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# Neurophysiological Substrate of Obsessive Compulsive Disorder: An Evidence from Topographic EEG

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## ABSTRACT

**Obsessive- compulsive disorder has recently been found to be associated with various biochemical markers; this has revived interest in its biological basis. Most of the work to date has emphasized on the role of neurotransmitters involved and neurophysiological arousal.**

**In this presentation, evidence from electrophysiological study: Topographic EEG mapping, has focussed on a possible left hemispheric dysfunction associated with this disorder. Thirty patients diagnosed as primary obsessional disorder according to DSM - III R, were examined by a topographic EEG. Their age ranged between 16 - 45 years, with a mean of 32. 1 years; 19 were males and 11 females. 50 % of our patients (15 cases) showed evidence of left hemispheric dysfunction where two had hyperactive foci in the temporal region. 20 % (6cases) showed evidence of right hemispheric involvement;: 4had a right hemispheric dysfunction and two had right hemispheric hyperarousal. 10% (3cases) had normal records; 13.3% (4cases) showed generalized cerebral dysfunction and 6.7% (2 cases) had borderline records. Our results indicate a more significant association between the left hemisphere and the obsessional symptomatology.**

## INTRODUCTION

Obsessive compulsive disorder is a syndrome characterized by recurrent, intrusive thoughts (obsessions), usually accompanied by repetitive purposeful behaviour (compulsion) such as ritualistic washing or checking.

OCD patients generally recognize their symptoms as senseless and egodystonic, and in most cases, struggle against performing their compulsive rituals.

Modern ideas about OCD emerged in the 19th century. Charcot and Margan coined the term "onomatomania" for patients who were disabled by "imperative ideas", and in 1890 Culere noted a link between epilepsy and Charcot& Magnan's onomatomania (Tuke, 1894).

Although Freud (1894) attributed the origin of obsessive states and neurosis to repressed memories of sexual guilt, other writers continued to regard them as organic in origin. Tuke (1894) considered that they were the result of abnormal cortical functions, a view supported by other authors (Wrxberg, 1938; Schilder, 1938; Beech & Perigault, 1974; Villa & Beech, 1977; Okasha, 1988).

Obsessional disorders have received considerable attention from psychologists over the last 10-15 years, al-

though most of the effort has been directed towards techniques, whil attempts to understand the mechanisms of the disorder have been less evident (Beech & Vaughan, 1979).

Current behavioural theories centre on the notion that an obsession is a learned behaviour which becomes established through its anxiety relieving properties. However, this simple explanation fails to deal with many puzzling features of the disorder, such as why the performance of rituals often increases rather than decreases anxiety, or how altered mood, rather than environmental experience, serves to activate pathological behaviour. An alternative approach to explaining the phenomena of obsessional disorder has involved the search for signs of physical abnormalities. A number of workers have suggested the possibility of neurological basis for OCD (Schilder, 1938; Bear & Fedio, 1977).

Recently, OCD has been found to be associated with various biochemical markers; this has revived interest in its biological basis. The evidence for a biological substrate for OCD has been gradually mounting (Turner *et al.* , 1985). However, the implications have been more for the neurotransmitter involved-serotonin- than for the site of dysfunction (Yaryura-Tobia & Bhavagan, 1977). Luria (1966) believed in the localization of functions of specific areas of the brain, in contrast with the generalists who believed that the whole cortex is implicated in all such phenomena. The evidence for localization of dysfunction in OCD has recently gained support from cerebral glucose metabolism studies (Baxter *et al.* , 1977), CT scans (Behar *et al.* , 1984), electrophysiological considerations (khanna *et al.* , 1987) and psychosurgical evidence (O'Callaghan *et al.* , 1982; Bridges *et al.* , 1973; Hassler, 1980).

In order to substantiate the hypothesis of a possible cortical dysfunction, we studied thirty patients (19males, 11 females) diagnosed as primary obsessional disorder according to DSM-III R.

