Intraoperative monitoring of visual evoked potential: introduction of a clinically useful method

Clinical article

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Object. To obtain a clinically useful method of intraoperative monitoring of visual evoked potentials (VEPs), the authors developed a new light-stimulating device and introduced electroretinography (ERG) to ascertain retinal light stimulation after induction of venous anesthesia.

Methods. The new stimulating device consists of 16 red light–emitting diodes embedded in a soft silicone disc to avoid deviation of the light axis after frontal scalp-flap reflection. After induction of venous anesthesia with propofol, the authors performed ERG and VEP recording in 100 patients (200 eyes) who were at intraoperative risk for visual impairment.

Results. Stable ERG and VEP recordings were obtained in 187 eyes. In 12 eyes, stable ERG data were recorded but VEPs could not be obtained, probably because all 12 eyes manifested severe preoperative visual dysfunction. The disappearance of ERG data and VEPs in the 13th eye after frontal scalp-flap reflection suggested technical failure attributable to deviation of the light axis. The criterion for amplitude changes was defined as a 50% increase or decrease in amplitude compared with the control level. In 1 of 187 eyes the authors observed an increase in intraoperative amplitude and postoperative visual function improvement. Of 169 eyes without amplitude changes, 17 manifested improved visual function postoperatively, 150 showed no change, and 2 worsened (1 patient with a temporal tumor developed a slight visual field defect in both eyes). Of 3 eyes with intraoperative VEP deterioration and subsequent recovery upon changing the operative maneuver, 1 improved and 2 exhibited no change. The VEP amplitude decreased without subsequent recovery to 50% of the control level in 14 eyes, and all of these developed various degrees of postoperative deterioration of visual function.

Conclusions. With the strategy introduced here it is possible to record intraoperative VEPs in almost all patients except in those with severe visual dysfunction. In some patients, postoperative visual deterioration can be avoided or minimized by intraoperative VEP recording. All patients without an intraoperative decrease in the VEP amplitude were without severe postoperative deterioration in visual function, suggesting that intraoperative VEP monitoring may contribute to prevent postoperative visual dysfunction. (DOI: 10.3171/2008.9.JNS08451)

Key Words • anesthetic agent • electroretinography • intraoperative monitoring • visual evoked potential • visual function

The goal of surgery to the optic pathways (that is, the optic nerve, chiasm, tract, radiation, and lateral geniculate body) is preservation or improvement of visual function. However, some patients experience postoperative visual deterioration.8,15 Accurate monitoring of intraoperative VEPs may contribute to preventing postoperative visual deterioration.

Although attempts have been made since the 1970s to monitor intraoperatively the VEPs elicited by flash stimulation, difficulties in obtaining stable VEP recordings rendered their clinical usefulness unclear.1,2,4,5,11,12,14,18,19 and the method is now considered unreliable. We began using intraoperative VEP monitoring in 1987; however, we encountered many instances in which the VEPs disappeared either after frontal scalp-flap reflection or during surgical manipulation of structures other than the optic pathway.16 We suspected that the VEPs disappeared because of deviation of the light axis after frontal scalp flap reflection and that this was attributable to the inflexibility of the hard light-stimulating disc bearing the LEDs, and we proceeded to make 3 improvements.

Abbreviations used in this paper: AVM = arteriovenous malformation; ERG = electroretinography; ICA = internal carotid artery; LED = light-emitting diode; SHA = superior hypophyseal artery; VEP = visual evoked potential.
Our first improvement was the development of a new light-stimulating device to guarantee retinal stimulation even if the scalp flap is reflected. Our second improvement was the introduction of ERG. Using ERG, we ascertained the arrival of the light stimulus at the retina even when the VEP could not be recorded. In other words, when reproducible VEPs could not be obtained, ERG made it possible to determine whether the reason was due to technical problems or preexisting visual dysfunction. Also, we could ascertain whether the VEP disappearance was due to extracranial procedures such as scalp flap reflection or to intracranial maneuvers and could alert the surgeon to the possibility of damage. Third, to avoid the effects of inhalation anesthesia on VEP recordings, we used total intravenous anesthesia with propofol. We now report our experience with 100 patients (200 eyes) who underwent surgery after the introduction of our 3 improvements.

