Evaluation of Clinically Applied Visual Evoked Potential (VEP) in Ophthalmological and Neurological Diseases

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VEPs were recorded in 222 cases of different disease groups and in 42 cases of the control group using a Nicolet CA 1000 system. The latency time of N1, P1, N2, and P2 from the prominent surface peak and the P1-N2 amplitude at full field pattern reversal VEP. The score of each disease group was compared with those of the control group. The results are as follows:
1. Functional amblyopia, optic neuritis, and optic nerve atrophy patients presented a significant decrease in amplitude in comparison to normal subjects.
2. In the P1 implicit time, optic neuritis, and optic nerve atrophy patients presented a marked delay. Functional amblyopia patients presented a moderate delay while other disease group patients presented normal to mild delays.
3. More than half of the optic neuritis and optic nerve atrophy patients presented a detraction of the P1 wave form.

Key words: visual evoked potential, functional amblyopia, P1 implicit time, P1 wave, optic neuritis, optic nerve atrophy.

INTRODUCTION

Visual evoked potentials (VEPs) have been in use for many years in the objective evaluation of disorders of visual pathways and in the electrophysiological research of visual processing. The bio-electrical signals, obtained by presentation of a visual stimulus in the visual field center, reflect the status of the neural conduction in disease or under certain experimental conditions. The daily work in a clinic shows that finally a small number of questions only is crystallized out concerning problems where VEPs are of a real usefulness. The most important question is, when a patient is complaining of a blur, whether there is an optic neuropathy or not. A second important question is the differentiation between reduced visual acuity due to an organic disease and one due to an amblyopia. The third question concerns the possibility of visual acuity estimation with VEP when a patient is suspected of feigning reduced visual acuity. All three questions mentioned above turn around our efforts to affirm or to exclude an organic disease of the optic pathways, at least, between retinal center and

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The purpose of this study is to provide information about the applications of the VEPs in common optic neuropathies and in related problems. For a better understanding of different types of disease, we will try to compare objective findings (VEP) between each disease group and normal subjects.

MATERIALS

In the present study, we examined the VEPs of patients in 9 disease groups, diagnosed by the Department of Ophthalmology and Neurosurgery during the 14 months from May, 1984 to July, 1985 in Kyung Hee University Hospital. The pathological groups included functional amblyopia, optic neuritis, optic nerve atrophy, intracerebral hematoma, cerebral concussion, multiple sclerosis, space-occupying lesion, cerebral infarction, and post-traumatic sequelae.

The control group, aged between 10 to 45 years, had normal visual acuity and had no organic or psychological diseases.

METHODS

1) Stimulation

A checkerboard pattern on a 12 inch TV monitor was presented to the subjects at a distance of 1 m.

The check setting on the screen was 32 min x 32 min with each square having a visual angle of 23.5 minute.

The monocular stimulation of each eye was recorded in all subjects while overall luminance was kept constantly.

2) Recording

The subjects were comfortably seated in the same room and were asked to fix their eyes on the center of the TV monitor and to pay attention to the patterned stimulus.

The results were recorded for each eye by the same examiner. The VEP was recorded by placing the active electrode over 5 cm above inion along the midline, the inactive electrode over the forehead zero and the ground over the vertex.

The normal wave consists of an initial negative deflection, N₁ at 69.59±3.82, a positive deflection, P₁ at 92.02±4.09, a second negative wave, N₂ at 127.28±12.16 and a second positive wave, P₂ at 187.76±24.39 milliseconds.

The P₁-N₂ amplitude was 9.89±3.29 microvolts (Fig. 2).

2) Disease group

The abnormal findings are classified into 3 kinds; the first is delay of latency, the second is decrease of amplitude, and the third is destruction of P₁ wave.

As compared with each peak, implicit
time, optic neuritis and optic nerve atrophy patients showed a marked delay of $P_1$ (Table 1) (Fig. 3). As compared with the other groups, optic neuritis and optic nerve atrophy presented a significant decrease in amplitude (Fig. 4). $P_1$ destruction is that a $P_1$ wave is not formed definitely and over 50% of optic neuritis and optic nerve atrophy showed $P_1$ destruction (Table 2).

**DISCUSSION**

With the development of computers which efficiently perform averaging, the visually evoked potential has been used to examine the electrical activity of visual pathway abnormalities. Many researchers and clinicians have previously established a correlation between VEP and clinical diagnosis.

