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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. 1**a). 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. 3**a). 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. 4**a) 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.

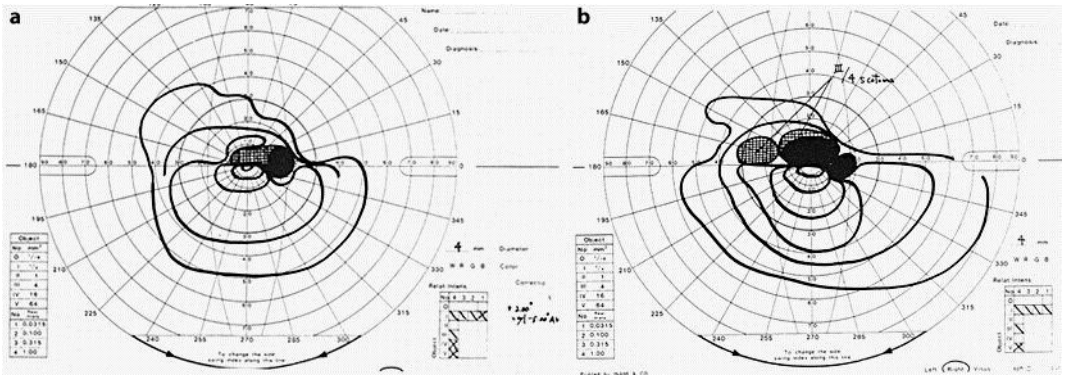

Fig. 1. Goldmann perimetry performed on the day after vitrectomy and again more than one year after surgery. The V-4 isoptor is constricted in the superior and temporal-superior visual field, and the internal isoptor shows a superior hemianopsia on the following day (**a**). The superior hemianopsia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

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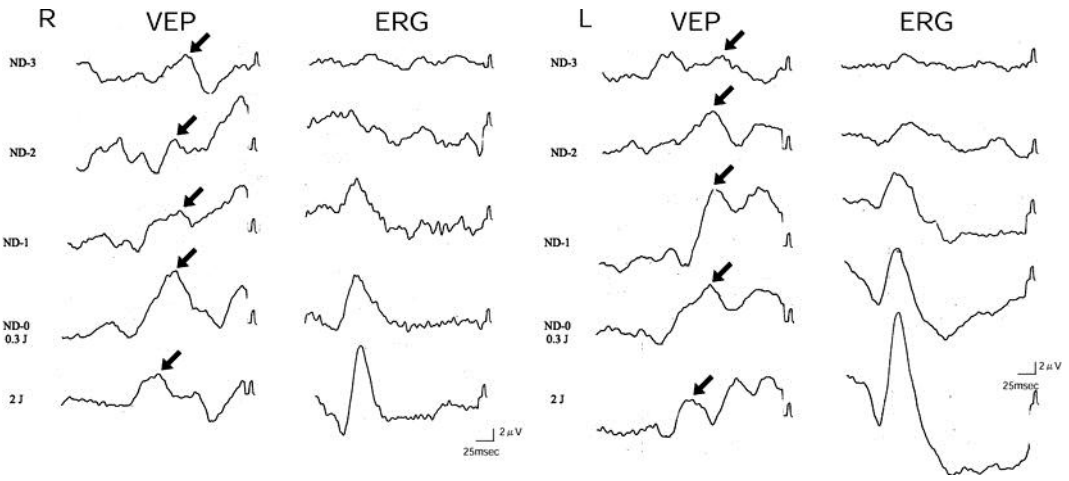

