committee. Experimental glaucoma was produced by laser treatment of the trabecular meshwork using a method reported previously. For this study, the right eye was the glaucoma eye in each animal, and the left remained normal.

Animals were sedated with ketamine hydrochloride intramuscularly (10 mg/kg) and intraocular pressure was measured under topical proparacaine hydrochloride anesthesia with an applanation tonometer or a calibrated pneumatonograph. Optic disc examination was performed stereoscopically with a slit lamp and contact lens. Fundus photography was carried out under intravenous sodium pentobarbital anesthesia (initial dose 25 mg/kg). Color stereoscopic photographs were made of the optic disc, and black and white photographs of the retinal nerve fiber layer were taken with red-free light, with both being used to judge the degree of optic nerve damage.

Monkeys were injected intramuscularly with 10 mg/kg body weight ketamine hydrochloride prior to electrophysiologic testing. An endotracheal respiration tube was inserted after topical application of xylocaine to the trachea. During the session, the animals were paralyzed with periodic intravenous injections of atracurium besylate (initial dose of 1 mg/kg, follow-up doses of 0.5 mg/kg as needed to maintain paralysis), and were mechanically respirated with room air via an endotracheal tube at a rate sufficient to maintain normal oxygen delivery. They were placed in a prone position on foam padding, and a padded head support directed the animals' gaze forward.

Apparatus

Alternating checkerboards were generated using the UTAS-E1000 electrophysiology system (LKC Systems, Inc., Gaithersburg, MD) and displayed on a CRT monitor. As shown in Figure 1, a condensing lens focused a small image of the screen in the film plane of a Zeiss fundus camera from which the 35 mm camera had been removed. When the monkey's fundus was focused in the fundus camera viewer and the crosshairs were positioned over the fovea, a foveally centered, 30° diameter image of the stimulus was thus automatically focused on the fundus.

Experimental Procedures

The animals were electrically grounded using a skin electrode attached to the right hind leg. Simultaneous VEPs and PERGs were recorded to seven check sizes, which varied from 15' to 15° in 0.3 log steps, and to a full field reversal. Check contrast varied between 95% for the 15° checks and 78% for the 30' checks, the difference in contrast attributable to the greater stray light produced by the smaller check sizes. We could not reliably measure contrast for the smallest check size (15'), due to the 15' integration area of the photometer. The screen produced an average retinal illuminance of 173 (human) trolands.

Pattern electroretinogram: PERGs were recorded using a jet electrode referenced to the forehead (the Fz VEP electrode). The monkey's pupils were dilated with mydriacyl (1% tropicamide), and the corneas were anesthetized with proparacaine hydrochloride. The monkey's retina was focused and aligned after placement of the electrode, to compensate for any changes in corneal refractive power caused by the contact lens, and to insure that overall image quality was good. Monkeys were dark-adapted for at least 20 min after they were aligned in order to allow full recovery of the PERG amplitude after exposure to the bright aligning light. The normal eye was tested first 50% of the time, and the glaucomatous eye was tested first 50% of the time. Unless full intensity-response functions to the flash ERG were measured, the flash ERG and oscillatory potentials were measured directly after the PERGs and VEPs were recorded, using a hand-held stroboscopic flash lamp (Grass Instrument Co., Quincy, MA) at a distance of approximately one meter.

Several investigators have reported that far-field PERG artifacts from the fellow eye can approach the PERG of the stimulated eye in amplitude. More recently, Arden and colleagues have confirmed that far-field artifacts are measurable with certain electrode configurations, but at much lower amplitudes than the stimulated eye response. All of the studies reporting large artifacts used gold foil electrodes, which are quite sensitive to eyelid and eye movement artifacts that can mimic bioelectric potentials.

In the current study, eyelid and eye movement artifacts were eliminated by paralyzing the monkeys. To determine whether the PERGs that we were re-
ERGs were recorded from the other animals using a single flash from a hand-held strobe stimulator.

