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Symptom-specific EEG power correlations in patients with obsessive–compulsive disorder

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Abstract

Neurophysiological studies in patients with obsessive–compulsive disorder (OCD) consistently revealed frontal alterations of cortical activity but otherwise showed inhomogeneous results, conceivably due to variable subgroups with diverse pathomechanisms involved. The aim of this study was to investigate quantitative electroencephalography (EEG) in patients with OCD as compared to healthy controls and to correlate neurophysiological data with clinical variables. EEGs were digitally recorded from 18 unmedicated patients (8 male, mean age 32.4±11.8 years, Yale–Brown Obsessive–Compulsive Scale (Y-BOCS) 15.3±7.9) and 18 matched healthy controls, and analysed quantitatively. The mean frequency of EEG background activity and absolute power in delta, theta, alpha and beta frequency bands were calculated. Mean frequency of background activity was significantly lower in patients as compared to controls (−1.44/s, $p<0.01$), predominantly for the frontal electrode positions. Power spectra revealed increased delta- and decreased alpha-/beta-power in the group of patients ($p<0.05$, patients vs. controls). Correlation analyses showed significant positive correlations of EEG-power with the Y-BOCS sub-scores "obsessions", and negative correlations with the sub-scores "compulsions" (Spearman's correlations, $r_s=+0.48$ to $+0.70$, and -0.47 to -0.6, respectively, $p<0.05$). The data provide evidence of a dysfunction of frontal cortical activity in patients with OCD. The opposite correlations of neurophysiological data and clinical features, i.e. obsessions and compulsions, are suggestive of pathophysiological differences based on the presence of the respective cardinal symptoms of OCD.

Keywords: Obsessive–compulsive disorder; OCD; Obsessions; Compulsions; Quantitative electroencephalography; qEEG; EEG power spectra

1. Introduction

Obsessive–compulsive disorder (OCD) is a chronic condition characterized by the presence of recurrent and often disabling obsessions and compulsions, experienced as intrusive and inappropriate (Stein, 2002). Nowadays there is growing evidence for a neurobiological basis of OCD (Insel, 1992; Stein, 2000). Functional neuroimaging studies with PET, SPECT, or fMRI (Baxter et al., 1988; Machlin et al., 1991; Hollander et al., 1995; Breiter et al., 1996; Saxena et al., 1998; Saxena and Rauch, 2000) support the involvement of the frontal–subcortical circuitry including orbitofrontal hyperactivity.

Functional alterations of cortical activity have also been shown in neurophysiological studies. Early EEG studies in patients with OCD, simply based on visual inspection, have reported a higher rate of abnormal patterns with unspecific slow wave abnormalities (Pacella et al., 1944) and a diffuse non-specific theta-activity (Insel et al., 1983). Although most of the more recent quantitative EEG studies revealed abnormalities predominantly in frontal and frontotemporal regions (Jenike and Brotman, 1984; Prichep et al., 1993; Kuskowski et al., 1993; Locatelli et al., 1996; Karadag et al., 2003), the reported changes were not homogeneous and were partly conflicting. The observations comprised reductions in absolute delta and beta power with a corresponding increase in relative alpha

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Table 1
Demographics and clinical characteristics of the study population: patients with obsessive–compulsive disorder (OCD) and matched healthy controls (HC); clinical scores (Clinical Global Impression/CGI, Hamilton Depression Rating Scale/HAM-D, Beck Depression Inventory/BDI, Yale–Brown Obsessive–Compulsive Scale/Y-BOCS) of the patient group; data presented as mean±S.D. or *n*, where applicable

	OCD	HC
n	18	18
Sex (female/male)	10/8	10/8
Age (years)		
Mean±S.D.	32.4±11.8	33.3±11.3
Range	17–57	21–60
Disease duration (years)		
Mean±S.D.	9.0±5.2	
Range	1.5–22	
CGI	4.9±2.0	
HAM-D	7.2±4.9	
BDI	12.6±8.2	
Y-BOCS	15.3±7.9	
Sub-scores:		
– Obsessions	8.4±6.2	
– Compulsions	6.8±4.9	

power (Kuskowski et al., 1993), or an increase in relative delta– but a decrease in relative alpha – power (Locatelli et al., 1996). Using the neurometrics method, other groups were able to differentiate OCD subgroups, characterized by the pattern of EEG power topography, in terms of the patients' responses to serotonin reuptake inhibitors (Prichep et al., 1993; Hansen et al., 2003). OCD subtypes, defined either clinically by the individual constellation of symptoms or with respect to treatment response, might be a consequence of different pathophysiological patterns, leading to variable and sometimes inconsistent neurophysiological findings.

