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A High Resolution Quantitative EEG Power Analysis of Obsessive-Compulsive Disorder

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Abstract

Introduction: The findings of the quantitative EEG power-spectral studies in obsessive-compulsive disorder (OCD) have so far been mostly inconsistent. Moreover, none of the studies has been a high resolution one.
Aim: The aim of the study was to examine the band power of delta, theta, alpha, beta1 and beta2 bands with high resolution EEG data in patients with obsessive- compulsive disorder and compare it with that of normal controls. It was hypothesized that there will be no significant group difference of individual band power between patient and control group.
Methods: Raw EEG data were acquired from 64 channels using a linked ear reference. We obtained EEG power values for 20 adult OCD patients (10 males; 10 females) and 19 appropriately matched healthy controls across the abovementioned bands. We used Advanced Source Analysis (ASA, ANT software b.v. Netherlands; version-3.0.0.5.) program for analysis of power.
Results: OCD patients had significantly higher power in comparison to controls which was widespread in the theta frequency, predominantly left sided fronto-temporo-parietal in delta and alpha and only left frontal in beta2 bands.
Conclusion: Increased band power in obsessive-compulsive disorder patients in all these bands which have been shown to be associated with cognitive processing, may reflect increased processing load in this group of patients with recruitment of wide area of cerebral hemisphere (German J Psychiatry 2007; 10: 29-35).

Keywords: Obsessive-compulsive disorder, quantitative EEG, band power, increased cognitive processing

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Introduction

In the past two decades, there has been a considerable advance in the understanding of the biological underpinnings of obsessive-compulsive disorder (OCD) with emergence of various hypothetical models. Currently, there is little confusion over a significant neurobiological basis of OCD. Genetic, neurochemical, neuropsychological and neuroimaging studies have shown certain consistent abnormalities in OCD. After repeated failures of structural neuroimaging methods in finding consistent structural abnormalities in psychiatric disorders, evolving functional brain-imaging techniques nowadays represent the most powerful tools for characterizing in vivo human anatomy, neurophysiology and neurochemistry at modest temporal and spatial resolution. Recent functional neuroimaging studies in OCD population have consistently found abnormalities the frontal cortex and basal ganglia structures (Saxena & Rauch, 2000).
Quantitative EEG (QEEG) being a physiological imaging technique can be used to delineate abnormal functioning of brain regions with remarkable temporal precision. However, the findings of the quantitative EEG power-spectral studies in OCD population have so far been mostly inconsistent. In one of the first quantitative EEG studies using power-

spectral analysis on 10 patients with OCD with a limited montage, Flor-Henry et al. (1979) reported relatively decreased variability in the temporal region. In this study, however, no frontal lead was used.
Khanna (1988) found decreased log power in the non dominant frontal-midline and posterior temporal regions. This study suggested right temporo-frontal hyper functioning to be associated with OCD and stressed the importance of the non-dominant fronto-temporal regions in this connection, with regards to both the localization by changes and by the nature of the activity observed (beta activity).
Bennasar et al (1991), in their study of power spectrum on OCD patients, found increased beta power in the frontal region. However, Perros et al. (1992) found significantly increased relative power in the theta-2 band in the left temporal and central regions and significantly reduced variability in frontal and temporal regions.
Kuskowsky et al. (1993) recorded EEG from 13 unmedicated and nondepressed patients with DSM-III-R obsessive-compulsive disorder (OCD) and from 10 age-matched controls. Quantitative analysis of the EEG revealed lower log absolute power in the delta, beta 1, and beta 2 bandwidths for OCD patients at frontal and right-hemisphere locations. Moreover, OCD patients displayed greater hemispheric asymmetries in EEG activity based on difference measures of EEG power from homologous electrode pairs, indicative of severe right hemisphere EEG hypoactivity.