They fall into two major symptoms: rituals and ruminations and all have experienced symptoms for at least one year. Their age ranged between 16-45 years, with a mean age of 32.1 years. Those with suspected organic lesions or secondary psychiatric disorders were excluded. All the patients were subjected to a semi-structured psychiatric interview (Ain Shams psychiatric sheet), neurological and physical examinations, topographic EEG mapping using 16 channels Dantec-Siegen Machine, where 16 scalp electrodes were applied according to the International 10-20 system with earlobes reference and FPz ground, to collect 16 channels EEG data. All the EEG channels were recorded with a common average reference.

Three to five minutes of EEG data were collected for each of the eyes closed, eyes open and photic stimulation.

The segments without artifacts were selectively recorded for mapping.

With EEG data, Fast Fourier Transformation performs spectral analysis to produce topographic mapping in the from of colour maps that show the distribution of brain electrical activity within selected frequency band.

We found that 90% of the cases (27 cases) showed TEEG abnormalities and 10% (3 cases) had normal records.

70% of our patients (21 cases) showed evidence of hemispheric lateralization, where 15 cases (50%) showed left hemispheric dysfunction predominantly posterior quadrant, where two had temporal hyperactive foci. Six cases (20%) showed evidence of right hemispheric involvement where two had right hemispheric hyperarousal and 4 showed hemispheric dysfunction.

No hyperactive foci were detected in the right hemisphere.

Four cases (13.3%) had generalized cerebral dysfunction and two cases (6.7%) showed borderline records (Table 1).

# TABLE I

*Topographic EEG Mapping in Obsessive Compulsive Disorder*

| Changes | RT HEM. | | LT HEMI | | GENER. | | BORDER. | | NORMAL | |
|---|---|---|---|---|---|---|---|---|---|---|
| | No | % | No | % | No | % | No | % | No | % |
| Dysfunction | 2 | | 2 | | | | | | | |
| Hyperarousal | | | | | | | | | | |
| Focal Changes | 2 | | Zero | | | | | | | |
| Dysfunction | 2 | | 11 | | | | | | | |
| Hyperactive Foci | | | 2 | | | | | | | |
| TOTAL | 6 | 20% | 15 | 50% | 4 | 13.3% | 2 | 6.7% | 3 | 10 % |

30 cases Age range=16-45 ys Mean=32.1 ys
Sex: 19 males 11 females

| TOTAL NUMBER = 30 cases | % |
|---|---|
| Normal 3 | 10 % |
| Borderline 2 | 6.7 % |
| Generalized 4 | 13.3 % |
| Right hemisphere 6 | 20 % |
| Left hemisphere 15 | 50 % |

