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Fast epileptic discharges associated with ictal negative motor phenomena

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HIGHLIGHTS

- This study reports ictal and negative motor phenomena occurring during intracerebrally recorded seizures.
- Positive motor seizures were associated with a prominent alpha–beta band discharge while negative motor seizures were associated with a gamma band discharge (>45 Hz).
- Both positive and negative ictal motor phenomena can be observed in the primary motor cortex depending on the properties of the epileptic discharge.

ABSTRACT

Objective: Focal motor negative phenomena have been described in seizures primarily involving "negative" motor areas (opercular pre-motor and medial pre-motor regions) and the rolandic region (post-central or pre-central). The localizing value of such signs and the mechanisms by which an epileptic discharge may generate negative phenomena remain debated.
Methods: Ictal positive and negative motor phenomena occurring during seizures affecting the rolandic area were studied in a patient having intracerebral recordings (stereo-electro-encephalography, SEEG) for drug resistant epilepsy.
Results: During the video-SEEG and EMG recording, nine positive and 27 negative motor seizures were recorded. All were generated within the same area (right opercular central area, Brodmann Area 4). The 2 different types of clinical seizure were differentiable by their power/frequency spectrum: positive motor seizures were associated with a prominent alpha–beta band discharge while negative motor seizures were associated with a gamma band discharge (>45 Hz).
Conclusions: We propose that within the primary motor cortex, high frequency sustained discharges may disrupt the ongoing excitatory drive to the peripheral motoneurons and produce negative motor signs, while sustained lower frequency discharges (alpha and beta bands) may activate the cortico-nuclear or cortico-spinal pathway and produce positive motor signs.
Significance: Both positive and negative ictal motor phenomena can be observed in the primary motor cortex depending on the properties of the epileptic discharge.

1. Introduction

Ictal negative motor phenomena are a rare but important manifestations of focal epilepsy (So, 1995). The mechanisms and localisation of these phenomena remain debated. Moreover, they offer a helpful model to understand the relationship between

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mechanisms of normal cortical motor function and the abnormal patterns of function during epileptic seizures that may give rise to clinical signs. When associated with focal epilepsy, negative motor phenomena comprise mainly epileptic negative myoclonus (ENM), and focal atonic seizures (FAS). Negative myoclonus manifests as brief (<400 ms) and repetitive lapses in postural tone of a body segment (Tassinari et al., 1995). ENM has been described in focal seizures originating either in the primary sensori-motor cortex (Noachtar et al., 1997), or in the lateral pre-motor cortex(Baumgartner et al., 1996). They are thought to have the same physiological substrate as the electrical silent period that follows the motor potential evoked by Transcranial Magnetic Stimulation (TMS) of the primary motor area (Luders et al., 1995). A period of neuronal inhibition has also been observed after cortical electrical stimulation with a single pulse corroborating this mechanism (Alarcón et al., 2012). Any transient focal disruption of the background level of excitatory drive that is normally continuously delivered from the primary motor cortex to spinal alpha motoneurons could potentially give rise to this sign (Hallett, 1995; Tassinari et al., 1995; Noachtar et al., 1997).

Focal atonic seizures (FAS) are focal seizures in which ictal paresis or paralysis of a body part occurs without preceding tonic or clonic signs (So, 1995). This paresis can be shown electrophysiologically as a decrease or interruption of electromyogram (EMG) activity, sometimes following a somatotopic progression (Thomas et al., 1995; Matsumoto et al., 2000; Meletti et al., 2003; Villani et al., 2006; Shi and Zuo, 2012). The localizing value of such seizures, and their pathophysiological substrate, are debated. One hypothesis is that they may result from epileptic activation of the so-called cortical negative motor areas (Luders et al., 1995; Meletti et al., 2003). These areas form part of the motor cortex, and are thought to exert a physiological motor inhibition because peri-cortical electrical stimulation in their vicinity has been shown to produce a prolonged interruption in voluntary movement and EMG activity (Luders et al., 1995; Enatsu et al., 2013). FAS have also been associated with epileptic discharges in positive motor areas of the primary motor cortex (pre-central gyrus) (Matsumoto et al., 2000; Shi and Zuo, 2012). Other authors have suggested that not only the spatial location but also the electrical characteristics of the epileptic discharge could affect the resulting clinical manifestation (Wendling et al., 2003).

