Clinical Neurophysiology 125 (2014) 1819–1825

Contents lists available at ScienceDirect

Clinical Neurophysiology

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

Event-related mu-rhythm desynchronization during movement observation is impaired in Parkinson's disease

T. Heida [a,*], N.R. Poppe [a], C.C. de Vos [b,c], M.J.A.M. van Putten [b,c], J.P.P. van Vugt [c]

[a] MIRA Institute for Biomedical Engineering and Technical Medicine, Faculty of Electrical Engineering, Mathematics and Computer Science, Biomedical Signals and Systems Group, University of Twente, Enschede, The Netherlands
[b] MIRA Institute for Biomedical Engineering and Technical Medicine, Faculty of Science and Technology, Department of Clinical Neurophysiology, University of Twente, Enschede, The Netherlands
[c] Medisch Spectrum Twente, Department of Neurology and Clinical Neurophysiology, Enschede, The Netherlands

ARTICLE INFO

Article history:
Accepted 23 January 2014
Available online 2 February 2014

Keywords:
Parkinson's disease
Event-related synchronization
Event-related desynchronization
Mu-rhythm
Mirror neuron system

HIGHLIGHTS

- EEG alpha and beta band desynchronization (i.e. mu-rhythm ERD) during movement observation was largely absent in Parkinson's patients.
- Mu-rhythm ERD impairment may be a marker for Parkinson's disease.
- Evaluating mu-rhythm ERD functionality may reveal pathological processes.

ABSTRACT

Objective: Patients with Parkinson's disease often experience difficulties in adapting movements and learning alternative movements to compensate for symptoms. Since observation of movement has been demonstrated to lead to the formation of a lasting specific motor memory that resembled that elicited by physical training we hypothesize that mu-rhythm desynchronization in response to movement observation is impaired in Parkinson's disease.
Method: In a pilot study with nine patients with Parkinson's disease at a Hoehn and Yahr stage of I or II and eleven age-matched controls, we tested this hypothesis by comparing the event related desynchronization (ERD) patterns from the EEG recorded during the observation of hand action and baseline videos.
Results: Healthy subjects showed normal bilateral ERD of the mu-rhythm. In patients with Parkinson's disease this distinct ERD pattern was lacking.
Conclusion: The results of this study suggest that event-related mu-rhythm desynchronization is impaired in Parkinson's disease, even at early stages of the disease.
Significance: Studying event-related mu-rhythm desynchronization dysfunction in Parkinson's disease patients may enhance our understanding of symptoms as impaired motor learning.

1. Introduction

Idiopathic Parkinson's disease (PD) is a progressive neurodegenerative disease resulting in multiple motor symptoms which differ in type and severity depending on disease progression, and show a large variability between patients. PD symptoms are associated with basal ganglia dysfunction caused by the loss of dopamine-producing cells in the substantia nigra pars compacta. The basal ganglia are involved in enabling practiced motor acts and in gating the initiation of voluntary movements by modulating motor programs stored in the motor cortex and elsewhere in the motor hierarchy (Marsden, 1982; Mink, 1996; O'Reilly and Frank, 2006). Parkinson's patients often experience difficulties in adapting movements and learning alternative movements to compensate for symptoms. Observation of movement has been demonstrated to lead to the formation of a lasting specific motor memory that resembled that elicited by physical training (Stefan et al., 2005).

* Corresponding author. Address: University of Twente, Biomedical Signals & Systems, Zuidhorst 214, P.O. Box 217, 7500 AE Enschede, The Netherlands. Tel.: +31 53 4892759; fax: +31 53 4892287.
E-mail address: t.heida@utwente.nl (T. Heida).

http://dx.doi.org/10.1016/j.clinph.2014.01.016
1388-2457/

We hypothesize that impaired motor learning in PD might partly reflect deficits in the neuronal responses to movement observation.

Non-invasive studies using positron emission tomography and fMRI studies have found that action observation activates, often bilaterally, the rostral part of the inferior parietal lobule (IPL) and the posterior part of the inferior frontal gyrus (IFG) as well as the ventral premotor area (PMv) and the anterior intraparietal area (AIP) (Alegre et al., 2010; Cattaneo and Rizzolatti, 2009; Chaminade et al., 2005; Fogassi and Ferrari, 2011; Iacoboni and Dapretto, 2006; Kessler et al., 2006; Keysers and Gazzola, 2006; Nishitani and Hari, 2000; Rizzolatti and Craighero, 2004). During electroencephalography (EEG) event related desynchronization (ERD) of mu-rhythm activity, i.e. a decrease in alpha (8–12 Hz) and beta power (13–30 Hz), occurs over these areas during movement observation. Mu-rhythm ERD during observed movement has been suggested to be an indicator of activity in the so-called mirror neuron system (MNS) (Alegre et al., 2010; Pfurtscheller and Lopes da Silva, 1999). Mirror neurons are a class of neurons that become active both when individuals perform a specific motor act and when they observe a similar act performed by others (Di Pellegrino et al., 1992; Fadiga et al., 1995; Rizzolatti et al., 2009). The MNS has been implicated in several social behaviors varying from motor learning (e.g. through imitation) to social recognition and empathy (Iacoboni and Dapretto, 2006). The MNS has been most widely studied in non-human primates using direct neuronal recordings. There is no consensus on the exact location of the MNS in humans; the areas mentioned above have been suggested to be part of this system. Subcortical areas involved in motor behavior such as basal ganglia and cerebellum were also found to be activated during observation of hand movements (Alegre et al., 2010; Decety et al., 1994; Devos et al., 2003; Frey and Gerry, 2006; Kessler et al., 2006; Kühn et al., 2004). For example, local field potentials in the subthalamic nucleus, measured in PD subjects during surgery for deep brain stimulation, show changes in activity during movement observation coherent with changes occurring in the motor cortex (Fogassi and Ferrari, 2011; Marceglia et al., 2009).

