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Advanced dynamic statistical parametric mapping with MEG in localizing epileptogenicity of the bottom of sulcus dysplasia

Midori Nakajima [a,e], Simeon Wong [b], Elysa Widjaja [c], Shiro Baba [a], Tohru Okanishi [d], Lynne Takada [a], Yosuke Sato [a], Hiroki Iwata [a], Maya Sogabe [a], Hikaru Morooka [a], Robyn Whitney [a], Yuki Ueda [e], Tomoshiro Ito [e], Kazuyori Yagyu [e], Ayako Ochi [a], O. Carter Snead III [a], James T. Rutka [f], James M. Drake [f], Sam Doesburg [h], Fumiya Takeuchi [g], Hideaki Shiraishi [e], Hiroshi Otsubo [a,*]

[a] Division of Neurology, The Hospital for Sick Children, Toronto, ON, Canada
[b] Neurosciences and Mental Health, Sickkids Center for Brain and Mental Health, Toronto, ON, Canada
[c] Diagnostic Imaging, The Hospital for Sick Children, Toronto, ON, Canada
[d] Department of Child Neurology, Seirei Hamamatsu General Hospital, Shizuoka, Japan
[e] Department of Pediatrics, Hokkaido University, Graduate School of Medicine, Sapporo, Japan
[f] Division of Neurosurgery, The Hospital for Sick Children, Toronto, ON, Canada
[g] Center for Advanced Research and Education, Asahikawa Medical University, Asahikawa, Japan
[h] Department of Biomedical Physiology and Kinesiology, Simon Fraser University, BC, Canada

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HIGHLIGHTS

- Advanced dynamic SPM (AdSPM) was applied to the bottom of sulcus dysplasia (BOSD).
- AdSPM localized epileptogenic BOSD more accurately than by the single moving dipole (SMD) method.
- AdSPM localizes BOSD both with and without an overlapping SMD-derived dipole cluster.

ABSTRACT

Objective: To investigate whether advanced dynamic statistical parametric mapping (AdSPM) using magnetoencephalography (MEG) can better localize focal cortical dysplasia at bottom of sulcus (FCDB).
Methods: We analyzed 15 children with diagnosis of FCDB in surgical specimen and 3 T MRI by using MEG. Using AdSPM, we analyzed a ±50 ms epoch relative to each single moving dipole (SMD) and applied summation technique to estimate the source activity. The most active area in AdSPM was defined as the location of AdSPM spike source. We compared spatial congruence between MRI-visible FCDB and (1) dipole cluster in SMD method; and (2) AdSPM spike source.
Results: AdSPM localized FCDB in 12 (80%) of 15 children whereas dipole cluster localized six (40%). AdSPM spike source was concordant within seizure onset zone in nine (82%) of 11 children with intracranial video EEG. Eleven children with resective surgery achieved seizure freedom with follow-up period of 1.9 ± 1.5 years. Ten (91%) of them had an AdSPM spike source in the resection area.
Conclusion: AdSPM can noninvasively and neurophysiologically localize epileptogenic FCDB, whether it overlaps with the dipole cluster or not.
Significance: This is the first study to localize epileptogenic FCDB using MEG.
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1. Introduction

1.1. Focal cortical dysplasia at bottom of sulcus

Focal cortical dysplasia at bottom of sulcus (FCDB) is defined as a focal cortical dyplasia (FCD) type II located at the bottom of a

Abbreviations: AdSPM, advanced dynamic statistical parametric mapping; BEM, boundary element model; BOSD, bottom of sulcus dysplasia; dSPM, dynamic statistical parametric mapping; FCDB, focal cortical dysplasia at the bottom of sulcus; MEG, magnetoencephalography; SMD, single moving dipole; SNR, signal to noise ratio; SPM, statistical parametric mapping.

* Corresponding author at: Division of Neurology, The Hospital for Sick Children, 555 University Avenue, Toronto, ON M5G 1X8, Canada.
E-mail addresses: hiroshi.otsubo@sickkids.ca, ghiotsubo@gmail.com (H. Otsubo).

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sulcus. FCDB is intrinsically epileptogenic and provokes early onset focal seizures, with a high frequency of seizures during active periods, and occasionally with hospitalization for severe exacerbation (Harvey et al., 2015). Complete resection of FCDB, however, results in seizure freedom in up to 90% of selected cases (Chassoux et al., 2010).

1.2. MRI for FCDB

A recent study showed that current MRI technology diagnosed 18 (86%) FCDB out of 21 small FCD (128–3093 mm^3) (Besson et al., 2008). Multimodal MRI has been largely responsible for recent advances in identification of structural abnormality in FCDB (Besson et al., 2008; Thesen et al., 2011; Mellerio et al., 2015; Hong et al., 2017).

However, the association between MRI-based identification of FCDB and their epileptogenicity has not been fully investigated. MRI-based diagnosis for FCDB remains challenging as structural abnormality is not equivalent to neurophysiological abnormality.

