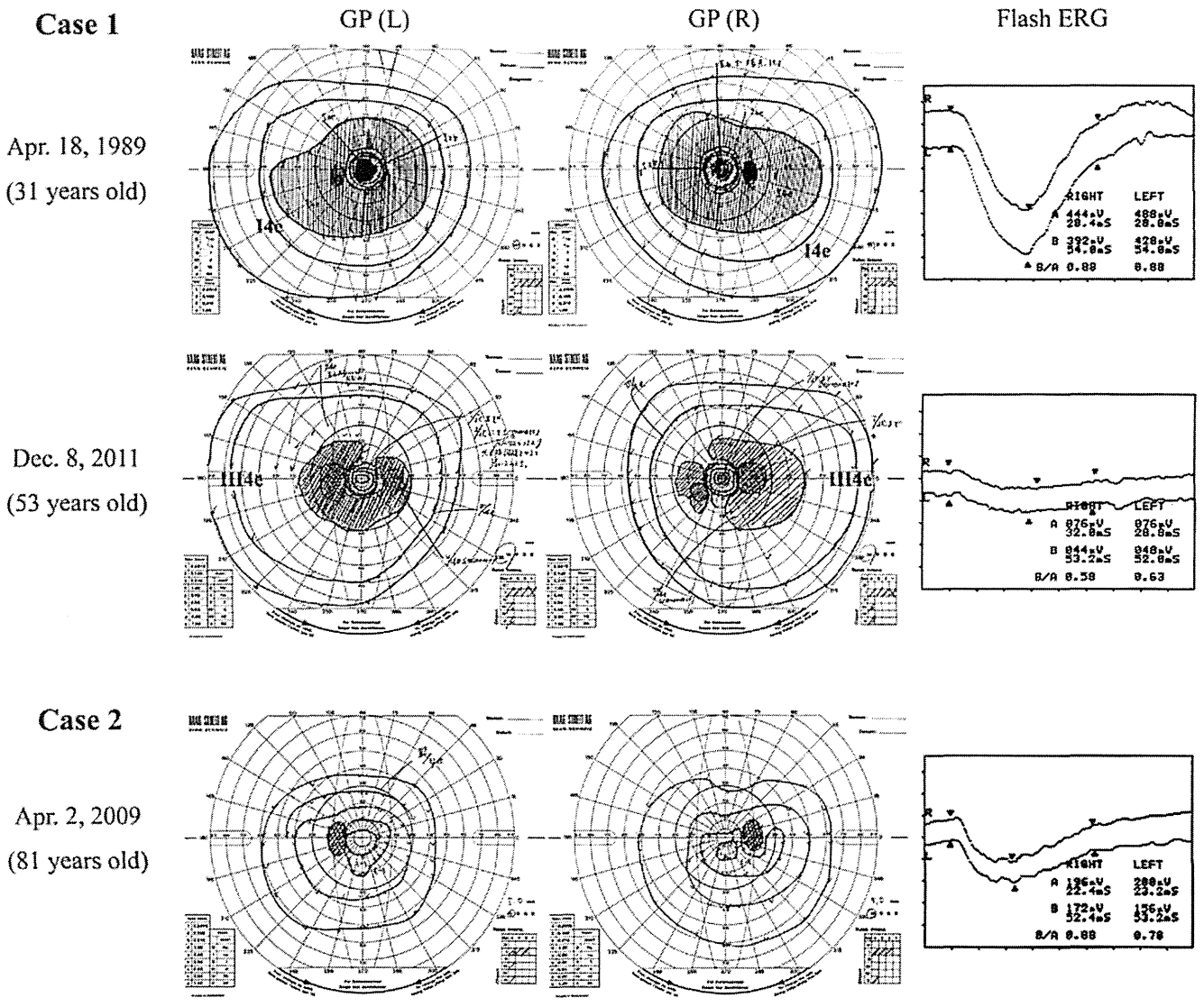


Figure 3. Results of Goldmann perimetry (GP) and flash electroretinography (ERG). In case 1, retinal sensitivity decreased and amplitude of ERG was attenuated during the 2-decade follow-up. In case 2, the visual field and the ERG findings were maintained relatively well in spite of her great age. This Figure was made with modification of Figures 2, 3, and 8 in Sato et al[3] with permission.

Figure 2. Results of fundus photography, fluorescein fundus angiography (FA) and optical coherence tomography (OCT) in case 1. At the initial visit (April 1989), cystic changes were observed in the fovea that became ambiguous 2 decades later; vision, however, did not improve. This figure was made with modification of Figures 1 and 6 in Sato et al,[3] with permission.

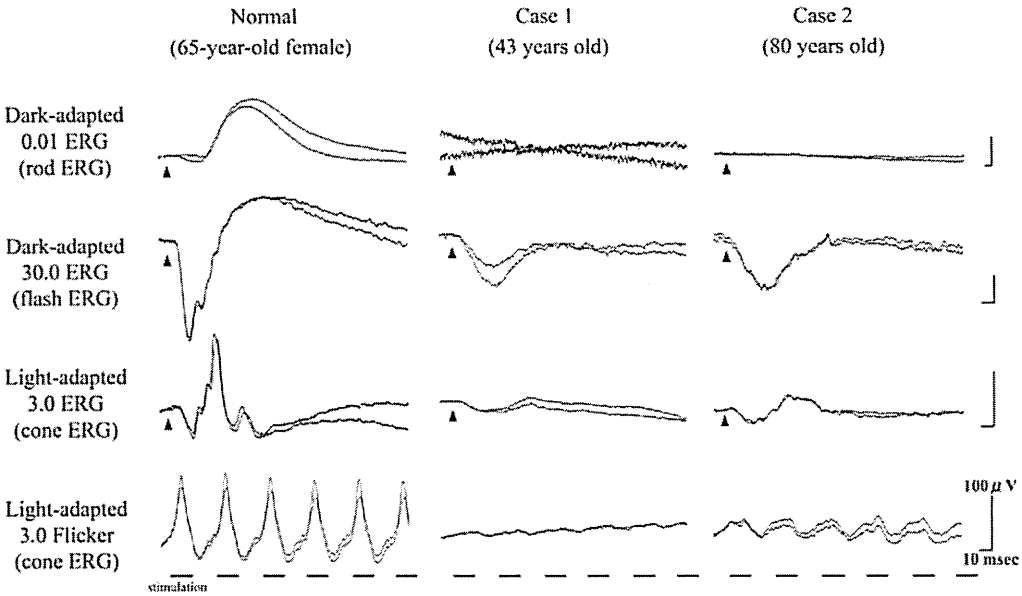


Figure 4. International Society for Clinical Electrophysiology of Vision (ISCEV)-standard electroretinography (ERGs) images. The 2 cases showed nonrecordable rod responses and significantly prolonged flash ERGs. Flicker ERGs derived from the middle- and long-wavelength-sensitive (M- and L-) cone systems were attenuated in these cases. These findings were consistent with enhanced S-cone syndrome. This figure was made with modification of Figure 4 in Sato et al[3] with permission.

