Jpn J Ophthalmol 2004;48:215–221
DOI 10.1007/s10384-003-0042-y

LABORATORY INVESTIGATION

Amplitude of the s-Wave of Multifocal Electroretinograms Can Indicate Local Retinal Sensitivity in Glaucomatous Eyes

Ken-ichi Murai, Yutaka Tazawa, Masaki Kobayashi, and Akira Hayasaka

Department of Ophthalmology, Iwate Medical University School of Medicine, Morioka, Japan

Abstract

Purpose: To determine whether the amplitude of the s-wave on a multifocal electroretinogram (mfERG) is correlated with the degree of visual field depression in eyes with glaucoma.

Methods: Twenty patients (20 eyes) with glaucoma,ages 46 to 69 years, were studied. Twenty healthy volunteers (20 eyes) with normal intraocular pressure and with no eye diseases served as controls. The retinal sensitivities of the upper and lower visual fields of the glaucomatous eyes were determined with a Humphrey Field Analyzer. The severity of retinal sensitivity depression was rated as mild (Group A), intermediate (Group B), or severe (Group C). To record the s-wave, mfERGs were elicited by pseudorandom stimulation, with the stimulus alternating according to a binary m-sequence for base periods (bpds) of 13.3, 26.7, 53.3, 106.7, and 213.3 ms. The mfERGwaves recorded from the upper and lower visual field were summed separately.

Results: In the control group, the s-wave in the summed mfERG was observed in all visual field halves at all bpds 53.3 ms or longer. The s-wave amplitude at a bpd of 213.3 ms was significantly larger than that at a bpd of 53.3 ms ($P < 0.05$). The s-wave was also present in the glaucoma patients' eyes, and the s-wave amplitude increased as the bpd increased. At bpds of 53.3, 106.7, and 213.3-ms, the mean s-wave amplitudes in Groups B and C were significantly smaller than those in the control group ($P < 0.05$, 0.01, and 0.05, respectively). At bpds of 53.3 and 106.7 ms, the mean amplitude of the s-waves in Group C was significantly smaller than that in Group A ($P < 0.05$). At a bpd of 106.7 ms, a significant correlation was observed between the retinal sensitivity and the s-wave amplitude ($P < 0.05$).

Conclusions: The significant correlation between the retinal sensitivity and the amplitude of the s-wave at a bpd of 106.7 ms supports the suggestion that the s-wave originates from the retinal ganglion cells and their axons. The amplitude of the s-wave may serve as an objective indicator of the severity of retinal ganglion cell damage. **Jpn J Ophthalmol** 2004;48:215–221 © Japanese Ophthalmological Society 2004

Key Words: ganglion cell, glaucoma, multifocal electroretinogram, s-wave, visual field

Introduction

Different components of the multifocal electroretinogram (mfERG) originate from the neural activity of different cells in the outer and inner layers of the retina, including the retinal ganglion cells (RGCs).[1–5] The s-wave, recently reported by us,[5] is a positive wavelet that is present on the descending limb of the first positive wave (P1) of the first-order kernel of the mfERG and is best seen at longer base periods[6] (bpds) of stimulation. The s-wave is important because its properties in normal eyes suggest that it probably originates from RGCs.[5] This suggestion is supported by its absence in eyes with optic neuritis, and its return after the eyes recover from optic neuritis. In addition, we have

Received: September 19, 2003 / Accepted: December 16, 2003
Correspondence and reprint requests to: Yutaka Tazawa, Department of Ophthalmology, Iwate Medical University School of Medicine, 19-1 Uchimaru, Morioka 020-8505, Japan
e-mail: ytazawa@iwate-med.ac.jp

found that the amplitude of the s-wave is significantly smaller in eyes with primary open-angle glaucoma (POAG) than in normotensive eyes (unpublished data). However, we did not find a significant correlation between the s-wave amplitude and the sensitivity of the visual field determined by a Humphrey field analyzer (HFA). Thus, we were not able to unequivocally conclude that the s-wave originated from the RGCs.

Because visual field defects in eyes with glaucoma result from damage to the axons of the RGC, we hypothesized that the decrease in the amplitude of the s-waves would correlate with the severity of the visual field depression. To test this hypothesis, we recorded mfERGs from patients with glaucoma with different degrees of visual field defects in the upper and lower halves of the visual field. We then compared the amplitude and implicit time of the s-waves recorded from the upper and lower visual field halves with the sensitivity of the retina in these corresponding areas.

