SHORT REPORT

Ictal magnetoencephalographic discharges from elementary visual hallucinations of status epilepticus

M Oishi, H Otsubo, S Kameyama, M Wachi, K Tanaka, H Masuda, R Tanaka

J Neurol Neurosurg Psychiatry 2003;**74**:525–527

Purpose: To report the rare opportunity to study ictal magnetoencephalography (MEG) in a 26 year old man with simple partial status epilepticus that presented as elementary visual hallucinations (EVHs) in the right upper visual field.

Methods: The patient described his EVHs as "snowing on TV," "flickering lights," and "rotating coloured balls" that continued for several days. MEG and simultaneous EEG were recorded twice: during an episode of EVHs (ictal recordings) and after EVHs were controlled by medications (interictal recordings).

Results: During EVHs, MEG showed continuous periodic epileptiform discharges over the left posterior superior temporal region, while simultaneous EEG showed rhythmic theta waves and sporadic spikes over the left temporal region. The MEG discharge consisted of a three phase spike complex. Equivalent current dipoles (ECDs), modelled from spike complexes, localised in the left superior temporal area. After drug treatment controlled the EVHs, interictal MEG and EEG showed rare spikes over the same left temporal region. The average ictal ECD moment (mean (SD)) (128.7 (32.8 nAm)) was significantly weaker than the average interictal ECD moment (233.0 (63.9) nAm) ($p<0.05$).

Conclusions: The continuous, periodic, and clustered discharges seen on ictal MEG were the sources of EVH. The weaker ictal ECD sources were frequently not detected by scalp EEG, while the stronger interictal sources, presumably originating from an extensive interictal zone, were sufficiently large to be seen as EEG spikes.

Magnetoencephalography (MEG) analysis typically uses an equivalent current dipole (ECD) model overlaid onto magnetic resonance (MR) images to localise sources of intracranial activity. As magnetic fields are comparatively unaffected by the different electrical conductivities of the brain, cerebral spinal fluid (CSF), skull, and skin, MEG can accurately localise the sources of intraneuronal electric currents that contribute to extracranial magnetic fields. MEG provides unique information about epileptogenic zones reflected by interictal spikes in patients with partial epilepsy.[1,2] Ictal MEG studies, however, are rare, as head movements interfere with the accuracy of MEG source localisation.[2,3] In the few successful ictal studies, MEG showed clustered spike sources from ictal ECDs alone and from colocalised ictal and interictal ECDs in epileptogenic zones.[3,4]

Patients may experience simple partial status epilepticus (SPSE) as visual hallucinations.[5] Elementary visual hallucinations (EVHs) consist of geometric figures, simple shaped figures, or flashes. EVHs are common in partial temporo-occipital lobe seizures.[6]

We describe a patient with SPSE manifested as EVHs, in whom we simultaneously recorded MEG and EEG during an EVH episode and after EVHs were controlled by drugs.

CASE REPORT

A 26 year old right handed man had complex partial seizures consisting of initial ictal blindness, headache, loss of consciousness, and at times clonic convulsion of the right upper limb since he was 8 years old. His episodes occurred a few times a year between age 8 and 24 and were controlled with valproic acid and zonisamide. His developmental milestones were normal, and he had no family history of seizures.

At 24 years of age, he had generalised tonic status epilepticus and was intensively treated with a barbiturate. Thereafter he frequently complained of having EVHs, which he described as "snowing on TV screen," "flickering lights," and "rotating multiple coloured balls" in the right upper quadrant visual field. At times, the EVHs continued for several days. Carbamazepine was started for the EVHs.

The neurological findings, including visual acuities and fields, were normal. MR images (MAGNEX Epios15; Shimadzu, Kyoto, Japan), consisting of 1.5T high resolution, 1 mm thin slices, showed no abnormalities. Long term video EEG showed sporadic spikes and medium amplitude rhythmic theta waves over the left middle and posterior temporal regions during EVHs.

SIMULTANEOUS MEG AND EEG

We recorded MEG and simultaneous EEG twice: during an episode of EVH and after the EVHs disappeared. For MEG we used a helmet shaped neuromagnetometer consisting of 204 planar-type gradiometers (Neuromag 204; 4D-Neuroimaging, Helsinki, Finland) in a magnetically shielded room. For EEG, we placed scalp electrodes according to the International 10–20 system. We collected both MEG and EEG data digitally at a 300 Hz sampling rate with a low pass filter of 130 Hz. We analysed the data off line with a bandpass filter of 3–45 Hz. For each MEG spike, we calculated the single ECD source using a spherical model. We evaluated the dipole moments of the MEG spike sources and overlaid the ECDs, with respect to three anatomical fiducial points, onto MR images. We used the Mann-Whitney U test for statistical analysis and set the level of significance at $p<0.05$.