Drake et al. (1996) compared EEG spectral measures in 20 patients meeting DSM-III-R criteria for obsessions and compulsions and 12 neurologically intact unmedicated controls. Among the patients, 10 had comorbid Tourette's syndrome. The EEG was recorded from 11 electrodes. The results showed that both left frontal and right frontal variability was significantly reduced in OCD patients in compared to controls. Consistent with literatures suggesting neurophysiological disturbances in OCD, this study, too, supported it by showing frontal lone dysfunction in OCD patients in comparison to neurologically intact controls.

Tot et al. (2002) did a recent study on OCD patients using quantitative EEG methods. The study included 22 right handed OCD subjects as the study group and 20 right handed pair-wise matched healthy subjects as control group. The authors used 12-channels for analysis of alpha, beta, delta and theta bands. Hemispheric asymmetry and regional differences for 3 brain regions: frontal, Temporal and parietal, were evaluated with frequency analysis. The analysis showed a considerable increase in delta and theta activity and a decrease in alpha activity of left fronto-temporal regions. A left temporal decrease in beta activity was significant. The study found that patients with OCD showed important fronto-temporal dysfunction, predominantly in the left hemisphere.

In another recent study, Karadag et al. (2003) obtained quantitative analysis of the EEG in patients with OCD. The study revealed a decreased beta and an increased theta power at fronto-temporal regions. The patients who had higher scores in doubting test (Maudsley Obsessive Compulsive Questionnaire) and more severely ill patients shared similar q-EEG features. The relative theta powers were significantly increased and alpha powers were significantly decreased in these patients, particularly in the frontotemporal region. It was suggested that the q-EEG may be useful in investigating the OCD patients with heterogeneous characteristics.

In summary, the results of different quantitative EEG studies in OCD patients have so far been mostly inconsistent except the finding of an increase in theta power, which has come up more or less consistently across studies. The reason for these inconsistencies is varied and includes lack of a control group, differences in EEG methods, medication differences and heterogeneity of the samples (one at least was largely made up of patients co-morbid for Tourette's).

To the best of our knowledge none of the studies has been a high resolution one. All studies used limited number of electrodes while EEG sampling of information. Moreover most of the study included heterogeneous population and did not strictly match healthy controls.

The aim of the study was to examine the band power (relative) of delta (0.5- 3.5 Hz), theta (4-7.5 Hz), alpha (8-12Hz), beta1 (12.5-20Hz) and beta2 (20.5-30Hz) bands in patients with obsessive-compulsive disorder and compare it with strictly matched normal controls using a high resolution EEG data. Given the inconsistencies of findings in the existing literature and exploratory nature of the study using a high resolution EEG technique, we hypothesized that there will be no significant group difference of individual band power between patient and control group.

Subjects & Methods

This study was conducted at the Center for Cognitive Neuroscience at the Central Institute of Psychiatry (CIP), Ranchi, India. The institute review committee approved the study.

Participants

Patients of both sexes, irrespective of medication status, aged between 18 and 50 years, giving informed consent and having a diagnosis of OCD according to DSM-IV (American Psychiatric Association 1994) were recruited using purposive sampling technique from outpatient facilities of the institute. Eleven patients (55%) were either drug naïve (5 out of 11 patients) or drug free for at least 2 weeks and 9 patients (45%) were receiving an SSRI (3 fluoxetine, 4 sertraline, and 2 fluvoxamine). Neither of them had any family history of significant neurological illness. Considering the high lifetime prevalence of co-morbid depression with OCD (Rasmussen and Eisen 1988) we also included those patients in whom onset of depressive symptoms (if present) had taken place after the onset of OC symptoms and was essentially mild (score $\leq$ 18) as per the 17-item Hamilton rating scale for depression (HDRS) (Hamilton 1960).
Healthy controls with nil significant personal and familial history, giving informed consent, and matched with patients for age, gender and handedness were recruited from the department personnel and post graduate students. We used

general health questionnaire-5 (GHQ-5) (Shamsunder et al., 1986) score 0 for screening of controls.
All subjects were right-handed as assessed by the sidedness bias schedule (SBS) (Mandal et al., 1992). Yale Brown obsessive compulsive scale (Y-BOCS) (Goodman et al., 1989) was applied on the patient population for quantification of psychopathology.
According to the above-mentioned inclusion criteria, 20 OCD patients and 19 healthy controls were recruited.

