OBSESSIVE COMPULSIVE DISORDER: IS IT A PROBLEM OF COMPLEX MOTOR PROGRAMMING?*

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SUMMARY

44 subjects with Obsessive compulsive disorder (OCD) and 40 normals were compared using an experimental paradigm involving recording of the bereitschaftspotential. A decreased onset latency and increased amplitude was found in the OCD sample as compared to normals. A neurophysiological substrate for the bereitschaftspotential has been proposed. The implications of these findings in OCD as compared to Gilles de la Tourette syndrome, and for a focal neuro-physiological dysfunction have also been discussed. The findings of this study implicate a dysfunction in complex motor programming in OCD, with the possibility of this dysfunction being in the prefrontal area.

Introduction

Obsessive compulsive disorder (OCD) has been an area of scientific scrutiny since the time of Sir Aubrey Lewis who was 'struck by the variety of problems and the difficulty of stating them' (Lewis 1936). Earliest attempts to explain obsessional states are described in the Malleus Maleficarum as witchcraft, devils and possession (Kramer and Sprenger 1951). Earlier theories stressed the psychological origins of OCD (Freud 1955). Although Schilder (1938) stressed the possibility of an organic basis, this approach has gained momentum only in recent years. Evidence for a biological substrate has accumulated from genetic, neuro-psychological and psychosurgical studies and single case reports (Jenike 1983, Lieberman 1984, Turner, Beidel and Nathan 1985).

Cummings and Frankel (1985) sought to compare Gilles de la Tourette syndrome (GTS) and OCD to derive a neurological hypothesis for obsessive-compulsive phenomenon. Many patients with GTS had obsessions and compulsions. The clinical similarities between these two disorders, their occurence among members of the same families, and the fact that both can be observed as symptoms of known basal ganglia disease have led them to suggest that they may share common neurological mechanisms. They have gone on to hypothesise that tics and vocalisations of GTS are aberrant manifestations of simple motor programmes that are spontaneously generated by the basal ganglia and that obsessive-compulsive phenomena represent such abnormal complex motor plans.

A compulsion is a repetitive and seemingly purposeful behaviour which is performed according to certain rules or in a stereotyped fashion. It is associated with a sense of subjective compulsion coupled with a desire to resist it. An obsession on the other hand is an ego-dystonic recurrent persistent idea, image or impulse (American Psychiatric Association 1980). The role of volition in both of these phenomena seems clear. To put it differently, these

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phenomena may represent complex motor plans which are experienced subjectively (obsessions) or executed motorically (compulsions).

One of the paradigms which involves most of the components required in complex motor programme (Marsden 1982) is the bereitschaftspotential (Kornhuber and Deecke 1964). This is a slow DC potential generated during the volitional process of a motor task. The literature on the bereitschaftspotential (BP) in psychiatric patients is scant. There is a single report in OCD in which this potential was not observed in recordings from two patients (Cazzullo et al 1981). To try and test the hypothesis put forward by Cummings and Frankel (1985) we decided to use this paradigm in a sample of OCD subjects and compare the findings with a normal control sample.

Material and Methods

44 subjects who met DSM III criteria for OCD (American Psychiatric Association) 1980 were taken as the patient sample. The subjects had been drug free for a mini-

mum period of one month prior to the experiment. They were selected from the Out Patient Department of the National Institute of Mental Health and Neurosciences, Bangalore, after obtaining informed consent. Patients who had depression were also included in this sample if (a) there was a gap of atleast 2 months between the onset of depression after OCD, (b) the depression as clearly secondary to the obsessive compulsive phenomenon and (c) no melancholic or psychotic features were present (modified from Insel et al 1983). Subjects with a concomitant history suggestive of drug abuse or dependence, mental retardation, metabolic disorders, hypertension or with a recurrent or past history of any neurological disorder were excluded from this study. Only subjects in the age group 18 to 55 years were taken.

