SUMMARY

The overall pervalence 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 pervious estimates. In Egypt we have up to one million sufferers. (OKasha, 1990).

Current behavioural theories centre on the nation that an obsession is a learned behaviour which becomes established through its anxiety reliving properties. However, this simple explaination fails to deal with many puzzling features of the disorder, such as whey the performance of rituals often increases rather 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 seen to resemble the fixed action patterns described by lorenz that they are hard wired into the brain circuitry. The rilualized aspect of the behavior and its statling uniformity along with the fact that childern and adults show identical symptoms, suggest biological preprograming. (Okacka, 1990).

The disease is more pravalent in relatives suggesting a genetic causes. It bas shown repeatedly that O.C.D. occurs in associations with many neurological disorders: Sydeham'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. smaller caudate volumes.

Position 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 stared in the basal ganglia are somehow triggered by abnormally functioning inferior frontal lobes. The intiating 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 evidences of hemispheric lateralization, where 50 % showed left hemispheric dysfunction and 20 % showed right hemispheric dysfunction from which 13.3 % showed non - specific generalized cereberal dysfunction and 6.7 % had borderline record. They interpreted their findings as pointing to a significant association between left hemispheric dysfunction and

obsessional symptoms. However in explaining their finding of a right hemispheric dysfuncion; 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 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 carebral 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.