EEG data acquisition

Raw EEG data were acquired through Nihon-Kohden Neurofax electroencephalograph EEG-1100K from 64 channels using Ag-Ag/cl electrodes placed according to international 10/10 system using a linked ear reference. The sampling rate was 1024Hz/sec/per channel and AD conversion was 16 bits. Skin resistance at each site was kept <5 KΩ. Eye movement potentials were recorded using two electrodes placed 1 cm lateral to the outer canthus of each eye. All subjects were asked not to smoke or take caffeine 3 hrs prior to the recording. Twenty minutes of continuous resting EEG was recorded with subjects sitting on a reclining chair with eyes closed in a light and sound attenuated room.

EEG data processing

An experienced EEG reviewer selected more than 2 minutes of continuous, artifact free, awake-state EEG data. The EEG files were subsequently opened with Advanced Source Analysis (ASA, ANT software b.v. Netherlands; version-3.0.0.5.) operations for analysis of power. We calculated absolute power ($\mu V^2/Hz$). Artifact rejection procedure was again carried out with the help of the artifact rejection program in the software. The study used a 50 Hz notch filter, had set a low cut filter at 0.16 Hz and a high cut filter at 70 Hz as a part of data processing. The software had an in-built Welch periodogram for windowing. Five frequency bands were defined as follows: delta (0.5- 3.5 Hz), theta (4-7.5 Hz), alpha (8-12Hz), beta1 (12.5-20Hz) and beta2 (20.5-30Hz) bands. Exact 2 minutes of artifact free EEG data was taken for quantitative analyses and was divided into multiple 2 secs epochs. The epochs were made to overlap for 0.5 secs, both at the beginning and at the end. Power analysis was done selecting 2 secs block lengths for already defined epoch events. We were forced to exclude 16 channels from the original set of 64 channels since we could not get 2 minutes of artifact free data from them. Thus, finally, the following 48 electrodes were taken for power analysis:
Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T7, T8, P7, P8, F1, F2, Fc1, Fc2, C1, C2, Cp1, Cp2, P1, P2, Af3, Af4, Fc3, Fc4, Cp3, Cp4, F5, F6F, c5, Fc6, C5, C6, Cp5, Cp6, P5, P6, Af7, Af8, Ft7, Ft8, Tp7, Tp8.
All the electrodes were ear referenced.

Statistical procedures

Statistical analysis was done using Statistical Package for Social Sciences (SPSS, Inc., Chicago, Illinois) version 11.0. As analysis for normal distribution of power values with Kolmogorov-Smirnov test with Shapiro-Wilk correction revealed significant non-normal distribution, all power scores were log transformed to achieve gaussianity.
Independent t test was applied to study the group difference of spectral power values for delta, theta, alpha, beta1, and beta2 bands between patient and control group across 48 channels. Since our study is the first high resolution quantitative EEG study on OCD population and essentially investigational in nature, we did not calculate regionwise averaging. Instead, we decided to apply t test on each channel power value. However, to reduce the possibility of type I error because of multiple dependent variables, Bonferroni's correction was made to the level of significance. The new level of significance for power data taken was $P < 0.01$. Exponential function of all the mean and standard deviation values were calculated from the log power data.
However, a level of significance (α) of < 0.05 (two tailed) was taken to consider a result statistically significant for socio-demographic and clinical data. Chi-square test (for categorocal variables) and independent t test (for continuous variables) were applied for studying group difference of socio-demographic data.
The study population in the present study was a dichotomy as to the medication status. Therefore, subsequent Mann-Whitney U test was performed to address the impact of medication status.