Subjects and Methods

Informed consent was obtained from each of the subjects after a thorough explanation of the purpose and procedures of this study. The procedures used conformed to the tenets of the Declaration of Helsinki.

Control Group

Twenty age-matched volunteers (20 eyes) served as controls. Their ages ranged from 35 to 65 years with a mean ± SD of 54.2 ± 5.8 years. The mean refractive error was −2.75 ± 1.68 dioptors (D), and the best-corrected visual acuity was 1.0 or better in all eyes. Slit-lamp biomicroscopy and indirect ophthalmoscopy showed no abnormalities of the anterior segment, optic media, or ocular fundus. The mean intraocular pressure (IOP) at the time of the mfERG recordings was 16.0 ± 0.87 mmHg with a range from 10 to 19 mmHg. The HFA test was not performed on the control group.

Glaucoma Group

The glaucoma group was made up of 20 patients (20 eyes); 10 patients (10 eyes) had POAG, and 10 patients (10 eyes) had normal-tension glaucoma (NTG). The mean age of the subjects was 58.5 ± 6.9 years with a range from 46 to 69 years. The mean refractive error (spherical equivalent) was −1.73 ± 1.21 D with a range from −3.0 to +3.0 D. The best corrected visual acuity was 0.8 or better in each eye. Examination of the anterior segment and the optic media by slit-lamp microscopy and the fundus by indirect ophthalmoscopy revealed no ophthalmological abnormalities other than incipient senile cataracts and those associated with glaucoma.

Glaucomatous cupping, a cup/disk (C/D) ratio over 0.7, of the optic disk was seen in all eyes. The IOP at the first visit ranged from 20 to 26 mmHg (mean, 22.4 ± 2.0 mmHg) for the POAG eyes, and from 15 to 20 mmHg (16.6 ± 1.1 mmHg) for NTG eyes. The interval between the first visit to the time of the mfERG recording ranged from 2 to 18 years (5.9 ± 4.4 years). One to two times per day of one to three kinds of antiglaucoma eye drops (mean, 1.3) had been prescribed during this period.

The IOP at the time of the mfERG recordings was 21 mmHg or lower in all of the eyes. Prior to the mfERG recordings, none of the patients had used any topical pilocarpine hydrochloride or oral carbonic anhydrase inhibitors and none of the patients had undergone ophthalmic surgery.

mfERG Recordings

The Visual Evoked Response Imaging System (VERIS; MAYO, Nagoya, Japan) was used for the mfERG recordings.[5–6] A bipolar Burian-Allen contact lens electrode was used to pick up the mfERGs, and the ground electrode was attached to the right earlobe. The other eye was occluded.

Before the mfERG recordings, the corrected visual acuity of each eye was measured. Then, the pupils were dilated with topical 0.5% tropicamide and 0.5% phenylephrine hydrochloride (Mydrin-P; Santen, Osaka, Japan). The refractive error was determined with an autorefractometer, and the spherical equivalent of the refractive error plus 3.0 D (viewing distance) was placed in front of the eye.

After light-adaptation for 15 min to 252 lux ambient room light, the cornea and conjunctiva were anesthetized by topical 0.4% oxybuprocaine hydrochloride (Benoxil; Santen) and 4% lidocaine hydrochloride (Xylocaine; AstraZeneca, Osaka, Japan).

The stimulus consisted of 37 hexagonal white (200 cd/m^2) and black (4 cd/m^2) elements arranged concentrically on a 17-inch (~43 cm) cathode ray tube (CRT) monitor. The black and white elements were alternated according to the binary m-sequence at 75, 37.5, 18.75, 9.4, and 4.7 Hz, that is, at base periods[6] (bpds) of 13.3, 26.7, 53.3, 106.7, and 213.3 ms, respectively. When the bpd was 26.7 ms or longer, gray elements (66.6 cd/m^2) were interposed between the white and black elements at a frequency of 75 Hz (Fig. 1). The white-to-gray contrast was 50%. The luminance of the screen around the stimulus elements was set at 40 cd/m^2. At each bpd, four sets of recordings, each lasting 30 s, were made (2 min in total). The recording was started at the shortest bpd, and a recording was made with the next longer bpd, with 1- to 2-min intervals between two successive sessions.