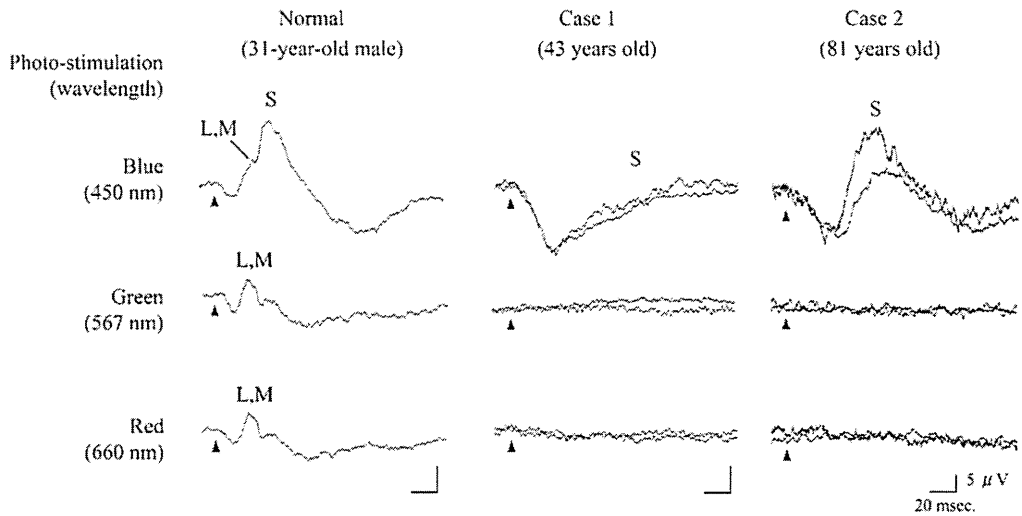


Figure 5. Color electroretinography (ERGs) images. The ERGs were recorded using 3-colored LED-built-in electrode (Kuniyoshi et al. Doc Ophthalmol 2003;106:311-8). The ERGs were elicited by 3 kinds of stimulus, namely, blue, green, and red light under the yellow background light. Luminance of the yellow background light was 670 cd/m$^2$ and duration of the stimulus was 2 milliseconds. The ERG waveform elicited by blue stimulus in the normal subject showed double-peaked, namely, rapid b-wave, which was derived from the L- and M-cone systems (L, M) followed by slow b-wave derived from the S-cone system (S). In cases 1 and 2, large b-wave with slow peak time was recorded with blue photostimulation, whereas no response was recorded with green and red photo stimuli. The intensity of the color stimulus was decided to elicit the rapid b-wave (L, M) with almost the same amplitude as in the normal subject. This Figure was made with modification of Figure 5 in Sato et al[3] with permission.

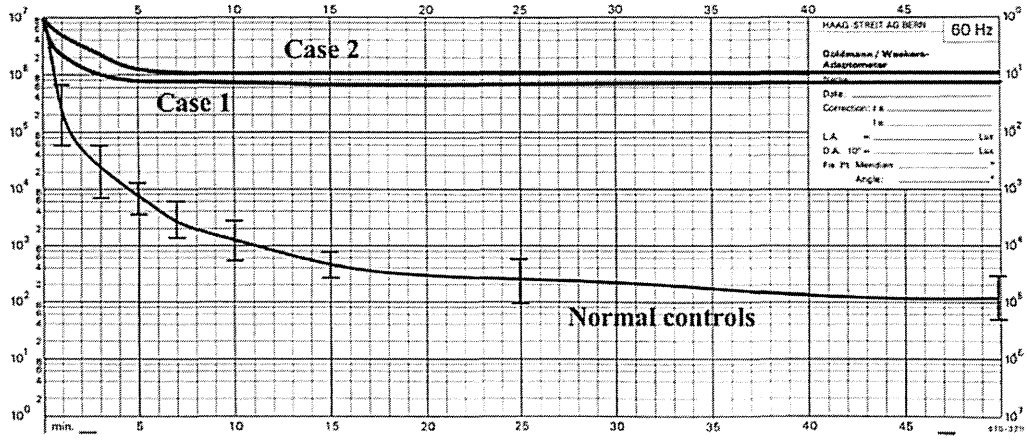


Figure 6. Results of Goldmann-Weekers dark adaptometry. Lower line indicates averaged value and its standard deviation resulted from normal controls.

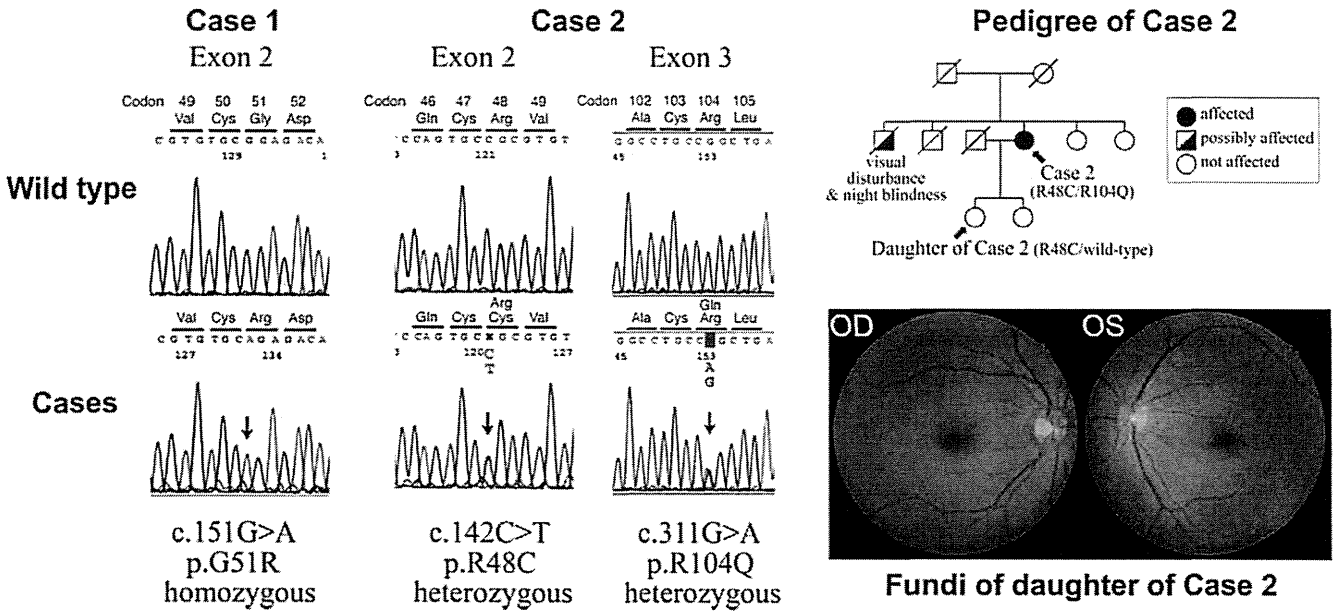


Figure 7. Results of DNA sequencing of the *NR2E3* gene in cases 1 and 2 (left), the pedigree of case 2, and fundus photographs of the daughter of case 2 (right). Mutation analysis identified a novel homozygous missense mutation of p.G51R, which resides in the DNA-binding domain (DBD) in NR2E3 protein in case 1. In case 2, heterozygous missense mutation of p.R48C and p.R104Q were identified, and the former is a novel mutation. A daughter of case 2 revealed heterozygous missense mutation of p.R48C and normal fundus appearance. In mutation screening of the *NR2E3* gene, all coding exons including exon/intron boundaries were amplified using polymerase-chain reaction (PCR) with primer pairs followed by sequencing. The primers and protocols used for PCR, and the procedures of PCR amplification and purification were the same as reported previously.[5]