Results

The patient sample included 10 male and 10 female subjects with a mean age 31.45 years (SD 8.91). The control group included 10 males and 9 females, with a mean age 29.85 years (SD 7.78). In patient group, mean age at onset of illness was 23.90 years (SD 6.89) while mean duration of illness was 7.60 years (SD 6.80). Mean duration of treatment in those who were taking medications was 8.28 months (SD 8.40). Mean Y-BOCS obsession score in the patient group was 15.45 (SD 2.23), compulsion score was 12.40 (SD 4.22) and mean Y-BOCS total score was 27.85 (SD 5.44). The mean HDRS score was 11.30 (SD 3.65).

Analysis of delta (0.5-3.5 Hz) power showed that the two groups differed significantly in terms of mean power across left frontal (F5, Af7), right frontal (F2, Af8), left temporal (Fc5, Ft7, C5, Cp5, T7, Tp7), right temporal (Ft8, T8, Tp8), left parietal (C3, Cp3, P1, P3, P5) and right parietal (Fc2) regions. The OCD group had higher mean power than the control group in all regions (Table 1).

Analysis of theta (4-7.5 Hz) power revealed that the two groups differed significantly in terms of mean power across all channels except Fc1, C1, and Fc3. In theta band, too, the OCD group had higher mean power than the control group (Table 2).

Table 1: Group difference in delta power (μV2/Hz) across all channels

Channel	Patients (Mean ±SD) (N=20)	Normals (Mean ±SD) (N=19)	t (df =37)	P*
F3	49.84 + 2.77	23.71 + 2.11	2.584	.014
F4	60.02 + 3.08	32.72 + 2.89	1.730	.092
C3	13.62 + 3.13	5.52 + 1.95	3.000	.005*
C4	13.63 + 3.11	5.56 +1.95	2.985	.005*
P3	56.93 + 4.53	16.65 + 1.68	3.365	.002*
P4	35.52 + 2.93	13.91 + 1.67	3.450	.001*
O1	55.36 + 3.62	24.48 + 1.67	2.579	.014
O2	54.47 + 3.64	23.95 + 1.74	2.560	.015
F7	140.75 + 3.81	55.55 + 3.11	2.336	.025
F8	119.71 + 3.23	51.94 + 2.75	2.376	.023
T7	68.33 + 3.59	21.85 + 1.86	3.515	.001*
T8	50.03 + 2.98	20.9 + 1.67	3.169	.003*
P7	76.84 + 4.91	26.78 + 1.78	2.721	.010
P8	58.65 + 3.5	27.51 + 1.81	2.389	.022
F1	801.75 + 3.32	122.47 + 18.26	2.665	.011
F2	647.29 + 2.91	296.28 + 1.85	2.777	.009*
Fc1	510.1 + 4.22	223.77 + 1.95	2.274	.029
Fc2	452.1 + 3.17	181.34 + 1.67	3.168	.003*
C1	270.4 + 2.94	149.35 + 1.91	2.071	.045
C2	230.26 + 3.16	107.68 + 1.85	2.551	.015
Cp1	398.82 + 5.54	139.99 + 1.73	2.542	.015
Cp2	263.78 + 3.16	136.48 + 1.74	2.259	.030
P1	571.69 + 3.53	211.35 + 1.76	3.150	.003*
P2	458.15 + 2.68	258.50 + 1.98	2.096	.043
Af3	1424.39 + 3.13	569.35 + 2.64	2.696	.011
Af4	1204.84 + 2.58	702.82 + 2.43	1.832	.075
Fc3	496.16 + 4.84	220.77 + 2.13	2.028	.050
Fc4	444.34 + 3.09	221.05 + 2.03	2.297	.027
Cp3	435.42 + 4.31	155.99 + 1.74	2.872	.007*
Cp4	291.81 + 3.57	139.78 + 2.38	2.101	.042
F5	1365.26 + 3.7	416.09 + 2.42	3.309	.002*
F6	1217.07 + 3.15	493.44 + 2.57	2.673	.011
Fc5	919.38 + 4.1	330.89 + 2.25	2.754	.009*
Fc6	765.48 + 3.18	328.88 + 2.22	2.642	.012
C5	919.38 + 4.1	330.89 + 2.25	2.754	.009*
C6	765.48 + 3.18	328.88 + 2.22	2.642	.012
Cp5	740.85 + 5.43	232.08 + 1.9	2.804	.008*
Cp6	430.65 + 3.75	177.68 + 1.8	2.680	.011
P5	957 + 5.55	249.76 + 1.7	3.268	.002*
P6	556.41+ 3.36	230.56 + 1.69	2.918	.006*
Af7	3787.65 + 4.07	1178.38 + 3.3	2.787	.008*
Af8	3782.73 + 3.34	1276.53 + 2.8	3.016	.005*
Ft7	1520.96 + 4.42	497.75 + 2.7	2.744	.009*
Ft8	1068.27 + 3.37	443.15 + 1.84	2.833	.007*
Tp7	1087.79 + 5.13	335.53 + 1.86	2.939	.006*
Tp8	880.33 + 4.61	299.23 + 1.7	2.912	.006*

