Pattern-reversal visual evoked potentials in patients with hydrocephalus

SHAKIR M. ALANI, M.B., CH.B., M.R.C.P.

Department of Clinical Neurophysiology, National Hospital for Nervous Diseases, Queen Square, London, England

Pattern-reversal visual evoked potentials (VEP's) in response to whole- and half-field stimulation were studied in 10 patients with hydrocephalus. Abnormalities consistent with optic nerve dysfunction were recorded in four patients. Two patients had response asymmetry to half-field stimulation, which suggested dysfunction of the visual pathway in the right hemisphere. The remaining four patients had normal responses. Measurement of VEP's was repeated after the surgical treatment of hydrocephalus in four patients, and showed marked improvement in two of the three patients with preoperative abnormalities. This study suggests that, in patients with hydrocephalus, VEP's are more sensitive than clinical methods in detecting visual pathway dysfunction and that they can be useful in the follow-up monitoring of surgically treated hydrocephalic patients.

Key Words • hydrocephalus • normal-pressure hydrocephalus • visual evoked potentials • evoked potentials

Visual evoked potentials (VEP's) are now well established as a sensitive method of detecting abnormalities of the visual pathways. Because of the close anatomical relationship between the ventricular system and the visual pathways, several investigators have studied the effects of enlarged ventricles in hydrocephalus on visual function using the evoked potential, mostly in response to flash stimulation and in children. Half-field stimulation, necessary to demonstrate posterior visual pathway disturbances, has not so far been used in the investigation of patients with hydrocephalus. This study investigates the effects of hydrocephalus on whole- and half-field pattern VEP's and discusses possible mechanisms and clinical applications.

Clinical Material and Methods

The VEP's of 10 hydrocephalic patients (seven men and three women, aged 19 to 72 years) were recorded. Data from 50 healthy volunteers (27 men and 23 women, aged 18 to 63 years) served as controls. The mean (± standard deviation) P100 latency and amplitude for whole-field stimulation were 98.1 ± 5.2 msec and 11.2 ± 4.9 μV, respectively. The mean P100 latency and amplitude for half-field stimulation were 102 ± 4.7 msec and 6.4 ± 2.1 μV, respectively. The diagnosis of hydrocephalus was entertained clinically and confirmed by computerized tomography (CT) scanning. Only patients who were able to cooperate during recordings were studied. The relevant clinical details are shown in Table 1.

Hydrocephalus was of the communicating type in six patients. Five of these patients presented with progressive spastic paraparesis and ataxia; four of these five had memory loss and three had urinary incontinence. Only the left eye was examined in one patient (Case 3), a 61-year-old man who had lost vision in the right eye due to trauma in childhood. The sixth patient (Case 8), a 50-year-old man with a long-standing memory problem, had a 3-month history of headache. He was found to have bilateral papilledema and generalized cysticercosis. The only abnormality found on the CT scans was enlargement of the cerebral ventricles in all six patients.

Four patients had obstructive hydrocephalus. A 28-year-old woman with a 13-year history of temporal lobe epilepsy (Case 6) presented with recent left-sided headache and increased seizure frequency. A left temporal dermoid cyst was removed surgically. Case 1, a 35-year-old man with a 10-year history of intermittent headache which increased during straining, presented with episodes of sudden loss of vision in homonymous visual fields. This happened on the left or right side independently, and would last for a few weeks each time, followed
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<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Type of Hydrocephalus</th>
<th>Visual Acuity</th>
<th>Visual Field*</th>
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<tr>
<td>1</td>
<td>35, M</td>
<td>obstructive</td>
<td>6/6</td>
<td>6/5</td>
</tr>
<tr>
<td>2</td>
<td>50, M</td>
<td>communicating</td>
<td>6/5</td>
<td>6/5</td>
</tr>
<tr>
<td>3</td>
<td>61, M</td>
<td>communicating</td>
<td>blind</td>
<td>6/6</td>
</tr>
<tr>
<td>4</td>
<td>58, M</td>
<td>communicating</td>
<td>6/6</td>
<td>6/12</td>
</tr>
<tr>
<td>5</td>
<td>19, M</td>
<td>obstructive</td>
<td>6/6</td>
<td>6/5</td>
</tr>
<tr>
<td>6</td>
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<td>6/5</td>
</tr>
<tr>
<td>7</td>
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<td>6/9</td>
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<tr>
<td>8</td>
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</tr>
<tr>
<td>9</td>
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<td>6/12</td>
<td>6/12</td>
</tr>
<tr>
<td>10</td>
<td>44, F</td>
<td>communicating</td>
<td>6/6</td>
<td>6/6</td>
</tr>
</tbody>
</table>

* LHH = left homonymous hemianopsia.

by full clinical recovery. At the time of VEP recording, he had a left homonymous hemianopsia. A third ventricle colloid cyst was later removed. The remaining two patients (Cases 5 and 9) had aqueductal stenosis.

The fundi, visual acuity (measured with the Snellen chart), and visual fields (tested either by the Bjerrum screen using 3-mm red and 1-mm white objects, or by confrontation using white and red neurological pins) were examined shortly before recording of the VEP's. None of the patients had visual inattention.