Methods

We monitored ERG data and VEPs intraoperatively in 100 patients (200 eyes) treated between January 2004 and December 2007. All were at intraoperative risk for visual impairment. Fifty-three patients had brain tumors, 42 had aneurysms, and 5 had AVMs (Table 1). The trial protocol was reviewed and approved by the local independent ethics committee. Informed consent was obtained from all patients or their legal representatives before enrollment in the study.

Our new light-stimulating device consists of 16 red high-luminosity (100 mCd) LEDs (Fig. 1 upper, Unique Medical Co.). The LEDs are embedded in a soft round (2-cm diameter) silicone disc to avoid deviation of the light axis during frontal scalp-flap reflection. The luminosity of the device is changeable from 500 to 20,000 Lx (Fig. 1 center). The device incorporates a safety system that shuts it down if continuous illumination by the LEDs exceeds 4 seconds.

Anesthesia, induced with a bolus injection of propofol (1.5–2 mg/kg) and fentanyl (2 µg/kg), was maintained by the continuous infusion of propofol (6–10 mg/kg/hr) and an additional injection of fentanyl (2 µg/kg) every 60 minutes. After induction of anesthesia, transparent eye patches were placed on the closed eyes. Then the light-stimulating device was placed on the eyelids and covered with another transparent eye patch (Fig. 1 lower). Needle electrodes for ERG were inserted subcutaneously at the lateral canthus; the reference electrode was placed in the contralateral canthus (Fig. 1 lower). The VEP recording electrodes were inserted subcutaneously at a point 4 cm above and 4 cm lateral from the external occipital protuberance (inion); reference electrodes were introduced subcutaneously in the mastoid process bilaterally. Because electrodes could not be inserted subcutaneously in patients with lesions in the occipital lobe, a saucer-like electrode was placed on the brain surface. A signal processor (Synax 1100, NEC Medical Systems; or Neuropack, Nihonkoden) was used to record the VEPs. The duration of each stimulus was 20 msec, and the frequency was 1 Hz. As we performed summation of 100 responses, each recording session required 100 seconds. The analysis time was 200 msec. We used low- (20 Hz) and high-band pass (500 Hz) filters. Before the start of each operation, we recorded the control waveforms of bilateral ERGs and VEPs. The stimulus intensity was supramaximal for ERG; the usual luminosity was 2000–5000 Lx. In patients whose ERG and VEP recordings disappeared after frontal scalp flap reflection, the luminosity could be increased to a maximum of 20,000 Lx. If there was a change in the VEP and/or the ERG after reflecting the scalp flap, we replaced the flap slowly and adjusted the light axis of the device on the eyelid until reproducible waveforms were obtained. A minimum of 2 recordings was obtained to confirm reproducibility of the data.

The reproducibility of ERG and VEP recordings was evaluated after induction of inhalation and venous anesthesia (Fig. 2). We obtained 2 continuous ERG and VEP recordings in the same patient and the same eye without visual dysfunction to confirm the reproducibility of ERG and VEP data obtained in the absence of surgical procedures. After induction of inhalation anesthesia, ERG data were not reproducible, and the reproducibility of the VEP amplitude was poor. On the other hand, after induction of propofol anesthesia, the reproducibility of ERG data was excellent, and the reproducibility of the VEP amplitude was good. Total venous anesthesia with propofol facilitated the detection of slight VEP changes during surgery.

We paid attention to the largest negative peak that appeared ~100 msec after stimulus onset. The VEP latency was defined as the peak latency of the largest negative peak and the amplitude as the voltage difference from the preceding positive peak to the largest negative peak. The criterion for amplitude changes was defined as a >50% increase or 50% decrease in amplitude compared with the

<table>
<thead>
<tr>
<th>Type of Lesion &amp; Location</th>
<th>No. of Lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>tumor</td>
<td>53</td>
</tr>
<tr>
<td>parasellar</td>
<td>28</td>
</tr>
<tr>
<td>temporal</td>
<td>13</td>
</tr>
<tr>
<td>parietal</td>
<td>5</td>
</tr>
<tr>
<td>occipital</td>
<td>4</td>
</tr>
<tr>
<td>frontal</td>
<td>2</td>
</tr>
<tr>
<td>orbital</td>
<td>1</td>
</tr>
<tr>
<td>aneurysm</td>
<td>42</td>
</tr>
<tr>
<td>ICA</td>
<td>25</td>
</tr>
<tr>
<td>ACoA</td>
<td>10</td>
</tr>
<tr>
<td>MCA</td>
<td>5</td>
</tr>
<tr>
<td>multiple</td>
<td>2</td>
</tr>
<tr>
<td>AVM</td>
<td>5</td>
</tr>
<tr>
<td>temporal</td>
<td>3</td>
</tr>
<tr>
<td>parietal</td>
<td>1</td>
</tr>
<tr>
<td>occipital</td>
<td>1</td>
</tr>
</tbody>
</table>

* ACoA = anterior communicating artery; MCA = middle cerebral artery.