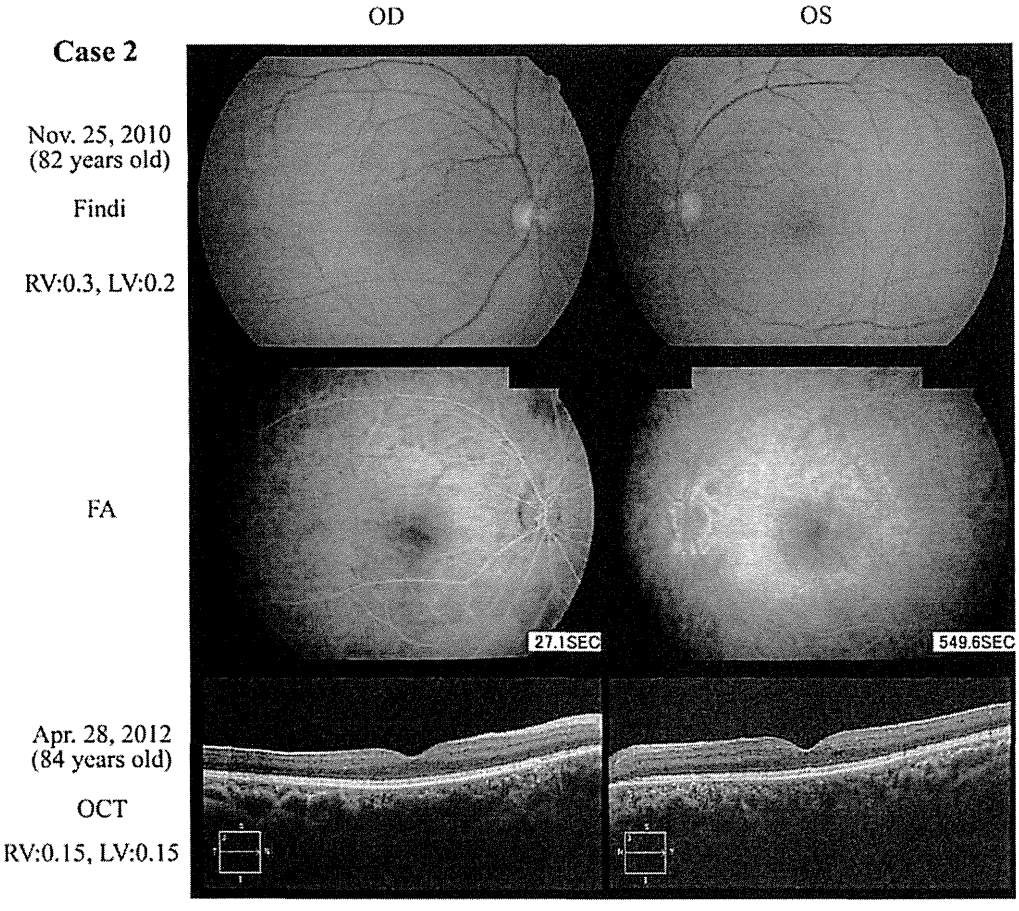


**Figure 8.** Results of fundus photography, fundus angiography (FA), and optical coherence tomography (OCT) in case 2. These photographs were taken after cataract surgery. The retinal degeneration was relatively mild with no pigmentation in both eyes. This Figure was made with modification of Figure 7 in Sato et al[3] with permission.

# *Improvement of visual acuity after transcorneal electrical stimulation in case of Best vitelliform macular dystrophy*

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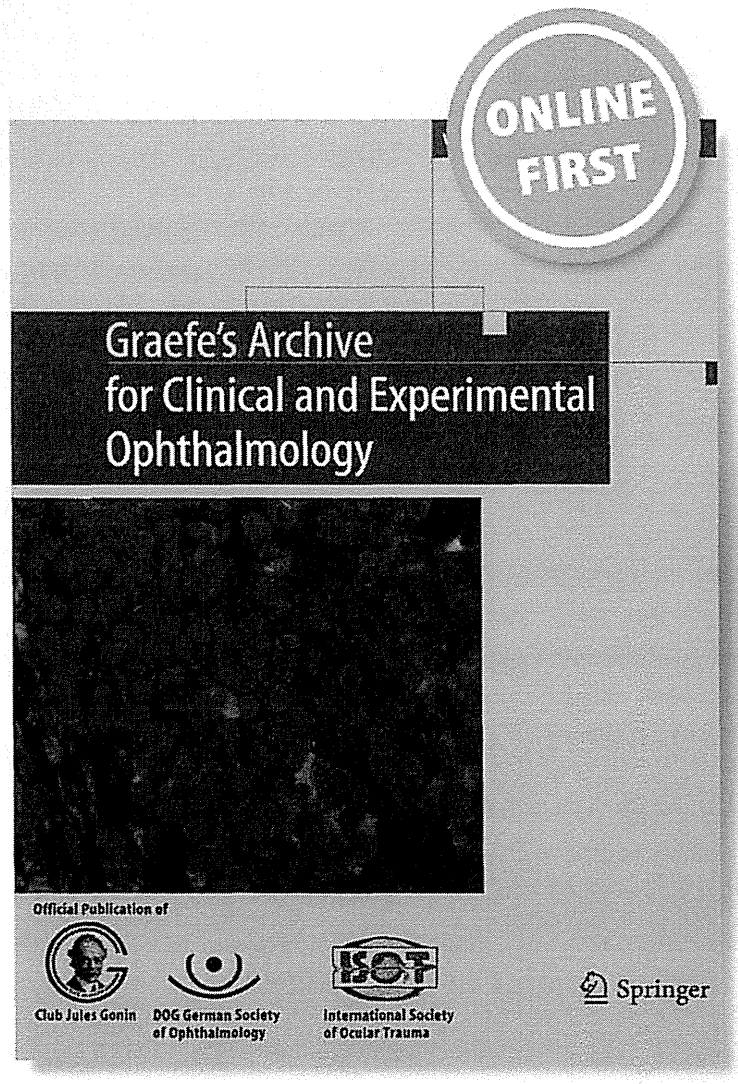


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CASE REPORT

# Improvement of visual acuity after transcorneal electrical stimulation in case of Best vitelliform macular dystrophy

**Naoki Ozeki · Kei Shinoda · Hisao Ohde · Susumu Ishida · Kazuo Tsubota**

Received: 13 September 2012 / Revised: 23 March 2013 / Accepted: 2 April 2013


**Abstract**
*Purpose* To report an improvement of the visual acuity after transcorneal electrical stimulation (TES) in a case of Best vitelliform macular dystrophy (BVMD).
*Patient and methods* A 26-year-old woman diagnosed with BVMD presented with reduced vision. Her best corrected visual acuity (BCVA) was reduced to 20/200 in the right eye, and TES was performed once a month for two sessions. The current of the biphasic pulses (anodic first; duration, 10 msec; frequency, 20 Hz) was delivered using a DTL-electrode, and the duration of the TES was 30 min.
*Results* The BCVA in the right eye slowly improved after the TES, and 6 months later the BCVA was 20/25. The results of Humphrey visual field tests (VF) and multifocal ERGs (mfERGs) were only slightly changed. Two years later, the BCVA decreased, and it was improved again after another session of TES with the same parameters of the electrical pulses.
*Conclusion* The improvement of the visual acuity in our case of BVMD indicates that TES should be tried in other cases of retinal dystrophy. Further clinical and laboratory studies on TES are needed.