Oscillatory potentials (OPs) were measured in two ways. Intensity-response and single-flash ERGs recorded in the conventional manner were digitally filtered by convolving the waveform with a rectangular spread function and subtracting the result from the waveform. The bandpass of this filter was 75–500 Hz. OPs were also elicited using a preconditioning flash of 3 log cd-s/m², delivered 30 sec before a stimulus of the same intensity. These responses were fed into two amplifiers, one having the filter characteristics used for intensity-response recording, the other having a high-pass of 75 Hz and a low-pass of 3000 Hz, to emphasize the high-frequency OPs. In both cases, the amplitudes of all the OPs obtained were summed.

**Histological Procedures**

The animals were sacrificed by perfusion of fixative through the aorta under deep pentobarbital anesthesia. After initial infusion of saline to clear red blood cells, we perfused 1.5% gluteraldehyde and 1% paraformaldehyde in cacodylate buffer (pH 7.4) followed by 5% gluteraldehyde and 4% paraformaldehyde in the same buffer. The eyes and optic nerves were removed, post-fixed in 1% osmium tetroxide in cacodylate buffer, and embedded in epoxy resin.

The method for estimating the number of remaining optic nerve fibers was identical to that described in Sanchez et al.43 Thick sections (1 μm) of optic nerve were stained with p-phenylenediamine, and mounted on glass slides. Each nerve cross-section was photographed, enlarged (×100) and printed on 16 × 20-inch paper. The nerve was divided into 16 labeled segments of roughly equal area. The area of the nerve bundles was measured for each segment using a planimeter. To estimate the density of nerve fibers, we photographed each nerve at ×100, four photographs per segment. Each photograph was placed in a digital image analyzer, viewing 2500 μm² areas at a time.

Each axon was counted and its minimum diameter measured. Photographs from the same segment of a nerve were analyzed together. The estimated total number of fibers in any given segment is simply the density for that segment multiplied by its measured neural area. The total number of fibers for any nerve is the sum of the total number of fibers in each of the 16 segments.

**Results**

**Histology**

The histological findings for all six monkeys are summarized in Table 1. Two of the monkeys (X33...
Table 1. Summary of clinical and histological data on glaucomatous monkey eyes

<table>
<thead>
<tr>
<th>Monkey</th>
<th>Cup/disc ratio</th>
<th>Nerve fiber layer</th>
<th>Intraocular pressure*</th>
<th>RE/LE fiber ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>W51</td>
<td>0.3</td>
<td>Normal</td>
<td>24 mm Hg</td>
<td>Poor fixation</td>
</tr>
<tr>
<td>T65</td>
<td>0.4</td>
<td>Normal</td>
<td>30 mm Hg</td>
<td>71%</td>
</tr>
<tr>
<td>W56</td>
<td>0.6</td>
<td>Mild diffuse atrophy</td>
<td>28 mm Hg</td>
<td>46%</td>
</tr>
<tr>
<td>U45</td>
<td>0.9</td>
<td>Diffuse atrophy</td>
<td>40 mm Hg</td>
<td>8%</td>
</tr>
<tr>
<td>X32</td>
<td>0.9</td>
<td>Diffuse atrophy</td>
<td>41 mm Hg</td>
<td>3%</td>
</tr>
<tr>
<td>X33</td>
<td>0.9</td>
<td>Diffuse atrophy</td>
<td>32 mm Hg</td>
<td>Not obtained</td>
</tr>
</tbody>
</table>

* Mean pressure over period during which damage occurred, which was consistently close to the pressure during testing.

and W51) were excluded from the optic nerve analysis, W51 because of poor optic nerve fixation, and X33 because of histological and electrophysiological evidence that the untreated eye had non-glaucomatous ocular disease. The remaining four animals showed a range of damage in the treated eye from mild to severe, with a good correspondence between the appearance of the optic disc and nerve fiber layer and the amount of damage estimated from the ratio of the number of optic nerve fibers in the treated vs. the untreated eye.