TABLE 1: Clinical findings in 100 patients undergoing VEP monitoring and ERG*
control level. The surgeon was immediately alerted when the VEP changed beyond these thresholds. In patients manifesting a change in VEP amplitude, changes in the latency of the largest negative peak were also studied.

We evaluated the ERG and VEP recording rate and examined the correlation between intraoperative VEP findings and postoperative visual function. Follow-up was performed at our Department of Ophthalmology ~2 weeks after surgery. All visual acuity data presented in this paper reflect corrected visual acuity.

Results

Recording ERG Data and VEPs

Stable and highly reproducible ERG and VEP data were obtained in 187 of 200 eyes (100 patients). In 12 eyes, the ERG data were stable, but VEP data could not be obtained, probably because all 12 manifested severe preexisting visual dysfunction (Fig. 3). The disappearance of ERG and VEP data in the remaining eye after frontal scalp flap reflection suggested technical failure due to deviation of the light axis. We encountered no complications attributable to ERG and VEP recording in the current series.

Representative Case

Case 1. This 52-year-old man presented with sudden-onset of severe headache and bilateral visual deterioration. His right visual acuity was hand motion; a visual field could not be obtained. His left visual acuity was 2.0; the visual field revealed temporal hemianopia. Three-dimensional CT angiography revealed a 9-mm ophthalmic aneurysm projecting superomedially, and the patient underwent right frontotemporal craniotomy for its obliteration. The ERG data were recorded on the right side. However, the right VEP was flat due to disturbance of the right optic nerve rather than technical failure. Both ERG and VEP recordings were reproducible on the left side. The right optic nerve was split by the aneurysm, and part of the aneurysmal wall was visible through the optic nerve. Two clips were applied to the aneurysm. The aneurysmal wall was incised for decompression of the right optic nerve; however, the right VEP remained flat and the left VEP did not change. Postoperatively, the patient’s right
visual acuity improved to 0.08; the visual field revealed nasal hemianopia. His left visual acuity remained unchanged, and the visual field improved to upper temporal quadrant hemianopia (Fig. 4).

**Correlation Between Intraoperative VEP Findings and Postoperative Visual Function**

In 1 of the 187 recordable eyes the VEP amplitude increased and postoperative visual function improved (Case 2). This patient had a pituitary adenoma that was treated via a transphenoidal approach.

Among 169 eyes without amplitude changes, 17 manifested improved postoperative visual function and 150 revealed no change. Both eyes exhibiting postoperative deterioration were in a patient (Case 3) with a temporal tumor; postoperatively, both had a mild visual field defect similar to quadrant homonymous hemianopia. This was the only false-negative finding (2 sides) in this series, giving a false-negative rate of 1.2%.

In 3 eyes, the VEP amplitude decreased transiently and recovered intraoperatively upon discontinuing a specific surgical maneuver (Table 2). In 1 patient with a pituitary adenoma the deterioration was attributable to retraction of the frontal lobe near the optic nerve. After internal decompression of the tumor, the VEP recovered. The other 2 patients had an ophthalmic segment aneurysm. In 1 patient with an ICA-opthalmic artery aneurysm, latency prolongation of ~10 msec was accompanied by a reduction in amplitude during dissection of the aneurysm from the optic nerve. After discontinuing the dissection, the VEPs recovered. In another eye, VEPs disappeared after aneurysmal neck clipping of an ICA-SHA aneurysm (Case 4) and recovered after clip removal. We considered the amplitude reduction to be due to dissection or retraction of the optic nerve in 2 eyes and to blood flow disturbance in the SHA in the third eye. Postoperative visual

![Fig. 3. Diagrams showing the preoperative visual acuities and fields of 12 eyes in which ERG could, but VEP could not, be recorded. In 11 of these eyes, corrected visual acuity was < 0.1 and in 10 the visual field was less than hemianopia.](image)

![Fig. 4. Case 1. Intraoperative ERG and VEP findings. It was possible to ascertain that the flash stimulation reached the retina by ERG. The right stimulating VEP was flat, but the left stimulating VEP could be recorded. We determined that the reason the right VEP was flat was due to preoperative visual dysfunction of the right optic nerve.](image)
function improved in 1 eye and did not change in the other 2 eyes (Table 2).