OBSESSIVE COMPULSIVE DISORDER

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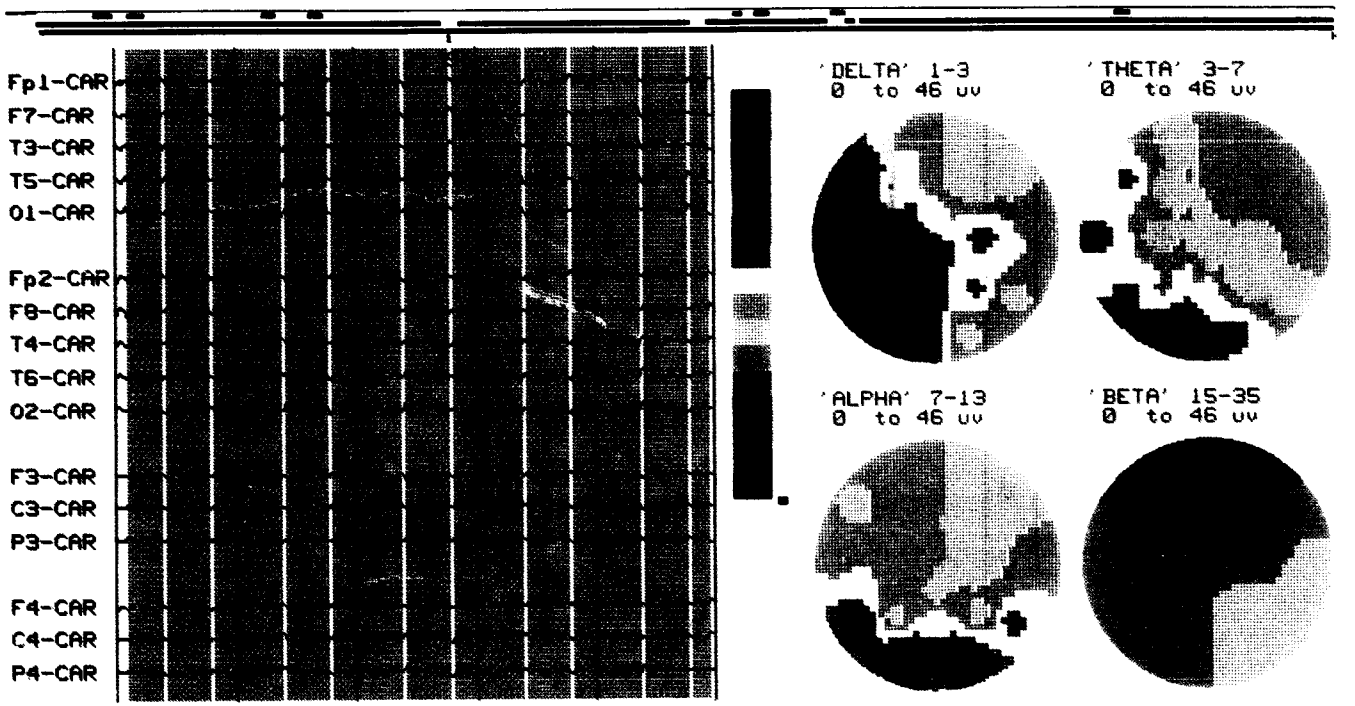


LEFT HEMISPHERIC DYSFUNCTION
PREDOMINANTLY TEMPORAL

OBSESSIVE COMPULSIVE DISORDER

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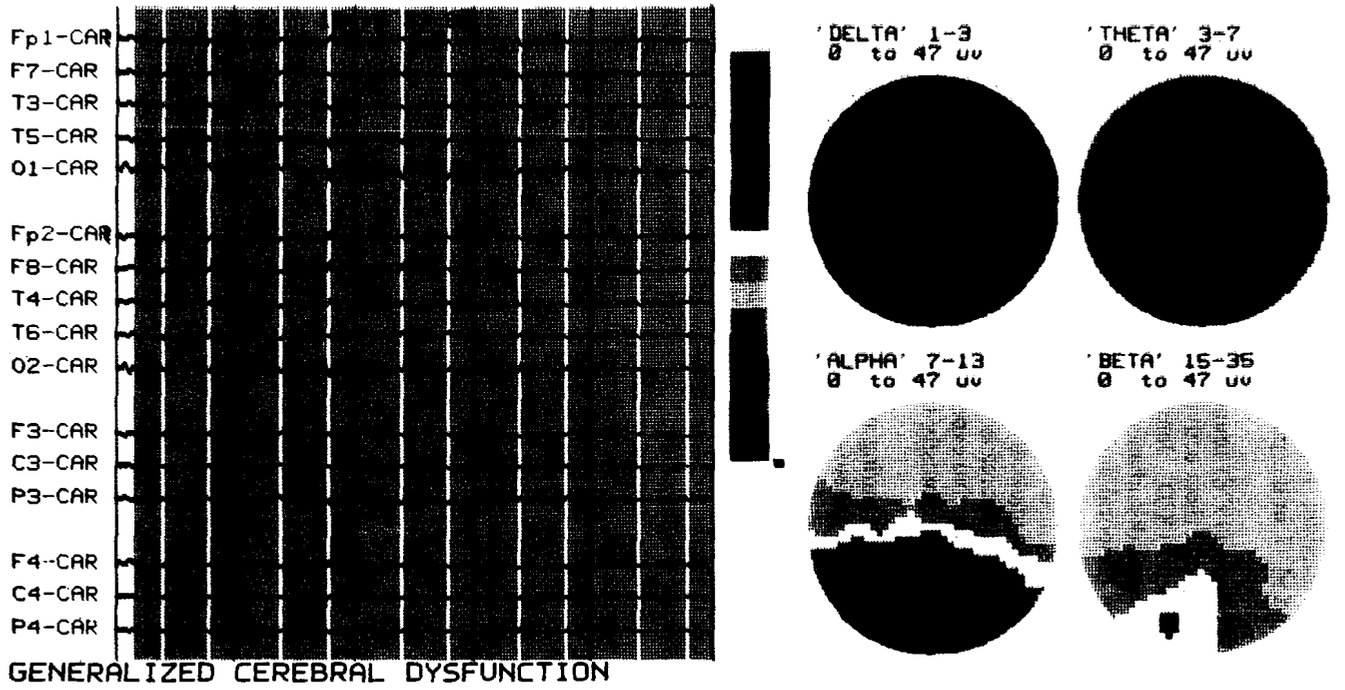


