# Obsessive-compulsive disorder, the brain and electroconvulsive therapy

***Obsessive-compulsive disorder, classified as an anxiety disorder, arises from malfunctioning of the fronto-striatal-pallido-thalamic-frontal circuit in the brain. As well as perseverative thoughts and acts, there are other signs of 'organicity'. Delay in treatment is often a result of patients' reluctance to seek help, and doctors' insufficient knowledge and experience in this clinical area. Electroconvulsive therapy is effective in severe and refractory cases.***

The film 'The Aviator' by Martin Scorsese about the famous 20th century American billionaire Howard Hughes brought obsessive-compulsive disorder (OCD) into a public domain. Other famous people suffered from this disorder including John Bunyan, Martin Luther, Samuel Johnson, Erik Satie, and probably Maurice Ravel. Senseless repetitive thoughts and actions form the core symptomatology, but less well known are the accompanying signs of 'organicity' such as cognitive impairment, 'soft' neurological signs, lack of drive and initiative, self-neglect, disinhibition and sometimes general functional decline. Stress and trauma often precipitate the onset, and high anxiety fuels the condition, while a pre-morbid personality can differ wildly. Hughes' end was as sad as it was telling – an adventurous, successful and rich man ended his life as a recluse, in squalor and dejection. What he might have benefited from is not a makeshift analytical couch, but vigorous psychiatric treatment, including electroconvulsive therapy (ECT).

More recently OCD has been viewed as part of a spectrum disorder that includes such conditions as trichotillomania, hypochondriasis, body dysmorphia, eating disorders, compulsive shopping, Gilles de la Tourette's syndrome, severe nail-biting, excessive gambling, severe nose picking, and bowel and urinary obsessions. All these conditions are characterized by some form of intrusions.

## Definition

The *Diagnostic and Statistical Manual of Mental Disorders, 4th edition* (DSM-IV) (American Psychiatric Association, 2000) classifies OCD as an anxiety disorder characterized by the presence of obsessions and compulsions that are a source of distress to the individual and do not form a part of another major mental disorder, e.g. schizophrenia or major depression.

Obsessions are defined as:

**'recurrent, persistent ideas, thoughts, images, or impulses that are ego-dystonic, i.e. they are not experienced as voluntarily produced, but rather as thoughts that invade consciousness and are senseless or repugnant. Attempts are made to ignore or suppress them'.**

Compulsions are defined as:

**'repetitive and seemingly purposeful behaviours that are performed according to certain rules or in a stereotyped fashion'.**

The *International Classification of Diseases (tenth edition)* (ICD-10) (World Health Organization, 1992) definition overlaps to a considerable degree. For a diagnosis to be made obsessional symptoms or compulsive acts, or both, must be present on most days for at least two successive weeks, be a source of distress or interfere with activities. A relationship between OCD and depression is emphasized, and a disorder is likely to run a more chronic course in the absence of depressive symptoms.

## An evolution of the concept: towards a neurophysiological model

Alarcón and colleagues (1994) traced the earliest descriptions of OCD to 15th century religious texts on demonology. Hartley made an attempt at a systematic explanation of obsessional ideation ('fixed and recurrent ideas') in 1749. In the first half of 19th century Esquirol described the 'reasoning monomanias or partial deliria', later the same century Kraft-Ebbing coined the term 'obsessive representation', Griesinger wrote about 'ruminative sickness' (Grübelnsucht) and Legrand du Saule described the 'touching madness'. In 1896 Freud (1955) emphasized the intrusive toilet training in anal stages of development, and subsequent submissive–hostile attitude towards authority figures, as the underlying aetiopathogenesis of 'obsessional neurosis'. But even he conceded that there might be a biological basis for this.

It was the 'Heidelberg school' that, among others, laid the foundation for a somatic basis of OCD. In 1938 Schilder, having critically reviewed the available literature, asserted that at least two thirds of such cases had organic causes. Later, Grimshaw (1964) reported that 19.4% of OCD patients had a history of neurological illness, as opposed to 7.6% in a control group. He went to suggest that 'obsessional behaviour is a general dispo-