The VEP amplitude decreased without subsequent recovery to 50% of the control level in 14 eyes in 8 patients (Table 3). In these eyes, the amplitude reduction was attributable to dissection or retraction of the optic nerve (1 eye each in 2 patients with parasellar tumors), dissection of the optic chiasma (2 eyes in a patient with a craniopharyngioma), dissection of the optic tract (2 eyes in a patient with a craniopharyngioma), optic tract ischemia (2 eyes in a patient with an ICA–posterior communicating artery aneurysm), removal of a temporal tumor (4 eyes in 2 patients with temporal tumors), and removal of an occipital AVM (2 eyes in a patient [Case 5]). Although further surgical maneuvers were immediately halted, the VEP amplitude did not recover up to 50% of the control level. Consequently, all patients manifested various degrees of postoperative visual disturbance (Table 3).

**Representative Cases**

**Case 2.** This 48-year-old man with a nonfunctioning pituitary adenoma presented with preoperative atypical bitemporal hemianopia and underwent transnasal transsphenoidal surgery. Intraoperatively the amplitude of the right stimulating and left recording VEP markedly increased; the left VEP did not change. Postoperatively, the visual acuity and field of both eyes were markedly improved (Fig. 5).

**Case 3.** This 56-year-old man had a radioresistant metastatic chondrosarcoma in the right temporal lobe. His preoperative visual function was normal. Bilateral intraoperative VEPs did not change. Postoperatively he developed a bilateral visual field defect similar to quadrant homonymous hemianopia (Fig. 6).

**Case 4.** This 48-year-old woman with an unruptured aneurysm at the junction of the right ICA-SHA underwent right frontotemporal craniotomy. We drilled the right anterior clinoid process to confirm the proximal neck of the aneurysm. The right VEP remained unchanged during drilling of the anterior clinoid process and optic canal. After clipping the aneurysm with an encircling clip the right VEP disappeared; the clip was removed immediately and the VEP reappeared 10 minutes later. When inspection with a micromirror confirmed that the SHA was not occluded, it was clipped with a slightly curved clip. The right VEP did not change until the end of surgery. This patient had no postoperative visual disturbance (Figs. 7 and 8).

**Case 5.** This 50-year-old woman had a left occipital AVM without any visual disturbance. As we exposed the entire left occipital lobe, VEPs were recorded from a surface electrode, resulting in an amplitude of 40 μV. During dissection of the nidus the amplitude decreased to 50% of the control level and there was bleeding from the removal cavity. During coagulation, the VEP decreased to 10% of the control level without a change in latency. Postoperatively, she developed homonymous hemianopia (Fig. 9).

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**TABLE 2: Profiles of 3 eyes in which VEPs deteriorated with subsequent recovery**

<table>
<thead>
<tr>
<th>Age (yrs), Sex</th>
<th>Lesion</th>
<th>Side</th>
<th>At Aggravation</th>
<th>At End of Op</th>
<th>Cause of VEP Deterioration</th>
<th>Preoperative → Postoperative</th>
</tr>
</thead>
<tbody>
<tr>
<td>66, M</td>
<td>pituitary adenoma</td>
<td>rt</td>
<td>disappeared, NA</td>
<td>80%, no change</td>
<td>retraction of rt frontal lobe</td>
<td>1.2 → 1.5</td>
</tr>
<tr>
<td>48, F</td>
<td>rt ICA-SHA aneurysm</td>
<td>rt</td>
<td>disappeared, NA</td>
<td>70%, no change</td>
<td>neck clipping</td>
<td>1.5 → 1.5</td>
</tr>
<tr>
<td>64, M</td>
<td>rt ICA-OphA aneurysm</td>
<td>rt</td>
<td>50%, 10 msec</td>
<td>100%, no change</td>
<td>dissection of rt optic nerve</td>
<td>1.5 → 1.5</td>
</tr>
</tbody>
</table>

* NA = not available; OphA = ophthalmic artery.
TABLE 3: Profiles of 14 eyes in which VEPs deteriorated without subsequent recovery to 50% of the control level

