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# Brain mapping in obsessive compulsive disorder

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The overall prevalence of O.C.D. ranged from 1.9 - 3.3 in five communities in U.S.A., which is a rate from 25 to 60 times greater than previous estimates. In Egypt we have up to one million sufferers. (Okasha, 1990). 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, serves to activate pathological behaviour. An alternative approach to explaining the phenomena has involved the search for neurological basis for O.C.D. (Schilder, 1938, Bear and Fedio, 1977). Many of the behaviours shown by O.C.D. patients seem to resemble the fixed action patterns described by Lorenz that they are hard wired into the brain circuitry. The ritualized aspect of the behavior and its striking uniformity along with the fact that children and adults show identical symptoms, suggest biological preprogramming. (Okasha, 1990). The disease is more prevalent in relatives suggesting a genetic cause. It has been shown repeatedly that O.C.D. occurs in associations with many neurological disorders: Sydenham's chorea, epilepsy, parkinsonian disease and toxic lesions of basal ganglia. A study by Rapoport (1989), showed with CAT Scans of the brain of O.C.D. patients smaller caudate volumes. Positron emission tomography showed O.C.D. patients had higher level of glucose metabolism in an area of the frontal lobe and in cingulate gyrus which connects the frontal lobe with the basal ganglia. Okasha (1990) had formulated a hypothesis about the possible biological basis of O.C.D. He suggested that latent behavioural patterns stored in the basal ganglia are somehow triggered by abnormally functioning inferior frontal lobes. The initiating impulses are conveyed to the basal ganglia by pathways mediated by serotonin. Successful drug treatment might alter the role of serotonin in those pathways. Brain Mapping with computerized EEG has revealed many abnormalities in O.C.D. Recently Okasha and Raafat (1990), have found that 90 % of the cases showed abnormal EEG and 10 % had normal records. 70 % showed evidence of hemispheric lateralization, where 50 % showed left hemispheric dysfunction and 20 % showed right hemispheric dysfunction from which 13.3 % showed non-specific generalized cerebral dysfunction and 6.7 % had borderline record. They interpreted their findings as pointing to a significant association between left hemispheric dysfunction and obsessive symptoms. However, in explaining their finding of a right hemispheric dysfunction; they suggested that, since depression is the most common complication of O.C.D., the previous finding could be interpreted as a psychobiological link between O.C.D. and

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affective illness. Within the context of our study, we have tried to replicate the previous findings. In agreement with previous research, we found left frontal dysfunction in ritual group in compared with normal one. We also found generalized cerebral dysfunction in rumination group when it was compared with normal group.

We also found right hemispheric dysfunction in the ritual group when it was compared with normal group.