

Pre-Surgical Mapping Of Visual Hemifield Response By MEG Prior To Tumor Resection: Outcome Study

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ABSTRACT

Visual evoked cortical magnetic field (VEF) waveforms were recorded from both hemifields in 20 patients with visual symptoms arising from parieto-occipital mass lesions, to identify preserved visual pathways. MEG VEF mapping was successful in 15/20 patients. Functional, though displaced or abnormal, responses were seen in 11/20 patients with disruption of pathway in 1/20. In summary, 3/20 patients (15%) had improved outcome either secondary to change in the surgical approach or the planned procedure, based on the MEG findings.

KEY WORDS Visual Evoked Response, Visual Evoked Field (VEF), MEG, MR-FOCUSS.

INTRODUCTION

The planning of treatment for structural lesions of the brain is facilitated by knowledge of the function of surrounding brain tissue. Mapping of function preoperatively helps to choose a surgical route to minimize neurologic deficits and define the limits of lesion resectability [Rezia 1997; Roberts 1995]. It also helps in preoperative counseling of the patient regarding the inherent risks. Direct cortical stimulation has been the gold standard for many years [Ganslandt, 1999], but increasingly noninvasive preoperative mapping has been used. Magnetoencephalography (MEG) has emerged as a powerful tool for noninvasive identification of eloquent cortex prior to planned neurosurgical operations [Gallen 1995]. MEG has been validated by several groups for preoperative localizations of sensorimotor cortex [Ganslandt, 1999], visual cortex [Harding, 1992], and language cortex [Papanicolaou, 1999].

We report an outcome study of 20 patients with parieto-occipital mass lesions causing visual symptoms and deficits in 14/20. All patients had visual evoked field response hemifield mapping by MEG to identify preserved visual pathways. Any alteration in the surgical approach/procedure based on the MEG findings in these patients was noted. All the patients were also followed postoperatively for any change in their visual deficits.

METHODS

Hemifield visual-evoked fields [Butler,1987] were recorded with 148 channel whole head MEG (4D Neuroimaging Magnes WH2500). Twenty patients (14 male, 6 female), (age range, 22- 84 yrs), with history of progressive visual impairment produced by a mass lesion seen by MRI were studied as part of their clinical care to document any residual functional visual cortex. The visual stimulus consisted of a 0.4-Hz black and white checkerboard pattern reversal image projected into the MSR via a system of mirrors. The size of the projected checkerboard was 1° (~2.5 cm) on a side with a visual angle of 11°. The patients were told to fixate on a target at the edge of the image. Each hemisphere was stimulated individually. Each run was performed twice.

Two hundred pattern reversal epochs were recorded with a 100-Hz high-pass filter and a sampling rate of 290.64 HZ. Data was filtered forward and backward using a 3-100 Hz bandpass with a 60-Hz notch filter. By visual inspection, peak latencies corresponding to the n75m, p100m, and n145m were identified and the single equivalent dipoles were calculated. The dipole selection criteria used were 1) correlation coefficient (R) of 0.98 or better, 2) root mean square (RMS) field values of at least twice the signal strength of the dipole moment (Q), and 3) confidence region (CR) of less than 1.0 cm³[Bowyer, 2003]. Results were overlaid on the patient's MRI scan

RESULTS

VEF waveforms were recorded from both hemi fields in all 20 patients. Fourteen out of 20 patients had preoperative visual field deficits, which was documented by perimetry in 9 of 14. Visual field deficits in 5 of 14 were recorded on clinical examination. Of the remaining 6 patients with no visual deficits on examination, VEFs were successfully recorded in 5 patients. In one patient, no reliable data could be obtained as the major peak latencies corresponding to the p100m and the n145m were obscured by artifact. Two of 5 patients showed superior displacement of the responses with normal latency and preservation of visual responses to left and right hemifield stimulation. The remaining 3 of 5 patients had normal VEFs. In the patients with clinically evident visual deficit (14/20), MEG data could not be interpreted in 4 patients because of presence of large signal artifact or noise. In total, technical problems prevented data interpretation in 5/20 patients. There was displacement of the ECD location in 8/14 patients and no response in 1/14 suggesting the disruption of the visual pathway in this patient. One patient had prolonged p100m latency without displacement of the response. The table below shows the outcomes for all 20 patients.

The mass lesions as identified by the MRI scan were resected or biopsied in all 20 patients. The surgical approach was altered based on the MEG report in 2 of the 15 patients analyzed. In another patient, the planned procedure was changed from a complete occipital pole resection to cyst drainage and biopsy which resulted in near total resolution of his hemianopia. Three patients showed postoperative improvement in the visual field deficits, confirmed by perimetry. These included 2 patients whose surgeries had been affected by the MEG results. None of the patients were noticed to have had any worsening in the visual deficits postoperatively.

