Clinical Neurophysiology 125 (2014) 1689–1699

Contents lists available at ScienceDirect

Clinical Neurophysiology

journal homepage: www.elsevier.com/locate/clinph

Decreased movement-related beta desynchronization and impaired post-movement beta rebound in amyotrophic lateral sclerosis

Nataša Bizovičar [a,*], Jurij Dreo [b], Blaž Koritnik [a], Janez Zidar [a]

[a] Institute of Clinical Neurophysiology, Division of Neurology, University Medical Centre Ljubljana, Slovenia
[b] Department of Neurology, University Medical Centre Ljubljana, Slovenia

ARTICLE INFO

Article history:
Accepted 25 December 2013
Available online 8 January 2014

Keywords:
Amyotrophic lateral sclerosis
Electroencephalography
Event-related desynchronization and synchronization
Alpha and beta rhythm
Voluntary movement
Sniffing

HIGHLIGHTS

- ALS patients have decreased alpha activity possibly due to pyramidal and thalamic degeneration.
- Decreased beta desynchronization could indicate altered motor network connections whereas unilateral post-movement synchronisation supports ALS-related corpus callosum degeneration.
- ALS affects movement-related cortical potentials and ERD/ERS measures via different mechanisms.

ABSTRACT

Objective: This study explored event-related desynchronization (ERD) and synchronization (ERS) in amyotrophic lateral sclerosis (ALS) to quantify cortical sensorimotor processes during volitional movements. We furthermore compared ERD/ERS measures with clinical scores and movement-related cortical potential (MRCP) amplitudes.
Methods: Electroencephalograms were recorded while 21 ALS patients and 19 controls performed two self-paced motor tasks: sniffing and right index finger flexion. Based on Wavelet analysis the alpha and beta frequency bands were selected for subsequent evaluation.
Results: Patients generated significantly smaller resting alpha spectral power density (SPD) and smaller beta ERD compared to controls. Additionally patients exhibited merely unilateral post-movement ERS (beta rebound) whereas this phenomenon was bilateral in controls. ERD/ERS amplitudes did not correlate with corresponding MRCPs for either patients or controls.
Conclusions: The smaller resting alpha SPD and beta ERD and asymmetrical appearance of beta ERS in patients compared to controls could be the result of pyramidal cell degeneration and/or corpus callosum involvement in ALS.
Significance: These results support the notion of reduced movement preparation in ALS involving also areas outside the motor cortex. Furthermore post-movement cortical inhibition seems to be impaired in ALS. ERD/ERS and MRCP are found to be independent measures of cortical motor functions in ALS.

1. Introduction

Amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disease characterized by the degeneration of upper motor neurons in the primary motor cortex and lower motor neurons in the brainstem and spinal cord (Wijesekera and Leigh, 2009). Proliferation of glial cells, extracellular matrix expansion, and interneuron dysfunction is also observed (Agosta et al., 2010a,b). Previous neurophysiological studies have provided some evidence of hyperexcitability of the motor cortex (Eisen et al., 1993; Prout and Eisen, 1994; Desiato and Caramia, 1997; Mills and Nithi, 1997) and of decreased interhemispheric inhibitory modulation of corticomotoneurons (Karandreas et al., 2007). Neuroimaging studies suggest a pathological mechanism involving the degeneration of multiple motor and extramotor neural networks (Rose et al., 2012). Eight of the nine published functional neuroimaging studies demonstrated increased activations in the sensorimotor and supplementary motor area during movement. This was interpreted as evidence of compensatory cortical reorganization in ALS (Kew et al., 1993, 1994; Konrad et al., 2002, 2006; Schoenfeld et al., 2005; Tessitore et al., 2006; Lulé et al., 2007; Stanton et al., 2007; Mohammadi et al., 2011).

* Corresponding author. Address: Institute of Clinical Neurophysiology, University Medical Centre Ljubljana, Zaloška 7, SI-1525 Ljubljana, Slovenia. Tel.: +386 51 364 190; fax: +386 1 787 30 34.
E-mail address: natasa.bizovicar@gmail.com (N. Bizovičar).

1388-2457/$36.00
http://dx.doi.org/10.1016/j.clinph.2013.12.108

The two most commonly used electroencephalographic (EEG) methods for studying central mechanisms of movement control are movement-related cortical potentials (MRCPs) and event-related desynchronization/synchronization (ERD/ERS) (Toro et al., 1994). These methods use different signal analysis approaches to quantify cortical processes during the preparation and execution of movements. While MRCPs are only able to detect brain activity that is time and phase locked to motor acts, ERD/ERS methods can extract frequency specific activity that is merely time-locked but does not need to be phase locked to the motor act (Pfurtscheller and Lopes da Silva, 1999; Lopes da Silva, 2010). ERD/ERS methods can therefore show an additional spectrum of brain activations that are not observable with more classical event-related potential methods. The capability of these oscillatory methods to help elucidate the pathophysiological consequences of neurodegenerative changes in ALS has not yet been sufficiently explored.

