Documenta Ophthalmologica (2005) 111: 149–157
DOI 10.1007/s10633-005-5362-4

Recording of both VEP and multifocal ERG for evaluation of unexplained visual loss

Electrophysiology in unexplained visual loss

Agnes B. Renner,[1] Ulrich Kellner,[1,2,3] Hilmar Tillack,[1] Hannelore Kraus[1] & Michael H. Foerster[1]
[1]*Augenklinik, Charité – Universitätsmedizin Berlin, Berlin, Germany;* [2]*AugenZentrum Siegburg, Siegburg, Germany;* [3]*RetinaScience, Bonn, Germany*

Accepted 21 November 2005

Key words: electrophysiology, electroretinogram, multifocal ERG, VEP, visual loss

Abstract

The purpose of this retrospective study was to determine the relevance of both visual-evoked potentials (VEP) and multifocal electroretinography (mfERG) to evaluate unexplained visual loss. Seventy-two consecutive patients (1996–2002) with visual disturbances of unknown origin underwent both VEP and mfERG (ISCEV standard). The mean age was 42.4 years (11.8–74.5) and median visual acuity 0.5 (no light perception – 1.0). Symptoms reported included visual acuity loss ($n=69$), visual field defects ($n=11$), disturbances of colour vision, light or dark adaptation ($n=10$). VEP and mfERG were normal in 43% ($n=31$). Both VEP and mfERG were pathological in 24% ($n=17$). In a further 18% ($n=13$) only the mfERG was pathological and in 15% ($n=11$) only the VEP was pathological. Macular dysfunction as detected with mfERG was present in 73% of 41 patients with at least one pathological test. Neuroimaging (MRI, CCT) and/or neurological examination was performed in 27/72 patients (38%), to account for unexplained visual loss, prior to the electrophysiological tests; these were normal in all patients. Electrophysiological tests revealed disturbances of the post-retinal visual pathway in only 3/27 patients. In 12/27 patients, mfERG revealed a macular disorder; in a further 12/27 patients VEP and mfERG were normal. The combined evaluation of VEP and mfERG is useful both to establish the area of dysfunction and the normality of the visual system. Electrophysiological testing prior to neuroimaging is recommended for patients where clear clinical signs of cerebral disorders are not evident. This reduces the frequency of unnecessary neuroimaging and associated radiation exposure.

Introduction

Unexplained visual loss is a diagnostic challenge for the ophthalmologist. On the one hand, detailed diagnostic procedures need to be applied to clarify the cause of visual disturbances and to provide an adequate cure. And on the other hand, patients with non-organic visual loss due, for example, to psychiatric disorders or malingering have to be detected early during the diagnostic process, to avoid unnecessary, time-consuming and costly diagnostic procedures. In some patients even after looking at case history and detailed ophthalmologic examinations, including the testing of visual acuity, refraction, visual field, swinging-flashlight-test, and biomicroscopy of the anterior and posterior segment, the visual loss remains unexplained. Possible explanations for this are post-retinal disorders of the visual pathway, or retinal dysfunction without apparent morphologic changes.

Visual-evoked potentials (VEP) provide an objective measure of the entire visual pathway and can be pathological in the presence of uncorrected refractive errors, ocular media opacities, maculopathy and dysfunction of the optic nerve or the central visual pathways. Because the VEP tests the entire visual pathway, a pathological VEP does not provide exact localisation of the defect. In contrast, the multifocal electroretinogram (mfERG) generated from the cones and bipolar cells of the posterior pole objectively evaluates the macula and allows for the localisation or exclusion of dysfunction of macular cones and bipolar cells.

Although the value of electrophysiological testing could be shown in adults and children [1–3], in our experience the ophthalmologist's first choice for additional diagnosis for patients with unexplained visual loss, is referral for neuroimaging or neurological diagnosis. Referring the patient for electrophysiology is often the ophthalmologist's last resort.

To assess whether the possible contribution of electrophysiological investigations is underestimated, we retrospectively analysed the data of 72 consecutive patients with unexplained visual loss who underwent both VEP and mfERG.