GENERALIZED CEREBRAL DYSFUNCTION

## DISCUSSION

In this study we found that 50% (15 cases) of our patients showed left hemispheric changes. 11 out of 15, had focal hemispheric dysfunction: four in the temporal region and 7 in the posterior quadrant.

Bingley & Persson (1978) found increased fronto-temporal theta activity in 5 of 35 subjects with OCD. Jenike & Brotman (1984) found disturbances predominantly in the temporal and fronto-temporal regions.

Zohar *et al.* (1988) found increased total cerebral blood flow markedly during imaginal flooding, but decreased even below relaxation levels in vivo exposure. These changes were found mostly in the left temporal region.

The greatest decreases during in vivo exposure was in the left parieto-occipital region.

They explained their finding on the basis that in vivo exposure, cerebral blood flow may represent a hypofunction of some parts of the brain, predominantly in the posterior left hemisphere.

None of our patients showed specific frontal lobe dysfunction as found in the work of Flor-Henry *et al.* (1979) who described left frontal lobe dysfunction in 11 subjects with OCD showing EEG abnormalities and neuropsychological test impairement.

Minski (1933) reported a case of OCD in a patient with left frontal lobe tumour.

Baxter *et al.* (1987) studied 14 subjects with OCD by PET and found an increase in left orbital gyrus and bilaterally in caudate.

Paunovic (1984) reported a patient who developed OCD after anterior dominant cerebral infarction.

Brikner (1940) reported compulsive repetition of the alphabet on stimulation of area 6 in patient with OCD.

O'Callaghan *et al.* (1982) found that lesions of the cingulate gyrus and lower medial quadrant of the frontal lobe have been found useful in OCD.

Latinen & Vikki (1973) have found that anterior septal ventral stereotactic cingulotomies below and in front of the knee of the corpus callosum were effective for tension but totally ineffective in OCD.

Changes in some of the early latency evoked potential (Shagass *et al.*, 1984) have been interpreted differently to implicate left hemisphere responsiveness, left frontal dysfunction and increased cerebral arousal.

However, such abnormalities in the EEG and similar neuropsychological tests were not found by Insel *et al.* (1984) in 18 cases of OCD.

Two cases of our patients had diffuse left hemispheric dysfunction, similar to the finding of Flor-Henry *et al.* (1979) who found dominant hemisphere dysfunction by computerized EEG.

Two of our patients had left mid-temporal hyperactive foci (sharp waves). Insel *et al.* (1983) found intermittent left temporal sharp waves in 2 of 18 cases with OCD.