Pattern-reversal stimulation was by 2/sec black and white checkerboard pattern reversal (luminance 86 cd/sq m and 1085 cd/sq m, respectively). The total field subtended 18° at the eye, each square subtending 34 minutes. Each eye was stimulated separately and patients were asked to fixate on the center of the screen for whole-field stimulation and on the left or right edge of the screen for half-field stimulation. With a sweep time of 256 msec, 128 sweeps were averaged by a Research Machines 380Z computer. The process was repeated as necessary to ensure reproducibility of the responses. Recording was by a horizontal occipital chain of five silver/silver chloride electrodes (referred to Fz) at 5-cm intervals, with the central electrode on the midline 5 cm above the inion. The recording amplifiers had a time constant of 0.3 seconds and a high-frequency cutoff (~3 dB) at 300 Hz.

Whole- and half-field responses were considered abnormal if the P_{100} component latency was greater than 2.5 standard deviations from normal mean, or if it was absent or greatly attenuated (less than 1 μV in amplitude).

**Results**

**Preoperative Results**

In response to whole-field stimulation, the P_{100} components were absent from both eyes in two patients. One of these (Case 1), at the time of recording, had a left homonymous hemianopsia. The other (Case 2) had full visual fields. The P_{100} components were grossly attenuated in recordings from the healthy eye in another patient (Case 3, Fig. 1), whose visual fields were also full. A fourth patient (Case 4) had a delayed P_{100} component from each eye; his visual fields were full.

In response to half-field stimulation, the P_{100} components were absent or greatly attenuated from the homonymous left half fields in two patients (Case 6, Fig. 2 left; and Case 5). Both had full visual fields. Responses to whole-field stimulation from each eye were normal in both patients.

Four patients (Cases 7 to 10) had normal VEP's. All had full visual fields.

**Postoperative Results**

The VEP's improved dramatically (judged by the return of P_{100} component latency and amplitude to normality) 1 week after the insertion of a ventriculoperitoneal shunt in Case 3 (Fig. 1) and 3 weeks after the removal of a left temporal dermoid cyst in Case 6 (Fig. 2 right). In Case 1, there was no significant change in the VEP's 3 weeks after the removal of a third ventricle colloid cyst, in spite of clinical improvement and full visual fields. The VEP's remained normal 1 week after the insertion of a ventriculoperitoneal shunt in Case 8.

**Discussion**

Although VEP's are best recorded over the occipital region ipsilateral to the stimulated fields, they have been shown to arise from the contralateral visual cortex in response to half-field stimulation. This has been attributed to the orientation of response generators in the visual cortex.³ The absence of P_{100} components in response to left half-field stimulation documented in two of the patients presented here (Cases 5 and 6) can therefore reasonably be attributed to posterior visual pathway dysfunction in the right cerebral hemisphere.

There is good experimental,¹⁴ radiological,⁹ and
pathological evidence of periventricular edema followed by gliosis and damage to white matter fibers in acute hydrocephalus. There is also evidence of decreased blood flow to white matter in the periventricular area during the stage of progressive hydrocephalus. Because of the close anatomical relationship of posterior visual pathway fibers to the periventricular area, this study suggests that the enlarged ventricle in hydrocephalus can damage these fibers, leading to the half-field VEP abnormalities described here.

The absence or gross attenuation of the P_{100} components in three patients and delay of P_{100} in one patient following whole-field stimulation are consistent with optic nerve dysfunction. These findings are in agreement with those of Hallday, et al., in compressive lesions of the anterior visual pathway. Optic nerve dysfunction in patients with hydrocephalus has been ascribed to compression of the optic nerve by the central herniation of the third ventricle.

The data presented here showed poor correlation between the clinical visual field and VEP findings. Only one of the patients with VEP abnormalities (Case 1) had a clear visual field defect (left homonymous hemianopsia). In this particular patient, the VEP abnormalities were more extensive than clinically expected. Previous studies have found that pattern-reversal VEP's can be more sensitive than clinical methods in detecting anterior visual pathway abnormalities. This study suggests that, in patients with hydrocephalus, such a statement could be extended to include the posterior visual pathway.

The postoperative VEP changes presented in this series cannot be used to make generalizations about VEP changes in surgically treated hydrocephalic patients because of the limited number of cases studied. However, this study adds to previous indications that VEP's could be useful in the follow-up monitoring of such patients.

This study has shown that VEP abnormalities in response to whole- and half-field stimulation are common in patients with hydrocephalus (six of the 10 patients in this series), even when the visual pathways are clinically normal. It suggests that these abnormalities are probably due to compression of either the
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posterior visual pathway in the cerebral hemisphere contralateral to the stimulated side (in patients with homonymous half-field abnormalities) or the optic nerves (in patients with whole-field abnormalities) by the enlarged ventricles. It also suggests that VEP's can be of value, with other methods, in monitoring surgically treated hydrocephalic patients.

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References


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Address reprint requests to: Shakir M. Alani, M.B., Ch.B., M.R.C.P., Department of Clinical Neurophysiology, St. James University Hospital, Beckett Street, Leeds LS9 7TF, England.