Pattern Electroretinogram

**Normal PERG data:** Figure 2 gives some typical examples of normal responses for various check sizes. Waveforms are shown for four check sizes ranging from 30° (uniform field) stimuli (bottom) to half-degree checks (top). Response amplitudes are measured from the first negative peak to the following positive peak. All waveforms are averages of 100 individual responses.

The uniform field response consists of a corneal-negative deflection followed by a larger positive wave and then another negative deflection. With increasing spatial frequency, the positive waveform becomes progressively broader, and the negative waveforms are diminished.

Figure 3 shows the variability of normal PERG responses. PERG mean values ± 2 standard deviations obtained using eight different check sizes are plotted in this figure for seven normal eyes. The variability is large even under our well controlled stimulus conditions. Figure 4 compares the overall variability to within-session variability data from a single monkey, for two check sizes. The squares are the data replotted from Figure 3 for the 15° and the 1° check sizes. The circles show the means ± 2 standard deviations of repeated measures recorded one after the other in the same session. Although the intrasubject variability was significant, the overall variability is larger than that observed in normal eyes.
variability is obviously less than it is across monkeys, one standard deviation still can be as large as 1 \mu V, with an average PERG amplitude of only 4 \mu V.

**PERG responses during acute IOP elevation:** As our monkeys typically had intraocular pressures greater than 30 mm Hg in their glaucomatous eyes, we conducted a control experiment to make sure that any abnormalities we might see in the PERG were due to glaucomatous damage rather than pressure elevation *per se*. First, we prepared a normal monkey for PERG measurements in the usual way. Then, after topical anesthesia with proparacaine hydrochloride, we inserted a cannula into his anterior chamber and controlled his intraocular pressure by raising or lowering a reservoir attached to the cannula. Figure 5 shows responses to 2° checks before pressure elevation (top), about 45 min after the pressure was raised to 38 mm (middle), and after the pressure was returned to normal (bottom). Acute pressure elevation had no detectable effect on the PERG amplitude. These results were confirmed in a second animal.

**PERG Amplitude Ratios**

Figure 6 shows PERG waveforms from the normal (solid lines) and affected (dashed lines) eyes of animal W56, who had about 46% as many optic nerve fibers in the glaucomatous eye as in the normal eye. Visual comparisons of the amplitudes for the two eyes show that the amplitudes are clearly reduced in the affected eye, but perhaps by different amounts at different check sizes.

In order to quantify the effect of glaucomatous damage on the PERG, we computed affected eye over normal eye amplitude ratios for each check size. For example, amplitudes of 6 \mu V in the normal eye and 3 \mu V in the glaucomatous eye would yield a ratio of 0.5. We found that the amount of amplitude loss depended on check size. In Figure 7, amplitude ratios are plotted as a function of check size for five monkeys. The individual means of the five functions have been superimposed on their combined mean by multiplying each function by the appropriate factor. The data are well described by a parabola with a minimum at a check size of about 2°.

**PERG-Histology Comparison**

The main purpose of this study was to compare both PERG responses and counts of optic nerve fibers between the normal and glaucomatous eyes of the unilaterally treated monkeys. The results strongly
Fig. 6. PERG waveforms shown here for the normal (solid lines) and affected eyes (dashed lines) of monkey W56, who had 46% of optic nerve fibers remaining in the glaucomatous eye. Responses shown for check sizes of 30° (full field), 4°, 1° and 15°.

Fig. 7. Normalized right eye/left eye PERG amplitude ratios for five monkeys as a function of check size. Small data symbols indicate individual animals and "x"s are averages of the five. Data are normalized by equating the mean amplitudes across check size for the individual animals. The curve is a parabolic least-squares regression curve. The shape of the curve suggests that the maximum PERG amplitude reduction occurs at intermediate check sizes. Amplitude ratios for 0.25° checks are not shown because data were not obtained for all monkeys.

