ELSEVIER

# Topographical Analysis of Motion-Triggered Visual-Evoked Potentials in Man

Yasushi Nakamura and Kenji Ohtsuka

*Department of Ophthalmology,*
*School of Medicine, Sapporo Medical University, Sapporo, Japan*

**Purpose:** The middle temporal (MT) area of the cortex of the monkey is involved in visual motion analysis. Previous studies using brain-imaging techniques have shown that the area around the anterior occipital cortex in man is homologous to the area MT of the monkey. In this study, we investigated the cortical components of motion-triggered visual evoked potentials and their topography in the visual cortex of man.

**Methods:** Visual evoked potentials to the onset of a visual motion stimulus (m-VEPs) were recorded from 5 normal subjects aged 25 to 34 years. A random dot pattern was used as the stimulus for the m-VEPs. The dots moved horizontally to the right and then to the left alternately for 500 milliseconds with interstimulus intervals of 1500 milliseconds. The speed of motion was varied in five steps from 5–25°/s. Fifteen electrodes were placed on the occiput around $O_z$ at 5-cm intervals. Color contour maps showing the distribution of voltage over the 15 electrodes at latencies ranging from 0–200 milliseconds with a 20-millisecond interval were made for each subject. These were coregistered with three-dimensional magnetic resonance images of the same brain to specify the topography of the main components of the m-VEPs in relation to the sulcal and gyral pattern of the visual cortex.

**Results:** We consistently observed a positive wave with a peak latency of about 100 milliseconds (P100) and a negative wave with a peak latency of about 150 milliseconds (N150) for all subjects. Topographical analysis showed that P100 was dominant in a relatively wide area caudal to $O_z$, whereas N150 was dominant in a relatively small area posterior to the right anterior occipital sulcus, which included the area corresponding to the area MT in man.

**Conclusions:** These findings suggest that N150 represents the activity of the area MT in the human visual cortex related to motion perception. **Jpn J Ophthalmol 1999;43:36–43** 

**Key Words:** Middle temporal area, motion-triggered VEP, three-dimensional MRI, visual motion perception.

## Introduction

Recent neurophysiological studies in the monkey have demonstrated the existence of two general information-processing streams in the primate visual cortex.[1–4] One pathway is considered to subserve exclusively form and color vision, and to lie ventrally and terminate in the temporal lobe (temporal stream). The other pathway is considered to be specialized exclusively for visual motion, and to lie dorsally and terminate in the parietal cortex (parietal stream). Neuroimaging studies of the human visual cortex revealed that the architecture of the human visual cortex is quite similar to that of the primate.[5–8] These streams in the visual cortex are thought to be cortical continuations of pathways that start in the retina. These pathways are known as the parvocellular (P) and the magnocellular (M) pathways, and they subserve form and color vision and visual motion, respectively.[9] The M pathway, which subserves visual motion, projects via layers 4b of the striate cortex (V1) to subdivisions of area V2 and to the middle temporal (MT) area in the superior temporal sulcus

Received: February 25, 1998

Address correspondence and reprint requests to: Kenji OHTSUKA, MD, PHD, Department of Ophthalmology, School of Medicine, Sapporo Medical University, S-1, W-16, Chuo-ku, Sapporo 060-8556, Japan



Published by Elsevier Science Inc.

0021-5155/99/$–see front matter
PII S0021-5155(98)00053-7

of the monkey.[1–4,9] Area MT is assumed to be an important neural region for visual motion perception.

Conventional visual-evoked potentials (VEPs) associated with the sudden reversal of the contrast of a pattern (p-VEPs) are assumed to represent the activity of the P pathway.[10–12] On the other hand, the VEPs associated with the onset of visual motion (m-VEPs) are considered to represent the activity of the M pathway.[12–15] The main element of m-VEPs appears to be a negative component with a peak latency of about 160–200 milliseconds.[12–15] This negative component of m-VEPs is thought to represent the activity of the parietal stream in the visual cortex. However, the topography of the main components of the m-VEPs on the human visual cortex is still not known.

In the present study, we examined the characteristics of the m-VEPs evoked by various stimulus speeds in normal subjects, and identified the main components representing the activity of motion processing in the human visual cortex. In addition, we specified the topography of the main components of m-VEPs in relation to the sulcal and gyral pattern of the cortex using three-dimensional magnetic resonance imaging (MRI) of the brain.

