Pattern ERGs and Visual Evoked Potentials in Maculopathies and Optic Nerve Diseases

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Normative data were obtained in 52 volunteers to both transient and steady-state checkerboard pattern reversal. Two sizes of checks subtending 15' and 31' of visual angle were used. The simultaneous recording of pattern ERGs (P-ERGs) and visual evoked potentials (VEPs) permitted the determination of the retinocortical time. That is, the transit time expressed in milliseconds between the activation of retinal structures and the arrival of the signal in the visual cortex. Three groups of patients with maculopathies, optic atrophy, and suspected optic nerve demyelination were also studied. Early maculopathies were characterized by delayed P-ERG b-waves and delayed VEPs but normal retinocortical time. Optic nerve demyelination was characterized by normal P-ERGs, delayed VEPs, and prolonged retinocortical time. Optic atrophy and established maculopathies were characterized by abnormal P-ERGs and VEPs. The implications of these findings were discussed.

Invest Ophthalmol Vis Sci 26:726-735, 1985

Simultaneous recording of pattern ERGs and visual evoked potentials (VEPs) has been utilized in the last few years to study disease of the macula and optic nerve.1-7 Maffei and Fiorentini8 recorded pattern ERG (P-ERG) to grating reversal in cats and demonstrated that section of the optic nerve abolished P-ERG. They noted that the disappearance of P-ERG followed the time course of ganglion cell degeneration. These authors proposed that pattern ERG represent ganglion cell activity. Maffei6 later noted preservation of flash ERG but absence of pattern ERG in a patient 1 month after a contusion of the left optic nerve. Sherman,4 on the other hand, reported that P-ERGs are normal in optic nerve disease and abnormal or absent in maculopathies. Sherman therefore proposes that the source of P-ERG is preganglionic and not related to ganglion cells activity. In view of this controversy and conflicting reports, the role of P-ERGs remains uncertain.

We studied 27 patients with various retinal and optic nerve disease to establish, in clinical practice, the role of simultaneous recorded P-ERGs and VEPs.

Materials and Methods

Pattern ERGs were recorded with a Burian-Allen contact electrode (Hansen Ophthalmic Development Lab; Iowa City, IA) placed on the cornea. The conjunctiva and the cornea were anesthetized with tetracaine hydrochloride 0.5% ophthalmic solution. After the electrode placement, each individual subject was refracted to his or her original visual acuity. Bipolar recordings were carried out between the stainless ring imbedded on the edge of the contact lenses and the silver-coated eye speculum of the electrode. Evoked potentials were recorded from silver-silver chloride electrodes applied to the scalp with collodion. Electrodes impedance was below 5000 ohms. One electrode was placed on the scalp at the midoccipital region, 5 cm above the external occipital protuberance. The ground electrode was placed at the vertex. Input from P-ERG and scalp electrodes was fed into preamplifiers adjusted to a bandwidth of 1 Hz to 300 Hz. The output was fed simultaneously into an oscilloscope and a minicomputer. The computer sampling rate was 3700 samples/sec. Each pattern ERG and VEP was the result of the summation of 100 responses. Pattern visual stimulation was carried out in a partially darkened room with averaged background light of 0.10 ft Lamberts or 0.342 candela/m². The output was fed simultaneously into an oscilloscope and a minicomputer. The computer sampling rate was 3700 samples/sec. Each pattern ERG and VEP was the result of the summation of 100 responses. Pattern visual stimulation was carried out in a partially darkened room with averaged background light of 0.10 ft Lamberts or 0.342 candela/m². The subject was seated comfortably in front of a translucent screen positioned at 1 m from the subject cornea. Pattern reversals were produced by a rapid displacement of a checkerboard through one square. The pattern was backprojected to the screen. The entire stimulating field subtended 22° and had a luminance of 34 ft Lamberts or 116.48 candela/m². Two size checks were employed routinely in every subject: 15' and 31' of arc. A contrast of 57% between dark and light checks was used. Transient P-ERGs and VEPs were obtained by a stimulation...
frequency of a complete pattern reversal every 600 msec. Steady-state ERGs and VEPs by a pattern reversal of every 62.5 msec or 8 Hz. The subject was instructed to fixate on a small red dot at the center of the field. Monocular stimulation was used with natural pupils. Patients with poor visual acuity and/or central scotoma were instructed to gaze directly toward the screen. An observer behind the screen directed the patient to maintain the gaze in an optimal position during the recording.

