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High frequency activity overriding cortico-cortical evoked potentials reflects altered excitability in the human epileptic focus

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HIGHLIGHTS

- We analyzed high frequency activity (HFA) of cortico-cortical evoked potentials (CCEPs) in epilepsy.
- The HFA power of early CCEPs in seizure onset zone (SOZ) increased more than that in non-SOZ.
- HFA overriding CCEPs may be a surrogate marker of cortical excitability in human focal epilepsy.

ABSTRACT

Objective: We aimed to clarify that high frequency activity (HFA) of cortico-cortical evoked potentials (CCEPs), elicited by single pulse electrical stimulation (SPES), reflects cortical excitability.
Methods: We recruited 16 patients with refractory partial epilepsy who had chronic subdural electrode implantation for presurgical evaluation. A repetitive SPES was given to (1) the seizure onset zone (SOZ) and (2) the control cortices (non-seizure onset zone: nSOZ). CCEPs were recorded from the neighboring cortices within SOZ and nSOZ. We applied short-time Fourier transform to obtain the induced responses for the timing of early (<50 ms after SPES) and late CCEP components and analyzed the logarithmic power change for ripple (<200 Hz) and fast ripple (>200 Hz) bands.
Results: Twenty-one clear CCEPs were recorded for both the SOZ and nSOZ. The HFA power of early CCEPs in SOZ significantly increased compared to that in nSOZ in both frequency bands, particularly in mesial temporal lobe epilepsy (MTLE).
Conclusion: Similar to the features of spontaneous pathological HFOs, the power of stimulus-induced HFAs in SOZ were greater than that outside SOZ, particularly in MTLE.
Significance: HFA overriding CCEPs can be a surrogate marker of cortical excitability in epileptic focus.
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Abbreviations: CCEP, cortico-cortical evoked potential; ECoG, electrocorticography; HFO, high frequency oscillation; HFA, high frequency activity; MTL, mesial temporal lobe; SOZ, seizure onset zone; SPES, single pulse electrical stimulation; STFT, short-time Fourier Transform.

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1. Introduction

The localization of the "epileptogenic zone" is a crucial factor in epilepsy surgery for patients with medically refractory partial epilepsy. While several non-invasive examinations, such as scalp electroencephalogram (EEG), magnetoencephalography, magnetic resonance imaging (MRI), F-18-fluorodeoxyglucose – Positron Emission Tomography (FDG-PET), and ictal single-photon emission computed tomography (SPECT), are useful to evaluate the epileptogenic zone, these results are occasionally insufficient (Lesser et al., 2010), requiring an additional intracranial EEG evaluation. In addition to conventional intracranial ictal EEG onset and interictal spike recordings, presumably epileptic high frequency oscillations (HFOs) have been recently recorded as possible surrogate markers of epileptogenesis even with macroelectrodes (Bragin et al., 1999; Urrestarazu et al., 2007; Zijlmans et al., 2009; Crepon et al., 2010; Jacobs et al., 2010). Epileptic HFOs are usually divided into ripple (R: 100–200 Hz) and fast ripple (FR: >250 Hz) by their frequencies. However, the analyses of spontaneous interictal HFOs are dependent on their occurrence and require technique expertise.

The measurement of cortical responses to single pulse electrical stimulation (SPES) is a straightforward way to evaluate epileptogenicity. We focused on the very early responses that are time-locked to the stimuli, specifically cortico-cortical evoked potentials (CCEPs), while others have focused on the late responses (e.g., delayed or repetitive responses) that are not time-locked and can occur with various delays (Valentin et al., 2002, 2005a,b; Alarcon et al., 2012). The CCEPs are typically composed of an early and a late negative component, which we labeled N1 and N2, respectively. By utilizing CCEP as a dynamic index of cortical excitability, the modulation of cortical excitability especially around the epileptic focus has been investigated (Matsumoto et al., 2005; Iwasaki et al., 2010; Enatsu et al., 2012b). CCEPs, or the early cortical responses to 1 Hz SPES, have been extensively employed to evaluate the cortico-cortical networks associated with various brain functions (Matsumoto et al., 2004, 2007, 2011, 2012; Greenlee et al., 2007; Lacruz et al., 2007; Conner et al., 2011; Swann et al., 2012; Terada et al., 2012; Matsuzaki et al., 2013; Entz et al., 2014), and seizure propagation (Enatsu et al., 2012a). Recently, we demonstrated that stimulus-induced high frequency activities (HFAs) on the CCEPs can be recorded in normal human cerebral cortices (Kobayashi et al., 2015; Usami et al., 2015). In the present study, we sought to clarify HFA overriding CCEPs and its involvement in human epileptogenesis. We hypothesized that HFA overriding CCEPs would be strongly associated with epileptogenicity. We compared the HFA overriding CCEPs in the seizure onset zone (SOZ) with that outside the SOZ.