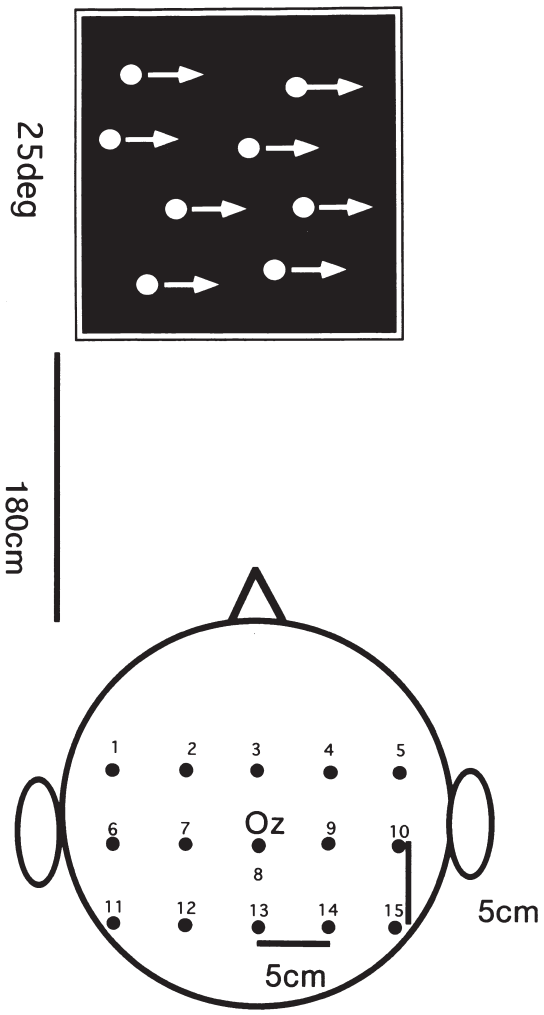


**Figure 1.** Schematic diagram of system for induction of motion-triggered visual evoked potentials. Array of 80 randomly located dots is projected by data projector onto stimulus field (25 × 25°) positioned 1.8 m in front of subject. Fifteen electrodes are placed on occiput around $O_z$ at 5-cm intervals. Electrodes are numbered from upper left to lower right. Linked earlobes served as reference electrode.

## Materials and Methods

### *Subjects*

M-VEPs were recorded from 5 normal subjects, 25–34 years of age (mean age = 28 years). All had a corrected Snellen visual acuity of at least 20/20 in both eyes. Informed consent was obtained from all subjects after the nature of the procedure was explained. Tenets of the Declaration of Helsinki were followed, and Institutional Human Experimentation Committee approval was obtained.

### *Visual Stimuli*

Visual stimuli were generated using a microcomputer. An array of 80 randomly located dots (0.75° in diameter, 21.0 cd/m$^2$, 97.7% contrast) was displayed in rapid succession (frame rate 70 Hz), and projected onto the stimulus field (25 × 25°) positioned 1.8 m in front of the subject by a data projector (SONY VPL-351QJ) (Figure 1). The dots moved to the right and to the left alternately with an interstimulus interval of 1500 milliseconds. A motion sequence consisted of the abrupt onset of motion, which continued for

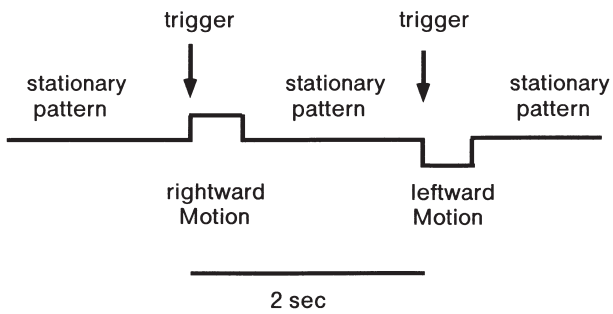


**Figure 2.** Visual stimulus paradigm. Motion sequence consisted of abrupt onset of motion continued for 500 milliseconds followed by abrupt offset and stationary phase of 1500 milliseconds; total time for one sequence was thus 2000 milliseconds.

Jpn J Ophthalmol
Vol 43: 36–43, 1999

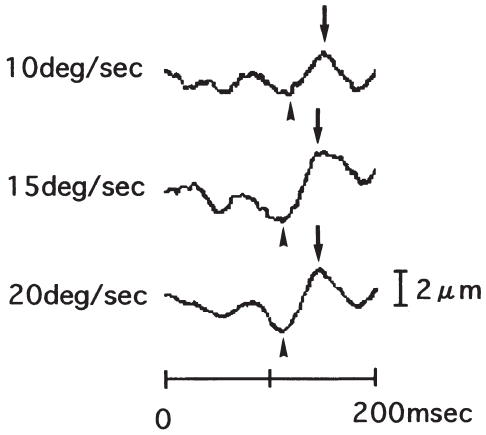


**Figure 3.** Example of m-VEPs induced by stimulus velocity of 10°/s (top), 15°/s (middle), and 20°/s (bottom). Upward deflections denote negative waves. Arrowhead and arrow indicate P100 and N150 waves, respectively.