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**Keywords** Phosphenes · Transcorneal electrical stimulation · Best vitelliform macular dystrophy

## Introduction

Electrical stimulation of the retina can be done with a contact lens electrode with the inactive electrode placed on the skin around the eye. Passing electrical currents between the two electrodes can evoke electrical phosphenes, and this method of stimulating the retina is called transcorneal electrical retinal stimulation (TES) [1, 2].

An improvement of the visual acuity, visual field (VF), and/or electrophysiological functions after TES has been reported in eyes with optic nerve diseases, retinal artery occlusion (RAO), and retinitis pigmentosa (RP) [3–5]. The recent published results of a large case series study showed a recovery of vision after optic nerve lesions by transorbital alternating current stimulation [6].

Best vitelliform macular dystrophy (BVMD) is characterized by an atrophy of the retinal pigment epithelium (RPE) which then affects the photoreceptors and leads to an impairment of central visual function. We present a case of BVMD whose visual acuity improved after TES.

## Subjects and methods

### Transcorneal electrical stimulation (TES) of retina

The cornea was anesthetized with 0.4 % oxybuprocaine hydrochloride and covered with 3 % hyaluronic acid and 4 % chondroitin sulfate (Viscoat, Alcon Japan, Tokyo, Japan), and a Dawson-Trick-Litzkow (DTL) electrode was placed on the cornea. A skin electrode was placed on the wrist. The electrical current pulses were delivered by a

stimulator (BPG-1,BAK Electronics, Inc., Mount Airy, MD, USA) through a stimulus isolation unit (BSI-2,BAK Electronics, Inc., Mount Airy, MD, USA). The current of the biphasic pulses (anodic first; duration, 10 msec; frequency, 20 Hz) was increased in steps to determine the threshold current necessary to elicit a phosphene. Then the current was increased until a phosphene was elicited that was perceived over the entire VF. This current was selected for the TES, and it was delivered continuously for 30 min for each TES session.

## Case report

A 26-year-old woman with BMVD presented with decreased vision OD (Fig. 1). She was diagnosed with BVMD when she was 10-years-old. From the patient's report, her vision was stable, but it was reduced for 1 week when she was 26 years old. She was examined at a private eye clinic, and her best-corrected visual acuity (BCVA) was 20/40 OD and 20/20 OS. The patient was then referred to the Keio University Hospital.

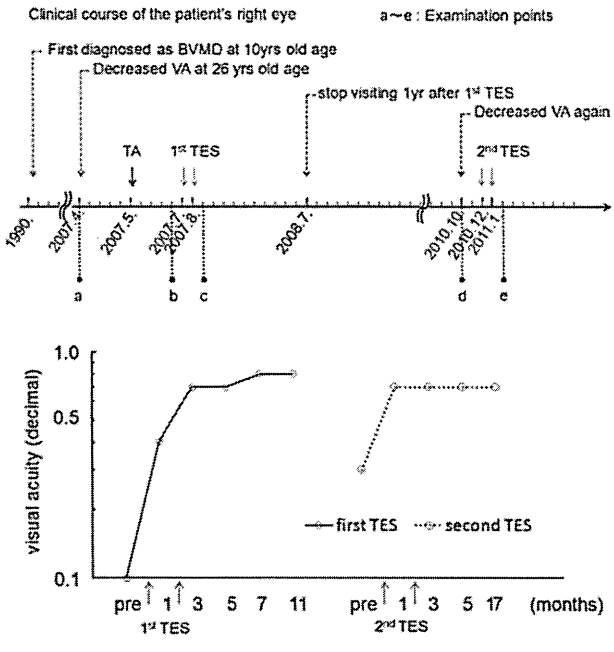


**Fig. 1** Results from the right eye of a patient with Best vitelliform macular dystrophy. **a** Clinical course of the BCVA in the right eye of our patient. Scale shows 1 month intervals unless otherwise indicated. BVMD: Best vitelliform macular dystrophy, TES: transcorneal electrical stimulation, TA: Subtennon injection of triamcinolone acetonide, VA: visual acuity. Point "*a*" is time when the data shown in Fig. 2a, b, c, d, and i were obtained. Point "*b*" is time when the data shown in Fig. 2e was obtained. Point "*c*" is time when the data shown in Fig. 2f and j were obtained. Point "*d*" is time when the data shown in Fig. 2g and k were obtained. Point "*e*" is time when the data shown in Fig. 2h and l were obtained. **b** Effect of transcorneal electrical stimulation (TES) on the best-corrected visual acuity (BCVA) in a patient with Best vitelliform macular dystrophy. After the first and second TES, the BCVA improved and was stable for several months. The *arrow* indicates the point when the TES was performed

Our examination showed that her BCVA was 20/40 OD and 20/20 OS. Ophthalmoscopic examination showed a 1.5-disc-diameter, yellowish macular lesion in both eyes (Fig. 2). Optical coherence tomography (OCT) showed an irregularity of the RPE, and a serous retinal detachment (SRD) in the macula of both eyes (Fig. 2). Her BCVA at this time was reduced to 20/200 OD. Perimetry showed a loss of sensitivity in the central 10° of the VF (Fig. 2). The amplitudes of the mfERGs were reduced and the peak latencies were delayed in the central areas corresponding to the decrease in sensitivity of the VFs (Fig. 2).

A sub-Tennon injection of triamcinolone acetonide failed to improve the BCVA and the SRD. Her visual acuity was measured with a Snellen chart at 5 m by an orthoptist who was masked to the diagnosis and any treatments. The patient had central fixation and her BCVA was measured 1 and 4 weeks after the sub-Tenon injection. The BCVA remained stable at 20/200, and no fundus change was observed.

Two months later, TES (250 μA, 170 μA) was performed twice with an interval of 1 month on the right eye. The procedures used conformed to the tenets of the Declaration of Helsinki, and an informed consent was obtained from the patient after an explanation of the procedures to be used. This study was approved by the Institutional Review Board of Keio University Hospital.

The patient had transient superficial keratitis immediately after each TES session, and otherwise there were no obvious changes by slit-lamp examinations and ophthalmoscopy. OCT showed no changes in the macular region (Fig. 2), but the patient reported an improvement of vision 1 month after the second session. The BCVA had improved to 20/30, and 6 months later, the BCVA in the right eye had improved to 20/25 (Fig. 1). The VFs and mfERGs showed only slight improvements (Fig. 2).