During the first MEG and EEG recording, the patient complained of persistent EVHs. The EEG recordings showed intermittent, medium amplitude, rhythmic theta waves with sporadic spikes over the left temporal region (fig 1A). In contrast, the MEG results showed essentially continuous, periodic (2–2.5 Hz) spike complexes over the left temporal region that persisted throughout the 20 minutes of recording (fig 1B). These identical and consistent MEG epileptic discharges had three phases (fig 1C): an initial spike, a second spike, and a wave.

Abbreviations: MEG, magnetoencephalography; EVH, elementary visual hallucination; ECD, equivalent current dipole; SPSE, simple partial status epilepticus

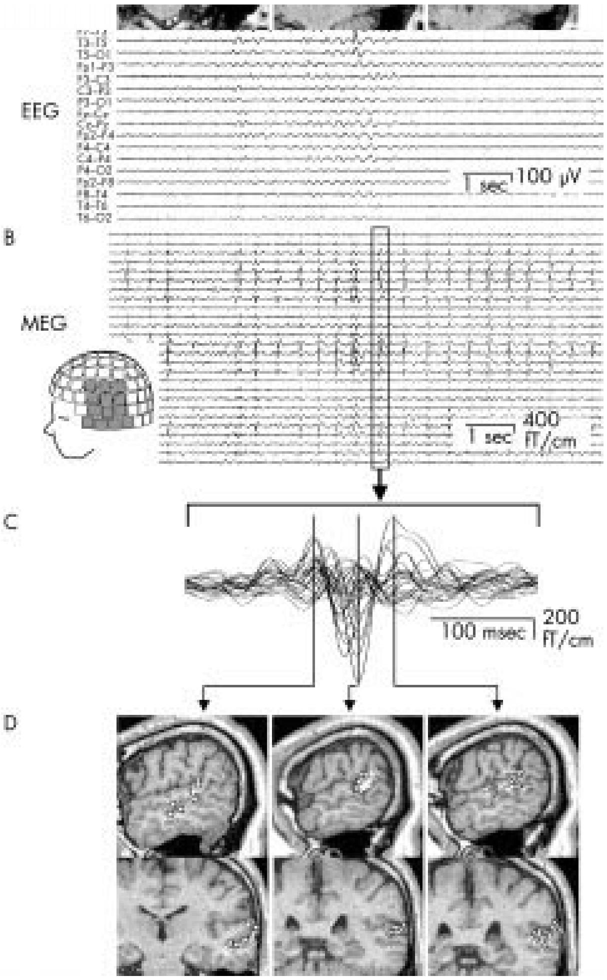

Figure 1 (A) Simultaneously recorded scalp EEG, (B) MEG data collected from the left temporal area during elementary visual hallucination in the upper right quadrant visual field. EEG showed intermittent medium amplitude theta rhythm with rare spikes over the left temporal region; MEG showed continuous spikes and waves over the left temporal region. (C) The overlaid waveforms of the MEG epileptiform discharges from the sample spike and wave in B. Three phases consisted of an initial spike, a second spike, and a wave. (D) Equivalent current dipoles (ECDs) on the patient's T1 sagittal and T1 coronal MR images. Squares, circles, and triangles represent ECD locations of the initial spikes, second spikes, and waves, respectively. Tails represent orientations of ECDs. The ECDs of the three phases were localised within the left superior temporal region.

We randomly selected 20 ictal MEG discharges and analysed each phase by the ECD method with a goodness of fit greater than 80%. We localised 14 ECDs from the initial spikes, 20 from the second spikes, and 16 from the waves (table 1). ECDs of initial spikes spread medially, adjacent to the grey and white matter junction (fig 1D). ECDs of second spikes had the smallest distribution and the strongest average moment of all phases. All ECDs clustered over the middle to posterior portion of the left superior temporal region.

When carbamazepine was increased, the patient stopped experiencing EVHs. During the second 20 minute MEG and EEG recording period, the patient reported no EVHs. Both MEG and EEG detected eight interictal spike discharges. Unlike the ictal recordings, no continuous MEG spike complexes were recorded. However, as with the ictal spikes, the eight MEG spike discharges consisted of three successive phases. We localised six ECDs from the initial spikes, eight from the second spikes, and five from the waves (table 1). All interictal ECDs were in the left temporal region.