The responses were amplified with a bandpass between 10 Hz and 300 Hz. Artifact removal was performed once to reduce noise. The signals were processed with the VERIS analysis software (VERIS Science 4.1.1.).

The 37 first-order kernels of the mfERG recorded from both normal and glaucomatous eyes were divided into two groups, 15 waves from the upper and 15 from the lower half

of the visual field (Fig. 2). The 15 waves for each half were summed to an all-trace wave, and the amplitudes and implicit times of the s-wave (Fig. 3) and the first positive wave (P1) of the all-trace waves were measured.

Visual Field Measurements

The retinal sensitivity profile of the eyes in the glaucoma group was determined by analyzing the data obtained by the HFA (Carl Zeiss Meditec, Dublin, CA, USA) with the Swedish Interactive Threshold Algorithm Standard 24-2. As with the mfERGs, the results for the upper and lower halves of the visual fieldwere analyzed separately. The difference from the normal value for the corresponding age in the mean retinal sensitivity was calculated for each half of the visual field. This difference (dB) was used to grade the severity of visual field depression: Group A (dB $>$ 0), Group B (0 $\geqq$ dB $>$ -4), and Group C (dB $\leqq$ -4) (Table 1).

Statistical Analyses

Post-hoc tests (Scheffe's method) were used to determine whether the s-wave amplitude, implicit time, and P1 amplitude at each bpd were significantly different among the three retinal sensitivity groups. The correlation between the retinal sensitivity (dB values) and the s-wave amplitude was analyzed by simple regression analysis. A P value of <0.05 was considered statistically significant.

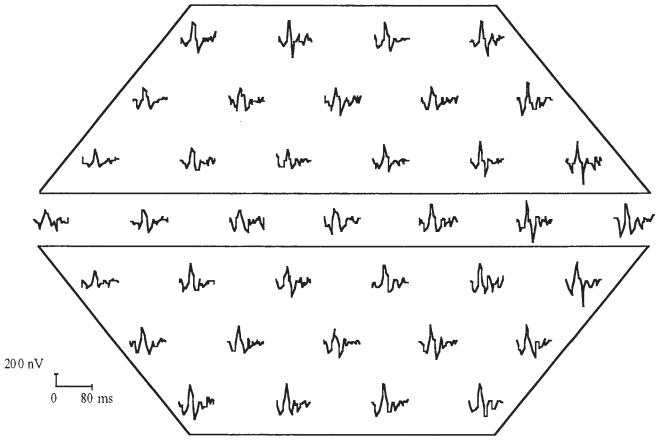

Figure 2. Grouping of multifocal electroretinograms into upper and lower halves.

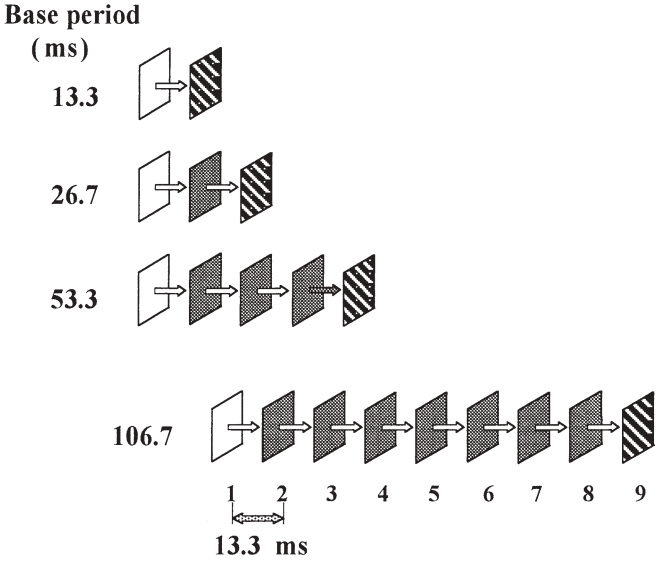

Figure 1. Stimulus pattern. To obtain base periods (bpds) longer than 13.3 ms, gray elements (66.6 cd/m^2) were interposed between the white and black (or white) elements at a frequency of 75 Hz. The elements illustrated with *striped patterns* are white or black according to a pseudorandom binary m-sequence.