We tested the hypothesis that the neuronal response to movement observation is impaired in PD by comparing the amount of event related desynchronization (ERD) from the EEG recorded during the observation of hand action and baseline videos of PD patients and age-matched healthy control subjects.

2. Methods

2.1. Study population

In this pilot study 9 Parkinson's patients (7 males, 2 females, average age 67.1 ± 8.6 years; the time since diagnosis ranged from 6 months to about 14 years; Hoehn and Yahr stage 1 (n = 3), and 2 (n = 6), see Table 1) and 11 age-matched healthy control subjects (6 males, 5 females, average age 61.5 ± 10.0 years) were included. All patients fulfilled the UK Brain Bank criteria for Parkinson's disease (Hughes et al., 1992). Patients with severe tremor, dyskinesias or those treated with deep brain stimulation were excluded in order to prevent EEG artifacts. Patients with known PD related dementia according to the clinical diagnostic criteria (Emre et al., 2007) and those using sedative medication were also excluded. Patients were allowed to take their usual anti-parkinsonian medication on the day of the experiment. All procedures conformed to the Declaration of Helsinki and were approved by the Medical Ethical Committee of the Medisch Spectrum Twente in Enschede, the Netherlands. All subjects gave written informed consent prior to participation in the study.

2.2. Measurement set up

Subjects sat comfortably in an armchair in an electrically and sound-shielded room watching hand action and baseline videos. The EEG was recorded using ASA acquisition software (ANT International BV, the Netherlands); 64 Ag/AgCl electrodes were positioned according the 10/10-system using a waveguard EEG cap. The signals were amplified, low-pass filtered (digital FIR filter 1350 Hz cut off) and sampled at 5 kHz (Refa, Twente Medical Systems International BV, the Netherlands).

2.3. Experimental protocol

A measurement consisted of six trials. During each trial, subjects watched a video consisting of eight fragments showing hand movements interspersed with seven baseline fragments. Subjects could rest between trials whenever desired. Presented hand actions included pinching, grasping, ball grasping, finger tapping, and hand turning (supination/pronation), randomly executed with left or right hand. As an example, Fig. 1A shows three frames of the video showing pinching movements executed with the left hand. A moving red ball over a black background was used as baseline video (Fig. 1B). It was expected that during the observation of this baseline video the brain returned to a resting state in which mirror neurons are deactivated. Patients were carefully watched to not perform any voluntary movements.

Each trial had a duration of approximately two and a half minutes with the hand action and baseline fragments having a length varying from 8 to 12 s. The various types of hand actions were randomly distributed over the six trials to prevent predictability. Each hand action appeared several times in a number of trials up to a maximum of six times.

2.4. EEG analysis

All analyses were performed off-line in Matlab (the Mathworks, Inc., 2009b). EEGlab (http://sccn.ucsd.edu/eeglab/; Delorme and Makeig, 2004) was used to create topoplots (i.e. a topographic map of a scalp data field in a 2D circular view); ANT-Matlab scripts

Table 1
Patient details (time in years).

Patient	Sex	Age	Disease duration	Hoehn & Yahr stage	Dominant motor symptoms	Antiparkinsonian medication
1	F	49	5	I	Tremor	None
2	M	62	4	II	Bradykinesia	Ropinirole
3	F	54	10	II	Tremor, bradykinesia	Levodopa/benserazide, pergolide, amantadine
4	M	72	2	I	Tremor	None
5	M	72	8	II	Tremor, rigidity	Levodopa/benserazide, pramipexole
6	M	68	7	II	Tremor, freezing, bradykinesia	Pramipexole
7	M	75	12	II	Freezing, bradykinesia	Levodopa/benserazide, pramipexole
8	M	73	7	II	Rigidity, bradykinesia, akinesia	Levodopa/benserazide
9	M	74	4	I–II	Tremor, bradykinesia	Levodopa/carbidopa, entacapone