500 milliseconds, followed by an abrupt offset and a stationary phase of 1500 milliseconds; the total time for one sequence was thus 2000 milliseconds (Figure 2). The speed of motion was varied in five steps (5, 10, 15, 20, and 25°/s). A laser dot fixation point (4 minutes of arc in diameter) was placed in the center of the stimulus field. Each subject was instructed to fixate on the dot during the experiment. Throughout the experiment, subjects were seated on a comfortable dental chair with neck support. All recordings were performed in a sound-attenuated, shielded chamber with a background luminance of 0.247 cd/m$^2$.

## *Recording*

Binocular m-VEPs were recorded from 15 unipolar leads (3 × 5) placed on the occiput around $O_z$ at 5-cm intervals, which covered an area of 10 × 20 cm on the occipital pole (Figure 1). Linked earlobes served as the reference electrode. A ground electrode was attached to the right wrist. The 15 electrodes were numbered from the upper left to the lower right. The onset of motion was used as a trigger for the recording. After amplification with a band-pass filter of 0.5–100 Hz, 128 motion sequences of 500-millisecond duration were averaged and digitized by a computer with a sampling rate of 2000 Hz.

## *Data Analysis*

The voltages of the m-VEPs from the 15 electrodes and the latencies of the major positive and negative peaks were analyzed on a microcomputer. Two-dimensional color contour maps were made for each subject, showing the distribution of voltage over the 10 × 20 cm area at latencies from the onset of motion ranging from 0 to 200 milliseconds with a 20-millisecond interval. The color contour maps consisted of 153 voxels (9 × 17 voxels). The voltage at each voxel was estimated from the voltages at the 15 electrodes with a computer (MS Excel® software and Macintosh computer 8500/132). Color corresponding to the voltage was allocated to each voxel by a computer (NIH image software and Macintosh computer 8500/132). The cortical area corresponding to each recording site was identified by a three-dimensional MRI of the visual cortex of each subject. $O_z$ and the four corners of the recording sites were marked to produce high signal intensity on $T_1$-weighted MRI. This method enabled us to coregister the color contour maps with the three-dimensional MRI maps of the visual cortex. Three-dimensional images of three different views (left posterior, posterior, and right posterior views) of the cortex were reconstructed from axial MRI scans (1-mm thick) for each subject. The color contour map was finally transformed to a three-dimensional one along the surface configuration of the three-dimensional MRI of the visual cortex using a computer, and coregistered with the three-dimensional MRI for each subject using five markers (four corners of the recording sites and $O_z$) on the MRI as landmarks. Using this method, we specified the localization of the main components of

**Table 1.** Peak Latencies of P100 and N150

| Velocity of Motion (°/s) | Subject 1 Latency (ms) | | Subject 2 Latency (ms) | | Subject 3 Latency (ms) | | Subject 4 Latency (ms) | | Subject 5 Latency (ms) | | Total Mean ± SD (ms) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | P100 | N150 | P100 | N150 | P100 | N150 | P100 | N150 | P100 | N150 | P100 | N150 |
| 5 | 119 | | 128 | | 107 | | 110 | | 105 | | 113 ± 9 | |
| 10 | 115 | 154 | 133 | 167 | 106 | 148 | 114 | 187 | 101 | 160 | 113 ±12 | 163 ± 15 |
| 15 | 117 | 151 | 120 | 151 | 106 | 162 | 112 | 142 | 108 | 145 | 112 ± 5 | 150 ± 7 |
| 20 | 113 | 146 | 102 | 152 | 105 | 140 | 110 | 162 | 106 | 135 | 107 ± 4 | 147 ± 10 |
| 25 | 105 | 144 | 82 | 155 | 106 | 141 | 110 | 156 | 97 | 130 | 100 ± 11 | 145 ± 10 |