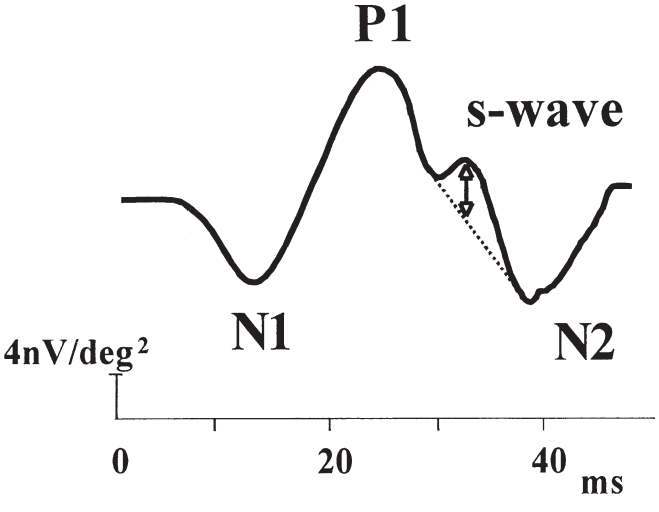

Figure 3. Method of measuring the amplitude and implicit time of the s-wave. The amplitude of the s-wave was measured as the height of a vertical line from the peak of the s-wave to its intersection with a line connecting the troughs of waves on either side of the s-wave peak. *P1*, first positive wave; *N1* and *N2*, negative waves.

Table 1. Grouping of visual field halves by dB values

Group	Number of halves	dB (mean ± SD)	Range (dB)
A (dB $>$ 0)	16	2.13 ± 1.65	0.16 to 4.48
B (0 $\geq$ dB $>$ -4)	11	-2.11 ± 1.32	-3.88 to -0.20
C (dB $\leq$ -4)	13	-13.29 ± 8.65	-29.92 to -4.44

dB, difference in mean retinal sensitivity from the normal value for the corresponding age.

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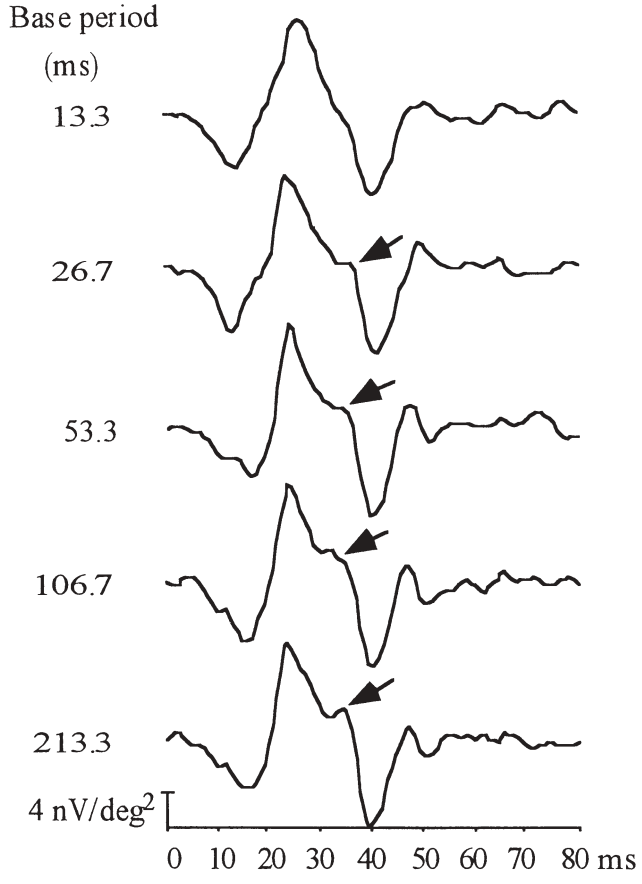

Figure 4. Examples of the averaged waveform of the first-order kernel elicited by different bpds in a normal control eye. In this case, an s-wave (*arrows*) was present on the descending limb of P1 for bpds ≥26.7 ms.