Flash ERGs were also obtained in every patient using Ganzfeld stimulation in accordance with the technique of Chatrian et al.9

Every subject was informed of the experimental nature of the test. The ethical principles of the Declaration of Helsinki10 concerning human experimentation were followed. Each patient underwent a complete neurophthalmologic examination, including visual field testing with Goldmann’s perimeter and tangent screen.

Results

Normal Subjects

P-ERGs and VEPs were studied in 52 normal volunteers. Their ages ranged from 20 to 84 yr with a mean of 39.3. They had a normal ophthalmologic examination.

The skewness, kurtosis, and shape of the distribution of the sample was determined for every parameter of the electrophysiologic data. The P-ERG and evoked potential latency and amplitude distribution was not Gaussian, therefore the data were transformed.11,12 Either the normal logarithm or the square root of the values were used for the transformation. The choice was based on whatever transformation produced a distribution closest to normalcy. The values of the peak latencies of P-ERGs and VEPs were transformed using the normal logarithm of the values, while the amplitude and the latencies differences between the two eyes were transformed using the square root of the values.

The limits of normal were established at the 98% tolerance limit.13 The measurements of P-ERGs and VEPs’ latencies for the right (OD) and left (OS) were highly correlated to each other (the correlation for ERG b-wave latency was 0.896; for the N70 latency, 0.80; and for P100, 0.91), therefore, the values of OD and OS were averaged and the mean used as a single entry for each individual.14,15

Transient P-ERGs (TP-ERGs) evoked by 15’ or 31’ checks were characterized by a negative-positive deflection. As shown in Figure 1, it was possible to identify a- and b-waves. In some normals, however, the a-wave was very small or absent. The amplitude

![Graph](image)

Fig. 1. Normal P-ERGs and VEPs to transient and steady-state pattern stimulation in a 36-year-old woman. The upper half of the figure shows the response to 15’ checks. The lower half of the figure shows the response to 31’ checks. RCT2 indicates retinocortical time 2, which is the transit time in milliseconds from the ERG b-wave to the N70 deflection. In the calibration signal, 10 refers to ERG amplitude; 15, to VEP amplitude. In this and all other illustrations, two trials are superimposed to show the reproducibility of the data.

and latency of each wave in tabulated in Table 1. Transient VEPs (T-VEPs) consisted of three major deflections P50, N70, and P100 (Fig. 1 and Table 1). Wave P100 was always present, while N70 was not identifiable in three subjects. Wave P50 was the most variable wave, being absent in eight subjects. Retinocortical time (RCT) was defined as the difference in milliseconds between the latency of VEP waves and ERG. Three RCTs were calculated. RCT1 = latency of P50 minus latency of a-wave; RCT2 = latency of N70 minus latency b-wave; RCT3 = latency of P100 minus the latency of b-wave. RCT1 could not be calculated in eight normal subjects. In subjects with
Table 1. Normative data to transient stimulation in 52 volunteers