Fig. 2. Simultaneously recorded flash VEPs and full-field single-flash ERGs. No significant differences were found between the two eyes in the amplitude and implicit times of N-70 and P-100 in the flash VEPs. The photopic ERGs recorded with skin electrodes showed no obvious differences between the eyes 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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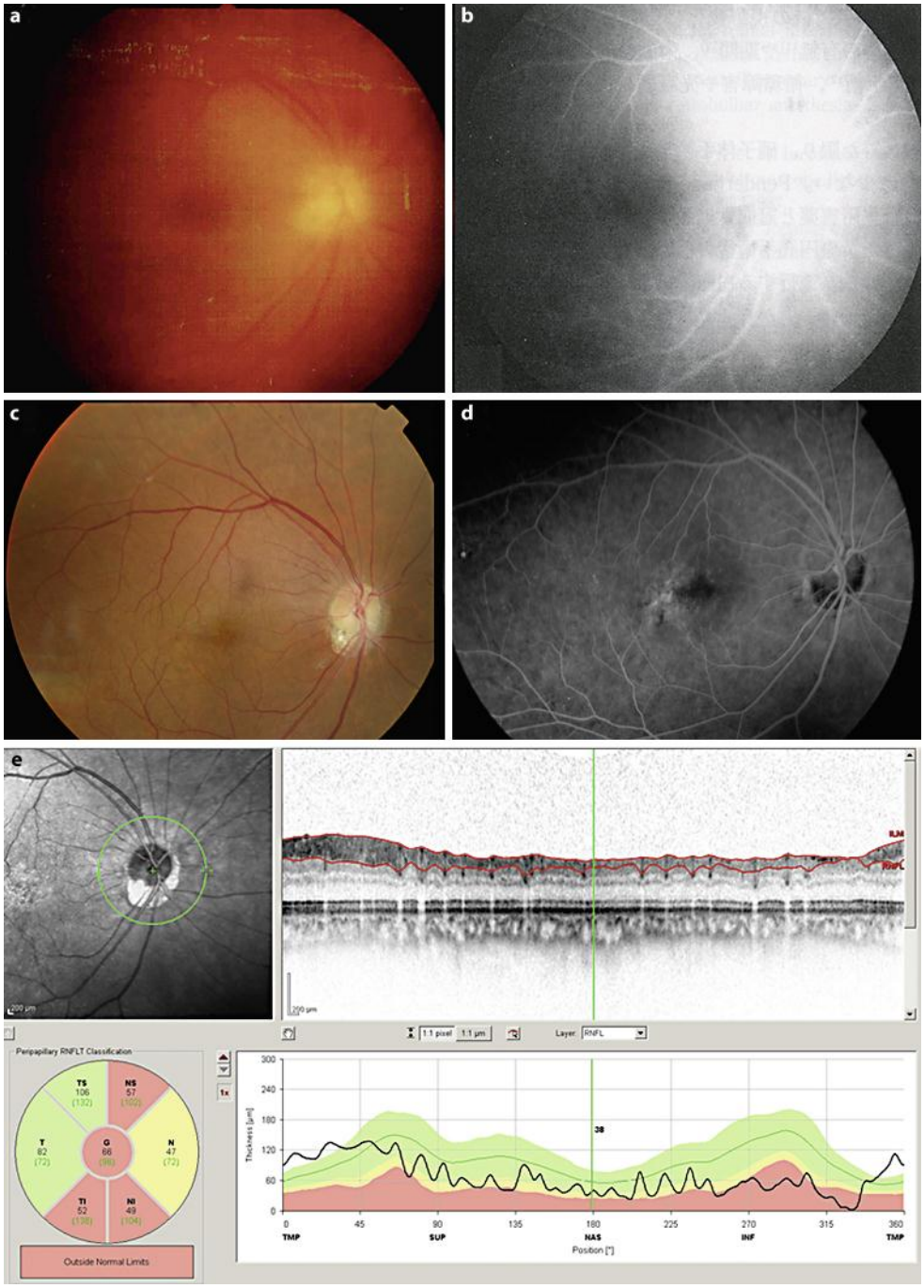