(*Bonferroni adjusted significance at P < 0.01)

Table 2: Group difference in theta power (μV^2/Hz) across all channels

Chan-nel	Patients (Mean ±SD) (N=20)	Normals (Mean ±SD) (N=19)	t (df =37)	P*
F3	7.72 ± 2.76	3.62 ± 1.85	2.799	.008*
F4	8.03 ± 2.84	3.58 ± 1.71	3.015	.005*
C3	2.67 ± 3.53	1.02 ± 1.54	3.171	.003*
C4	2.68 ± 3.53	1.02 ± 1.54	3.162	.003*
P3	11.25 ± 3.33	3.42 ± 1.91	3.818	.000*
P4	7.99 ± 3.47	3.00 ± 1.97	3.030	.004*
O1	14.32 ± 3.05	5.94 ± 1.74	3.097	.004*
O2	14.32 ± 2.98	5.80 ± 1.84	3.166	.003*
F7	12.52 ± 2.73	5.31 ± 1.72	3.296	.002*
F8	11.36 ± 2.66	4.98 ± 1.62	3.312	.002*
T7	10.74± 2.67	4.1 ± 1.41	4.048	.000*
T8	9.39 ± 2.81	4.04 ± 1.48	3.336	.002*
P7	16.76 ± 2.91	6.16 ± 1.8	3.592	.001*
P8	14.75 ± 2.92	6.45 ± 1.94	2.886	.006*
F1	123.87 ± 2.72	27.24 ± 5.06	3.531	.001*
F2	112.54 ± 2.8	49.21± 1.8	3.060	.004*
Fc1	82.83 ± 3.08	37.56 ± 1.96	2.648	.012
Fc2	84.67 ± 3.18	32.58 ± 1.81	3.216	.003*
C1	42.82 ± 3.53	21.06 ± 1.97	2.169	.037
C2	42.12 ± 3.74	17.19 ± 1.69	2.758	.009*
Cp1	60.69 ± 3.62	20.02 ± 1.67	3.501	.001*
Cp2	48.13 ± 3.77	19.10 ± 1.62	2.858	.007*
P1	116.28 ± 3.45	35.73 ±1.99	3.654	.001*
P2	102.69 ± 3.43	38.02 ± 1.85	3.157	.003*
Af3	146.1 ± 2.59	55.80 ± 1.76	3.813	.001*
Af4	135.92 ± 2.59	65.81 ±1.74	2.883	.007*
Fc3	68.74 ± 3.35	30.01 ± 1.98	2.613	.013
Fc4	65.63 ± 3.16	28.19 ± 1.52	3.012	.005*
Cp3	68.14± 3.46	23.41 ±1.54	3.552	.001*
Cp4	57.8 ± 3.58	22.68 ± 1.65	2.985	.005*
F5	133.17 ± 2.79	57.54 ± 1.97	2.996	.005*
F6	125.89 ± 2.68	54.64 ±1.61	3.333	.002*
Fc5	97.01 ± 3.00	36.29 ± 1.53	3.645	.001*
Fc6	91.09 ± 2.94	39.01 ± 1.52	3.202	.003*
C5	81.64 ± 2.93	29.85 ±1.45	3.868	.000*
C6	73.14 ± 3.04	29.58 ± 1.52	3.332	.002*
Cp5	119.44 ± 3.11	32.74 ±1.41	4.767	.000*
Cp6	83.86 ± 3.2	32.81 ± 1.65	3.243	.003*
P5	207.04 ± 3.37	60.93 ± 1.99	3.842	.000*
P6	130.50 ± 3.11	56.28 ± 2.00	2.772	.009*
Af7	234.51 ± 2.79	86.25 ±1.79	3.714	.001*
Af8	218.66 ± 2.69	89.51 ± 1.73	3.467	.001*
Ft7	164.51 ± 2.92	60.41 ± 1.62	3.726	.001*
Ft8	139.38 ± 2.77	57.25 ± 1.44	3.591	.001*
Tp7	199.82 ± 2.88	68.05 ±1.54	4.125	.000*
Tp8	186.74 ± 3.09	66.8 ± 1.73	3.588	.001*