Materials and methods

The study was performed at the Department of Ophthalmology at Charité Campus Benjamin Franklin. Inclusion criteria for the study were, (1) the presence of visual acuity loss of unknown origin in at least one eye confirmed by re-examination prior to electrophysiological testing or; (2) patients with visual acuity of 1.0 in both eyes but complaining about visual acuity loss or visual field defects; and (3) the recording of both VEP and mfERG. Patients who did not undergo both VEP and mfERG and patients to whom electrophysiological testing was applied to clarify a clinically suspected specific diagnosis (e.g. optic neuritis, retinal or macular dystrophy) were excluded. In this study, 72 patients, consecutively seen between 1996 and 2002 could be included and their records reviewed retrospectively.

Clinical examinations were conducted after explanation of the procedures was made and formal consent obtained. The research adhered to the tenets of the Declaration of Helsinki. All patients underwent a complete eye examination including best-corrected visual acuity, anterior and posterior biomicroscopy. Ocular pressure was measured in 47/72 patients. Colour vision was tested with the desaturated Panel D 15 test ($n = 57$). Visual field testing was performed with Goldmann ($n = 53$) or automatic perimetry ($n = 4$). Fluorescein angiography was carried out in 16 patients. RPE autofluorescence imaging of the fundus was performed on 5 patients as described previously [4].

Electrophysiological testing included VEP ($n = 72$), mfERG ($n = 72$) and full-field ERG ($n = 24$). The same technician recorded all examinations and the recording equipment remained constant throughout the study. VEP and full-field ERG recordings were performed according to ISCEV standards [5, 6], and mfERG recordings in accordance with the ISCEV guidelines [7]. The recording protocols have been described in detail elsewhere [8–10]. VEPs were recorded with a Nicolet Spirit (Fa. Nicolet, Madison, USA). A black and white checkerboard pattern was displayed on a monitor with a reversal rate of 2/s. The pupils were undilated and optimal correction of refractive errors was used. One hundred and twenty-eight recordings were averaged for each of the three checkerboard sizes. Patients were encouraged to fixate; their cooperation and online signals were observed by the same, fully trained, technician. Recordings were repeated when loss of concentration or multiple eye movements influenced the recordings, the decision being based on the experience of the technician. The normal ranges for the P100 latency were defined by calculating the median values and the 95% confidence intervals in one eye of 70 subjects of similar age. The VEP was considered as pathological when in at least one of three pattern sizes, the latency of P100 was delayed beyond the normal range (pattern size with normal value for P100 latency: $1.5° < 118$ ms, $0.32° < 117$ ms, $0.16° < 124$ ms).

MfERGs were recorded and analysed using the VERIS system (Tomey, Germany) [11]. Recording was performed with maximum pupil dilation (2.5% phenylephrine and 0.5% tropicamide) using a Jet contact lens electrode (Microcomponents SA, Division Universo Plastique,

Switzerland). Refractive errors were corrected. A black and white pattern of 61 hexagons was presented on a monitor (200 cd/m^2 for white, 99.3% contrast) for stimulation. The duration of data acquisition was 4 min, divided into eight 30-s sessions. Patients were encouraged to fixate; their cooperation and online signals were observed by the same, fully trained, technician. Recordings of one session or the complete mfERG were repeated when loss of concentration or multiple eye movements influenced the recordings, the decision being based on the experience of the technician. Data analysis (first order kernel) was performed with the VERIS system. The response elicited by the central hexagon (ring 1) and the averaged responses elicited by the concentric rings of the hexagons surrounding the centre (ring 2–5) were evaluated. Amplitudes and implicit times were determined for the first positive component (P1) of each trace, based on manual cursor placement. Amplitudes were expressed relative to their respective area (nV/deg^2). MfERG stimuli location and anatomical areas [12] correspond roughly as follows: ring 1 to the fovea, ring 2 to the parafovea, ring 3 to the perifovea, ring 4 to the near periphery, and ring 5 to the central part of the middle periphery. The normal ranges for P1 amplitudes and implicit times were defined by calculation of the median values and the 95% confidence intervals in one eye of 50 age-similar subjects. The mfERG was considered to be pathological when at least the P1 amplitude of one of the 3 inner rings was reduced below the normal range.