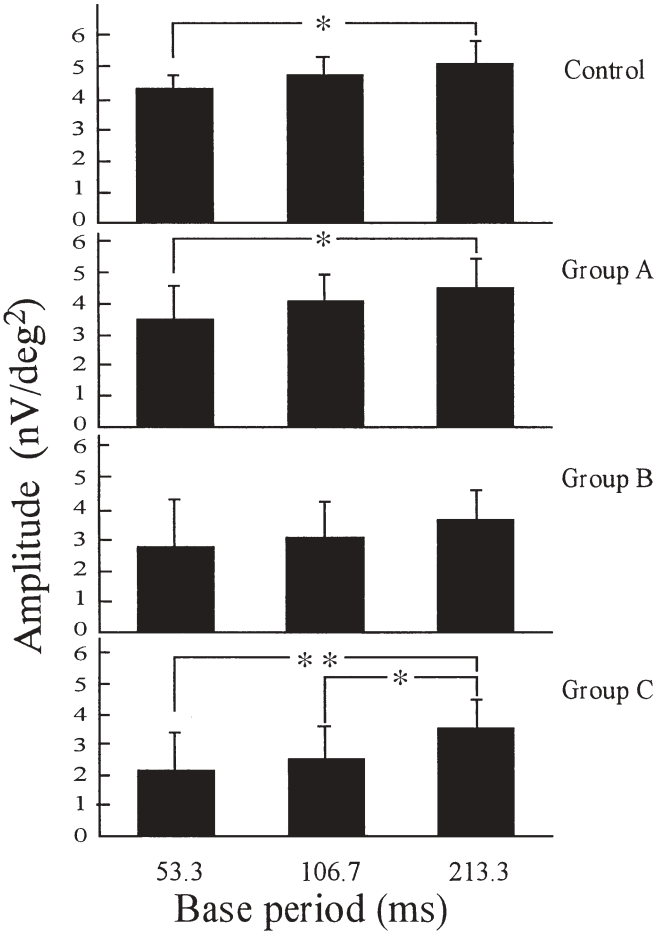

Figure 5. The mean amplitudes ± SD of the s-wave in normal control eyes and glaucomatous eyes (*Groups A, B, and C*) as a function of bpd. The longer bpds elicited larger average amplitudes in all groups. $*P < 0.05$; $**P < 0.01$.

Results

Control Group

The all-trace waves of the first-order kernels of the mfERGs recorded at each bpd from a control subject are shown in Fig. 4. The first-order kernel was made up mainly of an initial negative wave (N1), the first positive wave (P1), the small positive s-wave on the descending limb of the P1, and the subsequent negative component (N2). The s-wave was not observed in the recordings at bsp of 13.3 ms, but at a bpd of 26.7-ms, the s-wave was present in four (two each of the upper and lower halves) of the 40 visual field halves. At bpds longer than 53.3 ms, the s-wave appeared in all 40 visual field halves.

The mean (±SD) amplitudes of the s-wave at bpds of 53.3, 106.7, and 213.3 ms were 4.27 ± 0.47, 4.70 ± 0.65, and 5.07 ± 0.74 nV/deg^2, respectively (Table 2). The s-wave amplitude at the bpd of 213.3 ms was significantly larger than that at the bpd of 53.3 ms ($P < 0.05$; Fig. 5).

In the control group, the mean amplitude and implicit times of the s-wave in the upper visual field at all bpds did not differ significantly from those in the lower visual field.

s-Wave of mfERGs in Glaucoma Group

mfERGs recorded from a glaucomatous eye in Group A are shown in Fig. 6A. In this group, the s-wave was not present in any of the visual field halves at bpds of 13.3 or 26.7-ms. At a bpd of 53.3 ms, the s-wave appeared in 15 of the 16 halves, and at bpds above 106.7 ms, the s-wave appeared in all visual field halves. In Group A, when the bpd was increased, the amplitude of the s-wave increased (Fig. 5, 6A), as in normal eyes. The mean amplitudes of the s-wave at bpds of 53.3, 106.7, and 213.3 ms were 3.32 ± 1.10, 4.13 ± 1.03, and 4.50 ± 0.94 nV/deg^2, respectively (Table 2). The s-wave amplitude at the bpd of 213.3 ms was significantly larger than that at the bpd of 53.3 ms (Fig. 5, $P < 0.05$). The implicit time of the s-wave did not differ significantly among the different bpds.