40 normal healthy volunteers matched for age and sex formed the control group. Present or past history of psychiatric illness or neurological disorder, and history of any psychiatric illness in the first or second degree relatives were used as exclusion criteria.
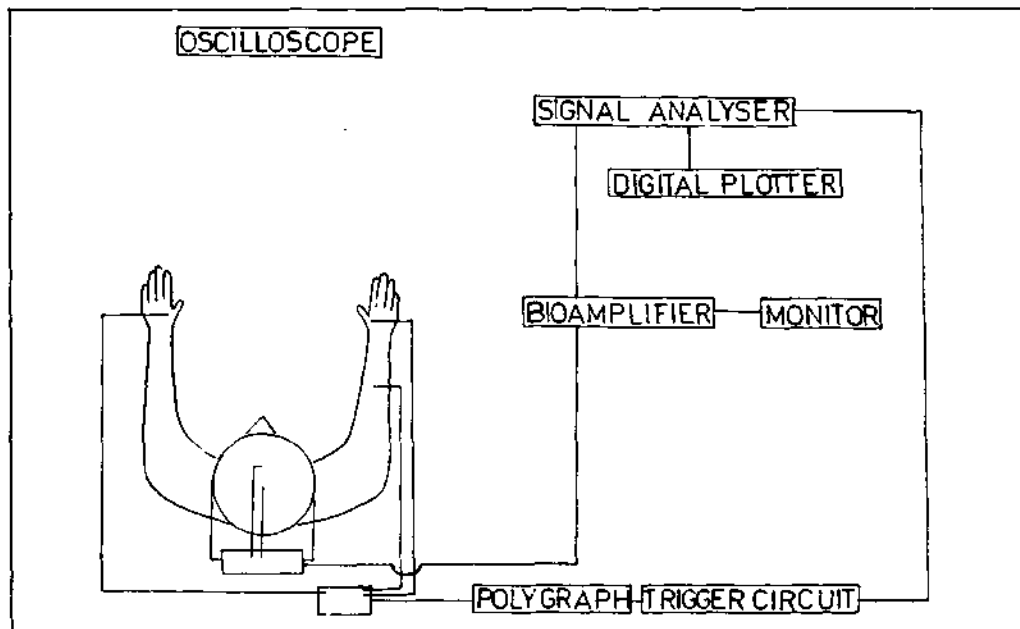
The methodology used for recording the BP (Figure 1) has been described earlier (Mukundan et al. 1986). The subject was seated in a sound proof room. Silver/silver chloride disc electrode were placed at Fz and Cz with linked ear lobes as the reference and the ground electrode on the nasion, using the 10-20 system (Jaspers 1958). For recording the EMG, the electrodes were placed on the right thenar eminence and forearm and grounded with an electrode placed on the left thenar eminence. The subject was asked to observe the 10 cm. screen of a Phillips PM3226 Oscilloscope placed directly in front at a distance of 1 metre. The subject was asked to observe the ± 1 cm. of the middle of the screen which was marked with bright yellow vertical lines. He was instructed to press two microswitches on both sides, on the arm rests, with the thumbs when a slowly moving trace was anywhere between these two lines. The subject was also asked to visually fixate on the oscilloscope and avoid eye blink or lateral eye movements. The microswitches required mild pressure and the excursion of the button was 2 mm. and could be operated by ramp movement. Bilateral responding was used to avoid any lateralising effect of the potential. The trace took 6 seconds to traverse the entire length of the screen and another one second to reappear at the other end.

The electroencephalogram was recorded on an AB621G Nihon Kohden Bioamplifer system with a modified time constant of 14 seconds and a high frequency cut off at 10 Hz. A polygraph channel of a Medicare unit picked up the muscle activity when the subject pressed the microswitches. This was fed into a trigger circuit with a threshold control so that a trigger was delivered at the onset of the muscle activity. This could be achieved by adjusting the sensitivity of the polygraph as well as the threshold control on the trigger circuit.

The electroencephalogram was recorded on an Iwatsu SM 2100B Single analyser with an analysis duration of 5 seconds, a data length of 1024 addresses per channel and a negative delay of 4 seconds. Serial averagings of 49 recordings were taken. A program was used for artefact rejection. The signal analyser was calibrated using the gain ratio for a 50 microvolt signal. Each subject was initially given a practice run of atleast ten trials.

The amplitude and onset latency of the BP were measured from the Fz and the Cz recordings. Amplitude was the maximum negativity reached before the premotion positivity started. The baseline activity in the initial 1 to 2 seconds was first determined from the mid-points of the wave form, and the onset of the BP was considered as the point of the baseline from which there was a continuous negative deflection of more than 2 microvolts, as this was found to be the maximal baseline shift.

Results

There were 44 subjects in the OCD sample, comprising 29 males and 15 females, with a mean age of 27.6 yrs. (SD = 5.1 yrs). The normal sample consisted of 26 males and 14 females with a mean age of 29.8 yrs. (SD = 5.8 yrs.). There were no statistically significant differences in the age and sex composition of these two groups.