*Bonferroni adjusted significance at P < 0.01

Table 3: Group difference in alpha power (μV^2/Hz) across all channels

Channel	Patients (Mean ±SD) (N=20)	Normals (Mean ±SD) (N=19)	t (df =37)	P*
F3	15.26 ± 3.23	7.13 ± 3.15	2.051	.047
F4	15.54 ± 3.21	6.79 ± 2.95	2.297	.027
C3	6.20 ± 3.35	2.26 ± 2.56	2.897	.006*
C4	6.24 ± 3.33	2.26 ± 2.57	2.921	.006*
P3	41.07 ± 3.29	16.12 ± 4.8	2.103	.042
P4	36.22 ± 3.7	15.16 ± 4.65	1.910	.064
O1	47.21 ± 3.19	27.77 ± 4.1	1.285	.207
O2	46.74 ± 3.27	28.26 ± 4.12	1.205	.236
F7	18.82 ± 2.91	8.31 ± 2.57	2.532	.016
F8	16.83 ± 2.98	7.37 ± 2.51	2.549	.015
T7	21.6 ± 2.75	10.00 ± 2.53	2.472	.018
T8	20.22 ± 2.83	9.98 ± 2.84	2.113	.041
P7	53.42 ± 3.18	29.58 ± 4.84	1.340	.189
P8	58.55 ± 3.33	36.65 ± 4.64	1.064	.294
F1	237.56 ± 3.29	47.42 ± 9.47	2.818	.008*
F2	238.53 ± 3.31	100.21 ± 3.35	2.249	.031
Fc1	168.11 ± 3.44	78.45 ± 3.55	1.903	.065
Fc2	176.78 ± 3.65	71.41 ± 3.3	2.267	.029
C1	91.13 ± 3.59	44.64 ± 3.33	1.794	.081
C2	97.76 ± 3.82	36.10± 2.88	2.565	.015
Cp1	160.53 ± 3.7	50.40 ± 3.08	2.960	.005*
Cp2	150.42 ± 3.77	57.52 ± 3.27	2.381	.023
P1	436.72 ± 3.43	125.61 ± 4.58	2.818	.008*
P2	411.91 ± 3.8	145.97 ± 4.16	2.346	.024
Af3	276.33 ± 3.24	109.88 ± 3.07	2.506	.017
Af4	269.67 ± 3.22	118.68 ± 3.00	2.255	.030
Fc3	132.87 ± 3.19	62.00 ± 3.53	1.966	.057
Fc4	132.98 ± 3.4	52.85 ± 2.72	2.572	.014
Cp3	191.85 ± 3.41	72.26 ± 3.28	2.521	.016
Cp4	176.02 ± 3.52	81.79 ± 3.23	1.965	.057
F5	219.36 ± 3.06	88.90 ± 2.65	2.684	.011
F6	213.41 ± 3.03	90.03 ± 2.68	2.564	.015
Fc5	168.04 ± 2.98	67.84 ± 2.86	2.641	.012
Fc6	152.98 ± 3.26	64.92 ± 2.55	2.503	.017
C5	173.28 ± 2.76	65.27 ± 2.62	3.077	.004*
C6	145.72 ± 3.15	65.22 ± 2.66	2.350	.024
Cp5	298.39 ± 2.98	104.91 ± 3.3	2.858	.007*
Cp6	238.56 ± 3.31	111.97 ± 3.38	1.957	.058
P5	720.83 ± 3.14	293.3 ± 5.31	1.970	.056
P6	568.39 ± 3.33	324.86 ± 5.22	1.213	.233
Af7	318.05 ± 2.93	137.72 ± 2.8	2.481	.018
Af8	299.74 ± 2.95	122.08 ± 2.65	2.718	.010
Ft7	261.6 ± 2.85	108.43 ± 2.75	2.668	.011
Ft8	223.65 ± 2.97	93.8 ± 2.49	2.693	.011
Tp7	493.29 ± 2.95	243.06 ± 3.91	1.800	.080
Tp8	515.89 ± 3.35	254.42 ± 3.97	1.705	.097