Table 2. Amplitudes and implicit times of s-waves of control eyes and eyes with glaucoma

Base period (ms)	Group	Amplitude (nV/deg^2)	Implicit time (ms)
53.3	Normal	4.27 ± 0.47 (40)	33.21 ± 2.22
	A	3.32 ± 1.10 (15) *	33.34 ± 1.86
	B	2.74 ± 1.61 (11) * **	34.15 ± 2.04
	C	2.13 ± 1.32 (13)	33.95 ± 2.14
106.7	Normal	4.70 ± 0.65 (40)	33.30 ± 1.95
	A	4.13 ± 1.03 (16) *	33.10 ± 2.44
	B	2.98 ± 1.22 (11) * **	34.41 ± 1.90
	C	2.45 ± 1.12 (13)	34.48 ± 1.30
213.3	Normal	5.07 ± 0.74 (40)	34.10 ± 2.32
	A	4.50 ± 0.94 (16) *	34.44 ± 1.65
	B	3.63 ± 1.03 (11) *	34.23 ± 2.03
	C	3.50 ± 0.95 (13)	34.10 ± 2.28

Numbers in parentheses indicate numbers of visual field halves from which the s-wave was detected.
*$P < 0.05$; **$P < 0.01$.

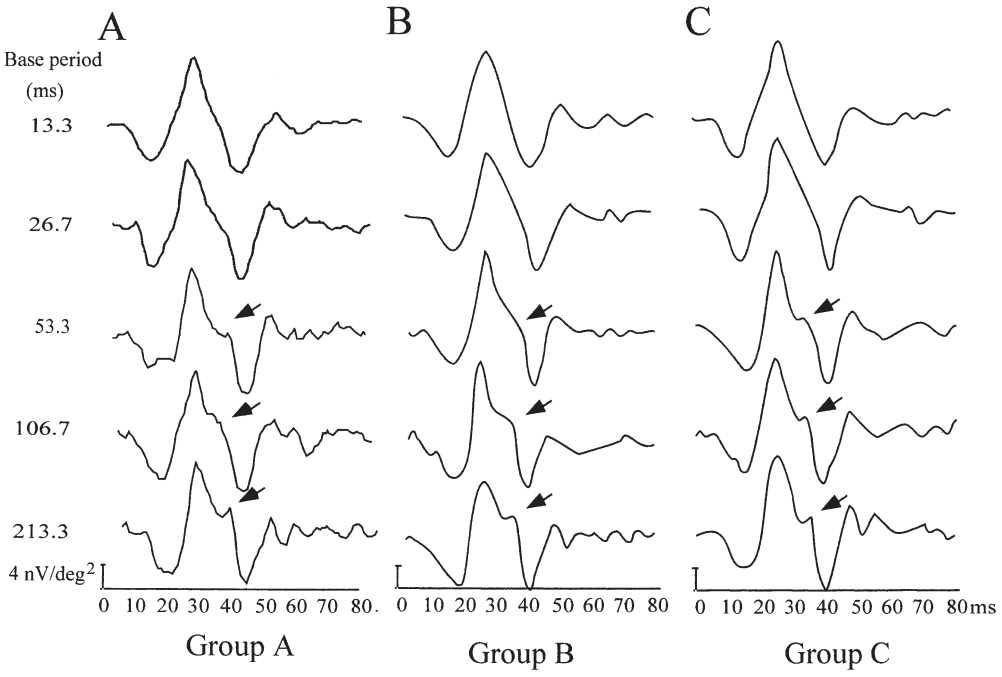

Figure 6A–C. Examples of the waveforms recorded from the halves in eyes of glaucoma patients. **A** Group A; **B** Group B; **C** Group C. *Arrows* point to the s-waves.

In Group B, the s-wave was not present at bpds of 13.3 or 26.7 ms but was present at bpds longer than 53.3 ms in all of the visual field halves (Fig. 6B). As in the control eyes, the mean amplitude of the s-wave in group B increased as the bpd increased, but the mean s-wave amplitudes were not significantly different among bpds (Table 2, Fig. 5). The implicit times of the s-wave were also not significantly different among the different bpds.

In Group C, the s-wave was also not present at bpds of 13.3 and 26.7 ms, but was present at bpds longer than 53.3 ms in all visual field halves (Fig. 6C). The mean amplitudes of the s-wave at bpds of 53.3, 106.7, and 213.3 ms were 2.13 ± 1.32, 2.45 ± 1.12, and 3.50 ± 0.95 nV/deg^2, respectively (Table 2). The s-wave amplitudes at bpds of 53.3 and 106.7 ms were significantly smaller than that at the bpd of 213.3 ms (Fig. 5, $P < 0.01$ and $P < 0.05$, respectively). The implicit times of the s-wave were not significantly different among the different bpds.