2. Materials and methods

2.1. Patients

We recruited 16 patients with medically intractable partial epilepsy who underwent chronic subdural electrode implantation for pre-surgical evaluation, including 8 patients of mesial temporal lobe epilepsy (MTLE), 7 of non-MTLE (nMTLE), and 1 of MTLE + nMTLE (dual pathology) (Table 1). In one patient who had dual pathology (Patient 9), two independent SOZs were located in the mesial temporal area and basal frontal area. Thus we cumulatively counted 17 SOZs across 16 patients. The SOZs were defined in the mesial temporal lobe (MTL) in 9 and in non-MTL regions in 8 by ictal electrocorticography (ECoG). The electrodes were made of platinum, and their recording diameter was 2.3 mm (Ad-Tech, Rachine, WI, USA) or 3 mm (Unique Medical Co., Ltd., Tokyo,

Table 1
Patient profile.

Patient	Age, sex, handedness	Epilepsy	Group of subanalysis	Etiology	Neurological examination	Recording hemisphere	Stimulus sites of SOZ	Stimulus sites of nSOZ
1	25M R	TLE	MTL	HS	Normal	L	FG	ITG
2	39F R	TLE	MTL	FCD	Normal	L	FG	ITG
3	17F R	TLE	MTL	FCD IB	Normal	L	FG	ITG
4	29M L	TLE	MTL	FCD IA + HS	Normal	L	PHG PHG	FG
5	38F R	TLE	MTL	HS	Normal	L	FG	ITG ITG
6	55M R	TLE	MTL	Diffuse astrocytoma grade II	Normal	L	PHG PHG	ITG ITG
7	41F R	TLE	MTL	FCD I + HS	Normal	L	PHG	ITG
8	27F R	TLE	MTL	FCD IA	Normal	L	FG	ITG
9	22M R	F-TLE	MTL (temporal) and nMTL (frontal)	Gliosis (frontal) + FCD IA (temporal)	Normal	L	FHG ORG	ITG ITG IFG
10	27M R	T-OLE	nMTL	FCD IIA	Constructive apraxia and unharmonious quadrant hemianopsia	R	OCG OCG	OCG
11	23F R	FLE	nMTL	FCD IA	Normal	R	IFG IFG	MFG
12	23M R	OLE	nMTL	FCD IIA	Quadrant hemianopsia	R	CU	LGG LGG
13	34M L	P-TLE	nMTL	Posttraumatic injury and ischemic change (parietal) + HS and dysplastic change (temporal)	Normal	R	MTG	MTG
14	28F R	PLE	nMTL	DNT	Normal	R	CG	CG
15	52M R	TLE	nMTL	Cavernous angioma	Normal	L	ITG	ITG
16	45 M corrected R	FLE	nMTL	Microdysgenesis and FCD	Mild gait disturbance	L	SFG	SFG

TLE, temporal lobe epilepsy; FLE, frontal lobe epilepsy; OLE, occipital lobe epilepsy; PLE, parietal lobe epilepsy; MTL, mesial temporal lobe; nMTL, non-mesial temporal lobe; HS, hippocampal sclerosis; FCD, focal cortical dysplasia; DNT, dysembryoplastic neuroepithelial tumor; FG, fusiform gyrus; ITG, inferior temporal gyrus; PHG, parahippocampal gyrus; ORG, orbital gyrus; OCG, occipital gyri; IFG, inferior frontal gyrus; MFG, middle frontal gyrus; CU, cuneus; LGG, lingual gyrus; CG, cingulate gyrus; SFG, superior frontal gyrus.

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