1.3. MEG for FCDB

The single moving dipole (SMD) method has been successfully employed to noninvasively localize the irritative zone for presurgical evaluation due to intrinsic epileptogenicity in FCD type II (Otsubo et al., 2001; Otsubo et al., 2005; Alshafai et al., 2014). However, we previously showed spatial discordance between MEG clusters and MRI-visible FCDB and proposed the underlying pathological mechanisms (Nakajima et al., 2016). We found two subsets of FCDB; (a) bottom of sulcus dysplasia (BOSD), where the SMD method shows dipoles remote from the MRI-visible lesion and; (b) BOSD+, where MEG clusters partially overlap with the MRI-visible lesion. BOSD is an epileptogenic lesion restrictedly existing only inside of a single sulcus with opposing walls. The SMD method drifted the MEG dipoles to areas remote from the MRI-visible lesions in BOSD due to closed-field effect, low density of neural cells, and small size of the BOSD. BOSD+, in contrast, features a pathological distant neuronal wiring extending from the MRI-visible lesion, suggesting that the epileptogenic zone is extended in these cases.

The SMD method can accurately localize spatial clusters of interictal dipoles in FCD type II. However, the complex neuropathological features of FCDB pose a significant challenge for the SMD method to accurately localize the epileptogenic zone for a subset of patients with FCDB.

1.4. Advanced dynamic statistical parametric mapping

Dynamic statistical parametric mapping (dSPM) generates spatiotemporal estimates of evoked potentials by combining the anatomical cortical surface derived from MRI with neurophysiological MEG information (Dale et al., 2000). Integration of noise normalization with the minimum norm estimate (MNE) allows dSPM to provide spatiotemporal estimates of source distribution with temporal resolution on the order of milliseconds (Dale et al., 1999; Dale et al., 2000; Fischl et al., 1999; Fischl et al., 2001; Liu et al., 2002). Studies of dSPM have shown advantages for localizing epileptiform activity in widespread spikes, relative to the single dipole method (Shiraishi et al., 2005a, 2005b; Tanaka et al., 2009). The cortical mesh utilized by the dSPM method also includes sulcal walls, and therefore may better model epileptic activity for FCDB, particularly at the bottom of sulci.

There are, however, operator-dependent biases in current dSPM analysis approaches. The operator-dependent selection of representative MEG spikes can affect the number, morphology, and threshold, and therefore raises questions of validity and robustness. The determination of the identified area of activated cortex is highly dependent on the threshold applied in dSPM (Tanaka et al., 2009).

In this study, we developed Advanced dSPM (AdSPM) to identify epileptogenic FCDB. To our knowledge, this is the first attempt to develop a new MEG analysis that addresses the shortcomings of dSPM and is intended for neurophysiological localization of epileptogenic FCDB. We hypothesize that AdSPM localizes epileptogenic FCDB with greater accuracy than the SMD method.

2. Materials and methods

2.1. Patients

This retrospective study was approved by the Hospital for Sick Children Research Ethic Board (#1000050641). We identified patients by reviewing electronic medical records between August 2008 and December 2016 at the Hospital for Sick Children. We identified 15 (9 male; mean age 8.8 ± 4.2 years) children who had (i) drug-resistant localization related epilepsy with diagnosis of FCD type II, (ii) scalp video EEG, (iii) MEG analysis, (iv) 3T MRI, and (v) with or without resective surgery and intracranial video EEG (IVEEG). The age ranged between 2 and 15 years with a mean age of 8.8 ± 4.2 years. Twelve were diagnosed as FCD type II by surgical specimen and the other three children were diagnosed by 3T MRI.

2.2. MRI

Images were acquired on a 3T Philips Achieva MR scanner (Philips Medical System, Netherlands) using an 8-channel phased array head coil in all children. Imaging consisted of axial and coronal fluid attenuation inversion recovery (FLAIR) (TR/TE = 10,000/140, slice thickness = 3 mm, FOV = 22 cm, matrix = 316 × 290), T2/PD (TR/TE = 4200/80/40, slice thickness = 3 mm, FOV = 22 cm, matrix = 400 × 272), and isometric 3D T1-weighted (TR/TE = 4.9/2.3 ms, slice thickness = 1 mm, FOV = 22 cm, matrix = 220 × 220). FCD type II on MRI was diagnosed by a neuroradiologist (EW) based on the presence of the following features: (1) focal cortical thickening, (2) blurring of the gray-white matter junction, (3) T2/FLAIR hyperintensity of subcortical white matter and cortex, and in some cases, tapering of abnormal white matter signal toward the ventricle. MRI-visible FCDB lesions were located at the bottom of the sulcus and did not extend to the most superficial cortex at the surface of the brain. We recorded the anatomical location and extent of the MRI- visible FCDB for further analysis.

2.3. Intracranial video EEG recordings

We determined the hemisphere and location to place the subdural grid electrodes based on 3D MRI, interictal/ictal scalp video EEG, lateralization and distribution of MEG dipole sources, MEG somatosensory evoked fields, and clinical symptoms. The technique of implantation of intracranial electrodes, mapping of the possible epileptogenic zone was done as described previously (Ochi et al., 2007; Akiyama et al., 2011; Okanishi et al., 2014). The Brainlab AG suite of intraoperative neuronavigation software (PatXfer 5.2 and iPlan 2.6, Feldkirchen, Germany) was applied to co-register the lesion, MEG dipoles, and grid and depth electrodes. Center-to-center spacing of the electrodes was 9–13 mm in the subdural grid and 7 mm in the depth electrodes. Subdural electrodes were 4 mm in diameter with an exposure of 2.3 mm (effective surface area: 4.2 mm^2); the surface area of depth electrodes was 8.3 mm^2 (Ad-Tech Medical Instrument, Racine, WI, U.S.A.). We implanted the depth electrodes by probe-guided

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