A typical BP recording is shown in figure II. Amplitude is represented in Figure III and latency in figure IV, for recordings from both the Fz and the Cz. The amplitude was higher for OCD as measured from the Fz ($T = 2.04$, $P < 0.05$) and Cz ($T = 2.01$, $P < 0.05$). The findings were more significant for the onset latency, OCD sample having shorter latencies as measured from both the Fz ($T = 7.07$, $P < 0.001$) and the Cz ($T = 9.54$, $P < 0.001$).
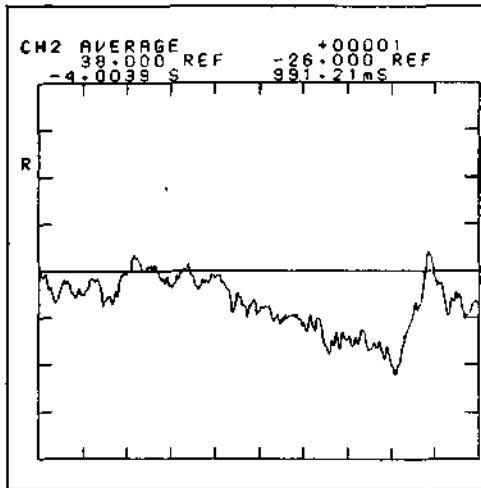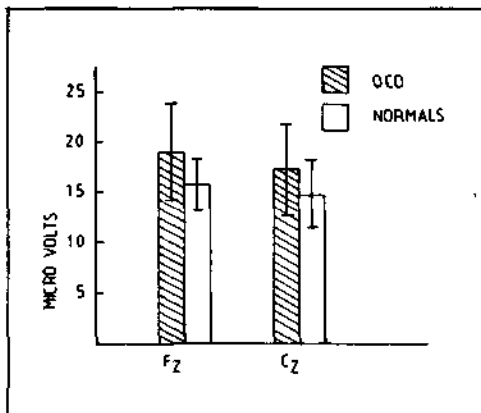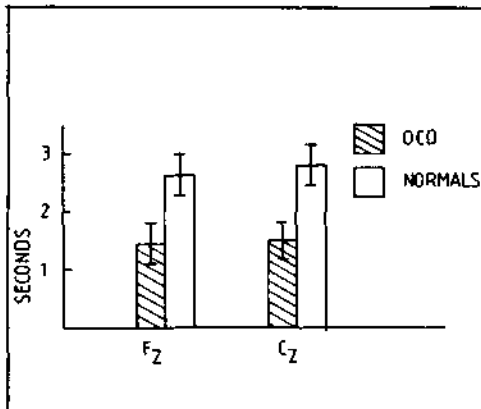
Fig. II. BP

Fig. III. Amplitude

Fig. IV. Onset Latency

Discussion

Onset latency has usually been taken as an important indicator of readiness in the BP; the longer the onset latency, the earlier the onset of readiness to act. The finding of this study would thus implicate a deficit in 'readiness' in OCD and lend support to Cummings and Frankel's (1985) hypothesis about an abnormality in complex motor programming in OCD.

The concept of complex motor programming has been put forth by Marsden (1982) and Humphrey (1983). This is presented in a modified form in figure V, with appropriate equivalents in the paradigm employed in this study. When external cues are involved, the first mental processes would involve their sensation and perception. The equivalent in this experiment is seeing the moving spot of light on the oscilloscope and using information from the association areas to judge or decide when it is in the middle of the screen. From here the pathways go to the lateral cerebellum and basal ganglia. The next, hypothesised part of this pathway is the ventrolateral thalamic area through which the fibres pass and there is evidence for involvement of some of the proposed structures in this pathway. Let us now come back to our findings and their implications in OCD. In GTS, Obeso et al. (1981, 1982) found the BP to be absent before tics, which Cummings and Frankel (1985) went on to regard as simple motor programs. We have presented earlier how obsessions and compulsions can be regarded as the subjective and motorically executived components in the motor programming circuit. Tics in GTS are apparently due to basal ganglia involvement and do not involve complex motor programming. Devinsky (1983) suggested the involvement of the periaqueductal grey matter and midbrain tegementum in this disorder. The evidence cited does not support any cortical involvement, and
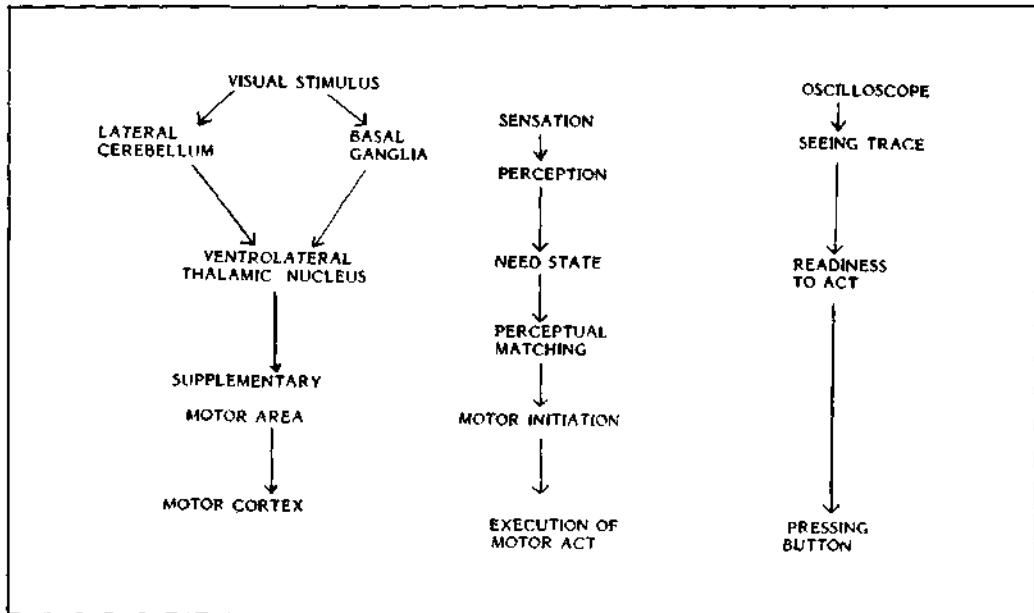
Fig. V