Full-field ERG recordings were carried out with maximum pupil dilation using a Nicolet Spirit and a Ganzfeld (Nicolet, Madison, USA). Stimulus duration was 0.1 ms. Following 30 min of dark adaptation four stimuli with increasing intensity (maximum light intensity: 10 cd·s/m^2) were used for the recordings in the dark. Light-adapted recordings were performed after 10 min of light adaptation in the presence of white background light of 30 cd/m^2 with white stimuli of maximum light intensity. No averaging was done. For comparison, age-related normal ranges for amplitudes and implicit times were determined by calculation of the median values and the 95% confidence intervals from single eyes of 70 subjects.

Results

Our study included 42 females and 30 males ranging in age from 11.8 to 74.5 years (mean 42.4 ± 15.9 years, median 48.3 years) at the time of their first visit. Reported symptoms appearing in 70 patients spontaneously and in 2 patients post-operatively (pars plana vitrectomy because of peripheral retinal detachment) included visual acuity loss ($n = 69$), visual field defects ($n = 11$) and disturbances of colour vision, light or dark adaptation ($n = 10$). The duration of the symptoms prior to the visit to our clinic ranged from a few weeks to several years (mean 1.6 ± 1.9 years; median 1 year). However, 25 patients could not give a more detailed answer than, "since a long time". Two patients had no symptoms and their visual loss was detected accidentally during routine eye examinations.

The median visual acuity was 0.5 (no light perception – 1.0). Visual loss was bilateral in 65/72 (90%) patients. One of the remaining patients had one eye enucleated; unilateral visual loss was noted in the other six patients. The anterior eye segments were normal in 80% of the patients, including 3 pseudophakic patients. In the remaining 14 patients (20%) an incipient cataract or mild corneal abnormalities were insufficient to explain the severity of visual loss. The posterior pole and optic disc were normal in 68% of patients. In the remaining patients, neither subtle alterations of the retinal pigment epithelium or epiretinal membrane formation (14 patients, 20%) or mild abnormalities of the optic disc (9 patients, 13%) correlated with the severity of the visual loss. Additional methods – such as fundus autofluorescence, fluorescein angiography or full-field ERG, performed on only a few of the patients, displayed only mild abnormalities – could not reveal any additional findings to clarify the diagnosis. The ocular pressure was normal in cases that where measured.

Both VEP and mfERG were recorded in all 72 patients. In 57% (41/72) the recording of both VEP and mfERG allowed for the localisation of the origin of the visual dysfunction. The VEP was abnormal in 28/72 (39%) of patients and the mfERG was abnormal in 30/72 (42%). Following the electrophysiological examination results, the patients were separated into 4 groups: VEP and mfERG pathological, VEP pathological,

mfERG pathological and VEP and mfERG normal. Patients with bilateral visual loss presented with similar findings in both eyes in nearly all cases. If an interocular difference was present, the results of the eye with the more severe visual loss were used for group selection.

A pathological VEP indicates a dysfunction of the optic pathway. In 24% both VEP and mfERG were pathological, locating the dysfunction in the macula. In 15% the VEP was pathological and the mfERG revealed normal results, indicating a dysfunction beyond the first and second neuron of the retina. In contrast, in 18% the VEP was normal and the mfERG pathological, indicating a macular dysfunction that does not affect the cells generating the VEP. In the largest group (43%), however, both VEP and mfERG were normal. Table 1 illustrates these four groups and further clinical results.

The mean age of patients was different between the four groups, with the normal group including the youngest patients. In contrast, the median visual acuity was similar in all groups. Most of the patients with normal VEP and mfERG also had normal colour vision and visual field, however, the patients in the other groups often presented with a pathological Panel D 15 test, or perimetry.

Additional clinical tests could not clarify the visual loss in the 31/72 patients with normal VEP and mfERG either. In 25/31 patients, a specific cause for the visual loss could not be established. Four/31 patients were suspected of aggravation, and in two further patients amblyopia due to uncorrected high myopia and hyperopia was suspected.