<table>
<thead>
<tr>
<th></th>
<th>15' Checks</th>
<th>31' Checks</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Limit*</td>
</tr>
<tr>
<td>ERG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a-wave latency</td>
<td>28.1</td>
<td>36.9</td>
</tr>
<tr>
<td>b-wave latency</td>
<td>47.5</td>
<td>56.0</td>
</tr>
<tr>
<td>b-wave amplitude</td>
<td>2.4</td>
<td>0.3</td>
</tr>
<tr>
<td>VEP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P50 latency</td>
<td>61.0</td>
<td>76.0</td>
</tr>
<tr>
<td>N70 latency</td>
<td>76.0</td>
<td>87.8</td>
</tr>
<tr>
<td>N70 amplitude</td>
<td>4.9</td>
<td>0.1</td>
</tr>
<tr>
<td>P100 latency</td>
<td>99.8</td>
<td>115.5</td>
</tr>
<tr>
<td>P100 amplitude</td>
<td>11.6</td>
<td>0.8</td>
</tr>
<tr>
<td>Retinocortical time</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RCT1</td>
<td>32.8</td>
<td>48.6</td>
</tr>
<tr>
<td>RCT2</td>
<td>28.0</td>
<td>46.0</td>
</tr>
<tr>
<td>RCT3</td>
<td>51.9</td>
<td>67.1</td>
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</tbody>
</table>

* Indicates the 98% tolerance limit of normality. † Indicates the 98% tolerance limit of normality for the difference between OS and OD.

no identifiable N70, the onset of wave P100 was used to calculate RCT2. The onset of wave P100 was arbitrarily defined as the point where the ascending part of the P100 wave transected the baseline. Both RCT2 and RCT3 were measurable in all normal subjects. The values of the RCTs are shown in Table 1.

Steady-state ERGs (SP-ERGs) and VEPs (S-VEPs) consisted of quasi-sinusoidal deflections as shown in Figure 1. The amplitude of the steady-state ERGs and VEPs was arbitrarily defined as the mean value in microvolts of the amplitude of the peak of the first three consecutive half-deflections measured as the distance from peak to peak. The values of the amplitude and amplitude differences between the right and the left eyes for both SP-ERGs and S-VEPs are tabulated in Table 2.

Patients

Twenty-seven patients with diseases of the retina and/or optic nerve were studied. The patients were subdivided into three groups. Group 1 consisted of seven patients with retinopathies preferentially affecting the macular region. Group 2 consisted of six patients with optic atrophy, and group 3 consisted of 14 patients with multiple sclerosis. Abnormalities of P-ERGs and VEPs were defined as latencies greater than the upper boundary of normalcy or amplitude smaller than the lower boundary of normalcy established for a given parameter at the 99% tolerance limit. Retinocortical times were considered abnormal only when RCT2 and RCT3 were prolonged above the upper boundary of normalcy. The two values were always coupled, that is, when RCT2 was prolonged, RCT3 was also prolonged. The present series is too small to determine whether or not the measurement of only one RCT is a sufficient criterion for abnormality.

Group 1: Attempts were made to select patients with unilateral maculopathies in order to compare in the same subject, the value of the normal eye and the affected eye. There were 5 patients with monocular disease, including the following: central serous chorioretinopathy (1), retinal infarct of the macular and perimacular region due to branch occlusion of the central retinal artery (1), central chorioretinal scar (1), and senile macular degeneration (2). All the patients had fluorescein angiography and fundoscopic photography to evaluate the status of the macular region.

Table 2. Normative data to steady-state stimulation in 52 volunteers

<table>
<thead>
<tr>
<th></th>
<th>15' Checks</th>
<th>31' Checks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Limit*</td>
</tr>
<tr>
<td>ERG</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average amplitude</td>
<td>2.5</td>
<td>0.8</td>
</tr>
<tr>
<td>VEP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average amplitude</td>
<td>8.6</td>
<td>2.4</td>
</tr>
</tbody>
</table>

* Indicates the 98% tolerance limit of normality. † Indicates the 98% tolerance limit for the difference between OS and OD.
Table 3. Percent of abnormalities