Fig. 8. (A) Comparison of right eye/left eye ratios of PERG amplitudes (black bars) and optic nerve fiber numbers (grey bars) for monkeys X32, U45, W56 and T65. All data are averages of check sizes of 4°, 2° and 1°. (B) Comparison of right eye/left eye ratios of PERG amplitudes (black bars) and optic nerve fiber numbers (grey bars) for monkeys X32, U45, W56 and T65. All data are averages of check sizes 8°, 15° and 30° (uniform field).

indicate that the PERG is related to ganglion cell function. The six monkeys tested all showed reduced PERGs in their glaucomatous eyes, and, except for one monkey (W51) whose histological data are unusable, the amount of reduction was consistent with the degree of damage apparent in the optic disc and nerve fiber layer. (W51 had a mean right eye/left eye PERG amplitude ratio of 0.60 but no apparent optic disc or nerve fiber layer abnormalities.) We were also able to get reliable optic nerve fiber counts in four of these monkeys. The relationship between fiber loss and PERG amplitude reduction is shown in Figure 8. There is a good correspondence between average PERG amplitude ratios and the right eye/left eye ratios of optic nerve fiber numbers for the same monkeys. This is illustrated for three check sizes that showed the greatest reduction in response (Fig. 8A). Figure 8B shows a similar analysis for the average of the three largest check sizes. There is still a good qual-
itative correspondence, but the PERG data consistently underestimate the degree of fiber loss. This underestimate can also be seen in a regression plot of the PERG ratios for all check sizes against the fiber number ratios (Fig. 9). The correlation between the PERG amplitude ratios and fiber number ratios is highly significant at 0.88 ($P < 0.0004$). However, if the PERG reductions were strictly proportional to the nerve fiber losses, the data points should fall on the dashed line. Instead, the regression line is shifted upward by about 0.14. This implies either that the last few ganglion cells in the retina can produce a highly disproportionate PERG response, or that part of the PERG is generated by neurons other than ganglion cells.

**Flash Electroretinogram**

Figure 10 shows flash ERG responses from a monkey who had only 8% as many axons in his glaucoma-damaged optic nerve as in his normal one. Nevertheless, the glaucoma (right) eye response is at least as large as the normal (left) eye response, confirming that glaucoma does not reduce the amplitude of the conventional ERG response. A statistical analysis of ten different comparisons (for five monkeys) of right/left eye flash ERG amplitudes showed that the amplitude was an average of 27 $\mu$V larger in the glaucomatous eye. This difference was nearly significant at the 0.05 level (two-tailed paired-comparison t-test; $t(9) = 2.14, P = 0.061$).

Complete intensity-response functions were recorded in two animals. No significant differences were found for the various Naka-Rushton parameters compared within animals. The ERG of U45, who had 8% of fibers remaining in the glaucomatous eye, had an $R_{\text{max}}$ of 243.9 $\mu$V compared to 215.6 $\mu$V in the fellow eye, a half-saturation constant (log K) of $-2.10$ vs. $-2.19$ in the normal eye, and a slope of 1.14 vs. 1.29. The ERG of W51, an animal with moderately reduced PERGs in which good histological data were not obtained, had an $R_{\text{max}}$ of 385.1 $\mu$V in the affected eye vs. 419.8 $\mu$V in the normal eye, a log K of $-2.12$ vs. $-2.16$ and a slope of 0.98 vs. 0.91 for the affected vs. normal eyes, respectively.

**Oscillatory Potentials**

Oscillatory potential amplitudes were reduced in the affected eyes when compared to those measured from the normal eyes in three out of five of the monkeys. Figure 11 illustrates OPs recorded from the normal (A) and affected (B) eyes of monkey X32,
who had lost 97% of nerve fibers in the experimental eye. From the optic nerve counts of four of the animals, it appears that a very large loss of nerve fibers must occur before a significant loss in OP amplitudes is observed. Figure 12 illustrates this relationship, and demonstrates that a monkey who had lost 92% of his optic nerve axons only had a 20% reduction in OP amplitude.