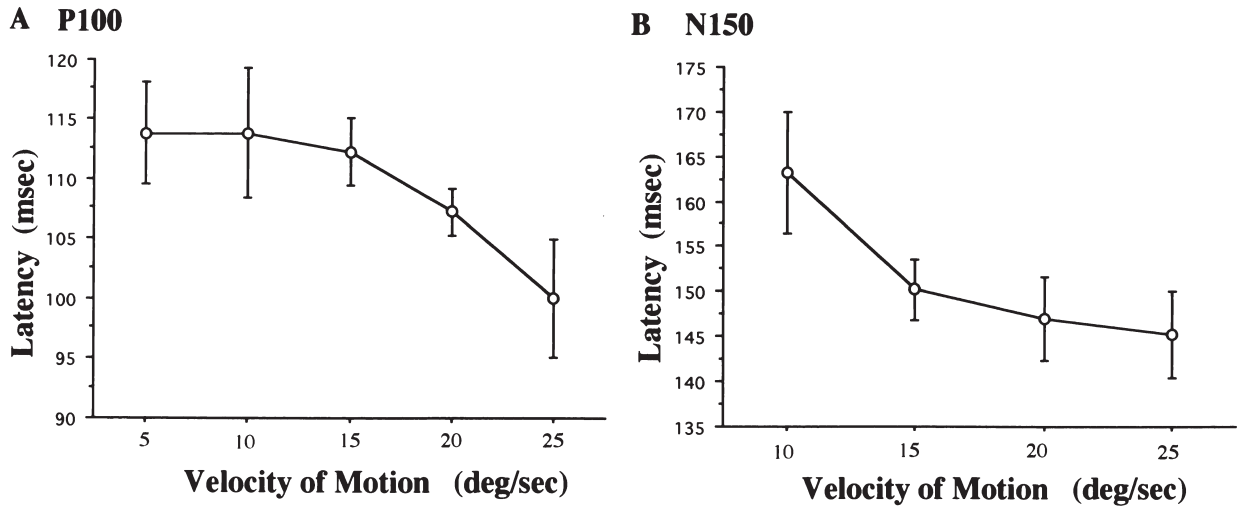


**Figure 4.** Relationship between peak latencies of P100 at $O_z$ and N150 at electrode 9 and velocity of motion. Each point represents mean value for 5 subjects, and error bars are standard deviation.

m-VEPs in relation to the sulcal and gyral pattern of the human visual cortex.

## Results

### *Peak Latencies and Amplitudes of m-VEPs*

The pattern of m-VEPs was essentially the same for the 5 subjects, and was dependent upon the velocity of motion. We observed a positive wave with a peak latency ranging from 82–128 milliseconds (P100) and a negative wave with a peak latency ranging from 130–187 milliseconds (N150) for all subjects (Figure 3). The mean latencies of P100 and N150 for the 5 subjects for a stimulus velocity of 25°/s were 100 ± 11 milliseconds (mean ± SD) and 145 ± 10 milliseconds, respectively. The mean difference in the peak latency between P100 and N150 for the 5 subjects was 43.0 milliseconds (SD = 13.4 milliseconds). Table 1 shows the latencies of P100 at $O_z$ and the latencies of N150 at electrode 9, which was located 5 cm to the right of $O_z$, for the 5 subjects. Figure 4 shows the relationships between the peak latencies of P100 and N150 and the velocity of motion. The latencies of P100 and N150 decreased as the velocity of motion increased. Table 2 shows the amplitudes of P100 at $O_z$ and amplitudes of N150 at electrode 9 in the 5 subjects. Figure 5 shows the relationships between P100 and N150 amplitudes and the velocity of motion. The amplitudes of P100 and N150 increased as the velocity of motion increased. N150 was dominant at stimulus velocity of 25°/s and relatively small at other stimulus velocities. N150 could not be observed at a stimulus velocity of 5°/s, whereas P100 was observed at 5°/s.

### *Topography of m-VEPs*

Figure 6 shows 15 traces of m-VEPs at each of the 15 electrodes at a stimulus velocity of 20°/s. P100 was

**Table 2.** Amplitudes of P100 and N150

| Velocity of Motion (°/s) | Subject 1 Amplitude (μV) | | Subject 2 Amplitude (μV) | | Subject 3 Amplitude (μV) | | Subject 4 Amplitude (μV) | | Subject 5 Amplitude (μV) | | Total Mean ± SD (μV) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | P100 | N150 | P100 | N150 | P100 | N150 | P100 | N150 | P100 | N150 | N150 | N150 |
| 5 | 1.26 | | 1.10 | | 1.05 | | 4.25 | | 3.75 | | 2.2 ± 1.5 | |
| 10 | 0.85 | 1.37 | 3.10 | 4.00 | 3.00 | 3.50 | 3.25 | 1.50 | 3.63 | 5.13 | 2.7 ± 1.0 | 3.1 ± 1.6 |
| 15 | 1.10 | 1.69 | 2.80 | 4.10 | 1.75 | 2.40 | 4.50 | 3.80 | 7.25 | 4.63 | 3.4 ± 2.4 | 3.3 ± 1.2 |
| 20 | 2.27 | 1.79 | 3.25 | 4.25 | 6.40 | 6.00 | 3.85 | 2.90 | 4.00 | 3.50 | 3.9 ± 1.5 | 3.6 ± 1.5 |
| 25 | 0.20 | 2.52 | 4.05 | 5.00 | 5.05 | 6.25 | 5.60 | 2.75 | 6.25 | 6.85 | 4.2 ± 2.3 | 4.6 ± 1.9 |