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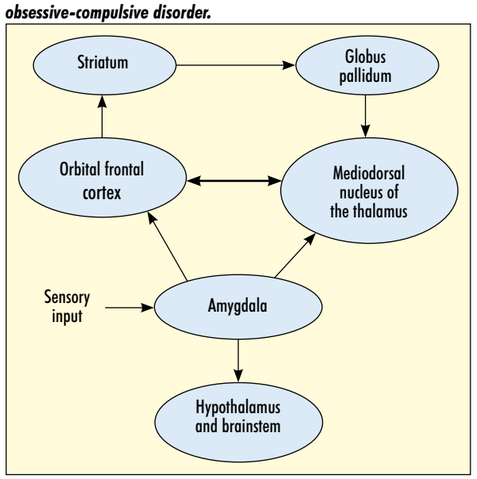
sition which can be released by the breaking of higher performances' and that 'neurological disease isolates subcortical perseveratory activity from higher cortical centres'. Modell et al (1989) have postulated that 'OCD symptoms occur when an aberrant positive feedback loop develops in the reciprocally excitatory frontothalmic neuronal interchange' as a result of inadequate inhibition by the limbic portion of the striatum. The failure of the fronto-striatal system in recognizing a completion of the task and inhibiting further response has been recognized by several researchers (Connolly and Burns, 1993; Rosenberg et al, 1997).

Zald and Kim (1996) have focused on a dysfunction of the orbital frontal cortex (OFC) and related neuronal circuits. The authors claim that OCD arises from an excessive disinhibition of the mediodorsal nucleus of the thalamus, pars magnocellularis (MDmc). This disinhibition might allow the information (normally inhibited by the thalamus) to pass freely through MDmc to OFC, which becomes overactive. Positive feedback loops involving the OFC-striatal-pallidal-MDmc structures become established, and this leads to a perseveration of information processed in the MDmc–OFC axis (*Figure 1*).

OFC has a strong association with the amygdala, which has been recognized as a locus of traumatic emotional memory that escapes categorization, sequencing and evaluation, hence its role in a post-traumatic stress disorder (Van der Kolk et al, 1996). Post-traumatic stress disorder shares a number of features with OCD – not least a sense of intrusiveness – and this requires further exploration.

Several neurotransmitters have been implied in the pathoneurophysiology of OCD: serotonin, acetylcholine and dopamine (Rauch and Jenike, 1993). Of these the serotonergic model has gained most currency in view of a positive response to treatment with serotonergic antidepressants. Such treatment appears to reduce metabolic overactivity in frontocingulate and striatal regions (McGuire, 1995).

***Figure 1. Some neuronal structures and circuits involved in obsessive-compulsive disorder.***




## Epidemiology

The prevalence of OCD in a world population is approximately 2–3% (Karno et al, 1988) and these rates seem to be consistent across the cultures. Onset occurs at a young age, usually 19.8 (+/- 9.6) years. Fewer than 15% of patients present with OCD after the age of 35 years. Commonly there is 10 years delay before an index clinical presentation and this might be because of a reluctance to report and seek help by those who suffer from it. In her illuminating book, *The Boy Who Couldn't Stop Washing*, Judith Rapoport (1990)* calls OCD sufferers the world's greatest actors who know well how to dissimulate and cover up their affliction. They suffer their anguish and mental pain in solitude and silence. Childhood onset often starts with counting, checking and repetitive movements; in adolescence washing predominates; in adulthood it is mostly ruminations. Any personality may be afflicted, and paradoxically a meticulous, orderly type is a minority. The symptoms wax and wane, and they are often chronic and debilitating, while an episodic course, precipitated by a life event, may be more susceptible to treatment and carry a better prognosis.

Prognosis depends on the duration of the condition, treatment and patient's cooperation, but complete remission is rare. Males with an earlier onset tend to have a worse prognosis than comparable female ones. In a 15-year study of outpatients with OCD Rasmussen and Tsuang (1986) found that 84% of the sample had a chronic or continuous course, 14% had a deteriorating course, and only 2% had an episodic course with symptom-free periods. Demal and colleagues (1993) reported an episodic course with partial remission in 24% of patients, a deteriorating course in 10%, a chronic unchanging course in 27%, and a continuous course with some improvement in 24%.

Obsessive-compulsive phenomena have been observed in post-encephalitic parkinsonism (Jellife, 1932), Sydenham chorea (Swedo et al, 1989), Gilles de la Tourette's syndrome (Pauls et al, 1986) and Huntington's disease (Cummings and Cunningham, 1992). These conditions share the location of pathology in the striatum and/or inferior prefrontal cortex.