<table>
<thead>
<tr>
<th>Type of disease</th>
<th>ERG</th>
<th>VEP</th>
<th>RCT</th>
<th>ERG</th>
<th>VEP</th>
<th>RCT</th>
<th>ERG</th>
<th>VEP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retinal</td>
<td>89</td>
<td>33</td>
<td>0</td>
<td>78</td>
<td>78</td>
<td>0</td>
<td>78</td>
<td>78</td>
</tr>
<tr>
<td>Optic atrophy</td>
<td>67</td>
<td>100</td>
<td>17</td>
<td>67</td>
<td>100</td>
<td>14</td>
<td>57</td>
<td>71</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>27</td>
<td>72</td>
<td>71</td>
<td>38</td>
<td>53</td>
<td>71</td>
<td>38</td>
<td>53</td>
</tr>
</tbody>
</table>

region. The visual acuity of the normal eye in each of these patients was 20/20 (corrected). The group also included two cases of bilateral senile macular degeneration. Transient P-ERGs were abnormal in 89% of cases with checks of 15' and in 67% of patients with checks of 31'. Steady-state P-ERGs similarly were abnormal in 78% and 58% of patients respectively (Table 3). Abnormalities usually consisted of absent or very small (less than 0.3 µV) TP-ERGs and (less than 0.8 µV) SP-ERGs (Figure 2–3). In four eyes, the
Fig. 3. Fluorescein angiography of OD in the same patient as in Figure 2. Note the dilated capillaries and extravasion of fluorescein in the macular area.

TP-ERG was present but delayed (Figure 4). T-VEP were either absent or delayed, however, in the four cases of delayed VEPs, the retinocortical times were normal. Correlation between visual acuity and transient ERGs and VEPs is shown in Table 4. One patient with senile macular degeneration limited to the right eye, and normal visual acuity, fundoscopic examination, and fluorescein angiography in the left eye had not only abnormal TP and SP-ERGs in the right eye, but also abnormal ERGs in the left eye. The abnormality in the left eye was confined to checks of 15'. These findings raise the possibility that pattern ERGs may be used for this early detection of macular lesions.

Group 2: Only patients with established optic atrophy were selected. One subject had bilateral optic atrophy; thus, seven eyes were studied. Optic atrophy was defined by the presence of pale optic disc, central scotoma, and visual acuity equal or worse than 20/200. Marcus Gunn phenomenon was also noted in five eyes. The etiology of the optic atrophy was as follows: meningitis (1), retrobulbar neuritis (3) (four eyes), optic nerve compression (1), and ischemic optic neuropathy (1). Transient and steady-state P-ERGs were abnormal (as defined above) in 67% of cases to 15' checks and in 57% of cases to 31' checks. T-VEPs to 15' and 31' were abnormal in all cases. S-VEPs to 15' checks were abnormal in all cases, while S-VEPs to 31' were abnormal in 71% of affected eyes (Fig. 5). Ganzfeld flash ERG were normal in each instance (Fig. 6).

Correlation between visual examination and P-ERGs is shown in Table 4. Two cases of optic atrophy with central scotoma and visual acuity of 20/200 had normal T-ERGs to both 15' and 31' checks. One of these patients had definite multiple sclerosis, the diagnosis of optic atrophy was first made in 1967. Visual acuity and central scotoma had remained stable during the ensuing 16 years. The other patient had bilateral optic atrophy and multiple sclerosis. Optic atrophy in the right eye had been present since 1950, while the left eye was functioning satisfactorily 3 months prior the diagnosis of optic atrophy. Visual acuity in the right eye was limited to hand motion, and the P-ERGs were absent. Visual acuity in the eye with normal P-ERGs was 20/200. A third patient who had the diagnosis of retrobulbar neuritis 2 months prior to testing is of particular interest. He had a central scotoma and vision limited to hand motion; the disc was pale. TP- and SP-ERGs were absent to 15' checks but present to 31' checks.