Neuroimaging (MRI or CCT) was performed, prior to the electrophysiological tests, in 25/72 patients (35%) because of visual loss; 7 of these 25 patients and two additional patients underwent neurological examination. Neuroimages and the results of the neurological examination were normal in all of these 27 patients. In only 3 of these 27 patients was the VEP abnormal and the mfERG normal, suggesting a dysfunction of the optic nerve or intracranial visual pathways. In contrast, in 9/27 patients both VEP and mfERG were pathological and in 3/27 patients, only the mfERG was reduced, both indicating a retinal disorder. In the remaining 12/27 patients VEP and mfERG were normal.

After electrophysiological testing, neuroimaging could only be recommended for 11/72 patients with pathological VEP latencies to exclude structural pathologies affecting the optic nerve or other parts of the central visual pathways. From 5 of these 11 patients the results from the MRI or CCT were available and were normal.

Of the 72, 11 patients could be re-examined by us. There were: 8 females and 3 males ranging in age from 20.1 to 64.5 years (mean 50.4 ± 15.1 years, median 58.5 years) at the time of their second visit. The time between the first and the second visit ranged from 1 month to 5.3 years (mean 1.6 ± 1.7 years, median 0.9 years). The median visual acuity was 0.5 at both visits. In the majority of cases both the VEP and the mfERG carried out during the second visit revealed nothing that had not been revealed during the first visit. In 2/11 patients the perimetry produced signs of simulation.

Table 1. Clinical and functional findings in patients with unexplained visual loss separated according to the results of VEP and mfERG

	VEP and mfERG pathological	Only VEP pathological	Only mfERG pathological	VEP and mfERG normal
Number of patients (%)	17/72 (24)	11/72 (15)	13/72 (18)	31/72 (43)
Mean age (years)	52.5 ± 12.4	42.6 ± 14.6	50.2 ± 8.8	33.6 ± 15.8
Median visual acuity	0.5	0.5	0.5	0.5
Colour vision defects	13/14	7/8	10/13	8/22
Visual field defects[a]	11/14	6/7	4/12	8/24
Full-field ERG pathological	2/5	2/3	2/5	6/11

[a]Visual field defects included scotoma (relative, absolute, central, paracentral) or concentric narrowing. VEP, visual-evoked potentials; mfERG, multifocal electroretinography.

The following case reports demonstrate the different VEP and mfERG results in patients with unexplained visual loss.

Case 1

An 11-year-old girl (Figure 1, patient JJ) presented with markedly reduced visual acuity in the right eye, manifesting immediately after a fall injury on the back of her head four days earlier. Neurological examinations including CCT were normal. Visual acuity was at OD 0.1 and OS 1.0. Biomicroscopy and funduscopy revealed regular morphology. VEP and mfERG were both normal in the right eye. The results could reassure the parents that there were no signs of ocular damage due to the accident. Child and family did not attend a recommended repeat examination in the event that visual loss persisted.

Case 2

A 49-year-old woman (Figure 1, patient RW) presented with visual acuity of 0.4 in both eyes. She reported having suffered from night blind-

