# Modification of the pattern-evoked magnetic fields associated with the location of the lesion along the visual pathways

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## 1 Introduction

Lesions along the visual pathways cause characteristic visual field deficits: Lesions affecting the optic nerve from the retina to the optic chiasm cause total loss of vision of the unilateral eye; compressive lesions of the optic chiasm cause loss of vision in the temporal halves of the bilateral visual fields (bitemporal hemianopsia); and lesions affecting the optic tract, the lateral geniculate body, or the optic radiation to the primary visual cortex cause loss of vision in the temporal half of the contralateral visual field (homonymous hemianopsia). The anterior part of the visual pathway, ending at the optic chiasm where neurons cross the hemisphere, transfers information from the temporal half visual fields of each eye. In contrast, the posterior visual pathway starting from the optic tract in each hemisphere of the brain is concerned completely with the contralateral temporal half of the visual field. Such anatomical characteristics provide clinically valuable information about the location of disturbances along the visual pathways even in a simple perimetry examination. The evoked potential method provides more sensitive results and can detect even subclinical lesions affecting the optic nerve such as optic neuritis, multiple sclerosis [1], or pituitary adenoma [2]. Pattern reversal is the most frequently used visual stimulation and using lateral half visual field stimulation, separate hemispheric responses can be identified more accurately. However, there is no clear clinical indication except for laterality for the posterior visual pathways. Visual evoked magnetic fields (VEFs) were investigated in case of representative lesions of the anterior and posterior visual pathways to find a more detailed relationship between the location of the lesion and the evoked responses.

## 2 Methods

### 2.1 Subjects

*Case 1: lesion of the anterior visual pathway*
A 60-year-old man complained of headache one month before the examination. Magnetic resonance imaging (MRI) identified a compressive lesion of the optic chiasm. His visual acuity and visual fields were normal. Biopsy was performed and the diagnosis was pituitary adenoma.

*Case 2: lesion of the posterior visual pathway*
A 14-year-old boy presented with automatism and complex partial seizures beginning when he was 8 years old. MRI found dysembryoplastic neuroepithelial tumor (DNT). He had undergone left parietal lobe lesionectomy at age 12 years. His visual acuity was 20/20 in each eye without correction. Humphery perimetry showed right lower quadrantanopsia.
Anatomical MRI was performed with a 1.5-T superconducting magnet device (Signa Advantage, GE Medical System). MR images were used to define the 'best fit sphere' necessary for source analysis, as well as combining the functional VEFs source localization with the brain anatomy of each subject.

### 2.2 Stimuli

Visual stimulation consisted of reversal of a green-black checkerboard pattern every 0.5 s. Individual checkerboard squares subtended 0.9° of arc at the subject's eye and the total stimulated field was 9°×9°. The brightness of the green squares was adjusted to 22 cd/m$^2$. The fixation point was 0.9° lateral to the center of the pattern edges, and left or right half visual field stimulations were presented monocularly. Recordings were made in a darkened magnetically shielded room and the contrast of the green and black squares was greater than 99%. Each trial was repeated at least twice to confirm reproducibility.

### 2.3 Magnetometry and source analysis

Magnetic fields evoked from the brain were measured by a 122 channel planar gradiometer MEG system (Neuromag 122, Neuromag, Ltd.) or a 306 channel MEG system consisting of a 204 channel planar gradiometer and a 102 channel axial magnetometer (Vectorview, Neuromag, Ltd). Sampling frequency and bandwidth were 1.25 kHz and 0.03 to 410 Hz for Neuromag 122 and 1 kHz and 0.1 to 333 Hz for Vectorview. Two hundred responses at a latency of -50 to 400 ms were averaged and recorded for each trial. Further processing of the recorded data used a digital bandpass filter of 2 to 40 Hz.

N75, P100, and N145 waves were identified visually in each evoked magnetic response waveform, and then confirmed by the iso-magnetic field contour map pattern at each latency.

Sources of VEFs were estimated by the single equivalent current dipole model based on a sphere conductor defined for each subject based on MRI. All sensor channels, except for noisy ones caused by non-biomagnetic sources, were used for source analysis. Sources were located on the MR images of each subject and compared to the anatomical structures of the brain.

## 3 Results

### 3.1 Lesion of the anterior visual pathway

Figure 1 shows the visual evoked responses for the left eye in Case 1. Table 1 summarizes latencies for both eyes. All N75-P100-N145 waves were uniformly delayed. Latencies in temporal half visual field stimulation were delayed more than those in nasal visual field stimulation.