Group 3: Fourteen patients with the diagnosis of definite or probable multiple sclerosis (criteria of McAlpine16) were studied. Patients with optic atrophy were excluded from this group. Most of these patients had normal ophthalmologic examination (Table 4). Five subjects had a small central scotoma due to a recent (less than 6 weeks) retrobulbar neuritis. TP-ERGs and SP-ERGs were abnormal only in approximately 21–38% of patients (Table 3). These abnormalities were present in patients with either central scotoma or pallor of the disc. Subjects with no visual difficulties had normal P-ERGs, although often their VEPs and RCTs were delayed. The typical pattern of patients with multiple sclerosis without disc temporal pallor and without impaired central vision was a normal pattern ERG with delayed transient VEPs and prolonged RCTs (Fig. 7). Steady-state stimulation was unrewarding in this group.

Discussion

Pattern-reversal ERGs to small and medium-size checkerboard patterns were reliably recorded simultaneously with VEPs. Transient P-ERGs were characterized by a negative a-wave followed by a positive
Fig. 4. A 63-year-old man with bilateral but asymmetrical senile macular degeneration. The upper half of the illustration shows the results to 15’ checks, the lower half of the illustration shows results to 31’ checks. In the less affected right eye (OD) steady-state stimuli evoked normal P-ERGs and VEPs but transient stimuli to 15’ checks evoked delayed ERGs and VEPs with a normal retinocortical time (left half of illustration). 31’ checks evoked normal response. Responses to steady-state are absent in the left eye (OS), response to transient stimuli are markedly depressed.

b-wave as described by Armington, Persson and Wanger, and Korth. The amplitude of the b-wave was unrelated to the size of the checks. Armington and Brigell similarly reported a lack of correlation between the amplitude of the b-wave and the spatial frequency of the pattern. Korth using onset and offset square wave stripe patterns, obtained P-ERGs with greatest amplitude at a spatial frequency of 3–4

Table 4. Correlation between visual examination and electrophysiologic data

<table>
<thead>
<tr>
<th>Number of eyes affected</th>
<th>Visual acuity</th>
<th>Central scotoma</th>
<th>Disc pallor</th>
<th>31’ TP-ERG AB</th>
<th>31’ TP-VEP AB</th>
<th>15’ TP-ERG AB</th>
<th>15’ TP-VEP AB</th>
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<tbody>
<tr>
<td>Maculopathies</td>
<td>9</td>
<td>20/200%–20/40</td>
<td>6</td>
<td>6</td>
<td>5</td>
<td>5</td>
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<tr>
<td></td>
<td></td>
<td>20/30–20/20</td>
<td>3</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Optic atrophy</td>
<td>7</td>
<td>HM–NLP</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FC–20/200</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>28 (?)</td>
<td>20/25–20/20</td>
<td>23</td>
<td>0</td>
<td>10*</td>
<td>23</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20/50–20/30</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>1</td>
</tr>
</tbody>
</table>

* Indicates pallor of the disc limited to the temporal side.
AB: abnormal; HM: vision limited to hand motion; NLP: no light perception; FC: vision limited to finger counting.
Fig. 5. A 36-year-old man with left optic atrophy secondary to retrobulbar neuritis 2 yr prior to present examination. Vision to the left eye is limited to hand motion. The upper half of the illustration shows the results to 15' checks; the lower half of illustration, the results to 31' checks. Note the complete absence of P-ERGs and VEPs to stimulation of the affected eye OS and normal responses to stimulation of the normal eye OD. In the calibration, marker 5 refers to ERGs amplitude; marker 10, to VEPs amplitude.

s'36

OS

VA = HANDS MOTION ONLY

OPTIC ATROPHY OS

OD

VA = 20/20

TRANIENT STEADY-STATE

CHECKS = 15'

b49

RCT2 = 22

P99.5

N71

CHECKS = 31'