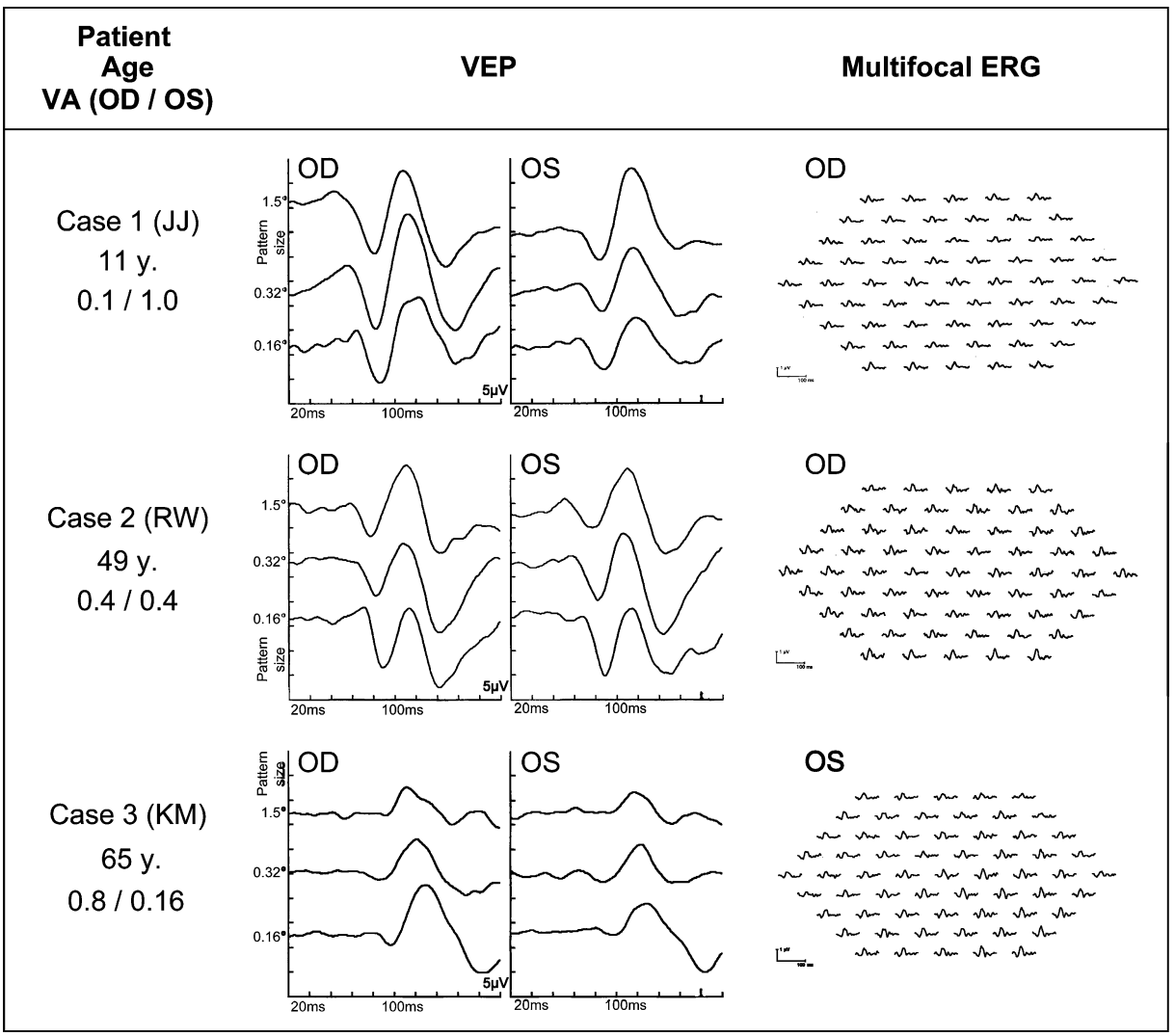

Figure 1. VEP and mfERG recordings from 3 patients with unexplained visual loss. For details see case reports 1–3 in the results section. VEP was tested with three pattern sizes (pattern size with normal value for P100 latency: 1.5° < 118 ms, 0.32° < 117 ms, 0.16° < 124 ms) in each eye. MfERG was recorded with 61 hexagons as described in the materials and methods section. VA – visual acuity.

ness, photophobia and visual field constriction for several years. Neurological examinations including CCT, prior to electrophysiology, were normal. Biomicroscopy and funduscopy revealed regular morphology. Visual field was constricted to about 20° in each eye, however, this constriction reduced to about 10° when tested binocularly. In addition, the visual field became narrower when the testing distance was doubled. Full-field ERG was completely normal, just as VEP and mfERG were. A non-organic origin for the visual acuity loss and the constricted visual fields could be determined.

Case 3

A 65-year-old male (Figure 1, patient KM) accidentally discovered visual loss along with reading problems in the left eye when he closed the right eye. He had been experiencing the difficulty for two weeks prior to reporting it. Visual field, performed by the referring ophthalmologist a day prior to our examinations, was normal in both eyes. Visual acuity was at OD 0.8 and OS 0.16. Biomicroscopy and funduscopy revealed regular morphology. Fluorescein angiography was normal. Colour vision defects were found in both eyes along the deutan/protan axis. MfERG was normal in the left eye. However, VEP revealed delayed latencies of P100 in pattern sizes 0.32° and 0.16° equally in both eyes. The patient was referred for neurological examinations including neuroimaging. All results were normal and visual loss remained unexplained with a suspicion of a residue of optic neuritis without multiple sclerosis.