Recent studies have shown that VEP may be very valuable in measuring the visual acuity of nonverbal subjects, in determining refractive errors in the study of amblyopia, in the detection of visual defects in multiple sclerosis, optic neuritis, macular degeneration, and color blindness. Delayed VEP to pattern stimulation

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**Table 1. Comparison of $P_1$ prolongation**

<table>
<thead>
<tr>
<th>Clinical Dx</th>
<th>Total</th>
<th>$&gt;$X±1 SD</th>
<th>$&gt;$X±2 SD</th>
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<tbody>
<tr>
<td>Normal</td>
<td>42</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>12</td>
<td>1</td>
<td>8.3</td>
</tr>
<tr>
<td>Post-traumatic sequelae</td>
<td>54</td>
<td>17</td>
<td>31.5</td>
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<tr>
<td>Cerebral concussion</td>
<td>82</td>
<td>34</td>
<td>41.5</td>
</tr>
<tr>
<td>Space occupying lesion</td>
<td>28</td>
<td>17</td>
<td>60.7</td>
</tr>
<tr>
<td>Intracerebral hematoma</td>
<td>8</td>
<td>4</td>
<td>50</td>
</tr>
<tr>
<td>Cerebral infarction</td>
<td>18</td>
<td>14</td>
<td>77.8</td>
</tr>
<tr>
<td>Functional amblyopia</td>
<td>8</td>
<td>6</td>
<td>75</td>
</tr>
<tr>
<td>Optic neuritis</td>
<td>6</td>
<td>5</td>
<td>83.3</td>
</tr>
<tr>
<td>Optic nerve atrophy</td>
<td>6</td>
<td>6</td>
<td>100</td>
</tr>
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</table>

* Mean $P_1$ latency ±1 standard deviation

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**Table 2. Comparison of $P_1$ destruction**

<table>
<thead>
<tr>
<th>Clinical Dx</th>
<th>Total</th>
<th>No. of $P_1$ destruction</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>42</td>
<td>0</td>
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</tr>
<tr>
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<tr>
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<td>0</td>
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<tr>
<td>Cerebral concussion</td>
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<td>4.7</td>
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<tr>
<td>Post-traumatic sequelae</td>
<td>58</td>
<td>4</td>
<td>6.9</td>
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<tr>
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<td>9</td>
<td>1</td>
<td>11.1</td>
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<tr>
<td>Space occupying lesion</td>
<td>32</td>
<td>4</td>
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<td>Functional amblyopia</td>
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<td>27.3</td>
</tr>
<tr>
<td>Optic neuritis</td>
<td>13</td>
<td>7</td>
<td>53.8</td>
</tr>
<tr>
<td>Optic nerve atrophy</td>
<td>19</td>
<td>13</td>
<td>68.4</td>
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</tbody>
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was considered to be pathognomonic for demyelinating optic nerve disease.\textsuperscript{3,6} However, the latency of VEP will be a pathological increase in compression of the optic nerve,\textsuperscript{7} glaucoma,\textsuperscript{2} and retinal disease affecting various parts of the sensory retina.\textsuperscript{8} In addition, demyelination in the optic nerve caused by optic neuritis and multiple sclerosis shows an irreversible delay in latency.

In our study, we obtained similar results. Over 90% of the patients with optic neuritis displayed the delay in latency to the reversing checkerboard pattern. However, less than 10% presented the $P_1$ delay in the case of multiple sclerosis. Such inconsistency for multiple sclerosis may have been caused by a lack of accurate laboratory data. As multi-
ple sclerosis is very rare in Korea, it is difficult to perform the diagnosis by laboratory data. Thus VEP of the patients suspected to have multiple sclerosis by clinical signs has been measured. Therefore the results are different from the data in other studies. Halliday points out that over 90% of the patients with clinically definite multiple sclerosis have the delay even in the absence of clinical signs of optic nerve damage.

Changes in the case of amblyopia are easily demonstrated by reversing the checkerboard pattern, small squares, low reversal frequency, and low contrast. In the study 75% of the functional amblyopes showed P1 delay, and their mean amplitude was lower than the results in other studies.

VEP is elicited mainly from the central part of the visual field and therefore represents the function of the macular area. As an increase in latency to pattern stimuli may also occur in retinal disease, the delayed VEP is not necessarily related to nerve fiber damage. Accordingly, the dysfunction of retinal elements must be considered in the diagnosis using VEP.

Consequently a pathological VEP is a strong indication of an abnormality in the visual pathway, mainly the optic nerve, if retinal function is normal.

VEP is particularly sensitive to the focus of the image. The amplitude was maximum using the focused image in the experiment.

This study demonstrated how the change in the amplitude and latency of VEP are related to the optic nerve, and showed that VEP is useful in diagnosis and determination of treatment.

REFERENCES