Table 1: *Case 1. Latencies of the N75-P100-N145 waves. LH: left half visual field, RH: right half visual field.*

| Eye | Stim. | N75 | P100 (ms) | N145 |
|---|---|---|---|---|
| L | LH | 130 | 176 | 226 |
| L | RH | 107 | 151 | 205 |
| R | LH | 102 | 169 | 219 |
| R | RH | 117 | 185 | 240 |

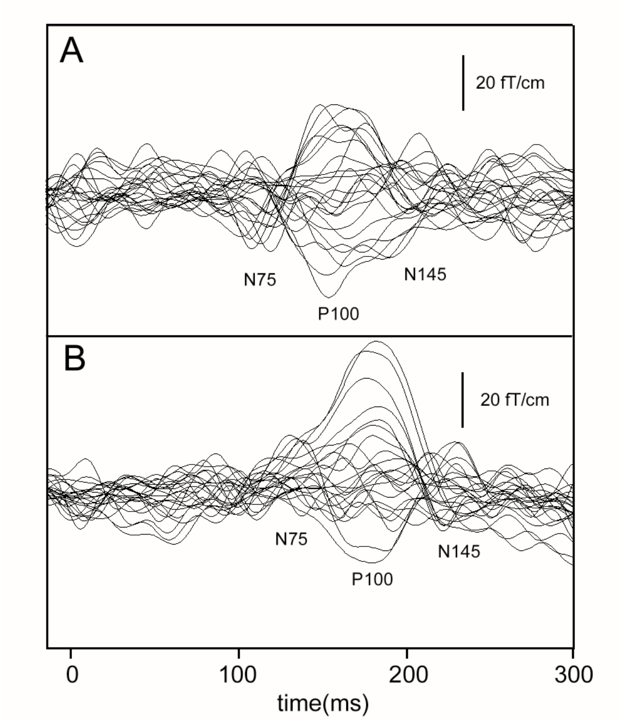


Figure 1: *Case 1. Visual evoked magnetic fields for A) right half, and B) left half visual field stimulation of the left eye.*

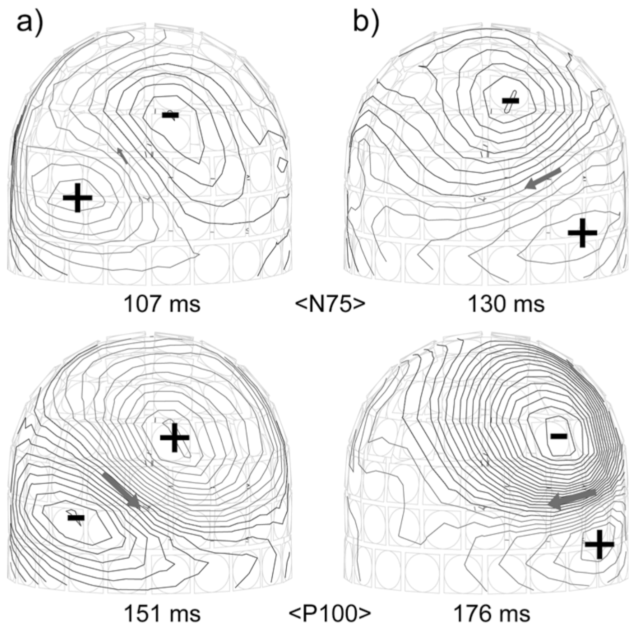


Figure 2: *Case 1. N75 and P100 iso-magnetic field contour maps at the peak latencies viewed from the rear of the subject for a) right half, and b) left half visual field stimulation of the left eye. 10 fT/cm step.*

Figure 2 shows iso-magnetic field contour maps at the peak latency of the N75 and P100 waves. Polarity of the N75 map in the left half visual field stimulation was reversed by the overlapping of the P100 wave.
Sources of the P100 wave were localized at the base of the calcarine fissure at the latencies shown in Figure 3. The P100 locations were shifted laterally if localized at the peak latency. P100 sources shown in Figure 3 were estimated at about 20 ms earlier to the peak latency.