To contribute to this discussion, we report the case of a patient who underwent a presurgical evaluation for medically intractable epilepsy with intracranial depth electrode recording using stereo-electroencephalography (SEEG). Both negative and positive focal motor seizures were recorded whereas ictal discharges were localized in the primary motor cortex (M1). In this study, we will compare the spatial and temporal characteristics of the epileptic discharges associated with positive and negative motor signs. The possible localizing value and pathophysiological significance of these findings will be discussed.

2. Patient and methods

2.1. Case history

A 32-year-old right-handed woman underwent presurgical evaluation for medically intractable epilepsy with stereo-electroencephalography (SEEG) in 2003. She had sustained a severe head trauma at the age of 8 with right fronto-temporal contusion. Her first seizure occurred at the age of 11. Since then, she has had frequent (twice a month) clusters of stereotyped simple focal seizures that are intractable to antiepileptic therapy. Her habitual seizure semiology consists of an initial sensation of throat constriction followed by a left inferior facial contraction, with no alteration of consciousness. More rarely, she may have bilateral facial contraction followed by elevation of the right shoulder. Secondarily generalized tonic clonic seizures occur once a year. She may also have rare isolated auditory illusions that occur independently from the previously described motor signs. Cranial magnetic resonance imaging (MRI) showed a region of atrophy, presumed secondary to her childhood head injury, affecting the right dorso-lateral pre-frontal region as well as the temporal pole (Fig. 1).

Interictal scalp EEG (according to the international 10–20 system) showed right rolandic spikes (maximum on C4). Numerous simple motor seizures (more than fifty) were recorded on ictal scalp EEG with simultaneous video recording. These were clinically characterized by brief left inferior facial contraction and more rarely by bilateral superior and left inferior face contraction followed by a tonic elevation of the right shoulder. After a cluster of seizures, she was anarthric with preserved understanding of complex orders. All seizures were electrically identical; ictal scalp EEG always showed a very reproducible unilateral pattern of high amplitude spike followed by a discharge (15 Hz) localized in the right central region (C4).

2.2. Intracerebral EEG recordings

Focal motor seizures were investigated using intracerebral multiple lead electrodes (10–15 contacts; length 2 mm; 1.5 mm apart; diameter 0.8 mm) as part of the presurgical evaluation. The electrodes were placed intracranially according to Talairach's stereotactic method (Bancaud and Talairach, 1973). The placement of electrodes was determined from available non-invasive information and hypotheses about localization of the epileptogenic zone (Fig. 1); these hypotheses included a right central-opercular origin (electrode OC), a possible involvement of the right superior temporal gyrus (electrodes T, C), of the right lateral and medial pre-motor regions (electrodes PM, SA) and of the pre-frontal and parietal opercular region (electrodes CC and OP). The spatial accuracy of the implantation and absence of any complication were confirmed by cranial computerized tomography (CT) scan after implantation, and subsequent cranial MRI after electrode removal. Fusion of the CT and MRI scans allowed an accurate anatomical localization of each electrode trajectory (Bartolomei et al., 2004). Signals were

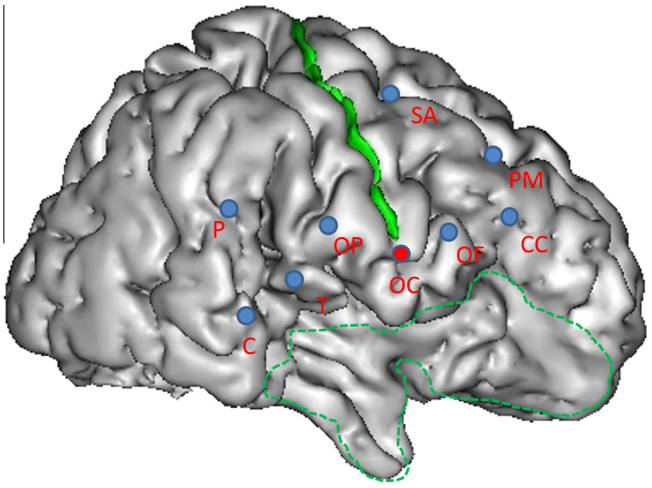

Fig. 1. 3D surface reconstruction of the right hemisphere showing the limits of the fronto-temporal post-contusive atrophy (green dotted line), the central sulcus (green), the entry points of the intra-cerebral electrodes (blue and red points). The following regions were explored with SEEG: opecular central (OC), opercular pre-frontal (OF), cingular (medial contacts of CC), pre-motor (SA and PM), parietal (P and OP), superior temporal (T and C). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)