Interictal and ictal MEG spike sources colocalised in the same left superior temporal region. However, the moments (mean (SD)) of the interictal second spikes (233.0 (63.9) nAm) and waves (169.1 (33.7) nAm) were significantly stronger than the moments of the ictal second spikes (128.7 (32.8) nAm) and waves (109.2 (55.7) nAm) ($p<0.05$).

DISCUSSION

To our knowledge, this is the first report of ictal MEG epileptiform discharges in the left superior temporal region during EVH of SPSE. Previous reports indicated that MEG spike sources, recorded in cortical myoclonus of epilepsia partialis continua, were localised in the precentral cortex.[7 8] The jerk-locked back averaging method confirmed the appropriate latency between MEG spike sources along the central sulcus and corresponding myoclonic jerks.[7] The clustered spike sources of the continuous periodic ictal MEG discharges recorded during EVH may function in the same way as those recorded in myoclonus, but have different manifestations because of their location near the optic pathway.

In a report on patients with epileptic visual auras, ictal scalp EEG showed temporo-occipital theta-delta waves, and intracranial EEG indicated beta waves or low voltage fast activities in the temporo-occipital region.[9] In a case report of a patient who experienced an EVH manifestation of SPSE, scalp EEG correlated a sharp/slow wave with a single flash episode.[10] In our case, interictal and ictal MEG spike sources colocalised in the same left superior temporal region, but the rare interictal MEG spikes, even though large, were not associated with EVHs. We suggest that the continuous and periodic property of the ictal MEG spike sources in the ictal symptomatic zone, close to the optic radiation in the left temporal region, probably provoked an epileptic EVH in the right upper quadrant of our patient's visual field.

All ECD localisations for the continuous ictal MEG spike complexes overlapped in a small area in the left superior temporal region. Simultaneous ictal scalp EEG, however, showed blunt theta waves. Several explanations could clarify these differences between MEG and EEG findings. Firstly, sampling

Table 1 Results of equivalent current dipoles (ECDs) of MEG polyspike and waves

	20 ictal discharges			8 interictal discharges		
	Initial spikes	Second spikes	Third waves	Initial spikes	Second spikes	Third waves
Number of ECDs	14	20	16	6	8	5
Moment (nAm)	95.3 (41.0)	128.7 (32.8)*	109.2 (55.7)*	145.2 (84.7)	233.0 (63.9)*	169.1(33.7)*
Positions (mm)						
X	–58.3 (9.0)	–61.9 (3.9)	–59.8 (6.8)	–57.7 (4.6)	–59.0 (3.9)	–57.1(5.7)
Y	–12.6 (8.4)	–19.0 (2.3)	–18.7 (9.4)	–5.7 (10.7)	–8.0 (9.4)	–16. (16.5)
Z	44.0 (8.7)	54.5 (4.3)	55.0 (7.0)	47.2 (10.9)	53.6 (2.2)	51.9 (2.7)

X, auricular line, positive right and negative left; Y, nasion-inion line, anterior positive and posterior negative; Z, from the centre toward the vertex of the head, positive superior and negative bottom; Values for the centre of the head (x, y, z) are all zero. *$p<0.05$. Data shown as mean (SD).

limitations of 10–20 scalp electrodes might prevent recording of the low amplitude discharges from tangential sources, and possibly existing radial sources, in the posterior portion of the superior temporal region midway between T3 and T5 scalp electrodes. High resistance conductors, consisting of brain, CSF, skull and skin, diminish lower spike discharges, and prevent them from reaching remote electrodes. Secondly, because those high resistance conductors have high frequency filtering effects, the shorter duration of the initial and second spikes within a very narrow area (less than 6 cm^2) may not show up on the scalp EEG.[11] Thirdly, MEG delineated generators only along the superior temporal sulcus. When the cortical surface that sources of electrical activity occupy is small, the attenuation on EEG is large.[11] The epileptiform discharges in a limited area results in lower amplitude and slower frequency on the scalp EEG.

The average moments of interictal MEG spike sources were stronger than those of ictal MEG spike sources, and the interictal EEG recordings showed more prominent spikes and waves than ictal EEG recordings. Simultaneous MEG and electrocorticography study showed that the percentage of spikes detected by MEG was proportional to the area of electrocorticographic epileptic discharges in the neocortical area.[12] The large interictal EEG spikes may have resulted from activation of an extensive, but non-ictal, cortical area.