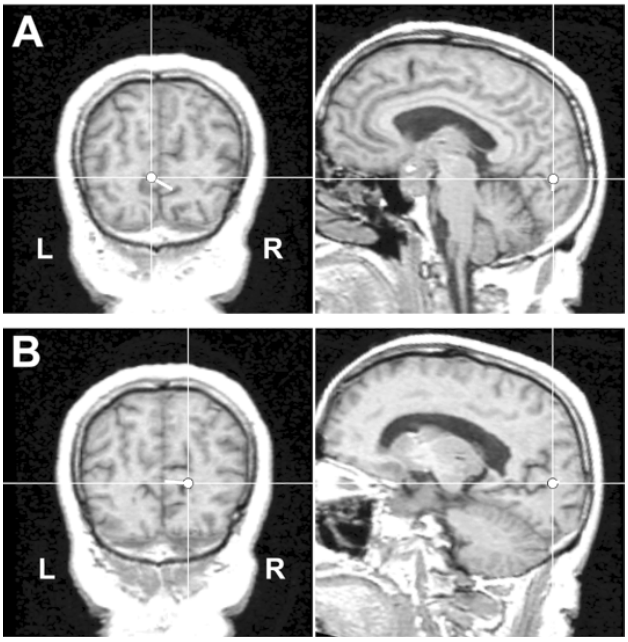


Figure 3: *Case 1. Estimated P100 dipoles for A) right half visual field stimulation at 134 ms, and B) left half visual field stimulation at 157 ms of the left eye.*

### 3.2 Lesion of the posterior visual pathway

Figure 4 shows the visual evoked responses for the right eye in Case 2. Table 2 summarizes latencies for both eyes. The P100 wave in the lesion hemisphere was markedly attenuated and delayed, whereas the N75 and N145 waves were almost unchanged.
Figure 5 shows the iso-magnetic field contour maps for the N75-P100-N145 waves. The dipole pattern of the early P100 wave in the lesion hemisphere was relatively weak and the N145 component in both hemispheres was not clear because of the overlapping of the P100 wave. The delayed P100 component was observed in the lesion hemisphere at a letency of about 200 ms, whereas the late N145 component was visible in the normal hemisphere.

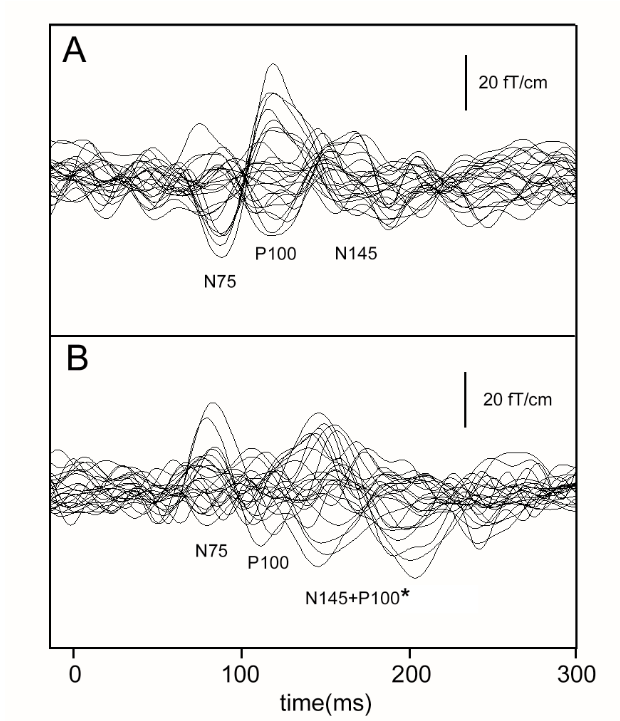


Figure 4: *Case 2.Visual evoked responses for A) left half, and B) right half visual field stimulation of the right eye. P100* indicates delayed P100 component.*