b47.5

RCT2 = 19

P98.5

N66.5

256 msec

256 msec

256 msec

c/deg. Similarly Hess and Baker21 and Baker and Hess,22 using grating pattern reversal, obtained ERGs with the greatest amplitude at 2-5 c/deg. This discrepancy may be related to the different nature of the stimulus and/or to relative narrow range of spatial frequency tested in our study. On the other hand, the latency of TP-ERGs and T-VEPs were dependent on the size of the pattern and were constantly shorter with 31' checks and longer with 15' checks. Korth19 studied the peak latency as a function of spatial frequency over 11 different spatial frequencies. He noted a linear steady increase with increasing spatial frequency for the pattern onset b-wave. The spatial frequency band-pass behavior of the P-ERG, with the changes in amplitude and latency, in relation to frequency and the shift of spatial tuning to lower spatial frequencies with increasing retinal eccentricity,21,22 indicates that the ERG arises from postreceptor neuronal cells. These physiologic studies in humans are complimentary to the animal data of Maffei,6,8 suggesting that P-ERG arises from ganglion cell activity.

Whether it is accepted that P-ERG reflects ganglion cell activity6,8 or a preganglionic neuronal activity,2-4 it is generally acknowledged that P-ERG is a valid index of postreceptor retinal processes.19-22 The simultaneous recording of TP-ERGs and T-VEPs permits the determination of the transit time from the retina to the cortex, ie, the retinocortical time. Occasionally, the a-wave and/or the P50 wave are absent in normal, therefore the retinocortical time cannot be computed. RCT2 and RCT3 were always measurable and are a reliable indicator of the transit time from retina to cortex.

The morphology of the steady-state P-ERGs and VEPs was similar to previously reported studies.1-8,21,22 However, our data is the first reported quantified normative values in a large group of normal subjects. Normative boundaries, utilizing modern statistical analysis,11,15 were set at the 98% tolerance level for
both latencies and amplitudes. These strict criteria of abnormalities permitted the identification of deviant electrophysiologic parameters with only a 2% error.

The simultaneous recording of P-ERGs and VEPs in the three groups of patients yielded interesting results. Two specific diagnostic parameters emerged: (1) delayed TP-ERG b-wave, delayed T-VEP N70-P100 with normal retinocortical time in early macular lesions; and (2) normal TP-ERG with prolonged T-VEP N70-P100 and prolonged retinocortical time in demyelination of the optic nerve. ERGs and VEPs were absent or markedly reduced in amplitude in cases of optic atrophy confirming previous reports by Fiorentini et al, Maffei, Bobak et al, and Dawson et al. Chronic lesions of the optic nerve causing optic atrophy are known to result in retrograde degeneration of ganglion cells. The absence of pattern ERG in these cases support the hypothesis of Maffei that this electroretinal event is generated in the ganglion cells.

However, two eyes with optic atrophy had normal transient and steady-state P-ERG. Optic atrophy in these cases was secondary to retrobulbar neuritis associated with multiple sclerosis. A third patient with recent onset of optic atrophy had normal P-ERGs to 31' checks and absent P-ERGs to 15' checks. These findings may indicate a gradual involvement of retinal cells with an earlier impairment of cells.
located in the foveola. The possibility that eye movements in patients with poor visual acuity due to a central scotoma may have interfered with the recording of the small pattern ERGs in the cases with absent P-ERGs must be considered. However, we believe that this explanation is not valid because P-ERGs were still absent when testing was carried out binocularly and the patient was therefore able to fixate with the normal eye. Although good fixation is necessary for accurate recording of P-ERGs, satisfactory fixation can be maintained in cooperative patients provided the recording time is kept short and the patient allowed to rest in between trials. Normal P-ERGs in optic atrophy were also reported by Sherman.\(^4\) His case 2 and case 4 had optic atrophy for longer than 4.5 yr. In these cases the preservation of P-ERG may either be explained by only partial axonopathy and retrograde degeneration with preservation of a number of ganglion cells sufficient to produce an ERG, or by the possibility that other postreceptor retinal cells constitute the source of the P-ERG. If the optic atrophy has developed recently, a normal P-ERG is to be expected because the retrograde degeneration of ganglion cells has not yet occurred.\(^24,25\)