Intergroup Analysis of the s-wave

The mean s-wave amplitudes at bpds of 53.3, 106.7, and 213.3 ms in Groups B and C were significantly smaller than

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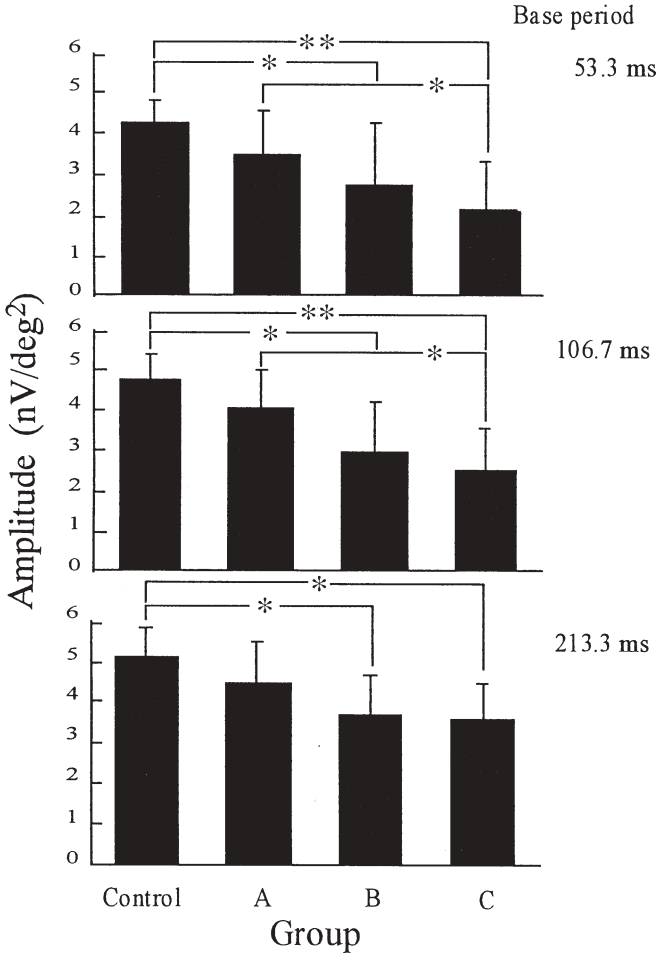

Figure 7. The mean amplitudes ± SD of the s-wave in normal control eyes and glaucomatous eyes as a function of the severity of the visual field damage. The amplitudes from the groups with severe visual field depression were smaller than those from the groups with mild visual field depression at 53.3, 106.7 and 213.3 ms bpds. $*P < 0.05$; $**P < 0.01$.

that in the normal control group ($P < 0.05$ and $P < 0.01$). The mean s-wave amplitude at bpds of 53.3 and 106.7 ms in Group C was significantly smaller than that in Group A ($P < 0.05$; Table 2; Fig. 7).

mfERG P1

The mean amplitudes and the implicit times of P1 of the first-order kernel were not significantly different among eyes in Groups A, B, and C for all bpds.

Retinal Sensitivities by HFA

The mean retinal sensitivity in Group A was 2.13 ± 1.65 dB for 16 half-fields (7 from the upper visual field halves, 9 from the lower halves); in Group B, it was −2.11 ± 1.32 dB in 11 half-fields (6 from the upper halves, 5 from the lower halves); and in Group C, it was −13.29 ± 8.65 dB in 13 half-fields (7 from the upper halves, 6 from the lower halves) (Table 1).

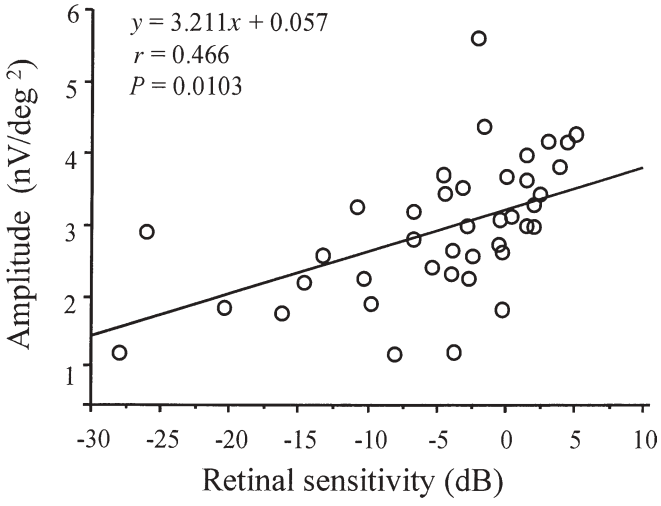

Figure 8. Correlation between the amplitude of the s-wave at a bpd of 106.7 ms and retinal sensitivity (*dB*) in halves of eyes of glaucoma patients. The amplitude of the s-wave decreased significantly ($P < 0.05$) with a decrease in the dB value.