*Bonferroni adjusted significance at P < 0.01

The OCD group had significantly higher mean power across left frontal (F1), left temporal (C5, Cp5), left parietal (C5, Cp1, and P1) and right parietal (C4) electrodes in alpha (8-12 Hz) band than the control group (Table 3).

Analysis of the beta 2 (20.5-30 Hz) band revealed that the OCD group had significantly higher mean power in left frontal Af7 ($t = 2.949$; $df = 37$; $P = 0.005$) in comparison to the control group.

In the beta 1 (12.5-20 Hz) band, no significant group difference was observed.

Mann-Whitney U test revealed no significant difference of power between those patients who were drug naïve/free and those who were taking medications.

Discussion

Methodological Considerations

Better spatial sampling is the first requirement for extracting more detailed information about cognitive processes from scalp recorded EEGs. It is well recognized that the optimum 3 dB point of the point spread function for conductance of potentials from the brain surface to the scalp averages about 2.5 cm (Gevins et al, 1995) and the 64-channel has got the benefit over the former 32-, or 19- channel record in the form of more spatial sampling of EEG data (Gevins et al, 1994). All of the quantitative EEG studies done so far on obsessive compulsive disorder have used lesser number of channels resulting in insufficient spatial resolution. The present study has improved upon the former studies by using 64-channel and thus getting satisfactory spatial resolution.

Unlike previous studies, the current study has strictly (pair wise) matched age, sex, and handedness while taking controls into the study. Thus, the possible confounding influence of all these factors could be controlled.

A matter of concern in studying patients with obsessive compulsive disorder is to control the effect of comorbid depressive symptoms, which have been the most common comorbid syndrome associated with obsessive compulsive disorder (Attiullah et al, 2000) with a lifetime prevalence which can be as high as 67% (Rasmussen & Eisen, 1988). The current study has only included those patients in whom onset of depressive symptoms had taken place after the onset of obsessive compulsive symptoms and was essentially mild (score < 18) as per 21-item HDRS scale.

Moreover, the current study has combined both visual inspection and computer assisted algorithms for rejection of artifacts which increased the accuracy of the procedure.

Discussion of Results

Patients with OCD had significant higher power in all the frequency bands except in beta 1. Moreover, the difference of power values was independent of mediaction status. This points towards an independent and robust electrophysiological abnormality in OCD patients.

Most robust difference was observed in theta band where there was diffuse increase in power across entire scalp. Bi-frontal, bi-temporal and bi-parietal increased power was noticed in the delta band also. However, the increase of power was seen in comparatively wider region in the left side than in the right side in delta frequency.

Slow wave predominance in OCD is well- known from the studies done by Pacella et al. (1944), Rockwell and Simons (1947), Bingley and Persson (1978), Insel et al. (1983). Increased power of the theta band in the left temporal and central region was also noticed in the study done by Perros et al. (1992). Karadag et al. (2003) found increased theta power in both fronto-temporal regions. However, in the present study, diffuse rather than regional increase in theta power was noticed.