<table>
<thead>
<tr>
<th>Age (yrs), Sex</th>
<th>Lesion</th>
<th>Side</th>
<th>At Aggravation</th>
<th>At End of Op</th>
<th>Cause of VEP Deterioration</th>
<th>Visual Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>67, F</td>
<td>Rathke cleft cyst</td>
<td>rt</td>
<td>20%, 10 msec</td>
<td>40%, no change</td>
<td>retraction of rt optic nerve</td>
<td>1.0</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.5</td>
</tr>
<tr>
<td>71, M</td>
<td>pituitary adenoma (recurrent)</td>
<td>rt</td>
<td>30%, no change</td>
<td>40%, no change</td>
<td>dissection of rt optic nerve</td>
<td>2.0</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td>2.0</td>
</tr>
<tr>
<td>36, M</td>
<td>craniopharyngioma (recurrent)</td>
<td>rt</td>
<td>30%, no change</td>
<td>40%, no change</td>
<td>dissection of optic chiasma</td>
<td>1.2</td>
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<td></td>
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<td></td>
<td></td>
<td>1.0</td>
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<tr>
<td></td>
<td></td>
<td>lt</td>
<td>30%, no change</td>
<td>40%, no change</td>
<td></td>
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<tr>
<td>41, F</td>
<td>craniopharyngioma</td>
<td>rt</td>
<td>disappeared, NA</td>
<td>10%, NA</td>
<td>dissection of rt optic tract</td>
<td>1.0</td>
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<td></td>
<td></td>
<td>0.03</td>
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<tr>
<td></td>
<td></td>
<td>lt</td>
<td>disappeared, NA</td>
<td>10%, NA</td>
<td></td>
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<tr>
<td>72, F</td>
<td>rt ICA-PCoA aneurysm (ruptured)</td>
<td>rt</td>
<td>disappeared, NA</td>
<td>40%, no change</td>
<td>temporary occlusion of ICA neck (90 sec x 3)</td>
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<td></td>
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<td></td>
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<td>0.9</td>
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<tr>
<td></td>
<td></td>
<td>lt</td>
<td>disappeared, NA</td>
<td>40%, no change</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11, F</td>
<td>rt temporal PNET</td>
<td>rt</td>
<td>30%, no change</td>
<td>30%, no change</td>
<td>removal of tumor</td>
<td></td>
</tr>
</tbody>
</table>

(continued)
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**TABLE 3: Profiles of 14 eyes in which VEPs deteriorated without subsequent recovery to 50% of the control level**

<table>
<thead>
<tr>
<th>Age (yrs),</th>
<th>Sex</th>
<th>Lesion</th>
<th>Side</th>
<th>At Aggravation</th>
<th>At End of Op</th>
<th>Cause of VEP Deterioration</th>
<th>Amplitude (control%), Latency (delay)</th>
<th>Visual Function</th>
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</thead>
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<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Preoperative → Postoperative</td>
<td></td>
</tr>
<tr>
<td>63, M</td>
<td>rt temporal glioblastoma</td>
<td>rt</td>
<td>20%, no change</td>
<td>20%, no change</td>
<td>removal of tumor</td>
<td>lt</td>
<td>30%, no change</td>
<td>30%, no change</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.2</td>
</tr>
<tr>
<td>54, F</td>
<td>lt occipital AVM</td>
<td>rt</td>
<td>10%, no change</td>
<td>10%, no change</td>
<td>removal of nidus &amp; hemo-stasis</td>
<td>lt</td>
<td>20%, no change</td>
<td>20%, no change</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.2</td>
</tr>
</tbody>
</table>

* PCoA = posterior communicating artery; PNET= primitive neuroectodermal tumor.