Jpn J Ophthalmol
Vol 43: 36–43, 1999

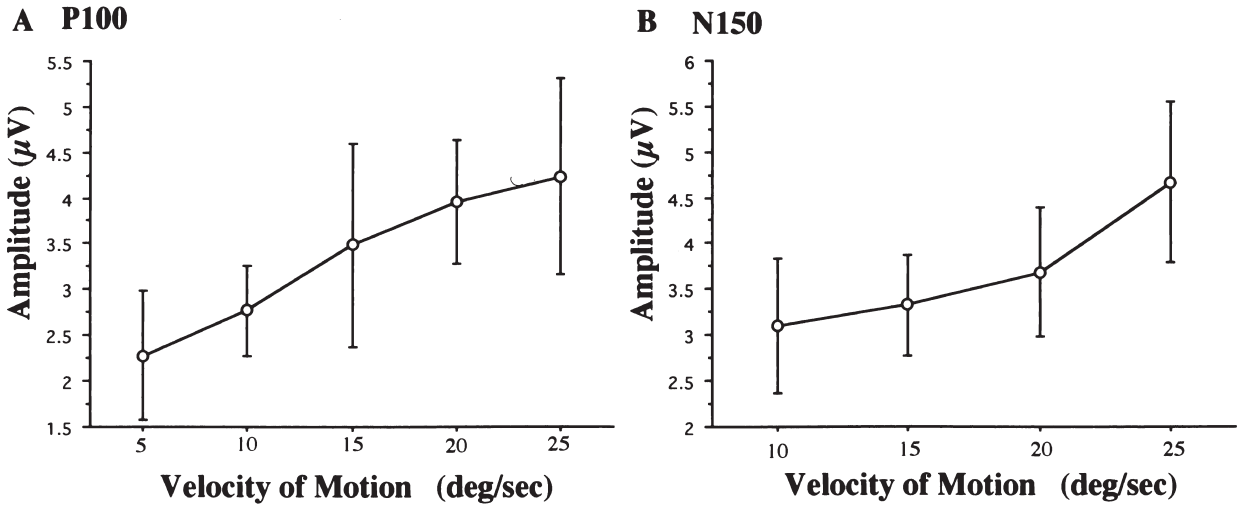


**Figure 5.** Relationship between amplitudes of P100 at $O_z$ and N150 at electrode 9 and velocity of motion. Each point represents mean value for 5 subjects, and error bars are standard deviation.

dominant in the area caudal to $O_z$ (electrodes 7, 8, 9, 12, 13, and 14). On the other hand, N150 was dominant in the area to the right of $O_z$ (electrodes, 8, 9, and 10). Figure 7 shows two-dimensional color contour maps indicating the distributions of voltage over the 15 electrodes at 6 different latencies from the onset of motion for a stimulus velocity of 25°/s. P100 was dominant in the relatively broad area caudal to $O_z$ (red area on the color contour map with 100 milliseconds latency). On the other hand, N150 was dominant in the relatively small area to the right of $O_z$ (purple area on the color contour map with 140 or 160 milliseconds latency). Figure 8 shows the three-dimensional MRI for three different views (left posterior, posterior, and right posterior views) of the cortex in one subject (upper panel), and a color contour map at a latency of 140 milliseconds superimposed on the surface configuration of the three-dimensional image of the cortex of the posterior view. The area where N150 (the purple area) was dominant was the area extending from the posterior pole of the striate cortex to the posterior portion of the middle temporal gyrus in the right cortex. The center of the N150-dominant area corresponded to the area posterior to the anterior occipital sulcus. N150 was not dominant in the left visual cortex. The results of the topographical analysis of P100 and N150 were essentially the same for the 5 subjects.

## Discussion

The results of the present study suggest that P100 and N150 are the principal components of the m-VEPs. Previous studies also reported a positive component of the m-VEPs with a peak latency ranging from 100–120 milliseconds and a negative component with a peak latency ranging from 160–200