The loss in OP amplitudes is not related to any changes in the flash ERG. OP amplitude intensity-response functions measured in U45 revealed that the asymptotic amplitudes were different by about 30 μV (108 μV in the affected eye vs. 140 μV in the fellow eye) and that there was a small elevation in the half-saturation constant in the affected eye of this animal (−1.21 vs. −1.38 log cd-s/m²). Animal W51, in which a good nerve fiber count was not obtained, showed a much larger change in OP maximum amplitude (213 μV vs. 341 μV in the affected vs. the normal eye, respectively). A large elevation in the half-saturation constant was also observed (−0.77 vs. −1.73 log cd-s/m² in the affected vs. the normal eye).

Visual Evoked Potentials

No significant change of VEP phase was observed when the affected eye was compared to the normal eye of monkeys T65, U45, W56 and W51. Except for the full-field condition, the VEP in the glaucomatous eye of monkey X32 was unrecordable.

Significant reductions of VEP amplitude, however, were seen for the glaucomatous eyes of monkeys X32 and U45 which had fewer than 10% of the optic nerve fibers left (t(15) = −14.5, P < 0.0004). The average amplitude ratios for the affected vs. normal eyes of these animals were 0.03 and 0.43, respectively, where a ratio of 1.0 indicates no difference between eyes. Although amplitude ratios of monkey W56 (with 46% of fibers left in the affected eye) were not significantly different from normal (t(7) = −1.225, P < 0.27), the mean ratio across check sizes was 0.877. Monkey T65, whose glaucomatous eye had 71% of its fibers remaining by histologic examination, had an amplitude ratio of 0.974. No preferential amplitude loss over the range of check sizes was observed for any of the animals.

The VEP severely overestimated the total number of remaining fibers in monkeys U45, W56 and T65. Figure 13 shows VEP waveforms for 4° and 30° check sizes for animals U45 and W56, who despite the fact that they had only 8% and 46% of optic nerve fibers remaining in their glaucomatous eyes, respectively, showed relatively minor amplitude losses in the VEP.

Figure 14 compares the number of fibers remaining to average VEP amplitude ratios across check size for monkeys X32, U45, W56 and T65, quantitatively confirming the observation illustrated in Figure 13.

Discussion

Our data imply a strong but nonexclusive relationship between PERG amplitude and retinal ganglion cell function. For stimulus check sizes between 1° and 4° of visual angle, the relative reduction of PERG amplitude is nearly proportional to the loss of optic nerve fibers. For check sizes larger than 4° and uniform field conditions, however, as much as 30 or 40% of the PERG response remains even when almost all of the optic nerve fibers (and, presumably, ganglion cells) have been destroyed. These data may reflect a larger luminance contribution for PERGs recorded using large check sizes. PERG responses to
small check sizes also seem to be relatively less affected by nerve fiber loss than moderate check sizes. This result may be due to the pattern of nerve fiber loss in glaucoma. Because the central field is relatively spared until late in the disease, the responses to the small check sizes, which presumably are generated by the high-resolution fibers that subserve the central field, would not be expected to correlate with the total loss in optic nerve fibers in glaucoma. This hypothesis is supported by our VEP data. The VEP P component, which largely reflects activity of the fibers subserving the macula, severely underestimated the loss of nerve fibers in these monkeys. A special nerve fiber analysis restricted largely to macular fibers showed an average of about 5% less fiber loss than the analysis for the entire optic nerve, which is also consistent with the idea that macular fibers tend to be relatively resistant to glaucomatous damage. Additionally, it is possible that the amplitudes from the smallest check sizes were overestimated because of relatively low signal-to-noise ratios.

