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Sleep influences the intracerebral EEG pattern of focal cortical dysplasia

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Summary
Objective: Focal cortical dysplasia (FCD) is able to generate an intrinsic pathological EEG activity characterized by a continuous or near-continuous spiking. Different patterns of discharge were described. We examined quantitatively the distribution of the intracerebral FCD patterns in relation to sleep in order to investigate whether this activity is independent of thalamocortical influences.
Methods: We analyzed the first sleep cycle of 5 patients with a diagnosis of FCD type II who underwent combined scalp-intracranial electroencephalography (EEG), and showed an intracranial EEG pattern typical for FCD. Three patterns of FCD intracranial EEG activity were identified in all 5 patients, and visually marked for a maximum of 30 min of each stage (wake, N1, N2, N3, REM): spike or polyspike exceeding 2 Hz (pattern 1), spike or polyspike interrupted by flat periods below 2 Hz (pattern 2) and discharges of >15 Hz low-voltage rhythmic activity with regular morphology (pattern 3). After marking, the percentages of the three patterns across the different stages were calculated.
Results: The three patterns of FCD were present between 45% and 97% of the total time analyzed. Pattern 1 was the predominant pattern in wakefulness (73—100%), N1 (76—97%) and N2

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(58—88.5%) in all patients, and in REM in 4 of 5 patients (91—100%). During N2 and N3, there was an increase in pattern 2 in all patients, becoming the predominant pattern in 3 of the 5 patients during N3 (63—89%). Pattern 3 was rare and only sporadically observed during N2 and N3. Wakefulness and REM sleep showed a similar pattern (pattern 1) with a slight amplitude reduction in REM sleep.

Significance: Despite the presence of an almost continuous discharge, sleep is an important modulator of the pathological EEG patterns found in FCD type II. This might suggest that dysplastic tissue is influenced by the thalamo-cortical control mechanisms involved in the generation of sleep.

Introduction

Focal cortical dysplasia (FCD) is a specific malformation of cortical development, often associated with medically refractory epilepsy (Andermann, 2000; Lerner et al., 2009; Semah et al., 1998) and intrinsic epileptogenicity (Battaglia et al., 2013; Chassoux et al., 2000; Palmini et al., 1995).

The intrinsic epileptogenicity of FCD translates into characteristic EEG patterns identified in scalp and intracranial EEG recordings: (1) spike and polyspike and waves with a rhythmic and subcontinuous occurrence, and absence of physiological background; (2) pseudoperiodic spikes or bursts of spikes interrupted by suppression of activity; (3) brief discharges of low-voltage, fast rhythmic activity with regular morphology; and (4) repetitive electrographic seizures with recruiting/derecruiting prolonged trains of rhythmic activity (Chassoux et al., 2000; Francione et al., 2003; Gambardella et al., 1996; Noli et al., 2013; Palmini et al., 1995; Tassi et al., 2002, 2012). Although these patterns are very characteristic for FCD, they were also described in Rasmussen encephalitis and dysembrioplastic neuroepithelial tumors (Chassoux et al., 2000; Palmini et al., 1995).

Sleep is well known to have substantial impact on epileptic discharges showing facilitation of spikes during NREM sleep compared to REM sleep (Malow et al., 1998; Montplaisir et al., 1987; Sammaritano et al., 1991). More specifically, in a very recent study, we showed that epileptic discharges such as interictal spikes and high frequency oscillations are facilitated by widespread high amplitude slow waves occurring during NREM sleep (Frauscher et al., 2015).

Information on the influence of sleep on the pathological EEG activity described in FCD is scarce. FCD type II is associated with higher spike rates during sleep compared to wakefulness in surface EEG (Chassoux et al., 2012) and depth electrode recordings showed an increase of fast discharges during NREM sleep, which tend to spread into contiguous non-lesional areas compared to wakefulness or REM sleep (Francione et al., 2003; Tassi et al., 2002, 2012). In addition, seizures in FCD type II occur predominantly during sleep compared to wakefulness (Chassoux et al., 2012; Noli et al., 2013), and the risk of sleep-related epilepsy (more than 70% of seizures occurring during sleep) (AASM, 2005) is 14 times higher than with other lesions and non-lesional epilepsy independently of the localization of the dysplastic lesion (Nobili et al., 2009).

FCD was suggested to have an intrinsic pacemaker. In order to determine whether epileptogenicity in FCD type II is a truly independent pacemaker or under thalamocortical influence, we investigated systematically and quantitatively the distribution of the various EEG patterns found in patients with FCD type II across the wake sleep cycle.

Methods

Selection of patients and nights of recordings

From a total of 57 medically refractory epilepsy patients who underwent presurgical evaluation with combined scalp-intracranial EEG at the Montreal Neurological Institute and Hospital between January 2010 and June 2014, we identified seven patients with a histologically confirmed diagnosis of FCD type II. Five of these seven patients showed the typical FCD EEG patterns during intracerebral EEG recordings. Demographic variables and data on epilepsy history, FCD localization, neuropathological findings, and seizure outcome were collected. The absence of the typical patterns is most likely explained in patient 1 by a sampling problem in a non-lesional FCD IIb (electrode was close, but not inside the lesion); patient 2 had an extensive mesiofrontal FCD IIa with 3 electrodes inside the lesion, but absence of the typical patterns (frequent discharges were present).

We analyzed the first night after 72 h post implantation (to avoid the effect of anesthesia), with no clinical seizures 4 h prior to the nocturnal recording in case of partial or 12 h in case of generalized seizures in order to keep the influence of seizures on the intracranial EEG patterns as low as possible, since there is no literature whether seizures might have an impact on the proportion of the different patterns. We do also not know if electrographic seizures could have an influence, but they generally have no visible impact on the EEG. As they are frequent in some patients, we decided they should not be an exclusion criterion in order not to have to exclude further patients. Data was recorded using the Harmonie EEG system (Stellate, Montreal, Canada).

Sleep staging and scoring of FCD EEG patterns

The first sleep cycle and its stages (wake, N1, N2, N3, REM) were manually scored according to AASM 2.0 (Berry et al., 2012) in 30 s epochs on the scalp EEG using a bipolar EEG montage (F3-C3, C3-P3, Fz-Cz, Cz-Pz, F4-C4, C4-P4,) by an electrophysiologist specialized in sleep medicine (BF). Following the studies described above (Chassoux et al., 2000; Francione et al., 2003; Gambardella et al., 1996; Noli et al.,