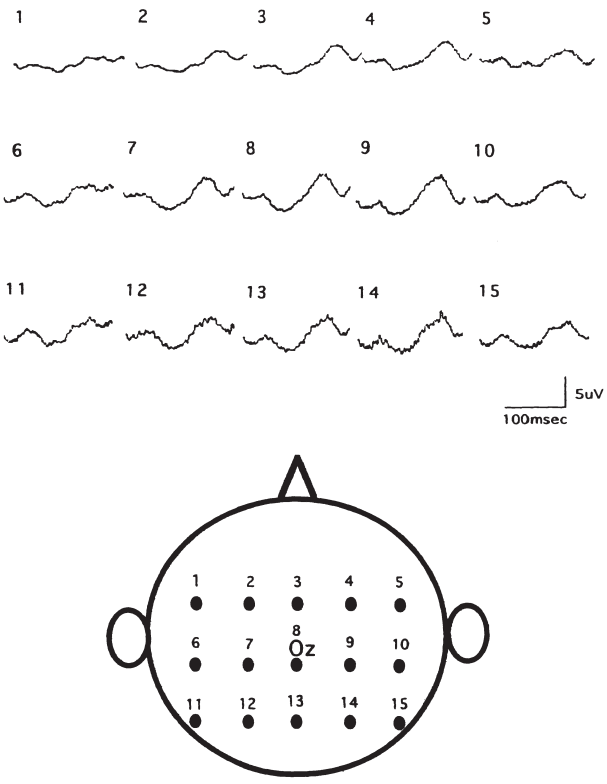


**Figure 6.** Examples of m-VEPs from 15 electrodes in one subject. Upward deflections denote negative waves.

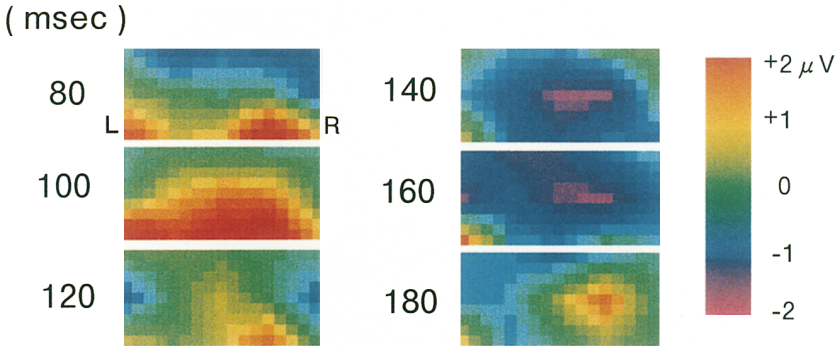


**Figure 7.** Two-dimensional color contour maps showing distribution of voltages over 15 electrodes at 6 different latencies from onset of motion at stimulus velocity of 25°/s. Each map consists of 153 voxels (9 × 17 voxels) having different colors corresponding to voltage at each voxel. Color code indicates voltage calibration.

milliseconds.[12–15] These latencies were relatively longer than those obtained in the present study. The stimulus velocities in the previous studies were relatively slower than those in the present study, which probably explains the discrepancy; our results showed that the peak latencies of the main components of m-VEP are longer with lower stimulus velocities. Thus, the differences in peak latencies between the present and previous studies are probably due to the differences in stimulus velocity.

A previous study using positron emission tomography and MRI demonstrated that the area homologous to the area MT of the monkey is situated ventrolaterally, just posterior to the meeting point of the ascending limb of the inferior temporal sulcus and the anterior occipital sulcus.[6] This region occupies the temporo-parieto-occipital pit, at the boundary of Brodmann's areas 19 and 37. Lesions located in this area have resulted in visual motion blindness (akinetopsia).[16–18]

The results of our topographical analysis of the human visual cortex in the present study indicated that the area where N150 was dominant extended from the posterior pole of the occipital cortex to the posterior portion of the middle temporal gyrus, and that the central portion of the N150-dominant area corresponded to the area posterior to the right anterior occipital sulcus. These findings suggest that the

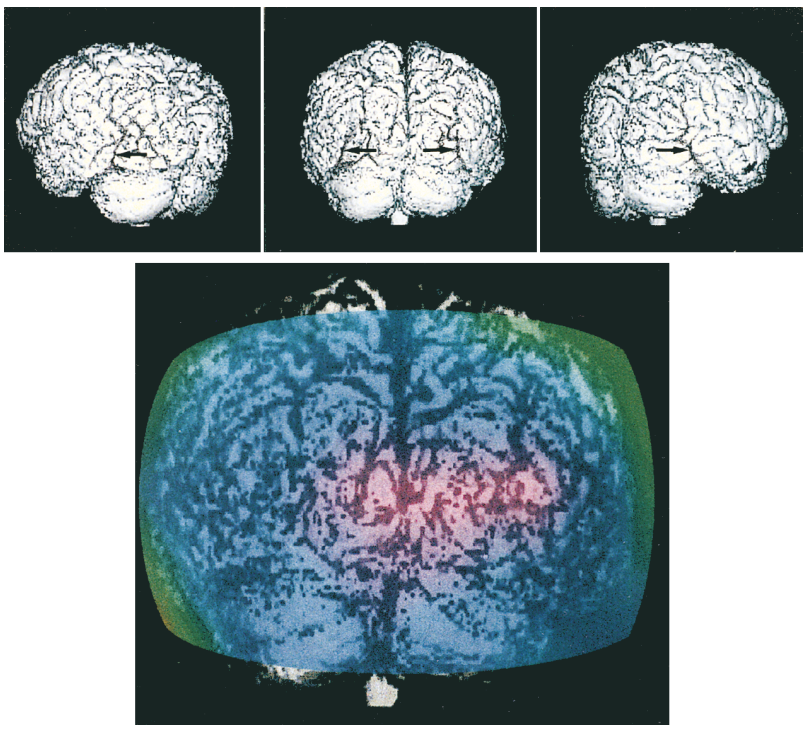

**Figure 8.** Three-dimensional reconstruction of cerebral cortex from magnetic resonance imaging scan (upper panel), and color contour map at latency of 140 milliseconds superimposed on surface configuration of three-dimensional image of cortex posterior view (lower panel). Upper left: view from left 40° to midline. Upper middle: posterior view. Upper right: view from right 40° to midline. Arrows indicate anterior occipital sulcus.