She returned to the Keio University Hospital 2 years later when her BCVA had decreased to 20/70 OD. The macular findings by ophthalmoscopy and OCT showed no changes (Fig. 2). TES was performed again; two sessions at 160 μA with a monthly interval on the right eye. One month later, the BCVA in the right eye improved to 20/30 (Fig. 1). The OCT image (Fig. 2), VFs, and mfERG responses (Fig. 2) were only slight changed. Quantitative analysis on the mfERG parameters failed to show significant changes. At the last examination at 17 months after the second TES session, her BCVA was 20/30. The fundus appearance was stable for more than 5 years in both eyes.

## Discussion

Our results showed that TES in a patient with BMVD improved the BCVA significantly for 2 months. Although

**Fig. 2** Fundus appearance and morphological and functional evaluation of the right macula of a patient with Best vitelliform macular dystrophy (BVMD). **a** and **b** Fundus photograph of the right (**a**) and left (**b**) eyes taken when the BCVA was 20/200 at the initial examination at point '*a*' in Fig. 1. **c** and **d** Horizontal crossed sections of time domain optical coherence tomographic (TD-OCT) images of the right (**c**) and left (**d**) eyes obtained on the same day as **a** and **b** at point '*a*' in Fig. 1. **e** Horizontally cross section TD-OCT image of the right eye before the first TES at point '*b*' in Fig. 1. **f** Horizontally cross sectional TD-OCT image of the right eye after the first TES at point '*c*' in Fig. 1. **g** Horizontally cross sectional Fourier domain (FD) OCT image of the right eye before the second TES at point '*d*' in Fig. 1. **h** Horizontally cross section FD-OCT image of the right eye after the second TES at point '*e*' in Fig. 1. After the first and second TES, no significant changes were observed in the OCT images. **i** Humphrey visual field of the right eye before the first TES at point '*b*' in Fig. 1. **j** Humphrey visual field of the right eye 2 months after the first TES at point '*c*' in Fig. 1. **k** Humphrey visual field of the right eye before the second TES at point '*d*' in Fig. 1. **l** Humphrey visual field of the right eye 2 months after the second TES at point '*e*' in Fig. 1

the BCVA was reduced 3 years after the TES, the vision improved again after another TES treatment (Fig. 1). These findings strongly suggest a causal relationship between the treatment and the visual improvement.

It has been reported that the mRNA and protein levels of IGF-1 [7], BDNF, CNTF, and Bcl-2 [8] were time-dependently up-regulated and Bax was down-regulated in the retina of Sprague–Dawley rats after TES. The levels of the mRNA and protein of IGF-1 and neurotrophins in these retinas gradually increased beginning several hours after the TES and reached a peak at around day 7. The levels were still significantly elevated at day 10 after TES [7]. This may explain why only two TES treatments were effective in improving vision for more than 12 months in our case.

Although the duration of the up-regulation of the ICG-1 system by a single TES session is limited, it might have had neuroregenerative effects as well, considering that VA remained improved for more than 12 months. Functional improvements for 3 months of the patient who was already at stationary stage of ION [3], RAO [4], and optic nerve lesions of various origins [6] support this hypothesis.

The optimal parameters of the pulse duration, current intensity, stimulation frequency, stimulation duration, waveform, and repetition times, were not determined. Morimoto et al. [9] reported that the optimal neuroprotective parameters were pulse duration of 1 to 2 ms/phase, current intensity of 100 to 200 μA, and stimulation frequency of 1, 5, and 20 Hz in rats. Inomata et al. reported that in monkeys the strength of the signals increased with longer stimulus durations, and the maximum signals were obtained when the stimulation frequency was between 15 and 20 Hz [10]. In healthy humans, Fujikado et al. studied the amplitude of pupillary reflex (PR) following TES, and reported that biphasic pulse trains (≥10 pulses) with a duration of 0.5 to 1.0 ms and a frequency of 20 to 50 Hz were effective [11]. We used these data to select the stimulation parameters for our patient.

Many investigators use a contact electrode for the stimulation electrode, whereas Fedorov et al. [6] used a skin electrode that was placed on the upper eyelid of patients with optic nerve lesions. This avoided corneal damage and can be considered for retinal diseases as well.

The mechanism for the improvement of the BCVA after TES in our case was not determined. However, we suggest that the reason why the OCT, perimetry, and mfERG findings did not have significant changes over time is because there may have been microstructural changes of the photoreceptors which were too minute to be detected by our OCT. The area of the functional improvement was limited and thus changes in the VFs and mfERGs could not detect it. More precise evaluations with electrophysiological or morphological techniques should help to determine the effect of TES. Further clinical and basic studies on TES are needed to establish TES as an accepted therapeutic modality for retinal dystrophy.

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# Acute Visual Field Defect following Vitrectomy Determined to Originate from Optic Nerve by Electrophysiological Tests

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**Key Words**
Ischemic optic neuropathy · Proliferative diabetic retinopathy · Multifocal electroretinogram · Multifocal visual evoked potentials · Photopic negative response

**Abstract**
***Purpose:*** To present our findings on the cause of an acute visual field defect (VFD) that developed in a patient on the day after vitrectomy for proliferative diabetic retinopathy. ***Case:*** A 50-year-old man complained of a blind area in the superior visual field that developed one day after vitrectomy. The patient had undergone uncomplicated vitrectomy for a long-duration vitreous hemorrhage associated with proliferative diabetic retinopathy. Residual vitreous hemorrhage hampered a clear view of the fundus. Goldmann perimetry showed a horizontal VFD in the superior field. The area corresponding to the VFD was examined by multifocal electroretinograms (mfERGs) and multifocal visual evoked potentials (mfVEPs). The amplitudes of the mfVEPs were reduced with prolonged implicit times especially when the superior hemifield was stimulated, while the amplitudes and implicit times were within the normal range when other parts of the visual field were stimulated. In addition, the full-field photopic ERGs and photopic negative responses were attenuated in the right eye. These findings suggested that the VFD did not originate from alterations in the retinal inner and middle layer but in the ganglion cells. The visual acuity improved to 1.2 but his optic disc became pale and the VFD remained unchanged more than 12 years after the surgery.

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***Conclusion:*** We suggest that vitrectomy can cause ischemic optic neuropathy by interfering with the circulation associated with diabetes mellitus. Evaluations by mfERGs, mfVEPs, and full-field photopic ERGs were helpful in making the diagnosis.

## Introduction

The visual function after vitrectomy depends on many factors, e.g., the underlying vitreoretinal disease, surgical procedures, and complications in either the anterior or posterior segments of the eye. Visual field defects (VFDs) are known to be a postsurgical complication, and they can be caused by retinochoroidal circulatory disturbances [1, 2], nerve fiber damage due to excessive exposure to dry air [3–5], optic nerve damage due to retrobulbar anesthesia [6–9], phototoxicity [10], and elevation of the intraocular pressure [11]. We report our findings in a patient who developed a severe VFD on the day following an uncomplicated vitrectomy for a vitreous hemorrhage associated with proliferative diabetic retinopathy [12]. A tentative diagnosis of ischemic optic neuropathy (ION) was made from the acute onset, superior hemianopsia, and the results of electrophysiological tests. We re-examined the patient after 10 years, and the VFD and the electrophysiological results remained unchanged. We conclude that our original diagnosis was correct, and also that the electrophysiological findings were critical in determining the pathological site of the VFD.

