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Movement preparation and cortical processing of afferent inputs in cortical tremor: An event-related (de)synchronization (ERD/ERS) study

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HIGHLIGHTS

- We show that patients suffering from isolated cortical tremor present a greater mu event-related desynchronization (ERD) compared to controls, while beta event-related desynchronization/synchronization (ERD/ERS) is unchanged.
- This reveals increased neuronal recruitment during motor preparation resulting from abnormal excitability of thalamo-cortical loops.
- Intracortical abnormalities in isolated cortical tremor are different and less severe than with patients suffering from cortical myoclonus associated to epileptic symptoms.

ABSTRACT

Objective: We sought to characterize cortical activity related to motor control in patients presenting with isolated cortical tremor, in order to determine whether or not myoclonus-related impairments are a source of event-related desynchronization/synchronization (ERD/ERS) disruption.
Methods: Nine patients presenting with isolated cortical tremor were compared with controls. Mu and beta ERD/ERS were computed over the scalp and brain surfaces using 128-channel electroencephalographic (EEG) recording during voluntary and passive finger extensions. We recorded somatosensory-evoked potentials following median nerve stimulation and performed myoclonic jerk-locked back-averaging of EEG activity.
Results: Back-averaging revealed a cortical premyoclonic spike in all patients. Five of the nine patients had exaggerated SEPs. The amplitude of mu ERD was greater in patients. Beta ERD/ERS did not differ from that seen in controls. Localizations of mu and beta ERD/ERS did not differ from controls and were identified in pre- and post-central sensorimotor cortical areas.
Conclusions: The present results suggest a hyperexcitability of the cortico-subcortical loops responsible for movement preparation and execution. Post-movement inhibition related to cortical processing of afferent input is unaffected in isolated cortical myoclonus.
Significance: Intracortical abnormalities can differ in patients suffering from cortical myoclonus, according to whether or not the individuals have associated epileptic symptoms.

1. Introduction

Cortical tremor is a variant of cortical reflex myoclonus (Ikeda et al., 1990); it consists of involuntary movements provoked by action and posture (for a review, see Cassim and Houdayer, 2006). Myoclonus itself is defined as a sudden, brief, jerky, shock-like, involuntary movement arising from the central nervous system (Fahn et al., 1986). Cortical myoclonus can be a clinical feature of a range of heterogeneous disorders, such as progressive myoclonic epilepsy (PME), juvenile myoclonic epilepsy and several neurodegenerative diseases (for a review, see Shibasaki and Hallett, 2005). It can also be observed as an isolated symptom, such as in cases of cortical reflex myoclonus or cortical tremor. Different degrees of severity exist. Cortical tremor can cause heterogeneous disabilities, from light troubles to great functional discomforts (see patient in Houdayer et al., 2007). Little is known about the physiopathology of this disorder. It may be caused by hyperexcitable areas of the

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sensorimotor cortex generating cortical spikes which precede subtle jerks in distal muscles during action (Ikeda et al., 1990). Giant somatosensory-evoked potentials (SEPs) as well as abnormal long-latency reflexes (LLRs) are often observed (Ikeda et al., 1990; Mima et al., 1998; Toro et al., 1993). These features may be related to impairment in sensorimotor integration, leading to cortical hyperexcitability.

Cortical activity related to motor control or sensory afferents can be studied using electroencephalography (EEG) in general and the event-related desynchronization/synchronization (ERD/ERS) technique in particular (Derambure et al., 1993; Pfurtscheller, 1977; Pfurtscheller and Aranibar, 1977). Cortical oscillations in the mu (8–12 Hz) and beta (13–25 Hz) bands are strongly related to motor behavior and sensory afferent processing, i.e. they react to contralateral movement and sensory stimuli (Chatrian et al., 1959; Gastaut et al., 1952; Jasper and Andrews, 1938). Mu desynchronization, which corresponds to an attenuation of the signal amplitude preceding and during voluntary movement, may be related to cortical activity related to movement preparation and execution (Neuper and Pfurtcheller, 1999; Pfurtscheller and Berghold, 1989; Steriade and Llinas, 1988). Beta ERS corresponds to a brisk, intense amplitude increase following movement termination. It may be related to an active inhibition period induced by sensory processing, since the same sort of beta ERS patterns can be induced by both active and passive movements (Alegre et al., 2002; Cassim et al., 2001) or by sensory stimuli (Houdayer et al., 2006; Pfurtscheller, 1981; Pfurtscheller et al., 2001; Salenius et al., 1997; Salmelin and Hari, 1994; Schnitzler et al., 1997; Stancak et al., 2003). Moreover, beta ERS is not observed anymore under ischemia (Cassim et al., 2001) and is greatly altered in patients presenting with sensory deafferentation (Reyns et al., 2008). Furthermore, beta ERS begins during a period of reduced cortical excitability (Chen et al., 1998). Our hypothesis is thus that beta ERS would be related to an active cortical inhibitory period related to processing of afferent input. Indeed, some authors have determined the origin of beta ERS as either the pre- or post-central gyrus (Crone et al., 1998; Gaetz and Cheyne, 2006; Jurkiewicz et al., 2006; Pfurtscheller et al., 1996; Salenius et al., 1997; Salmelin and Hari, 1994; Szurhaj et al., 2003) in the sensorimotor areas. Mu and beta ERD/ERS analysis is therefore a robust technique for monitoring cortical activity related to movement preparation and execution as well as post-movement cortical afferent processing.