Jpn J Ophthalmol
Vol 43: 36–43, 1999

N150 component of the m-VEPs resulted from the activity of the area MT in the human visual cortex. However, the area where N150 was dominant in the present study extended to the posterior pole of the occipital cortex. Several studies have reported motion-selective activity located superior and posterior to area MT.[6,18] This area in the human visual cortex might correspond to area V3 of the monkey, where approximately half the cells are motion- and direction-selective.[19] Shipp et al[18] demonstrated that area V3 is related to residual motion perception in a patient with bilateral lesions of the area MT. Therefore, it is possible that N150 also represents the activity of area V3.

N150 was not dominant in the left cortex. This is assumed to be the result of the relative dominance of the right cortex for visuospatial functions,[20,21] although it is not known whether the right cortex is dominant in motion perception. A previous study[22] reported that visual deficits of motion interpretation and stereopsis are associated with right occipital-temporal lesions in the human brain. Recently, we reported that motion perception was severely damaged in a patient with an infarction of the right cortex around the anterior occipital sulcus.[23] These findings suggest that the area MT in the human right cortex is relatively dominant in motion perception.

On the other hand, the results of our topographical analysis indicated that P100 was dominant in the broad area caudal to $O_z$. A previous study suggested that a component of m-VEPs with a peak latency around 100 milliseconds reflects the activity of the striate cortex.[24] A positive component with a similar latency is also observed for p-VEPs, and its dipole source is localized in the striate cortex.[25] The 100-millisecond component of p-VEPs represents the activity in the striate cortex evoked by neuronal signals projected through the optic radiation. It is possible that P100 of m-VEPs represents the activity in the striate cortex evoked by neuronal signals projected through the M pathway. The M pathway subserving visual motion projects to area MT via the striate cortex.[1–4,9] The mean difference in the peak latency between P100 and N150 was 43 milliseconds. A previous study suggested the existence of parallel visual motion inputs into the striate cortex and area MT, a sequential input to area MT through the striate cortex, and a fast parallel input to area MT that bypasses the striate cortex.[26] The latency of the signal arrival in area MT through the fast parallel input is estimated to be about 35 milliseconds.[26] This value is markedly faster than the latency of N150. These findings suggest that N150 represents the activity of the area MT, which could be elicited by the sequential input through the striate cortex.

The relationships between the amplitude of m-VEPs and the stimulus velocity in the present study suggest that the preferred stimulus velocities for evoking N150 are faster than those for evoking P100. Previous neurophysiological studies indicated that area MT of the monkey contains motion-sensitive neurons and the distribution of preferred stimulus velocities had a peak close to 32°/s, and for most neurons in the MT the optimum stimulus velocities were in the range of 10–50°/s.[1,27–30] In the primary visual cortex, the preferred stimulus velocity of motion-sensitive neurons is relatively slower than that of the neurons in area MT, and range from 9–30°/s.[31,32] These neurophysiological findings support the assumption that P100 and N150 reflect the activity of the primary visual cortex and the area MT, respectively. The distribution of the preferred stimulus velocities of motion-sensitive neurons in the primary visual cortex or area MT are correlated with the speed characteristics of P100 or N150.

The authors thank Prof. Takashi Nakagawa for his valuable comments on this manuscript and Dr. Hiroshi Maekawa for technical assistance. This work was partially supported by a Grant-in-Aid for Scientific Research from the Japanese Ministry of Education, Science, Sports and Culture (No. 08672027).