## Case Report

A 50-year-old man underwent uncomplicated vitrectomy on September 22, 1998 for a vitreous hemorrhage of 2 months duration which was associated with proliferative diabetic retinopathy. His preoperative best-corrected visual acuity (BCVA) was hand movements in the right eye and 1.2 in the left eye. He underwent conventional pars plana vitrectomy, and no complications were encountered during the surgical procedures. His blood pressure increased to 176/107 mm Hg just before the surgery, but it decreased and became stable between 116–140/70–90 mm Hg intra- and postoperatively.

The patient complained of a blind area in the superior visual field of the right eye on the day after the vitrectomy (fig. 1a). His decimal BCVA was 0.02 in the right eye. The residual vitreous hemorrhage hampered a clear view of the fundus. On the second day after surgery, flash visual evoked potentials (VEPs) and full-field single-flash electroretinograms (ERGs) were recorded simultaneously [13]. The recording electrodes for the ERGs were attached to the surface of the lower eyelids to avoid using a contact lens electrode.

The implicit times of the flash VEPs were slightly delayed in both eyes and no difference was found between the eyes (fig. 2). The amplitudes of the a- and b-waves of the full-field ERGs were normal but the oscillatory potentials were slightly reduced in both eyes. However, no differences were found between the eyes (fig. 2). At that time, we did not evaluate the photopic negative response (PhNR) because its origin had not fully been determined.

Blood tests showed no abnormalities in the erythrocyte sedimentation rate, blood coagulation factor, C-reactive protein, and complete blood count. The results for antinuclear antibody were negative. ION was suspected because of the acute onset, horizontal hemianopsia, normal full-field ERGs, and diabetes.

Oral carbazochrome and kallidinogenase were started. The fundus became more visible one week after the surgery, and the BCVA improved to 0.7. Ophthalmoscopy showed localized edema adjacent

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to the optic disc (fig. 3a). The arm-to-retina time of the fluorescein angiography (fig. 3b) was delayed, and an island-like hypofluorescence surrounded by a hyperfluorescent region was present inferior to the optic disc.

Multifocal ERGs (mfERGs) and multifocal VEPs (mfVEPs) were recorded approximately two weeks after the surgery according to the ISCEV standard [14, 15]. The amplitudes of the mfERGs (fig. 4a) were within the normal range over the central retinal area, while the amplitudes of the mfVEPs (fig. 4b) were reduced and the implicit times prolonged especially those elicited by stimulating the superior hemifield of the right eye. These findings suggested that the VFD did not originate in the retinal inner and middle layer but was of ganglion cell origin.

The visual acuity improved to 1.2 in one month and has been stable for 12 years in the right eye, but the optic disc gradually became paler especially in the inferior region (fig. 3c). Fluorescein angiography (fig. 3d) showed a delayed arm-to-retina time and a semicircular hypofluorescent region inferior to the optic disc. The VFD remained unchanged for more than 12 years after the surgery (fig. 1b). Optical coherence tomography (Spectralis OCT, Heidelberg Engineering, Germany) performed 12 years after the vitrectomy demonstrated a selective atrophy of the nerve fiber layer inferior to the optic disc in the right eye (fig. 3e).

A re-examination of the PhNR of the photopic ERGs recorded at the initial examination showed that it was selectively reduced in the right eye (table 1 and table 2). These findings strongly supported our initial diagnosis of ION.

## Discussion

Several mechanisms have been reported to explain the VFDs after vitreous surgery: phototoxicity due to the bright light from the operating microscope or endoillumination [16, 17], intra- or postoperative fluctuations of the intraocular pressure and/or systemic blood pressure [18–20], mechanical stress on the optic nerve during the creation of a posterior vitreous detachment [21], chemicophysical stress on the retina by dry air during fluid-air exchange [3], retinal damage due to panretinal photocoagulation [22], optic nerve damage due to retrobulbar anesthesia [6–9], and damage to the optic nerve because of the compromised circulation associated with diabetes mellitus [23–25].

Our case was initially diagnosed with ION because of the acute superior horizontal VFD. The attenuated mfVEPs corresponding to the VFD and normal mfERGs suggested that the pathological site was not in the outer and middle layers of the retina but the ganglion cells and/or optic nerve. This supported our initial diagnosis.

Little information is available of cases that developed ION after vitrectomy [18, 26]. Pendergast et al. [18] reported on a 73-year-old woman with coronary artery disease who developed ION 4 months after vitrectomy. Taban et al. [26] reported on two cases, a 65-year-old woman with hypertension and diabetes mellitus who developed ION at 3.5 weeks after vitrectomy, and a 94-year-old man with hypertension whose visual acuity was found to be reduced on postoperative day 34. Both were diagnosed with ION but the etiology of the ION was not determined. Taban et al. [26] also found 190 cases that developed a VFD after vitrectomy, and approximately 20% of these had evidence of optic nerve damage, relative afferent pupillary defect, or optic nerve pallor. They stated that in spite of the fact that the etiology of the VFD remains undetermined, VFD as a complication of vitreous surgery is relatively common. We suggest that circulatory disturbances associated with diabetes mellitus might have played some part in our case.

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No obvious difference was found between the mfERGs from the superior and inferior retina which also supports our suggestion that the VFD did not originate in the inner and middle retinal layer but was of ganglion cell and/or optic nerve origin. Furthermore, the selectively reduced PhNR in the right eye strongly supported this idea, although we did not use this test in 1998. The PhNR has been reported to be a sensitive test to determine functional alterations of ganglion cells, and its clinical application has been extended [27–29]. Our case highlights the importance of the PhNR in differentiating ganglion cell damage in patients with VFD after surgery. It is, however, difficult to determine whether the ganglion cells or optic nerve was the exact origin in the present case. We believe that it is more likely that the ganglion cell damage was related to ION.

In summary, electrophysiological evaluations were helpful in making a diagnosis in our case. The mfERGs, mfVEPs, and PhNR were useful in determining the pathological site of the VFD that occurred after vitrectomy.

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## Disclosure Statement

No author has a financial or proprietary interest in any material or method mentioned.

Table 1. Amplitude and implicit time of the P-100 in flash VEPs

| Stimulus intensity | Right | | Left | | R-L | |
|---|---|---|---|---|---|---|
| | Amp., μV | Imp.T., ms | Amp., μV | Imp.T., ms | Amp., μV | Imp.T., ms |
| 0.3 J | | | | | | |
| ND-3 | 3.4 | 130 | 2.4 | 138 | 1.00 | –8.00 |
| ND-2 | 5.4 | 117.5 | 5 | 130 | 0.40 | –12.50 |
| ND-1 | 6.3 | 127.5 | 11.6 | 130 | –5.30 | –2.50 |
| ND-0 | 9.9 | 126.3 | 8.9 | 125 | 1.00 | 1.30 |
| 2.0 J | 4.9 | 105 | 5.6 | 105 | –0.70 | 0.00 |

Amp. = Amplitude; Imp.T. = implicit time.