Epstein & Bailine (1971) found temporal lobe spikes and theta waves in the sleep EEG during stage 1 and REM of three subjects with OCD.

Links between OCD and epilepsy have been noticed in few case reports. Napoleon had epilepsy and compulsive ritual of counting the number of windows in the building he passed. Parcella *et al.* (1944) reported two patients who developed petit mal few months after showing obsessive compulsive symptoms.

Another subject developed typical OCD when GME stopped suddenly after 15 years (Garmany, 1974). Stereotyped thoughts or forced thinking have been demonstrated as part of an epileptic aura (Penfield & Jasper, 1954), and obsessive compulsive symptoms may appear briefly during the aura of a fit associated with temporal lobe pathology or reappear transiently in the post-ictal phase

(Brikner *et al.* , 1940). Kettl& Marks (1986) reported two cases in which typical OCD developed in teenage patients shortly after the onset of epilepsy, one patient had GME, and the other TLE. Both patients were free of obsessive compulsive symptoms before the onset of epilepsy.

20% of our patients (6 cases) showed right hemispheric changes.

Four cases had right hemispheric dysfunction. This finding was not supported by many authors who studied subjects with OCD.

Although, khanna *et al.* (1987) found decreased power in the non-dominant fronto-medial and temporal regions. Hassler (1980) reported that right sided disruption of thalamofrontal pathway was in some cases sufficient to produce clinical recovery.

Some patients required subsequent operation in the dominant hemisphere.

We suggest that since depression is the most common complication of OCD (Goodwin *et al.* , 1969) and that in our previous study of brain mapping in affective disorder (Okasha *et al.* , 1988) we found that most of the depressed patients had right hemispheric dysfunction, this finding could be interpreted as a psychobiological link between OCD and affective illness, even in those obsessionals who do not manifest depressive symptoms. However, the nature of a link between OCD and affective illness is not clear.

Neither DST nor the sleep EEG abnormalities predict response to antidepressants in OCD (Insel *et al.* , 1984).

One explanation for the apparent psychobiological link between the two disorders is that patients with chronic OCD develop episodic depressions and that rather than displaying the affect common in major depressive disorder, these episodes are manifested as an exacerbation of obsessions and rituals.

Many patients with OCD are ill for years before they seek treatment, some apply for help when they become overtly depressed, some when their obsessive compulsive symptoms are more severe.

In both cases, the acute episode superimposed on a chronic disorder may be a from of affective illness.

Two cases of our patients showed right hemispheric hyperarousal, a finding which could not be supported by other authors, although it may suggest the presence of high cortical arousal as an evidence for anxiety associated with OCD.

The septo-hippocampal system model of Gray (1982) can explain some physiological basis of OCD. It was observed that increased emotional arousal precedes the onset of OCD. This may lead to oversensitivity of the hippocampal system which may mediate signals labelling previous mental stimuli as aversive. Similar studies in other anxiety disorders have generally found no abnormal results (Curtis *et al.* , 1982).

13. 3% of our cases (4 cases) showed non-specific generalized cerebral dysfunction and 6. 7% (2 cases) had borderline records, similar to the findings of Insel *et al.* (1983).

10% of our patients (3 cases) had normal records. These three patients did not appear to be differentiated from the remaining patients respecting age and clinical status.

However, these individual differences in clinical group indicated perhaps the need to take account of variables as subjective state at the time of recording and current information of obsessional states.

The data reported here and the studies cited previously, point to the prevalence of left hemisphere dysfunction and abnormal cognitive processing.

It is of interest to note that similar findings were also reported in psychotic disorders (Ciesielski *et al.* , 1981; Flor-Henry *et al.* , 1979; Ritter *et al.* , 1979; Shagass *et al.* , 1977; Saleut *et al.* , 1971; Duffy, 1986; Okasha *et al.* , 1988) which may suggest the existence of a link between OCD and psychosis.