Case 4

Four months ago, a 53-year-old male (Figure 2, patient YB) complained of visual loss in his left eye which manifested as a result of an accident where he suffered several broken ribs. Visual acuity was at OD 0.8 and OS 0.05. Biomicroscopy and funduscopy revealed regular morphology. Colour vision defects were present in the left eye along with a small relative scotoma in the centre. VEP showed small but symmetrical amplitudes in both eyes and latency of P100 was normal. However, the central area of the mfERG in the left eye showed reduced amplitudes, indicating a macular dysfunction. The reason for the maculopathy remained unclear.

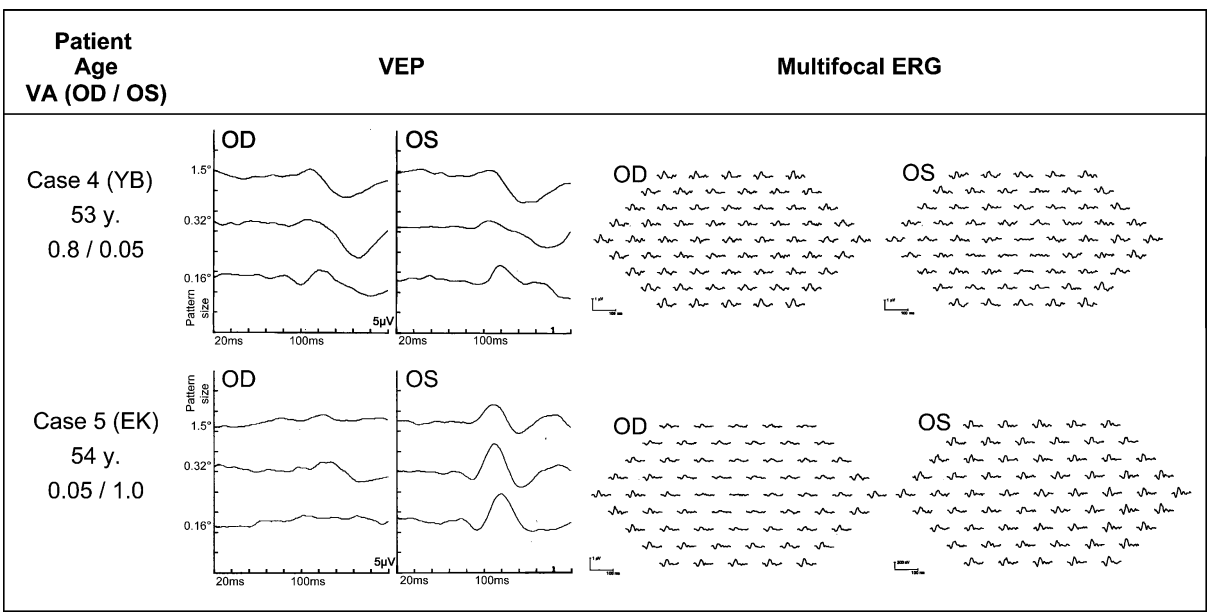

Figure 2. VEP and mfERG recordings from 2 patients with unexplained visual loss. For details see case reports 4–5 in the results section. VEP was tested with three pattern sizes (pattern size with normal value for P100 latency: 1.5° < 118 ms, 0.32° < 117 ms, 0.16° < 124 ms) in each eye. MfERG was recorded with 61 hexagons as described in the materials and methods section. VA – visual acuity.

Case 5

A 54-year-old female (Figure 2, patient EK) presented with visual loss in her right eye, which she had experienced for 6 months. Neuroimaging, prior to electrophysiology, did not reveal any pathology. Visual acuity was at OD 0.05 and OS 1.0. Biomicroscopy and funduscopy revealed regular morphology. Colour vision defects were found in both eyes (OD > OS) without any typical axis of confusion. An absolute scotoma was detected in the centre of the visual field including the blind spot in the right eye. VEP and mfERG were pathologic in the right eye but normal in the left eye. A macular dysfunction was diagnosed, however, the origin remained undetermined.