the tics and vocalisations perhaps occur due to the aberrant simple motor programs executed at this sub-cortical level. The neurotransmitter implicated in GTS is dopamine, with stimulation of supersensitive receptors producing tics (Devinsky 1983). The meager evidence based on CSF monoamine metabolite studies and drug response implicates the serotonergic system in OCD (Cummings and Frankel 1985) as proposed initially by Yarvura Tobias et al. (1977). Cummings and Frankel (1985) consider that since the primary dopaminergic and serotonergic cell masses are at the subcortical level, these findings do not invalidate their hypothesis. Dopaminergic subcortical involvement with impairment of simple motor programming has thus been shown in GTS; in OCD a serotonergic dysfunction resulting in faulty complex motor programming is likely to be present.

It would perhaps be too early to state exactly where the deficit in the complex motor programming circuit lay in OCD. When studying alcoholics Mukandan et al.

(1986) found a decreased latency with normal amplitude in the BP, which they interpreted as a volitional deficit. Absence of amplitude differences was interpreted as this parameter being a function of the force and complexity of the motor act. They also suggested that amplitude and latency most probably had different generators. Looking at their findings from a neurophysiological perspective, it is important to remember that chronic alcohol intake is related to cortical dysfunction (Lishman 1978), so much so that Tarter et al. (1984) have proposed a prefrontal deficit as a predisposing neuropsychological model for alcoholism. The delay in onset latency may thus be due to cortical dysfunction rather than the involvment of the basal ganglia. However, on the other hand, studies involving the basal ganglia have largely implicated differences in amplitude (Deecke 1985). It would be interesting to postulate that while the amplitude of the BP is a function of sub-cortical structure, onset latency is more under cortical control.

Khanna et al. (1985) reported a case

where OCD developed after head injury. Neuropsychological evaluation was suggestive of predominant bilateral frontal lobe dysfunction. There we had commented on the similarity between obsessions and perseveration. Both could be due to frontal dysfunction; the latter being differentiated by an inability to change set. A further comparison can be drawn with the premotor syndrome described by Luria (1966) as an early sign of involvement of the prefrontal cortex, which precedes a later stage of development of obvious perseveration.

Other evidence for frontal lobe involvement in OCD comes from psycho-surgery, where sectioning of frontal pathways is associated with a decrease in obsessionality (O'Callaghan and Carroll 1982). A dominant frontal dysfunction has been proposed by Flor-Henry et al. (1979) based upon electrophysiological and neuropsychological studies. Isolated case reports of frontal lobe lesions producing obsessive-compulsive phenomenon also exist (Jenike 1984). The evidence implicating the basal ganglia in OCD is more meager. OCD was more common in patients with von Economo's encephalitis, where the primary pathology was subcortical (Cummings and Frankel 1985).

The aim of this paper has been three fold. Firstly, with a review of the relevant literature, we have tried to expound a hypothesis about the neurophysiological substrate of the BP, using complex motor programming as a paradigm. Secondly, we have tried to show how such a paradigm can be applied to psychiatric subjects. Finally, with reference to our sample, we have been able to show a deficit in complex motor programming in OCD. The exact site of this deficit in the context of the OCD sample is difficult to define at this juncture, but the frontal lobe seems to be a strong candidate, as has been discussed. Nevertheless, further techniques will have to be used, and this study replicated, before any definite conclusions can be categorically drawn.

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