In the present study, we wanted to establish whether or not the above-mentioned cortical processing activity is impaired in cortical tremor, even during movements that are not disturbed by myoclonic jerks. To this end, we studied mu and beta ERD/ERS related to active and passive index finger extension. Passive movements allowed us to analyze cortical oscillatory changes related to movement-induced reafferentation.

2. Methods

2.1. Patients

Over a 12-month period, we recruited patients complaining of isolated tremor (i.e. in the absence of any concurrent, progressive neurological disease) and in whom neurophysiologic exploration indicated cortical myoclonus. Nine patients (8 females and 1 male, aged 19–75, mean age ± SD: 55.7 ± 17.6 years old) participated in the study. In all cases, the action tremor involved both arms and sometimes predominated on one side. Electromyographic (EMG) polygraphy revealed that the tremor was caused by short-duration, distally predominant positive and negative myoclonus, induced by muscle activation and with a fair degree of rhythmicity (Fig. 1). No EMG activity was observed at rest. This EMG pattern corresponds to cortical tremor as defined by Ikeda et al. (1990). Long latency reflexes were tested using median nerve stimulation. None of these patients had any C reflex response.

None of the patients had progressive diseases that could otherwise have accounted for action tremor neither they had an otherwise significant personal medical history. All patients had normal magnetic resonance imagery (MRI). Five patients were on drug treatment during the experiment (see Table 1 for the details concerning each patient). Patients were compared to 12 healthy volunteers (3 females and 9 males, aged from 28 to 74; mean age ± SD: 51.5 ± 16.5). One patient and one control subject were left-handed. All subjects gave their informed, written consent to participation and the study was approval by the local investigational review board, in accordance with the 1964 Declaration of Helsinki.

2.2. EEG and EMG recording

EEG was recorded with a 128 Ag–AgCl electrode cap, according to the international 10-05 system. AFz was used as the ground electrode. During recordings of index finger movements, the reference was placed on the left mastoid. For recordings of SEPs and cortical activity related to myoclonus, the reference was placed on the mastoid ipsilateral to the recorded arm. EMG activity of the dominant *extensor indicis proprius* (EIP) muscle and the bilateral *first dorsalis interosseus* (FDI) and *extensor carpi radialis* (ECR) muscles were recorded using surface Ag–AgCl electrodes. The

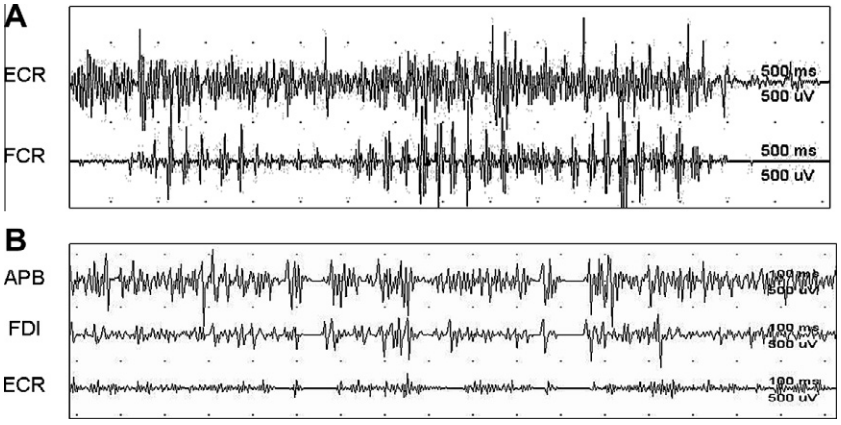

Fig. 1. EMG polygraphic results in a typical patient. (A) EMG activity of the ECR (upper line) and FCR (lower line) with the hands outstretched. (B) Activities of the APB, FDI and ECR muscles during co-contraction of each muscle.