DISCUSSION

This outcome study demonstrates that MEG can be used to elucidate the visual pathway integrity in patients with visual field deficits including many patients with substantial or even complete homonymous visual field loss. Displaced ECD responses or VEFs with prolonged latencies can be used as a guide to preserve vision in patients with visual field deficits when planning the surgical approach. This information may be the key factor in deciding whether to perform a large resection or merely a biopsy. This was illustrated by one patient in this series who had a complete homonymous hemianopia due to a cystic neoplasm. His visual field deficit nearly resolved with drainage of the cyst. Thus the abnormal responses signify the presence of an intact, though affected, visual field pathway. The presence of functional visual cortex in MEG should prompt consideration of a surgical approach which would retain the functionally important cortex. It should be borne in mind that the prolonged latencies may represent peritumoral edema causing the visual changes rather than direct invasion of the neoplasm.

In our study, MEG VEF mapping was successful in 15 of 20 patients. In 5 of the 20 patients, technical problems such as artifacts from metallic implants prevented successful recordings. Preoperative MEG recording of visual hemifield stimulation successfully identified preserved

visual pathways in 11 of 20 patients with visual field deficits. This provided necessary information to plan surgery in 3 patients which resulted in visual improvement in these patients.

| Patient | MRI findings | MEG – Ipsilateral findings | Surgery | Outcome | Pathology |
|---------|---|---|---|--|--|
| 59 F | Left Parieto-occipital area mass | Superomedial displacement – Localise to Calcarine cortex | Tumor Resection | No visual deficits- pre or post op | Giant cell Glioblastoma Multiforme |
| 42 M | Left parietal and occipital lobe mass | Prolonged p100m latency without displacement | Tumor Resection | R homonymous hemianopia, unchanged postop | Glioblastoma with necrosis |
| 55 M | Left parietal and posterior frontal cortex lesion | Normal response. | Biopsy | No visual deficits – pre or post op | Cerebral cortical necrosis with vasculopathy |
| 75 M | Large Right parietal and cerebellar mass | Posteroinferior displacement of responses - Localise to R occip tip. | Tumor resection | L homonymous hemianopia, improved - L inf. Quadrantopia postoperatively | Metastatic carcinoma (Lung) |
| 49 F | 2 cm left parasagittal necrotic mass lesion | No significant displacement of the response. Localizes inferior to the cystic mass lesion. | Tumor resection | No visual deficits –pre or post op | Carcinoma, Non-small cell, metastasis |
| 54 M | Right parieto-occipital lobe and posterior right temporal lobe mass | Area activated by left visual hemifield stimulation is considerable smaller than right – uncertain significance | Tumor resection and right occipital lobectomy | L homonymous hemianopia unchanged postop | Glioblastoma multiforme |
| 72 F | Left temporal and parietal mass lesion and surrounding edema. | Superior displacement | Tumor resection | No visual deficits – pre or post op | Metastatic Papillary Adenocarcinoma, |
| 84 M | Left parietal hemorrhagic mass lesion | Obscured by artifact | Tumor resection | R inferior quadrantopia, lost to follow up | Glioblastoma multiforme |
| 64 M | Right occipital cystic mass lesion. | Mesial displacement of hemifield VER and SSER - preserved with prolonged latencies | Tumor resection with clot evacuation | L homonymous hemianopia, unchanged postop | Metastatic renal cell CA with hemorrhagic necrosis |
| 66 M | Right temporo-occipital mass. | Superior displacement | Tumor resection | L homonymous hemianopia unchanged postop | Glioblastoma multiforme |
| 47 M | Left parieto-occipital cystic lesion. | Normal response | Tumor resection | No visual deficits – pre or post op | Glioblastoma multiforme |
| 71 M | R posterior temporoparietal and occipital mass | Obscured by artifact | Tumor resection | L homonymous hemianopia, unchanged post op | Glioblastoma multiforme |
| 71 M | Right occipital, temporal and splenial mass | Anterolateral displacement | Tumor resection | L homonymous hemianopia, unchanged post op | Malignant Non-Hodgkins Lymphoma |
| 55 F | Right parieto-occipital mass. | Obscured by artifact | Tumor resection | L homonymous hemianopia, unchanged postop | Metastatic adenocarcinoma |
| 51 M | Right occipital cystic mass with an enhancing mural nodule | Superior displacement of responses, along calcarine cortex | Tumor resection | L homonymous hemianopia, improved immediately post op to near resolution | Malignant astrocytoma |
| 22 M | Mass lesion in the posterior region of the brain involving atrium | No measurable evoked response, contralateral findings normal | Tumor resection | L homonymous hemianopia, unchanged post op | Pilocytic astrocytoma |
| 67 M | Left parietal lobe mass lesion | Preserved but delayed response, displaced downward | Subtotal tumor resection | R homonymous hemianopia, unchanged postop | Glioblastoma |
| 61 M | 2 cm left occipital ring enhancing tumor with a necrotic center | 1.5 cm anterior and 1 cm superior displacement | Abscess drainage | R homonymous hemianopia improved post op to inf. quadrantopia | Abscess, bacterial |
| 48 F | R temporo-parieto-occipital mass with edema and meningeal spread | Weak response, No source identified | Tumor resection | No visual deficits – pre or post op | Papillary adenocarcinoma |
| 58 F | Intraxial mass in the left occipital lobe | Displaced downward, lies along the inferior and medial rim of the cystic mass lesion. | Tumor resection | Incomplete R homonymous hemianopia, unchanged postop, | Metastatic squamous cell CA (lung) |

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