## Clinical presentation and neuropsychology

### Persevarations

#### Obsessions

The most frequent content of obsessions are thoughts about danger or harm to the self (e.g. fear of contamination), feelings of doubt, aggressive or sexual impulses and somatic concerns. Fear of having contracted acquired immunodeficiency syndrome (AIDS), a sense of having committed some impropriety or offence, or passed on some illness to others or having oneself some serious illness, are among the examples. In early onset, counting is common. Urges to perform violent or embarrassing acts, e.g. jumping in front of the car, injuring a child or shouting blasphemies in church, have also been reported.

*This book is highly recommended for those who want to understand the disorder, the sufferers and problems encountered in treatment and care.

#### Compulsions

These are stereotyped actions that the person feels compelled to carry out. Most frequently performed are handwashing, checking, cleaning teeth, taking showers, changing clothes and attempts at bowel emptying. Compulsions may cause problems in themselves as frequent washing may lead to eczema, or frequent teeth brushing may lead to gum lesions. Sometimes, as in the case of Howard Hughes, fears of contamination and frequent washing are at odds with the external chaos or squalor.

In contrast to delusions, obsessions and compulsions are viewed as irrational, yet irresistible for the individual afflicted. Some patients report short-lived relief from anxiety after performing the rituals.

### Cognitive impairment

Christensen and associates (1992) found a significant deficit in non-verbal memory and visuo-spacial abilities among OCD patients. A controlled study by Jurado et al (2002) highlighted the patients' impairment in 'temporal ordering' and 'feeling-of-doing' judgment, suggestive of their lack of self-awareness of the performance. Difficulty in shifting cognitive sets and persevarative errors found in OCD patients are consistent with orbito-frontal deficits (Head et al, 1989; Spitznagel and Suhr, 2002). Cavedini et al (2002) compared a decision-making process in OCD, panic subjects and controls, and found it impaired in the OCD group. The patients also show impaired ability to forget unpleasant material (Tolin et al, 2002).

Short-lasting amnesic episodes and confusion in three OCD patients with compulsive checking was reported by Thomasanterion et al (2002). If a biography of Howard Hughes is anything to go by, Brown and Broeske (1996) reported such amnesic episodes in his case.

### Movement abnormalities

These include slowness, hesitancy in movement initiation, loss of motor fluency, motor stereotypies, involuntary and mirror movements, and abnormalities of gait (McGuire, 1995).

Slowness is an independent feature, and not a result of perseverative symptoms 'taking a long time'. It is akin to parkinsonian bradykinesia and may be related to a disturbance in basal ganglia. It could be accompanied, as in parkinsonism, by bradyphrenia.

### Problems with volition and motivation

Loss of psychic self-motivation, behavioural inertia, a loss of the emotional component of experiences, and a kind mental emptiness has been found in OCD, as a result of organic lesions of basal ganglia (Laplane, 1994). The most severe form of inertia is abulia – a lack of spontaneous, goal-directed behaviour. It is seen predominantly with lesions of the basal ganglia and of the frontal lobes, the brain structures involved in OCD. It can be easily overshadowed by more noticeable perseverative symptoms or by anxiety, but its impact could be seriously debilitating. It can be mistakenly interpreted as depression, but lack of depressive cognition and biological symptoms of depression should help in differential diagnosis. The treatment might include dopamine agonists, but further research is needed in this area.

## Psychiatric comorbidity and misdiagnosis

There is a high rate of co-morbidity with OCD. Depression and anxiety have been reported in two thirds of patients (Rasmussen and Eisen, 1994) and bipolar affective disorder and cyclothymia may also co-exist (Kruger et al, 2002). There is a high life-time prevalence of specific phobia (22%), social phobia (18%) and panic disorder (12%) (Rasmussen and Eisen, 1991). Autistic traits are reported in 19% of OCD patients (Bejerot et al, 2001). Co-morbid post-traumatic stress disorder renders OCD less responsive to treatment (Gershuny et al, 2002). Obsessive-compulsive symptoms in a setting of schizophrenia seem to carry a poor prognosis (Fenton and McGlashan 1986). In cases with prominent abulia a diagnosis of 'process schizophrenia' is the most frequent clinical error made by doctors (including psychiatrists). This may be because many psychiatrists have only limited experience with OCD (Rapoport, 1990). The consequences of such misdiagnosis could be devastating for the patient.

## Aetiology

Traumatic head injury may affect neuronal circuits that underlie OCD symptomatology (Grados, 2003).

Infection, particularly a streptococcal type, has been linked with occurrence or exacerbation of OCD through autoimmune reactions (Murphy et al, 2001).