Fig. 3. Fundus photograph, fluorescein angiogram, and optical coherence tomographic images. **a** Fundus photograph taken one week after surgery showed localized edema adjacent to the optic disc. **b** Fluorescein angiogram obtained on the same day as that in **a** shows a delayed arm-to-retina time 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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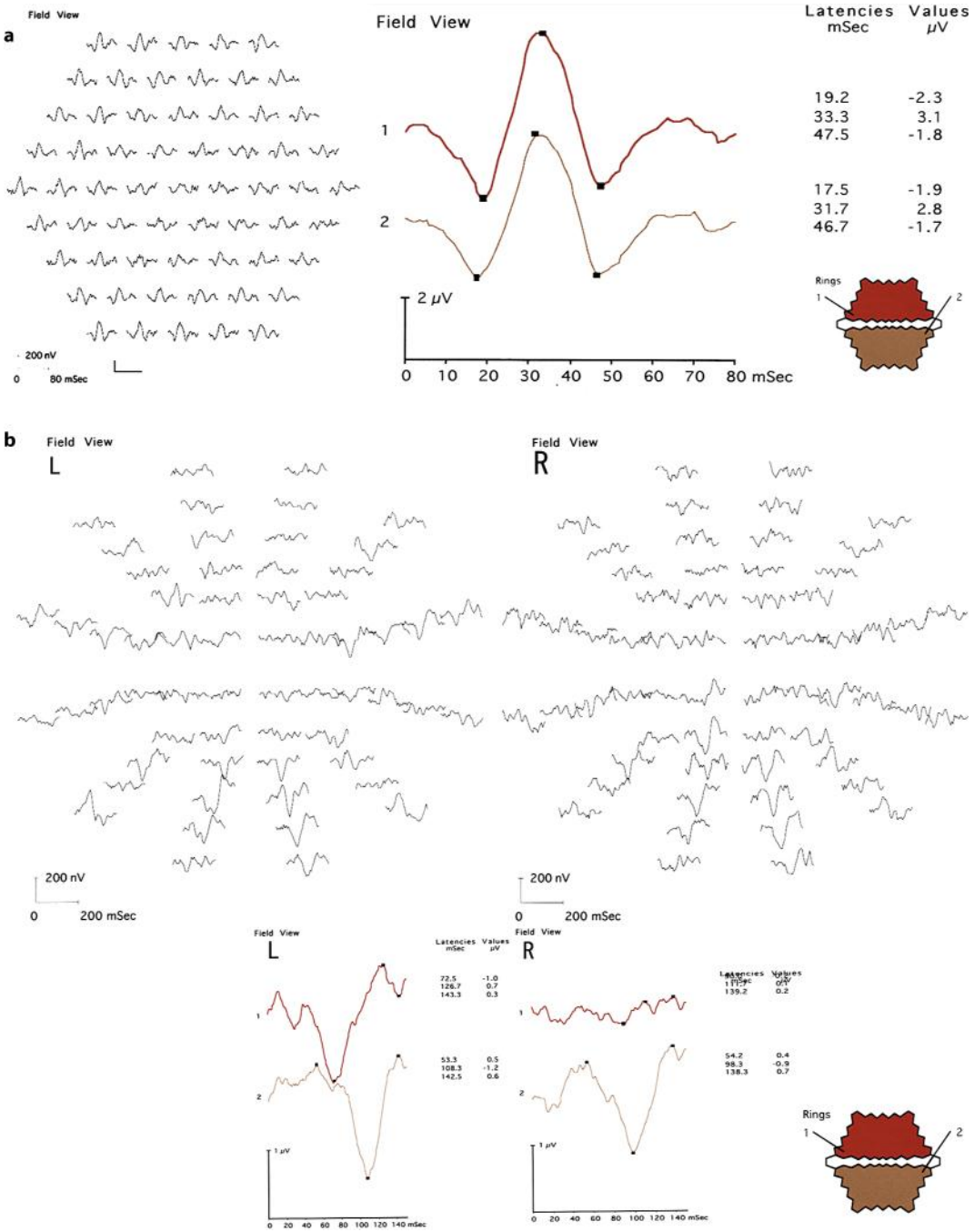

Fig. 4. Multifocal ERGs and VEPs recorded one week after the vitreous surgery. **a** The amplitudes and the implicit times of the mfERGs from the right eye are within the normal range. **b** The mfVEP showed amplitude reduction and delayed implicit time especially from superior hemifield in the right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

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