Our findings are consistent with the reports of other investigators that the PERG contains luminance-specific and pattern-specific components. In addition, Harrison et al recently found that a significant PERG response remained in a human eye after complete section of the optic nerve. Current density profile studies suggest that the source of the non-ganglion cell component of the PERG is more distal than that of the ganglion cell component, but still different from that of the flash ERG.

A primary reason for the recent intense interest in the PERG is the possibility of developing a clinical test of ganglion cell function. Results of many studies have suggested that the PERG provides the earliest evidence for glaucomatous damage, and hence may prove to be the most sensitive measure for managing glaucoma. Marx et al found abnormalities in PERG amplitudes that preceded clinically significant cupping of the optic nervehead in the eyes of primates with laser-induced glaucoma. The PERG is reduced in populations of human subjects who have glaucoma. In a study of 32 patients with primary open-angle glaucoma, Trick showed that the mean of PERG amplitudes recorded from the patient population was significantly lower than the mean PERG amplitudes recorded from age-matched normal subjects. Our study, however, suggests that managing individual patients with the PERG will be difficult at best because of the large variability inherent to the PERG. PERG variability was high even under stimulus conditions that were much better controlled than is clinically feasible. We had precise control of stimulus focus and direction of gaze, and we completely eliminated eye movements. Despite this, the normal range of PERG amplitudes extends nearly to zero. Our data had an internal control, a fellow normal eye that could be used to correct for some variations of normal PERG amplitude. In clinical practice, however, open angle glaucoma is nearly always a binocular disease for which such an internal control is not available.

Recent work on the PERG has focused not on the amplitude of the positive potential (P), but rather on the size of the second negative potential (N). Holder has shown a selective reduction of N amplitude in patients who had optic nerve disease other than glaucoma, and reductions of both the positive and negative potentials in patients who had glaucoma. Also, Weinstein et al have reported selective losses of N amplitude in their population of 12 glaucoma patients (seven with open-angle glaucoma and five with narrow-angle glaucoma) and in some of

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Fig. 13. Visual evoked potential (VEP) waveforms for the normal (solid lines) and affected eyes (dashed lines) of monkeys who had 8% (left panel) and 46% of fibers remaining (right panel). Responses are shown for 4° and 30° check sizes.

Fig. 14. Average right eye/left eye ratios of VEP amplitudes (black bars) and optic nerve fiber numbers (grey bars) for monkeys X32, U45, W56 and T65. The VEP ratios are substantially higher than the nerve fiber ratios for three of the four monkeys.
their 22 ocular hypertensive patients, although they note that P1 was also reduced in some glaucoma patients. Unfortunately, their normal control population was significantly younger than their patient populations, leaving open the possibility that some of the effect was due to age-related rather than glaucoma-related changes.

In our study, the temporal parameters of the stimulus precluded an examination of N2 in all of our subjects except W56, who showed a 54% loss of optic nerve fibers and a 37% loss of 10 Hz PERG amplitude in the glaucomatous eye. In this animal, PERGs were also recorded to 2 Hz alternating checkerboards with check sizes of 30', 1°, 2°, 4° and 8° of arc. At this temporal frequency, in agreement with the results of Trick et al., no significant difference in P1 amplitude was observed between eyes for any of the check sizes or for the mean P1 amplitude calculated across check sizes (2.50 μV vs. 2.32 μV in the normal vs. the glaucomatous eye). We also found no difference in the N2 amplitudes (5.84 μV vs. 6.02 μV, respectively), or in the P1/N2 ratios, calculated using the method of Weinstein et al (0.43 vs. 0.39, respectively). Our results were obtained on only one animal; however, they cast doubt on the reliability and sensitivity of the N2 PERG potential for detecting glaucomatous optic nerve damage.

Key words: pattern ERG, glaucoma, VEP, optic nerve histology, monkey

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