## References

1. Zeki S. Functional organization of a visual area in the posterior bank of the superior temporal sulcus of the rhesus monkey. J Physiol Lond 1974;236:549–73.
2. Maunsell JH, Newsome WT. Visual processing in monkey extrastriate cortex. Ann Rev Neurosci 1987;10:363–401.
3. Van Essen DC, Anderson CH, Felleman DJ. Information processing in the primate visual system: an integrated systems perspective. Science 1992;255:419–23.
4. Merigan WH, Maunsell JH. How parallel are the primate visual pathways? Ann Rev Neurosci 1993;16:369–402.
5. Zeki S, Watson JD, Lueck CJ, Friston KJ, Kennard C, Frackowiak RS. A direct demonstration of functional specialization in human visual cortex. J Neurosci 1991;11:641–9.
6. Watson JD, Myers R, Frackowiak RD, et al. Area V5 of the human brain: evidence from a combined study using positron emission tomography and magnetic resonance imaging. Cereb Cortex 1993;3:79–94.
7. Tootell RB, Reppas JB, Kwong KK, et al. Functional analysis of human MT and related visual cortical areas using magnetic resonance imaging. J Neurosci 1995;15:3215–30.
8. Tootell RB, Dale AM, Sereno MI, Malach R. New images from human visual cortex. Trends Neurosci 1996;19:481–9.
9. Livingstone MS, Hubel DH. Psychological evidence for separate channels for the perception of form, color, movement, and depth. J Neurosci 1987;7:3416–68.
10. Arden GB, Barnard WM. Effect of occlusion on the visual

evoked response in amblyopia. Trans Ophthalmol Soc 1979; 99:419–26.

11. Wanger P, Persson HE. Visual evoked responses to pattern-reversal stimulation in childhood amblyopia. Acta Ophthalmol 1980;58:697–706.
12. Kubova Z, Kuba M, Juran J, Blakemore C. Is the motion system relatively spared in amblyopia? Evidence from cortical evoked responses. Vision Res 1996;36:181–90.
13. Kuba M, Kubova M. Visual evoked potentials specific for motion-onset. Doc Ophthalmol 1992;80:83–9.
14. Bach M, Ullrich D. Motion adaptation governs the shape of motion-evoked cortical potentials. Vision Res 1994;34:1541–7.
15. Kubova Z, Kuba M, Spekreijse H, Blakemore C. Contrast dependence of motion-onset and pattern-reversal evoked potentials. Vision Res 1995;35:197–205.
16. Thurston SE, Leigh RJ, Crawford T, Thompson A, Kennard C. Two distinct deficits of visual tracking caused by unilateral lesions of cerebral cortex in humans. Ann Neurol 1988; 23:266–73.
17. Zeki S. Cerebral akinetopsia (visual motion blindness). Brain 1991;114:811–24.
18. Shipp S, de Jong BM, Zihl J, Frackowiak RS, Zeki S. The brain activity related to residual motion vision in a patient with bilateral lesions of V5. Brain 1991;117:1023–38.
19. Felleman DJ, Van Essen DC. Receptive field properties of neurons in area V3 of macaque monkey extrastriate cortex. J Neurophysiol 1987;57:889–920.
20. Mesulam MM. A cortical network for directed attention and unilateral neglect. Ann Neurol 1981;10:309–25.
21. Weintraub S, Mesulam MM. Right cerebral dominance in spatial attention. Further evidence based on ipsilateral neglect. Arch Neurol 1987;44:621–5.
22. Vania LM. Selective impairment of visual motion interpretation following lesions of the right occipito-parietal area in humans. Biol Cybern 1989;61:347–59.
23. Konno S, Nakamura Y, Ohtsuka K, Nakagawa T. Disturbance of visual motion perception after right occipitotemporal infarction. Neuro-Ophthalmol 1996;16:S294.
24. Probst T, Plendl H, Paulus W, Wist ER, Scherg M. Identification of the visual motion area (area V5) in the human brain by dipole source analysis. Exp Brain Res 1993;93:345–51.
25. Maier J, Dagnelie G, Spekreijse H, van Dijk BW. Principal components analysis for source localization of VEPs in man. Vision Res 1987;27:165–77.
26. Ffytche DH, Guy CN, Zeki S. The parallel visual motion inputs into areas V1 and V5 of human cerebral cortex. Brain 1995;118:1375–94.
27. Maunsell JH, van Essen DC. Functional properties of neurons in middle temporal visual area of the macaque monkey. I. Selectivity for stimulus direction, speed, and orientation. J Neurophysiol 1983;49:1127–47.
28. Mikami A, Newsome WT, Wurtz RH. Motion selectivity in macaque visual cortex. I. Mechanisms of direction and speed selectivity in extrastriate area MT. J Neurophysiol 1986; 55:1308–27.
29. Rodman HR, Albright TD. Coding of visual stimulus velocity in area MT of the macaque. Vision Res 1987;27:2035–48.
30. Lagae L, Raiguel S, Orban GA. Speed and direction selectivity of macaque middle temporal neurons. J Neurophysiol 1993;59:19–39.
31. Orban GA, Kennedy H, Bullier J. Velocity sensitivity and direction selectivity of neurons in areas V1 and V2 of the monkey: influence of eccentricity. J Neurophysiol 1986;56:462–80.
32. Newsome WT, Mikami A, Wurtz RH. Motion selectivity in macaque visual cortex. III. Psychophysics and physiology of apparent motion. J Neurophysiol 1986;55:1340–51.