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Table 2. Amplitude and implicit time of the a- and b-waves and PhNR in each eye

| Stimulus intensity | Right | | | | | Left | | | | | R-L | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Amp., μV | | | Imp.T., ms | | Amp., μV | | | Imp.T., ms | | Amp., μV | | | Imp.T., ms | |
| | a-wave | b-wave | PhNR | a-wave | b-wave | a-wave | b-wave | PhNR | a-wave | b-wave | a-wave | b-wave | PhNR | a-wave | b-wave |
| 0.3 J | | | | | | | | | | | | | | | |
| ND-3 | 0 | 2.24 | 2.24 | 45 | 82.5 | 0.56 | 2.24 | 1.4 | 60 | 80 | −0.56 | 0.00 | 0.84 | −15.00 | 2.50 |
| ND-2 | 0.56 | 0.84 | 2.8 | 80 | 90 | 1.68 | 3.36 | 1.96 | 47.5 | 90 | −1.12 | −2.52 | 0.84 | 32.50 | 0.00 |
| ND-1 | 0 | 5.32 | 7.84 | 40 | 65 | 0.84 | 5.32 | 10.08 | 42.5 | 65 | −0.84 | 0.00 | −2.24 | −2.50 | 0.00 |
| ND-0 | 1 | 8.4 | 8.4 | 42.5 | 70 | 6.16 | 17.08 | 13.16 | 45 | 70 | −5.16 | −8.68 | −4.76 | −2.50 | 0.00 |
| 2.0 J | 6.16 | 14.56 | 12.04 | 37.5 | 60 | 7.28 | 17.08 | 23.8 | 45 | 67.5 | −1.12 | −2.52 | −11.76 | −7.50 | −7.50 |

Amp. = Amplitude; Imp.T. = implicit time.

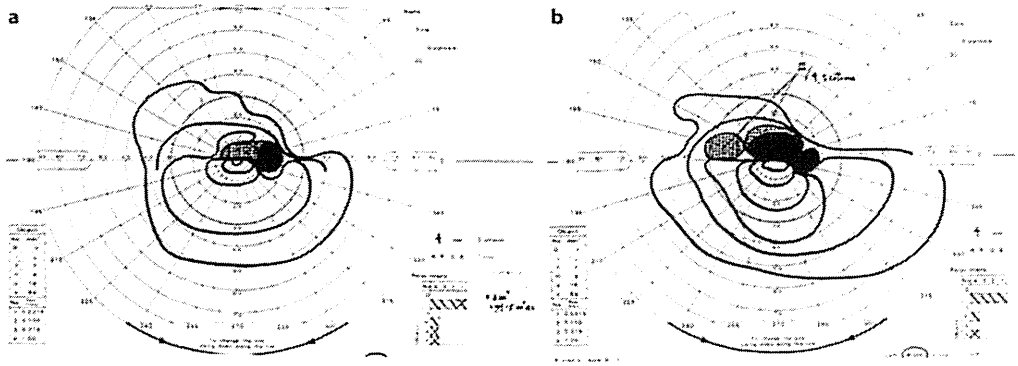


Fig. 1. Goldmann perimetry performed on the day after vitrectomy and again more than one year after surgery. The V-4 isoptor is constricted in the superior and temporal-superior visual field, and the internal isoptor shows a superior hemianopsia on the following day (**a**). The superior hemianopsia remained unchanged (**b**).

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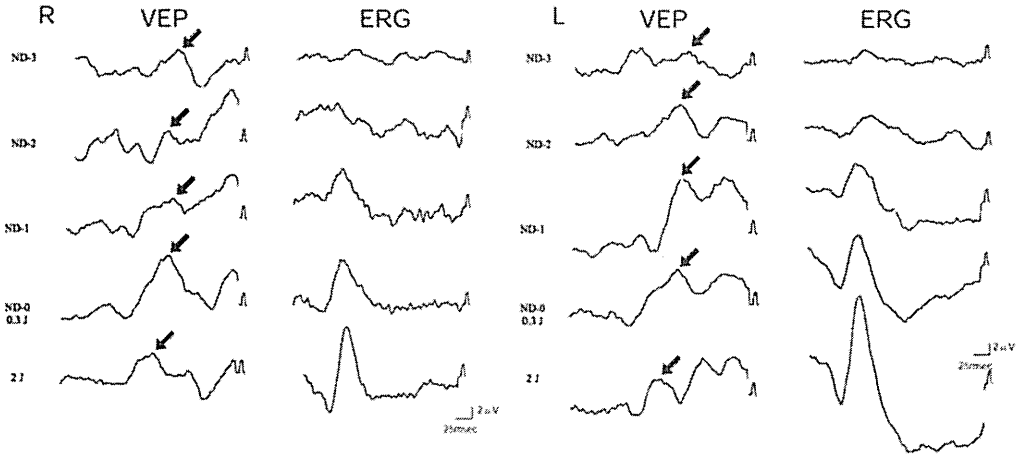


Fig. 2. Simultaneously recorded flash VEPs and full-field single-flash ERGs. No significant differences were found between the two eyes in the amplitude and implicit times of N-70 and P-100 in the flash VEPs. The photopic ERGs recorded with skin electrodes showed no obvious differences between the eyes in the amplitude and implicit times of the a- and b-waves, but the amplitudes of the PhNR were reduced in the right eye. The arrow points to P-100. The values of the amplitudes and implicit times are shown in table 1 and 2.