EVHs are frequently associated with occipital seizure activity.[13] However, primary visual auras are also common in posterior temporal EEG foci.[14] EVH consists of geometric figures, simple shaped figures, or flashes, provoked from the primary visual cortex, the striate cortex, adjacent white matter, and the temporo-occipital region. In our patient, the history of complex partial seizures, including ictal blindness, clonic convulsion of the right arm, and loss of consciousness, indicates an epileptic neural network in the centro-temporo-occipital lobe. His persistent EVHs express the continuous epileptiform discharges that MEG defined within the lateral temporal/medial occipital epileptic network. Though the mechanism underlying ictal variability is uncertain, electrical hyperexcitability associated with seizures reverberates within the neural networks, which operate together and inextricably to culminate in the expression of seizures.[15]

ACKNOWLEDGEMENT

We thank Kuniko Tsuchiya and Tohru Kondo for their technical assistances and Carol Squires for editorial service.

Authors' affiliations

M Oishi, S Kameyama, H Masuda, Department of Neurosurgery, National Nishi-Niigata Central Hospital, Niigata, Japan
M Wachi, K Tanaka, Department of Psychiatry, National Nishi-Niigata Central Hospital
R Tanaka, Department of Neurosurgery, Brain Research Institute, Niigata University, Japan
H Otsubo, Division of Neurology, Department of Paediatrics, The Hospital for Sick Children, Toronto, Canada

Competing interests: none declared.

Correspondence to: Dr M Oishi, Department of Neurosurgery, National Nishi-Niigata Central Hospital, 1–14–1 Masago, Niigata 950–2085, Japan; oishi@masa.go.jp

Received 15 September 2002
Accepted 10 November 2002

REFERENCES

1 **Otsubo H**, Ochi A, Elliott I, *et al*. MEG predicts epileptogenic zone in lesional extrahippocampal epilepsy: 12 pediatric epilepsy surgery cases. *Epilepsia* 2001;**42**:1523–30.
2 **Stefan H**, Schneider S, Feistel H, *et al*. Ictal and interictal activity in partial epilepsy recorded with multichannel magnetoencephalography: correlation of electroencephalography/electrocorticography, magnetic resonance imaging, single photon emission computed tomography, and positron emission tomography findings. *Epilepsia* 1992;**33**:874–87.
3 **Oishi M**, Kameyama S, Morota N, *et al*. Fusiform gyrus epilepsy: The use of ictal magnetoencephalography. Case report. *J Neurosurg* 2002;**97**:200–5.
4 **Shiraishi H**, Watanabe Y, Watanabe M, *et al*. Interictal and ictal magnetoencephalographic study in patients with medial frontal lobe epilepsy. *Epilepsia* 2001;**42**:875–82.
5 **Sowa MV**, Pituck S. Prolonged spontaneous complex visual hallucinations and illusions as ictal phenomena. *Epilepsia* 1989;**30**:524–6.
6 **Panayiotopoulos CP**. Elementary visual hallucinations in migraine and epilepsy. *J Neurol Neurosurg Psychiatry* 1994;**57**:1371–4.
7 **Shigeto H**, Tobimatsu S, Morioka T, *et al*. Jerk-locked back averaging and dipole source localization of magnetoencephalographic transients in a patient with epilepsia partialis continua. *Electroencephalogr Clin Neurophysiol* 1997;**103**:440–4.
8 **Watanabe Y**, Sato S, Nakamura F, *et al*. The practical benefits of magnetoencephalography in comparison with electroencephalography in a patient with epilepsia partialis continua. *No To Shinkei* 1995;**47**:357–62.
9 **Bien CG**, Benninger FO, Urbach H, *et al*. Localizing value of epileptic visual auras. *Brain* 2000;**123**:244–53.
10 **Walker MC**, Smith SJM, Sisodiya SM, *et al*. Case of simple partial status epilepticus in occipital lobe epilepsy misdiagnosed as migraine: clinical, electrophysiological, and magnetic resonance imaging characteristics. *Epilepsia* 1995;**36**:1233–6.
11 **Cooper R**, Winter AL, Crow HJ, *et al*. Comparison of subcortical, cortical and scalp activity using chronically indwelling electrodes in man. *Electroencephalogr Clin Neurophysiol* 1965;**18**:217–28.
12 **Oishi M**, Otsubo H, Kameyama S, *et al*. Epileptic spikes: Magnetoencephalography versus simultaneous electrocorticlgraphy. *Epilepsia* 2002;**43**:1390–5.
13 **Williamson PD**, Thadani VM, Darcey TM, *et al*. Occipital lobe epilepsy: clinical characteristics, seizure spread patterns and results of surgery. *Ann Neurol* 1992;**31**:3–13.
14 **King DW**, Ajmone Marsan C. Clinical features and ictal patterns in epileptic patients with EEG temporal lobe foci. *Ann Neurol* 1977;**2**:138–47.
15 **Spencer SS**. Neural networks in human epilepsy: evidence and implications for treatment. *Epilepsia* 2002;**43**:219–27.