Correlation between Retinal Sensitivity and s-Wave Amplitude

There was a significant correlation between the retinal sensitivity (dB) and the s-wave amplitude at a bpd of 106.7 ms (Fig. 8; $r = 0.466$; $P = 0.0103$). However, the correlations between retinal sensitivity and the s-wave amplitude at other bpds were not significant.

Discussion

A number of recent studies have shown that mfERGs contain components from the inner retinal cells, including the RGCs and their axons.[1–5] Many reports have also been published concerning changes in the pattern of the mfERG in cases of glaucoma.[7–14] In monkeys, Hood et al.[1] reported that the first-order kernel was made up of two positive peaks, and that the peak with the longer implicit time disappeared after an intravitreal injection of tetrodotoxin (TTX) and *N*-methyl DL aspartate (NMDLA). Because these agents suppress the spike potential of RGCs and the activity of amacrine cells, they suggested that this wave originated from RGCs and amacrine cells.

They also recorded a wave in the human mfERG that was similar in shape to that in the monkey.[15] They examined the relationship between the degree of visual field depression and the amplitude of this wave in the eyes of POAG patients with visual field depression. They did not find a significant correlation between the visual field depression and

the amplitude of this wave measured at the same retinal location. They stated that this absence of a significant correlation was because of differences in the extent of cellular damage in glaucomatous eyes; that is, some cases had damage only to RGCs, while others had damage extending to amacrine cells. They thus concluded that the mfERG cannot be used to obtain information on local visual field depression.[15]

Thienprasiddhi et al.[16] recorded multifocal visual evoked potentials (mfVEPs) from patients with glaucoma who had visual field depression limited to one hemifield. They found that mfVEPs could detect the glaucomatous visual field damage.

We studied the s-wave of mfERGs of patients with POAG and found that the s-wave of the first-order kernel was significantly smaller in eyes of patients with POAG than in eyes of normal subjects (unpublished data). This finding supports the view that the s-wave originated from the RGCs. However, a significant correlation between the s-wave amplitude and the severity of visual field depression was not found because we studied only eyes with mild visual field depression. In addition, we compared the sensitivity of the entire visual field to the all-trace s-waves in our analysis and found that when comparing the s-wave with the severity of visual field sensitivity, the mean dB value of the entire tested area was not an adequate index of the severity of visual field damage.

In glaucomatous eyes, the severity of the visual field depression often differs between the upper and lower halves; abnormal changes tend to appear in the upper visual field earlier than in the lower field.[17] Because of this difference in sensitivity, separate analyses were conducted for upper and lower visual field sensitivity (with HFA) and upper and lower s-wave amplitudes on the mfERG. Satomi et al.[18] used this method but found no significant correlation between visual field depression and amplitudes or latencies of N1 and P1 on the mfERG recorded from the upper and lower halves of glaucomatous eyes.

We found a significant positive correlation between retinal sensitivity (dB) and the s-wave amplitude at a bpd of 106.7 ms. This suggests that there is a correlation between the s-wave amplitude and the severity of the visual field damage. However, no such correlation was observed at a bpd of 213.3 ms. At present, the reason for this finding is unclear, but it was probably because the recording time (2 min) was not long enough, and because an increase in the bpd resulted in a lower number of summations of the mfERG. In future studies, this correlation may need to be assessed using recording times longer than 2 min when the bpd is longer than 106.7 ms. In addition, if the visual field were divided into smaller segments and the depression of sensitivity in each segment were compared with the s-wave amplitude in the corresponding segments, it might be possible to obtain a more detailed objective evaluation of local retinal function.

In conclusion, our results support the view that the s-wave of the first-order kernel on a human mfERG originates from the RGCs and their axons. The s-wave can probably serve as an objective indicator of the severity of not only glaucoma but also other diseases of the RGCs.

Acknowledgments. The authors thank Dr. Duco I. Hamasaki for his valuable suggestions and for editing the manuscript.

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