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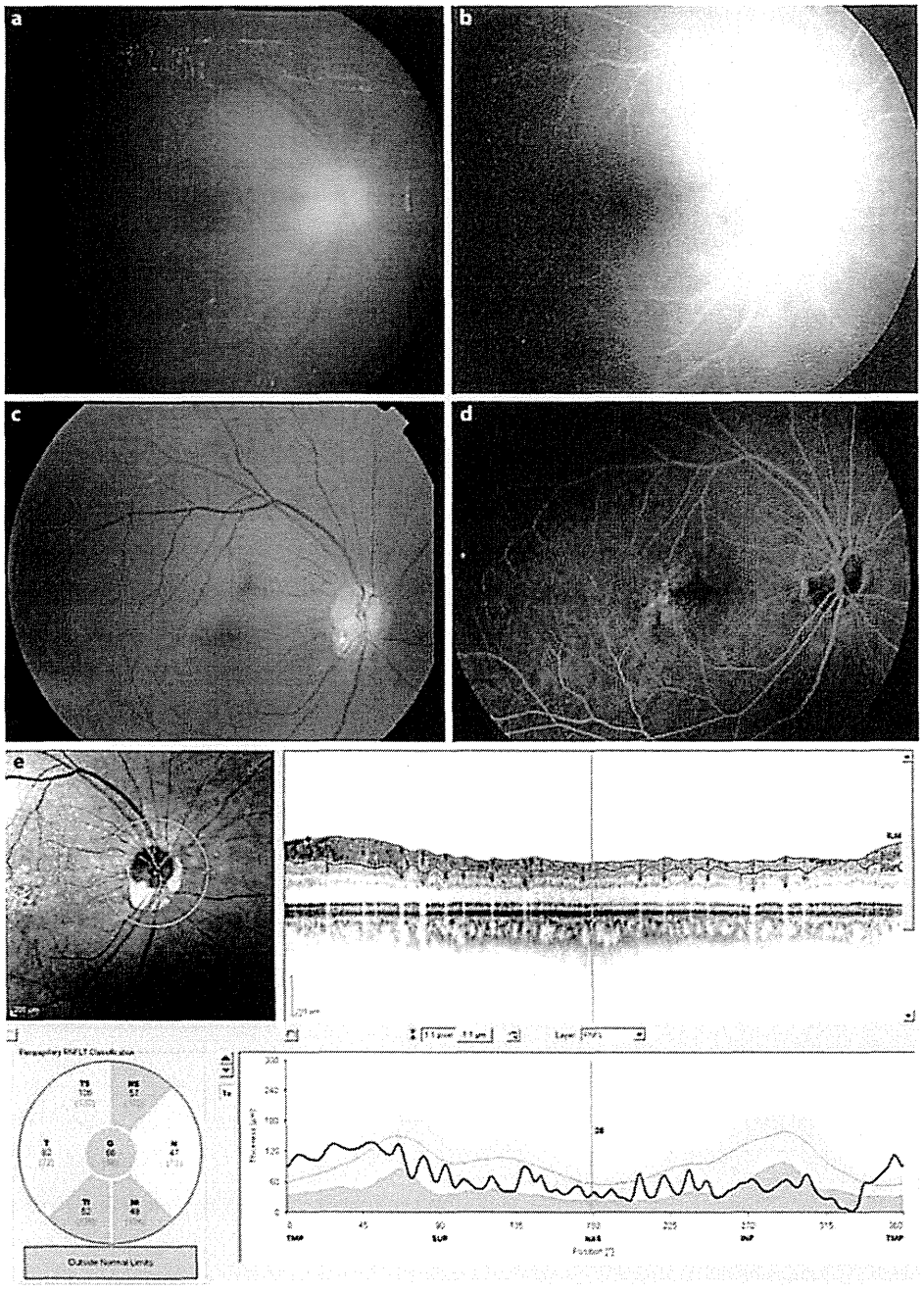


Fig. 3. Fundus photograph, fluorescein angiogram, and optical coherence tomographic images. **a** Fundus photograph taken one week after surgery showed localized edema adjacent to the optic disc. **b** Fluorescein angiogram obtained on the same day as that in **a** shows a delayed arm-to-retina time and island-like hypofluorescence surrounded by a hyperfluorescent region inferior to the optic disc. **c** Fundus photograph taken 10 years after surgery shows a pale optic disc especially in the inferior region. Visual acuity was 1.2. **d** Fluorescein angiogram obtained on the same day as **c** shows a delay in the arm-to-retina time and semicircular hypofluorescent region inferior to the optic disc. **e** The optic nerve fiber layer thickness analysis using optical coherence tomography (Spectralis OCT, Heidelberg Engineering, Germany) performed 12 years after vitrectomy showing selective atrophy of inferior nerve fiber layer around optic disc in the right eye.

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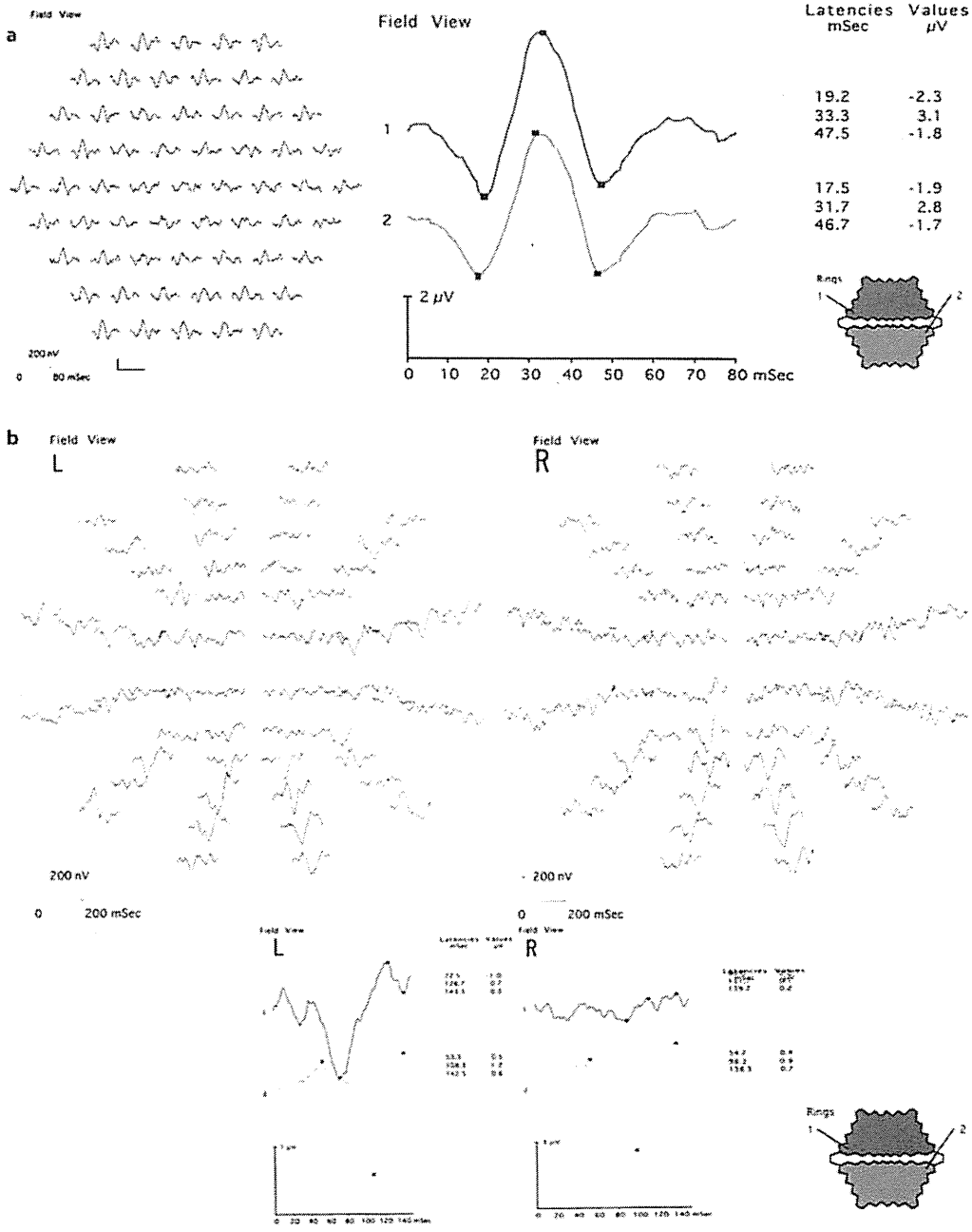


Fig. 4. Multifocal ERGs and VEPs recorded one week after the vitreous surgery. **a** The amplitudes and the implicit times of the mfERGs from the right eye are within the normal range. **b** The mfVEP showed amplitude reduction and delayed implicit time especially from superior hemifield in the right eye.

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