## CONCLUSION

There may be a possible relationship between the left hemisphere and OCD and we can consider that obsessive compulsive symptoms were the result of abnormal cortical functions with abnormal cognitive processing.

However, the relationship between OCD and specific areas of the brain has varying interpretations.

Further studies with new brain imaging techniques, neuropsychological tests, better sampling of patients and discrimination of symptoms might unlock further secrets of this disorder and would also reduce the current discrepancies of opinion.

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## ABSTRAIT

### Le Substrat Neurophysiologique de la Maladie Nevrose Obsessionelle: Une Evidence du EEG Topographique

La maladie de la nevrose obsessionelle a été recement trouvée avec beaucoup de marque biochemiques. Cela a revie l'intérêt à son base biologique. La plus-part des traveaux récents ont souligné le rôle des neurotransmitters contribuants et des neurophysiologiques. Dans cette presentation, les evidences de l'étude electrophysiologique (Topographic EEG mapping) ont mis l'acçent sur un dérangement possible du hemisphère gauche associé avec cette maladie. Trente malades diagnostiqués comme ayant une maladie obsessionelle primaire selon le DSM III R, ont été examinés par un EEG topographique . Leurs âges varient de 16 a 45 ans avec une moyenne de 32.1 ans. 19 sont des hommes et 11 des femmes.

50 % de nos malades(15 cas) ont montré l'evidence de dérangement du hemisphère gauche, deux ont des endroits hyperactifs dans la region temporale . 20% (6 cas) ont montré l'evidence d'atteinte du hemisphère droit. 4 ont un dérangement du hemisphère droit et deux ont un hypereveillement de ce même hemisphère 10 %(3cas) ont des records normals.

13.3 % (4 cas) ont un dérangement cerebral general et 67 % (2 cas) ont des borderline records.

Nos resultats montrent une association plus significante entre l'hemisphère gauche et les symptomes obsessionelles.

# الموجز

## الأسس العصبية الفسيولوجية لمرض الوسواس القهرى

وجد حديثا ان اضطراب الوسواس القهرى يكون مصحوبا بعلامات كيميائية ، وقد أحيى ذلك الاهتمام بأصوله البيولوجية . وقد أكدت معظم الأبحاث الحديثة على دور الموصلات العصبية والنشاط العصبى الفسيولوجى الزائد .

وقد ركزت هذه الدراسة على المصاحبات الكهروفسيولوجية - من خلال رسم المخ المقطعى - كدليل على احتمال وجود خلل وظيفى مصاحب لهذا الاضطراب فى النصف الأيسر من المخ.

وقد شخص ثلاثون مريضا طبقا للدليل الامريكى الثالث لتقسيم الامراض النفسية ، ثم فحصوا من خلال رسم المخ المقطعى الكهربائى . وقد كانت أعمارهم تتراوح بين ١٦ إلى ٤٥ عاما بمتوسط ٣٢,١ عاما ، ١٩ منهم من الذكور و١١ أنثى .

وقد أظهر ٥٠٪ من المرضى (١٥ حالة) دلائل اضطراب وظيفى فى نصف المخ الأيسر فى حين أن حالتين وجدت عندهم بؤرة نشطة فى المنطقة الصدغية ، وقد ظهر فى ٢٠٪ من المرضى (٦ مرضى) دور نصف المخ الأيمن ، وكان منهم ٤ حالات تميزت بالإضطراب الوظيفى للنصف الأيمن وحالتين تميزتا بزيادة الإثارة ، وكانت قياسات ١٠٪ من الحالات (٣ حالات) طبيعية ، وأظهر ١٣,٣ (٤ حالات) اضطرابا عاما فى المخ ، وحصل ٦,٧٪ (حالتين) على نتائج بينية .

ولقد دلت نتائج هذا البحث على وجود ارتباط ذى دلالة أعلى بين النصف الايسر من المخ وأعراض الوساوس القهرية .