**Discussion**

In 1973, Wright et al.\cite{21} first reported using VEP monitoring during surgery for orbital tumors to prevent postoperative visual disturbance. Although others have found intraoperative VEP monitoring useful,\cite{6,9,13,20} in many patients the acquisition of stable recordings was reportedly difficult, and the clinical usefulness of intraoperative VEP monitoring remained unclear.\cite{1,2,4,5,11,12,14,18,19} We started intraoperative VEP monitoring in 1987, but the monitoring has not been clinically useful.\cite{16}

Various light stimulators have been used.\cite{1,2,6,7,9,11–14,18–21} We found that when we used the hard-plate device featuring LED, the VEP disappeared in many patients after frontal scalp-flap reflection.\cite{16} To solve this problem, we embedded high-luminosity LEDs in a soft, round (2-cm diameter) silicone disc. At maximum, the luminosity of the new device is 20 times (20,000 Lx) that of the former device, and adjustments make it possible to stimulate the retina at supramaximal intensity. We usually apply a luminosity of 2000–5000 Lx. Our search of the literature found no reports of complications under the closed eyelid when ophthalmological surgery lasting 15 minutes was performed at 50,000 Lx under an operating microscope. Using a maximal luminosity setting of 20,000 Lx, the total amount of light to acquire a single VEP recording was 1/1000 of the total light exposure used in ophthalmological surgery. As we did not encounter any complications when we used the new device for VEP recording, we consider its use to be safe.

We introduced ERG to ascertain intraoperatively that the light stimulus reached the retina even in cases in which VEPs were not obtained.\cite{3} When retinal stimulation is confirmed, the inability to acquire VEP recordings in patients with preoperative visual disturbance is thus attributable to the preexisting visual dysfunction rather than light axis deviation. In the current series there were 12 eyes in which ERG could, but VEPs could not, be recorded; all manifested severe preoperative visual disturbance (Fig. 3). In 11 of these eyes, corrected visual acuity was < 0.1, and in 10 the visual field was less than hemianopia. Given that there are large individual differences in the VEP amplitude, we could not define the degree of visual disturbance at which the VEP disappears. However, our
data suggest that there is a level of visual dysfunction that results in the disappearance of the VEP.

Since the VEP travels from the retina to the occipital lobe via 3 synapses, in the presence of anesthetic agents, the VEP amplitude and latency demonstrate frequent and marked changes even without manipulation in and/or around the optic pathway.\textsuperscript{1,4,5,7,11,12,17} To overcome this phenomenon, we used total venous anesthesia with propofol;\textsuperscript{18} this reduced the amplitude and latency fluctuations and stable recording became possible (Fig. 2), although we continued to encounter amplitude fluctuations of up to 20% of the control. This indicates that it is necessary to confirm the reproducibility of VEP data before performing maneuvers that may endanger the optic pathway.

We examined the amplitude and latency of the largest negative peak that occurred ~ 100 msec after the start of light stimulation. Given that VEPs differ from individual to individual and are affected by existing visual impair-

\begin{figure}
\centering
\includegraphics[width=\textwidth]{image.png}
\caption{Case 2. \textit{Upper:} Preoperative MR image, and visual acuities and fields. \textit{Center:} Intraoperative VEP findings. The amplitude of the right stimulating and left recording VEP markedly increased. The asterisks indicate an increase in amplitude. \textit{Lower:} Postoperative MR image showing a residual tumor. The visual acuity and field of both eyes markedly improved postoperatively.}
\end{figure}
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ments, interindividual comparisons are difficult. However, in all 187 recorded eyes we found that if we restricted our evaluation to the same side in the same patient, reproducibility was adequate for the detection of amplitude and latency changes. As defined here, the criterion for VEP changes was a change in the amplitude from the control level. All 14 eyes that did not recover from VEP deterioration of < 50% of the control manifested postoperative visual disturbance. Latency evaluation was difficult in the presence of an amplitude decrease. We observed 10 msec or longer latency prolongation in only 2 eyes in this study; recovery was noted at the end of surgery. These results suggest that amplitude changes in particular may represent a landmark on intraoperative VEP recordings that alerts to visual disturbance and that the surgeon must be warned when the amplitude reduction approaches 50%.

Fig. 6. Case 3. Upper: Preoperative MR image, and visual acuities and fields. Center: Intraoperative VEP findings. Lower: Postoperative MR image, and visual acuities and fields. The patient developed a bilateral visual field defect similar to quadrant homonymous hemianopia.
In 17 eyes visual function was improved postoperatively; only 1 eye exhibited an intraoperative amplitude increase. In the other 16 eyes there were no amplitude changes, although we found that, in some patients, visual function was improved just after the operation. We posit that some time is required for the affected neural structures to recover functionally. There were 16 eyes that developed postoperative visual dysfunction. In 14 of these we observed various degrees of amplitude reduction; in the other 2 eyes in 1 patient the amplitude did not change. This was the only false-negative result (2 sides) in this series. Thus, our false-negative rate was 1.2%. Postoperatively, this patient developed mild bilateral visual field defects similar to quadrant homonymous hemianopia. Another patient manifested quadrant homonymous hemianopia including the central visual field. In that case, the VEP amplitude at the end of surgery was 50% lower than the control level. At present, our method of intraoperative VEP monitoring cannot detect partial visual field defects. However, if the amplitude did not decrease up to 50%, the surgeon can be relieved to understand that severe visual dysfunction will not occur.