Abnormal small or absent P-ERGs also were observed, however, in maculopathies. Similar abnormal pattern ERGs were described independently by Sherman\(^4\) and Trau et al\(^26\) in retinal diseases affecting the macula. Sherman\(^4\) suggested that these findings favor a preganglionic source for P-ERG. We are proposing an alternate explanation based on the importance of the macular region as a trigger to evoke pattern ERGs. Armington and Brigell\(^19\) used annular checkerboard stimuli to show that P-ERG was best evoked by stimuli directed to the macular region rather than the peripheral retinal region. They stated that "the cone receptors that initiated the response are most concentrated in the center of the field." In maculopathies, the stimulus either fails to trigger the cones or the preganglionic cells and therefore the ganglion cells are not activated. P-ERG evoked by small checks requires intact macular receptors, intermediate neurons, and ganglion cells. Pathologic states affecting the macular region and/or the ganglion cells will similarly affect the generation of P-ERG. In maculopathies, the stimulus is not coded or cannot be transmitted to the ganglion cells, and in ganglion cells degeneration, the ganglion cells are unable to process the coded message. The importance of an intact macular region was shown in one of our cases of senile macular degeneration. P-ERG to 15' and 31' checks were absent, but the use of larger checks of 1°4' evoked normal P-ERG presumably activating receptors in peripheral retinal fields. Transient P-ERG-VEP results were not significantly different than the results obtained with steady stimuli. However, there were some cases of maculopathies with abnormalities limited to either transient or steady-state ERGs. These findings suggest that the use of both stimuli may increase the diagnostic yield and raises the interesting possibility that the two stimuli may activate different channels.\(^27\)

Although maculopathies and optic nerve diseases associated with axonopathy and retrograde degeneration of ganglion cells have similar electrophysiologic findings, their diagnosis can be easily established by other neuroophthalmologic tests such as fundoscopic examination and fluorescein angiography. The importance of these electrophysiologic tests resides in the early detection of macular dysfunction. One of our cases of macular degeneration with subjective, fundoscopic, and angiographic evidence of unilateral involvement showed bilateral P-ERG abnormalities indicating subclinical involvement of the "spared" macula. The importance of P-ERG for the early detection of maculopathies was first suggested by Sokol.\(^28\)

The Multiple Sclerosis group includes patients with variable optic nerve pathology. Although it is reported that more than 90% of optic nerves of MS patients have demyelinating plaques,\(^29\) there is no available clinical method to assess the extent of the demyelination nor the amount of axonal degeneration present in the plaque. It is a parsimonious interpretation of the present data that most of the patients have a predominantly demyelinating lesion of the optic nerve. It is to be expected, however, that a small percentage of these patients have axonal involvement with various degree of retrograde degeneration. This small subgroup will have abnormal P-ERGs, while the majority of the patients with presumed pure demyelination have normal P-ERGs and delayed P-VEPs. To support this interpretation, there is the observation that MS patients with normal visual examination always have normal P-ERGs.

Another interesting feature of the present study was the demonstration of two types of abnormalities in cases of optic and/or retrobulbar neuritis. One type of abnormality consisted of normal P-ERG, prolonged VEP, and prolonged RCT, indicating demyelination of the optic nerve with preservation of the axons. Normal P-ERGs were also described in 28 patients with optic neuritis.\(^30\) The other type of abnormality consisted of abnormal P-ERGs and VEPs and was noted in cases with involvement of the axons and retrograde degeneration of the ganglion cells. Porciatti et al\(^31\) noted a similar dichotomy and suggested that the presence of a normal P-ERG is a better prognostic sign for functional recovery than
the absence of P-ERG. Further data are needed before the
prognostic significance of P-ERG can be deter-
mind definitely.

In conclusion, the utilization of simultaneous re-
cording of ERGs and VEPs provides diagnostic and
pathophysiologic insights into retinal and optic nerve
disease.

Key words: pattern ERG, visual evoked potentials, macu-
lopathies, optic atrophy, optic nerve

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visually evoked potentials from adult ambliopes in response to
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