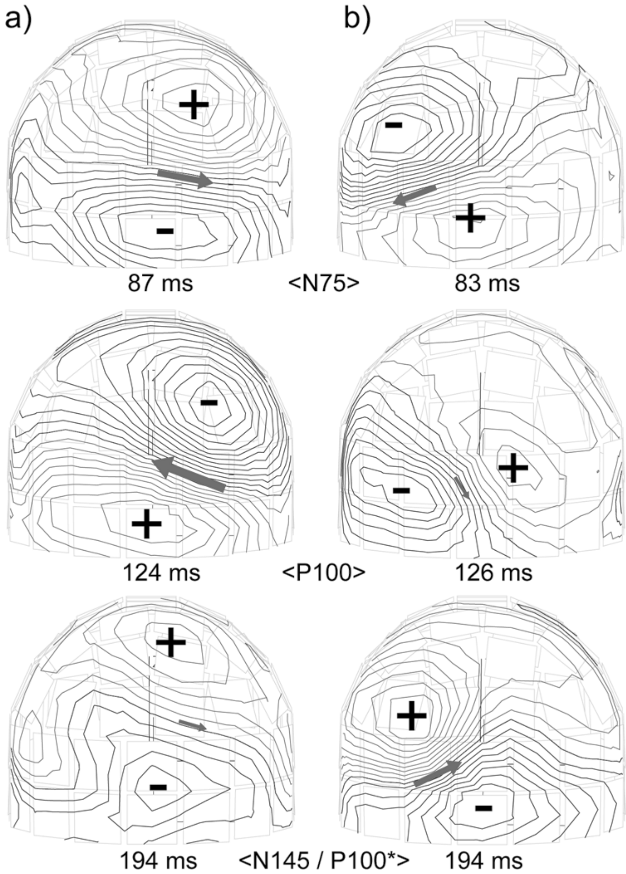


Figure 5: *Case 2. Iso-magnetic field contour maps for a) left half, and b) right half visual field stimulation of the right eye. 10 fT/cm step.*

Table 2: *Case 2. Latencies of the N75-P100-N145 waves. P100* indicates delayed component in the lesion hemisphere. LH: left half visual field, RH: right half visual field.*

| Eye | Stim. | N75 | P100 | P100* | N145 |
|---|---|---|---|---|---|
| | | (ms) | | | |
| L | LH | 74 | 114 | — | 147 |
| L | RH | 82 | 117 | 194 | 145 |
| R | LH | 87 | 118 | — | 155 |
| R | RH | 83 | 110 | 200 | 147 |

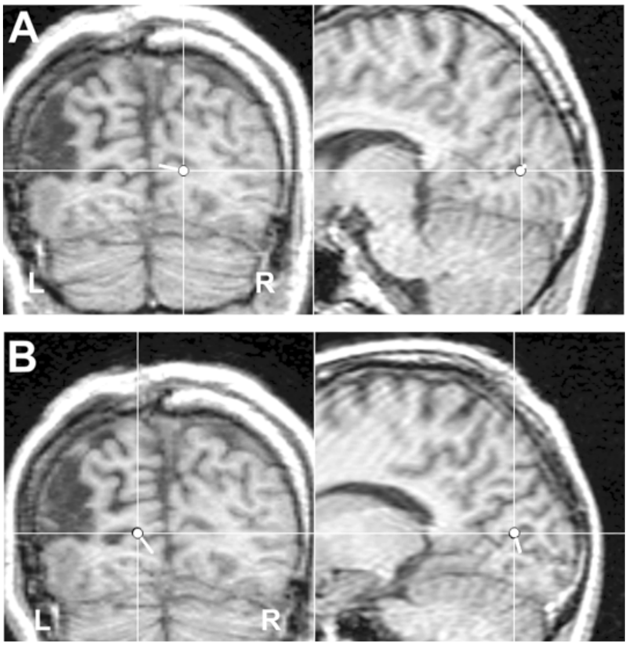


Figure 6: *Case 2. Estimated P100 dipoles for A) left half visual field stimulation at 124 ms, and B) right half visual field stimulation at 126 ms of the right eye.*

Sources of the P100 wave were localized at the base of the calcarine fissure in both normal and lesion hemispheres (Figure 6).

## 4 Discussion

The P100 wave was significantly attenuated and delayed in the lesion hemisphere in Case 2, whereas all components were uniformly delayed in Case 1. Sources of the P100 waves were localized at the base of the calcarine fissure regardless of the location of the lesion around the visual pathways. This result agrees with previous source localizations carried out for normal subjects [3, 4].
Our finding indicates that all components of the visual signal are transferred through the same anterior visual pathway. Therefore, any lesion affecting the anterior visual pathway causes uniform delay of the N75-P100-N145 waves.
In contrast, the components are transferred through different posterior pathways after arriving at the primary visual cortex (V1) [5]: P100 wave takes the dorsal pathway, and N75 and N145 waves take the ventral pathway. These waves are then back-projected to V1 after processing in the higher visual cortices and generate P100 and N75 and N145 waves at V1. This model can explain the drastic delay of the P100 latency in the lesion hemisphere and the minimal modification of the N75 and N145 waves in Case 2.
Modifications of the N75-P100-N145 components of the pattern-evoked magnetic fields are quite useful to evaluate lesions around the posterior visual pathways.

## References

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