

EDITORIAL

The Biology of Obsessive Compulsive Disorder

The overall prevalence of OCD ranged from 1.9-3.3% in five communities in USA, a rate from 25 to 60 times greater than previous estimates i.e. in Egypt we may have up to one million sufferers.

The work in ethology by Konard Lorenz who described nest building, grooming, courtship and defensive behavior patterns that appeared without learning models and he hypothesized that they are hard wired into the brain circuitry. Many of the behaviors shown by OCD patients seem to resemble the fixed action patterns described by Lorenz. It is obvious that cultural and physical stimuli account to some degree for a particular patient's symptoms, but the ritualized aspect of the behavior and its startling uniformity along with the fact that children and adults show identical symptoms suggest biological preprogramming.

The disease is more prevalent in relatives suggesting a genetic cause. About 20% of OCD patients display motor tics: involuntary movements that are usually blinks of the eye or facial grimaces. It has been shown repeatedly that OCD occurs in associations with several types of neurological disorders: Sydenham's chorea, epilepsy, Parkinson's disease, Tourette syndrome and toxic lesions of the basal ganglia. The basal ganglia are known to be way stations between sensory inputs and the resulting motor and cognitive outputs. It is possible that in OCD, disturbances in these way stations have somewhat short circuited the loop that normally connects sensory input and behavioral output, thereby releasing stored hard wired behavioral packages?

A study by Rapoport (1989) showed with CAT scans of the brain of OCD smaller caudate volumes. Another study showed that 20% of rheumatic fever with chorea displayed OCD which may suggest a dysfunction in the basal ganglia.

Positron emission tomography (PET) showed OCD patients had higher level of glucose metabolism in an area of the frontal lobe and in the cingulate gyrus which connects the frontal lobe with the basal ganglia.

Two different types of treatment of OCD may be effective: behaviour therapy mainly exposure-prevention and antiobsessional drugs. Behavior therapy does not contradict the biological basis. Ethnologists have shown that many fixed action patterns in animals can be extinguished by repeated training. Moreover, since the brain is both a biological organ and the recipient of sensory and psychological inputs, it is only to be expected that strictly psychological causes can have biological effects. Behavior therapy seems to be more effective in treating compulsions than obsessions.

On the other hand new drug treatments seem to be effective in reducing both obsessions and compulsions. Three drugs have been shown to have anti-OCD effects: clomipramine, Fluvoxamine and Fluoxetine. Their effects are not mediated through their antidepressive properties as most antidepressants are not helpful in OCD. The effectiveness of these drugs is probably related to the physiology of serotonin.

The role of serotonin, in human behaviour is not well understood, but there is evidence that it is important in suicide, appetite, aggression, depression among other functions. All these drugs are characterized by being potent reuptake inhibitors of se-

rotonin i.e. increasing its turnover. No one has shown any abnormality of serotonin either in the blood or spinal fluid in OCD patients and the precise mode of actions of these drugs remains to be elucidated.

To formulate a hypothesis about the possible biological basis of OCD, it seems possible that latent behavioural patterns stored in the basal ganglia are somehow triggered by abnormally functioning inferior frontal lobes. The initiating impulse is conveyed to the basal ganglia by pathways mediated by serotonin. Successful drug treatment might alter the role of serotonin in those pathways, thereby damping the spark from the frontal lobes.

We have to differentiate between impulse control disorders e.g. Trichotillomania, Kleptomania, pathological gambling where the act produces gratification as compared with obsessions where no gratification is established. But whether these disorders will respond to those antiobsessional drugs will be the subject of future trials and research.

Brain Mapping with computerized EEG has revealed high abnormalities in OCD. Recently Okasha & Raafat (1990) have shown a high incidence of left hemispheric dysfunction, mainly temporo-parietal.

Some authors according to the biological substrate of OCD divide it into many subtypes which will underline the method of therapy.

We have: (1) OCD and Tourette syndrome responding to serotonin drugs and neuroleptics; (2) Schizo-obsessives responding to neuroleptics; (3) OCD and depression, they are hard to treat, 50% may respond to serotonin drugs and further 20% with the addition of lithium. Adjuvant therapy with fenfluramine, clonidine and sometimes psychostimulants; (4) OCD & anxiety symptoms or panic attacks may respond to benzodiazepines and serotonin drugs. There is probably a serotonergic OCD, may be a dopamine OCD, a peptide OCD and OCD that is mainly related to environmental Factors.

OCD patients have a risk avoidance, are meticulous, have multiple rituals and they have high 5HIAA in the C.S.F. In the impulsive suicidal or homicidal patients, you have the reverse, with very low 5HIAA. It may be that serotonin systems may have something to do with a dimension that at one end may be impulsivity and the other end, harm avoidance, or obsessions and compulsions.

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