Discussion

In this retrospective study we report the results of recording both VEP and mfERG in a consecutive series of 72 patients with unexplained visual loss. Electrophysiological techniques provide objective evaluation of retinal and intracranial visual pathway function [1–3, 8, 13]. Visual acuity, Panel D 15 test and perimetry provide important information about retinal and intracranial visual pathway function as well, but these are subjective evaluation techniques and can be feigned by the patient.

Both VEP and mfERG could localise the area of dysfunction in 57% of patients and macular dysfunction could be documented by the reduced P1 amplitudes in the mfERG in most of these patients (30/41, 73%). In the majority of these patients (17/30, 57%), the VEP was pathological as well; this was due to the reduced pattern recognition of the dysfunctional macula. However, the VEP was unexpectedly normal in 13/30 (43%) patients with macular dysfunction.

In one quarter of the pathological patients (27%) disorders affecting structures of the optic pathway, beyond the retinal bipolar cells, were indicated by normal mfERG and delayed VEP latencies. Neuroimaging should be recommended for further analysis of the origin of the dysfunction in these patients.

It has to be noted that confirming a dysfunction is not equivalent to providing an exact diagnosis. The discrepancy between none or mild structural changes and the marked functional loss remained, but the location of dysfunction could still be determined. However, additional or repeated ophthalmologic examination failed to reveal adequate diagnosis in several patients.

Both VEP and mfERG were normal in 43% of patients and did not reveal any signs of dysfunction of the retina, optic nerve or intracranial visual pathways. One cannot exclude the fact that a disorder, which affects central visual function has been overlooked, however, it is unlikely that severe disorders of the posterior pole are not detected by either VEP or mfERG. If VEP and mfERG are normal in unexplained visual loss, severe dysfunction of the posterior pole or optic pathway can be excluded; this is reassuring for the examiner and the patient.

In our study neuroimaging was recommended in only 11/72 patients after the electrophysiological tests. It is of interest that 27/72 patients underwent neuroimaging or neurological examination prior to the electrophysiological diagnostic. In the majority of these patients the dysfunction could be either localised with the mfERG in the macular retina (12/27, 44%) or VEP and mfERG were normal (12/27, 44%). Neuroimaging was recommended in only 3/27 patients after the results of ophthalmologic examination. Neuroimaging and neurological examination did not result in any pathological findings. Electrophysiological testing prior to neuroimaging would have markedly reduced the number of examinations. One has to keep in mind that those patients, who were sent for neuroimaging resulting in pathological findings, were not referred for electrophysiological evaluation. If unexplained visual loss presents and clinical findings are suggestive for cerebral disorders (e.g. bitemporal visual field defects, sudden onset of headache), we recommend neuroimaging without delay. In all other cases, combined electrophysiological evaluation should be performed first to provide a basis to select patients for further diagnostic procedures including neuroimaging.

In this group of patients, in particular, one has to be aware that the results of both VEP and mfERG depend on focusing and fixation, which

can be influenced by patients who do not fully cooperate. Whereas malfixation at one spot will show a shift in the mfERG amplitudes, random fixation at different paracentral spots during a mfERG recording can reduce the amplitude of the central responses. Defocusing or fixation loss during VEP recording can cause a delay in latency. However, in our experience most patients cooperated very well and, when tested for the first time, did not know how to fake, to create intentional malingering. Recordings of mfERG and VEP were repeated when loss of concentration or multiple eye movements influenced the recordings. The majority of patients had either normal or abnormal results of both VEP and mfERG and the findings were nearly always similar in both eyes in bilateral visual loss. As such, we are confident that only a limited number of patients may have been successful in influencing the test results. It has to be emphasised, that in examining this group of patients special care had to be taken to monitor cooperation during the test. One possible way of testing cooperation is the simultaneous recording of VEPs and pattern ERGs (PERG) [14, 15].