Therefore, the aim of this study was to assess quantitative EEG parameters in patients with OCD compared to healthy controls, and to investigate, whether there are electrophysiological differences between the patients according to their clinical presentation in terms of the cardinal features "obsessions" and "compulsions".

2. Methods

The study was reviewed and approved by the local ethics committee of the Ludwig–Maximilians–University of Munich and was carried out in accordance with the Declaration of Helsinki. All subjects gave written informed consent for participation in this study, after the design and the procedures had been fully explained.

2.1. Subjects

We investigated 18 inpatients (10 female, 8 male) with obsessive–compulsive disorder (OCD), free of any additional psychiatric (axis I) or medical illnesses, diagnosed by experienced psychiatrists according to DSM-IV and ICD-10 criteria during hospitalization in the Psychosomatic Hospital Windach. The patients were compared with 18 age- and sex-matched healthy controls, mainly recruited from medical students and hospital personnel, who were free of any previous or current neuropsychiatric disorders, exposure to psychotropic medication, or a family history of neurological or psychiatric diseases.

The overall severity of the disease was estimated by the Clinical Global Impression Score (CGI). Signs and symptoms of OCD were clinically rated with the Yale–Brown Obsessive–Compulsive Scale (Y-BOCS) (Goodman et al., 1989b,c), additional depressive symptoms were assessed with the Hamilton Rating Scale for Depression (HAM-D, 17-item version; Hamilton, 1960) and the Beck Depression Inventory (BDI; Beck et al., 1961).

All study participants were right-handed according to the Edinburgh-Handedness Scale (Oldfield, 1971). At the time of the EEG-recordings, both patients and controls were free of any medication with a drug free period of at least 2 weeks prior to the study.

2.2. EEG data acquisition and analysis

For EEG recording, the patients were seated in a sound-attenuated, electrically shielded room in a reclining chair with eyes closed (wakeful-resting condition). Electrodes were placed via electrocaps according to the 10/20 system with Cz as reference and Fpz as ground electrode. Additional electrodes (above the left eye and at the left ocular canthis) were used to record the electrooculogram (EOG) simultaneously. Impedances of all electrodes were below 10 kΩ throughout the session. EEG recordings were obtained during 5 min eyes closed, resting condition using a computerised 19-channel acquisition system (brain electrical signal topography (BEST)) through amplifiers with bandpass from 0.16 to 70 Hz (50 Hz notch filter), digitized at a sample rate of 256 Hz, and were digitally stored for further processing and analysis off-line. Visual inspection for artifact detection was performed off line subsequently by two independent investigators. Any epochs with generalised or local biological or technical artifacts (e.g. muscle activity, electrode artifacts, eye movements/blinks) were identified and excluded. Furthermore, the subjects' wakeful–resting condition during recording was controlled for by the exclusion of any EEG epochs indicating somnolence or reduced alertness, which was

Table 2
Differences in absolute EEG power [μV^2] (OCD patients minus healthy controls) for the respective frequency bands: presentation of mean differences and of the significant differences per electrode position ($^{\dagger}p<0.05$, $^{\ddagger}p<0.01$)

	Delta	Theta	Alpha1	Alpha2	Beta1	Beta2	Beta3
F3-C3	$4.52^{\ddagger}$				$-2.44^{\dagger}$		
F4-C4	$6.59^{\dagger}$				$-2.14^{\ddagger}$	$-1.79^{\dagger}$	
F7-T3				$-4.17^{\ddagger}$	$-2.38^{\ddagger}$		
F8-T4	$8.54^{\dagger}$		$4.13^{\dagger}$	$-3.44^{\dagger}$	$-2.70^{\ddagger}$	$-2.55^{\dagger}$	
T3-T5	$2.64^{\dagger}$			$-30.08^{\ddagger}$	$-5.49^{\ddagger}$		
T4-T6				$-23.81^{\ddagger}$	$-5.03^{\ddagger}$	$-2.87^{\dagger}$	
C3-P3	$2.20^{\ddagger}$			$-18.41^{\ddagger}$	$-3.23^{\ddagger}$	$-1.60^{\dagger}$	
C4-P4				$-15.50^{\ddagger}$	$-2.64^{\ddagger}$		
P3-O1	$2.31^{\dagger}$			$-16.39^{\dagger}$	$-3.53^{\ddagger}$	$-2.16^{\dagger}$	
P4-O2	$2.05^{\ddagger}$			$-14.00^{\dagger}$	$-3.12^{\ddagger}$	$-1.62^{\dagger}$	
Mean	$3.87^{\dagger}$	−0.03	8.94	$-13.42^{\dagger}$	$-3.27^{\dagger}$	$-2.13^{\dagger}$	−0.68