Approximately 20% of relatives have clinical OCD, and further 15% have a subclinical form, with a childhood-onset having a particularly high genetic predisposition (Riddle et al, 1990). A higher incidence of anxiety and affective disorders has also been reported in the families of OCD patients.

Life events before onset are reported as more common and more severe in the group with low familial loading (Albert et al, 2002). Stress and accompanying anxiety can exacerbate symptoms and is paired with changes in cerebral blood flow. The parent–child relationship may play a role – there is limited evidence that parental overprotection plays a role in development of OCD, as a retrospective study has shown (Turgeon et al, 2002). It is important to avoid ascribing the disorder to the elusive 'faulty parenting'.

Childhood trauma, again ascertained retrospectively, is higher in OCD sufferers (Lochner et al, 2002).

Several of these aetiological factors (childhood infection, 'overprotective' mother, head injury, life events and stress) were hinted at in Scorsese's film.

## Brain imaging

Functional neuroimaging techniques such as single photon emission computed tomography and positron emission tomography have reported increased activity in the orbito-frontal and anterior cingulate cortex, or the neostratum, when compared with controls (Machlin et al, 1991; Perani et al, 1995). Symptomatic improvement was associated with a reduction in metabolism in areas previously overactive, e.g. caudate nucleus (Hansen et al, 2002). Structural brain imaging has been less convincing; an earlier report of bilateral reduction in volume of the caudate nuclei on computed tomography scan (Luxemburg et al, 1988) was not replicated with the use of magnetic resonance imaging (Kellner et al, 1991).

## Treatment

Pharmacological treatment includes the use of selective serotonin re-uptake inhibitors (SSRIs) such as sertraline, citalopram, fluvoxamine and fluoxetine (McGuire, 1995), with a response rate of 50%, compared with 35% on placebo. Augmentation with atypical antipsychotics such as risperidone and quetiapine has been reported (Sun et al, 2001; Mohr et al, 2002).

Behavioural/cognitive strategies including exposure and response prevention and cognitive-behavioural therapy have been successfully used in individuals, groups and families (Marks et al, 1988; Thienemann et al, 2001).

Reported efficacy of the above treatments seems to vary, but Dominguez and Mestre (1994) claim that irrespective of treatment modality only 30–50% reduction in symptoms is anticipated.

Aiming to 'interrupt reverberating circuit' Mitchell-Heggs et al (1976) reported definite clinical improvement in 89% of 27 OCD patients 16 months after stereotactic limbic leucotomy. More recently cingulotomy, transcranial magnetic stimulation, gamma knife procedure and deep brain stimulation have been used in treatment-refractory cases (Dougherty et al, 2002; Rasmussen et al, 2003).

### KEY POINTS

- Obsessive-compulsive disorder is a relatively common, serious and debilitating illness, often present for a decade before it is reported, diagnosed and treated.
- It is a neurobiological condition, accompanied by a number of 'soft' neurological signs and cognitive deficits.
- Medication, behavioural/cognitive strategies, or a combination thereof, effects a 30–50% reduction in symptoms.
- Electroconvulsive therapy should be tried for treatment-resistant cases before invasive methods are considered.

## ECT for OCD

Thus far there has been no report in the British psychiatric literature as to the efficacy of ECT in patients with OCD, but such reports have appeared in American and European literature (Beale et al, 1995; Rabheru and Persad, 1997). Several single cases of refractory OCD that responded satisfactorily to ECT have been reported (Wohlfart, 1996), including an elderly woman (Casey and Davis, 1994). One larger study of 32 patients with a severe disorder was reported, with most subjects showing considerable and lasting improvement after a course of ECT (Maletzky et al, 1994), independently of the improvement in depressive symptoms. The patients usually required more ECT for their OCD than for their depressive symptoms. Some patients relapsed on discontinuation and needed a maintenance ECT (Husain et al, 1993; Casey and Davis, 1994). This article had been already accepted for publication when the National Institute for Health and Clinical Excellence (NICE) published their guidlines for OCD (NICE, 2005). The guidelines do not mention the use of ECT.

## Conclusions

OCD is a brain dysfunction with considerable co-morbidity, and may have a devastating effect on the patient's life. Increasing public and medical awareness is vital for early diagnosis and treatment. Routinely this includes SSRIs and behavioural/cognitive strategies, but ECT and surgical methods are useful in refractory cases. The relationship between OCD, trauma and post-traumatic stress disorder needs further exploration. **BJHM**

*Conflict of interest: none.*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