There is an ongoing discussion of which test is more appropriate for testing macular function; more experience has been gathered from PERG compared to mfERG [15, 16] and the advantage of PERG is the additional information available on the retinal ganglion cells, which cannot be obtained by using a combination of mfERG and VEP. The advantage of mfERG, on the other hand, is the possibility of analysing the distribution of retinal dysfunction at the posterior pole; this cannot be obtained by using PERG. The mfERG may be used even with lower visual acuities than the PERG. One would expect that the combination of mfERG, PERG and VEP would be suitable to detect even more retinal dysfunctions in these patients compared to the use of only one or two methods. We restricted ourselves in this study to mfERG and VEP only because our experience with PERG is limited and we wanted to streamline the diagnosis of these patients.

In the present study, the combined evaluation of VEP and mfERG was useful in either establishing the area of dysfunction or the normality of the visual system in patients with unexplained visual loss. The combination of both methods revealed more pathologies than could be detected using one method alone. The results indicate that ophthalmologist should use all diagnostic possibilities to clarify unclear visual disturbances prior to considering neuroimaging. Electrophysiology is a time-consuming and highly specialized method, but it is non-invasive, safe and free of side effects. Patients would benefit from such a change in diagnostic priority because one could avoid subjecting at least some patients to unnecessary neuroimaging procedures and associated exposure to radiation.

Acknowledgement

We thank L. Udvarhelyi for his editorial assistance.

References

1. Corbett MC, Shilling JS, Holder GE. The assessment of clinical investigations: the Greenwich grading system and its application to electrodiagnostic testing in ophthalmology. Eye 1995; 9(Suppl): 59–64.
2. Hidajat RR, Goode DH. The clinical value of ophthalmic electrodiagnosis in children. Australas Phys Eng Sci Med 2001; 24: 172–76.
3. Woodruff SA, Fraser S, Burton LC, Holder GE, Sloper JJ. Evaluation of the electrodiagnostic investigation of children using the Greenwich grading system. Eye 2004; 18: 15–19.
4. Renner AB, Tillack H, Kraus H, et al. Morphology and functional characteristics in adult vitelliform macular dystrophy. Retina 2004; 24: 929–39.
5. Odom JV, Bach M, Barber C, et al. Visual evoked potentials standard (2004). Doc Ophthalmol 2004; 108: 115–23.
6. Marmor MF, Holder GE, Seeliger MW, Yamamoto S. Standard for clinical electroretinography (2004 update). Doc Ophthalmol 2004; 108: 107–14.
7. Marmor MF, Hood DC, Keating D, Kondo M, Seeliger MW, Miyake Y. Guidelines for basic multifocal electroretinography (mfERG). Doc Ophthalmol 2003; 106: 105–15.
8. Jandeck C, Kellner U, Kraus H, Foerster MH. Elektrophysiologische Untersuchungen entsprechend den ISCEV-Standards bei Kindern unter 10 Jahren. Ophthalmologe 1997; 94: 796–800.
9. Kellner U, Bornfeld N, Foerster MH. Severe course of cutaneous melanoma associated paraneoplastic retinopathy. Br J Ophthalmol 1995; 79: 746–52.
10. Kellner U, Kraus H, Foerster MH. Multifocal ERG in chloroquine retinopathy: regional variance of retinal dysfunction. Graefes Arch Clin Exp Ophthalmol 2000; 238: 94–97.
11. Sutter EE, Tran D. The field topography of ERG components in man – I. The photopic luminance response. Vision Res 1992; 32: 433–46.

12. Polyak SL. The Retina. Chicago: University of Chicago Press, 1941.
13. Bach M, Kellner U. Elektrophysiologische Diagnostik in der Ophthalmologie. Ophthalmologe 2000; 97: 898–920.
14. Holder GE. Pattern electroretinography (PERG) and an integrated approach to visual pathway diagnosis. Prog Ret Res 2001; 20: 531–61.
15. Holder GE. Electrophysiological assessment of optic nerve disease. Eye 2004; 18: 1133–43.
16. Hood DC. Assessing retinal function with the multifocal technique. Prog Ret Res 2000; 19: 607–46.

Address for correspondence: Agnes B. Renner, M.D., Augenklinik, Charité – Universitätsmedizin Berlin, Campus Benjamin Franklin, Hindenburgdamm 30, D-12200, Berlin, Germany Phone: +49-30-8445-2364; Fax: +49-30-8445-4450; E-mail: a.renner@berlin.de