Human EEG studies have demonstrated that alpha and beta oscillations show characteristic spatiotemporal patterns during sensorimotor processing (Neuper et al., 2006). The decrease of alpha and beta power relative to pre-movement baseline levels (ERD) in association with unilateral limb movements begins approximately 1.5–2 s before movement onset. It can be viewed as an electrophysiological correlate of an activated cortical network or of increased cellular excitability in the thalamocortical system (Pfurtscheller, 1981; Pfurtscheller and Lopes da Silva, 1999; Pfurtscheller et al., 1999; Neuper and Pfurtscheller, 2001). ERD is most prominent over sensorimotor areas contralateral to movement execution and extends bilaterally with movement initiation (McFarland et al., 2000). On the other hand increases in relative spectral power are hypothesized to result from a temporary synchronization of neural networks and are referred to as ERS (Pfurtscheller, 1992; Stancák and Pfurtscheller, 1995). In association with quick simple unilateral movements, ERS can be observed in the post-movement phase (starting approximately 1–2 s after movement cessation and is also called "beta rebound") (Jurkiewicz et al., 2006; Neuper et al., 2006). It has been hypothesized that ERS is produced by deactivated cortical areas and may represent simple idling activity and/or an active inhibition of the motor network (Pfurtscheller and Lopes da Silva, 1999; Cassim et al., 2001). There are presently only a few studies investigating electrocortical changes in ALS. Two have reported that MRCPs are reduced in patients with extensive upper motor neuron signs (pronounced spasticity) (Westphal et al., 1998; Inuggi et al., 2011). One reported that patients with low upper motor neuron burden (UMNB) scores generally had higher MRCPs than patients with high UMNB scores (Bizovičar et al., 2013).

While MRCPs mostly reflect neuronal activity in cortical motor areas, the generation of alpha and beta oscillations depends both on local and remote connections extending also outside the motor cortex (Schnitzler and Gross, 2005). The disruption of such connectivity due to disease or injury might influence the oscillatory activity of these networks. Bai et al. investigated beta ERD in primary lateral sclerosis and did not find any changes in beta oscillations (Bai et al., 2006). Two studies investigated beta ERD and ERS in terms of their applicability for EEG-based brain–computer interfaces in ALS (Bai et al., 2010; Kasahara et al., 2012). While Kasahara et al. showed that beta ERD is smaller in patients compared to controls this was only investigated with hand grasping motor imagery but not during actual movement execution (Kasahara et al., 2012). Riva et al. investigated ERD/ERS in ALS during actual movements but found no changes in ERD amplitudes. They did however report significant reductions of beta ERS in ALS. This latter finding was interpreted as indicating an impairment of the physiological cortical idling after movement (Riva et al., 2012).

The present study explores changes in absolute and relative spectral power in ALS and builds upon previous work (Westphal et al., 1998; Inuggi et al., 2011; Bizovičar et al., 2013) that focused purely on MRCPs. We investigated features of alpha and beta ERD/ERS in ALS to further elucidate possible changes in cortical regions outside the primary motor cortex. We additionally tested the hypothesis that ERD/ERS might be an independent measure of cortical dysfunction in ALS.

2. Methods

2.1. Study sample

We recruited 21 patients: mean age (SD): 64 (10); 13 male; 2 presented with bulbar and 19 with spinal disease onset. Inclusion criteria for patients were a clinical diagnosis of ALS according to Awaji-shima diagnostic criteria for laboratory supported probable and definite ALS (de Carvalho et al., 2008). The control group consisted of 19 healthy, age- and sex-matched volunteers (mean age (SD): 60 (9); 12 male, 7 female, with no history of neurological, psychiatric disease or respiratory dysfunction, and any other severe diseases, no use of neuroleptic drugs, drug or alcohol abuse. All participants were right-handed according to the Edinburgh Handedness Inventory (Oldfield, 1971). The study population was identical to the one used in a previous study on MRCPs in ALS (Bizovičar et al., 2013). Informed consent was obtained directly from each subject. The study protocol was approved by the Slovenian National Medical Ethics Committee.

2.2. Motor tasks

Subjects were seated in a comfortable chair in a darkened, sound-attenuated and electromagnetically shielded room in order to minimize sensory interference. They performed two self-paced (voluntary motor tasks): fast right index finger flexions (FF) while pressing on a small rubber balloon and a sniffing manoeuvre (SN), monitored by a pressure sensing nasal plug. Before each of the two experimental blocks, participants undertook a 10–15 min practice session, during which they were trained to perform movements every 5–10 s at 20% of their individual maximal strength.

2.3. EEG recording and processing

EEGs were recorded for approximately 30 min using a digital system (BrainAmp MR plus, Brain Products GmbH) with 30 Ag/AgCl cap-mounted electrodes. Impedances were kept below 10 kΩ. EEG signals were analog-filtered (0.013–250 Hz) and sampled at 500 Hz. All further analyses were performed off-line in BrainVision Analyzer 2 (Brain Products GmbH). Each recording was first visually inspected. Segments containing overt and common artefacts were excluded from further analysis. Eye movement artefacts were removed with an Independent Component Analysis (ICA) procedure. The recording reference was at FCz, but the signals were later digitally re-referenced to an average reference. Wavelet analysis was used to extract time–frequency power spectra and to determine the frequency bands that were most responsive to movement related spectral power changes. We used the Morlet wavelet to extract power values between 1 and 40 Hz in 80 linear frequency steps (the Morlet parameter was set at $c = 8$). Wavelets were later normalized with uniform scale power (unit energy normalization). We compared absolute spectral power densities (SPD) obtained with the Fast Fourier Transform (FFT) during the baseline "resting" interval (from −2500 to −1988 ms) and during movement execution (from −256 to +256 ms). Complex demodulation was used to extract a continuous time-domain signal for the most responsive frequency bands indicated by previous Wavelet analysis. The signal obtained by complex demodulation thus represents