Theta rhythms are one of the most intensively investigated phenomena in neurophysiological studies in terms of correlating the rhythm with cognitive functions. Distributed theta system in the human brain may be the system having most weight on integrative cognitive functions (Basar et al 2001). Hence, the diffuse significant increase of power values in the theta frequency domain may be considered as the electrophysiological marker of increased cognitive processing in OCD.

However, this relationship can be viewed from either way. It is expected that neuronal activation which is widespread across cortical areas are manifested in the slow frequency range. The behavior of a chain of neurons responsible for the generation and propagation of rhythmic activity over cortical tissue can be described by non-linear dynamics. Lower spatial frequencies are allowed by the boundary conditions to persist on larger cortical surfaces because of interference of waves traveling in different directions. Thus, larger surfaces allow for lower frequencies (Nunez, 1995). The same phenomenon could possibly be there for wide areas of neuronal activation (bi-fronto-temporo-parietal) seen in OCD subjects being manifested in the delta frequency band.

Increase of power in the slow frequency bands in OCD raises an important issue which can be seen from a different perspective. Interestingly, increased slow wave frequencies is also observed in case of other neurodevelopmental disorders as well, such as schizophrenia, ADHD etc. Compared with normal controls, schizophrenics have more slow activity in the form of delta, theta, and alpha 1 bands (Miyauchi et al. 1990). A recent meta-analysis of quantitative EEG studies done on ADHD children by Snyder & Hall (2006) also found increase in theta power in this group. The similarity of observation in our OCD sample too is a significant one and may point towards a shared electrophysiological dysfunction in neurodevelopmental disorders. This particular hypothesis needs to be taken up for further explanation by future quantitative electrophysiological studies.

In the current study, predominant increase in alpha power in patient group was noticed in the left temporo-central and left frontal region. This finding is in contrast with the finding obtained in the study by Tot et al (2002) who found lower frequencies of alpha activity predominantly at left fronto-temporal localizations. This finding is in agreement with predominant left sided increase of power (except theta power) in our study.

The finding of increased beta2 power in the frontal region is consistent with the finding obtained in the study of power spectrum by Bennasar et al (1991). In their study they also found increased frontal beta rhythm in OCD patients. However, in current study, the increase in power was noticed in the left frontal region only. This is also in harmony with predominant left sided increase of power (except theta power) seen in the present study.

So, in the current study, OCD patients had increased EEG power in comparison to controls, which is widespread in the theta frequency, predominantly left sided fronto-temporo-parietal in delta and alpha and only left frontal in beta2 bands. Interestingly, the findings of our study point towards a significant laterality effect, indicating a left brain dysfunction in OCD patient population. Previously, Flor-Henry et al (1979) also proposed the same in their study.

Considerable evidences have accumulated in recent times regarding the definite associations of theta, alpha, and delta frequency oscillations in the brain with cognitive processing (Basar et al, 2001). Like theta frequency which has been shown to reflect integrative cognitive processing (Basar, Schurmann & Sakowitz, 2001), frequency in the delta frequency, too, have been shown to be involved in similar process (Basar et al, 2001). Current research has shown that several forms of 'functional alpha' can be observed during sensory, cognitive, and motor processes (Basar et al, 2001).

The present work is the first high resolution q-EEG study done on OCD patients. Increased band power in obsessive-compulsive disorder patients in all those bands which have been shown to be associated with cognitive processing may reflect increased cognitive processing load in this group of patients with recruitment of wide area of cerebral hemisphere.
Future studies could address this issue by recording EEG while OCD subjects perform cognitive tasks. Simultaneous functional neuroimaging record will increase precision of this method. Further, reversibility of electrophysiological dysfunction with improvement of OC symptoms can also be studied. However, use of small sample size and presence of comorbid depression were the main limitation of our work despite taking statistical precautions.

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