Of the 3 eyes with intraoperative VEP deterioration and subsequent recovery upon changing the operative maneuver, 1 showed postoperative visual function improvement and 2 exhibited no change. We think that without intraoperative VEP monitoring resulting in a change in surgical maneuvers, these 3 patients might have developed postoperative visual disturbance. In Case 4, we detected blood flow insufficiency in the SHA; Goto et al. reported a similar case. Again, we think that without intraoperative VEP monitoring, this patient might have suffered postoperative unilateral blindness. Since VEP changes may result in the use of alternative operative maneuvers, we think that our method is clinically useful.

In 14 eyes the VEP amplitude deteriorated and did not recover intraoperatively to 50% of the control level. In 3 patients (6 eyes) with a temporal tumor or an occipital AVM, we were forced to continue the surgical procedures, and the development of postoperative visual dysfunction was unavoidable. In 2 patients (4 eyes) with craniohypophyseal glioma, the surgeon was aware of deterioration in the VEP amplitude but did not change the surgical maneuvers. Consequently, 1 patient, treated shortly after the introduction of our method, developed severe postoperative visual disturbance. Based on this experience we do not attempt total tumor removal in cases with VEP disappear-

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**Fig. 7. Case 4.** *Upper:* Three-dimensional CT angiogram (anterior view) revealing a medially projecting ICA aneurysm at the origin of the SHA. *Center:* Intraoperative photograph obtained at the first clipping with an encircling clip. *Lower:* Intraoperative photograph obtained at the second clipping with a slightly curved clip. The patency of the SHA was confirmed by a micromirror.

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**Fig. 8.** Intraoperative VEP findings. During drilling of the anterior clinoid process and the optic canal, the right VEP did not change. After clipping the aneurysm with an encircling clip, the right VEP disappeared. The clip was removed immediately and the VEP reappeared 10 minutes later. As inspection with a micromirror confirmed that the SHA was not occluded, it was clipped with a slightly curved clip. The right VEP did not change until the end of surgery.
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In the other 4 eyes without amplitude recovery to 50% of the control level, the surgeon changed the operative procedures; however, these patients manifested postoperative visual dysfunction whose degree may have been greater in the absence of intraoperative VEP monitoring. Since the method described here was able to detect the dysfunction of the optic nerve, optic chiasma, optic tract, temporal lobe, and occipital lobe, it can be used to detect abnormalities in the entire optic pathway. At present, it is not possible to predict the recovery of an amplitude reduction on intraoperative VEP recordings. Therefore, if there is an intraoperative VEP decrease, its cause must be ascertained; if the surgical maneuver is responsible for the decrease, it must be changed immediately.

We applied our VEP stimulating and recording conditions in 100 patients (200 eyes). At present, 100 seconds are required to obtain 1 waveform; this represents a serious disadvantage and means that real-time monitoring is not yet possible. Further study is necessary to reexamine the stimulating and recording conditions in efforts to shorten the time required for the acquisition of reproducible waveforms.

Fig. 9. Case 5. Upper: Preoperative MR image, and visual acuitities and fields. Center: Intraoperative VEP findings. Lower: Postoperative MR image, and visual acuitities and fields. During dissection of the nidus the amplitude decreased to 50% of the control level, and there was bleeding from the removal cavity. During its coagulation the VEP decreased to 10% of the control level without a change in latency.
Conclusions

With our method, which involves an improved light-stimulating device and simultaneous ERG/VEP recording after total venous anesthesia is induced, we were able to acquire satisfactory intraoperative VEP recordings in eyes without severe preoperative visual dysfunction. In some patients, postoperative visual deterioration could be avoided or minimized by intraoperative VEP recording. Changes in intraoperative VEP findings, especially in the VEP amplitude, were well correlated with postoperative visual function, suggesting that intraoperative VEP monitoring may contribute to prevent postoperative visual dysfunction.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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