Memory functioning in obsessive-compulsive disorder

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A number of studies have reported neuropsychological deficits in obsessive-compulsive disorder (OCD). These have mainly implicated frontal or temporal dysfunction. In this study, we compared the performances of OCD patients and normal subjects using a factorial interpretation of the Wechsler Memory Scale. Our results do not demonstrate significant memory impairment in OCD patients but point to the possibility of frontal lobe dysfunction as a factor in the pathophysiology of OCD.

Keywords: Memory – Neuropsychology – Obsessive-compulsive disorder (OCD) – Wechsler Memory Scale (WMS)

INTRODUCTION

Patients with obsessive-compulsive disorder (OCD) sometimes complain of memory disturbance. Mnesic functions have classically been associated with left temporal lobe dysfunction (Delaney et al., 1980) but the complex circuits which connect limbic structures to the frontal lobe are also of considerable importance (Fuster, 1989). According to the classical model (Mishkin, 1982), memory seems to be related to two pathways, the first involving the amygdala and the connections with the medial frontal cortex, the second involving the hippocampus, anterior thalamic nuclei and their connection with the DLPC and temporal cortex. Both systems are thought to be of importance in recognition memory (Bachevalier and Mishkin, 1986), but DLPC dysfunction has also been associated with deficits in selective association and immediate learning (Sass et al., 1987; Helkala et al., 1988) and recall memory impairment in DLPC damaged patients (Grafman et al., 1986) as well as in schizophrenics (Goldberg et al., 1989, 1990).

A link between frontal lobe dysfunction and cognitive deficits has been reported in OCD (Flor-Henry, 1974, 1983; Malloy, 1987; Modell et al., 1989; Rosenthal and Fedio, 1975). More recent studies have also identified non-verbal memory deficits (Christensen et al., 1992), difficulties in neuropsychological tasks involving spatial abilities (Hymas et al., 1991) and in reproduction of complex figures (Cox et al., 1989). Impairment of visual, memory and intellectual processes on the Luria-Nebraska battery (Moses et al., 1983; Bellini et al., 1989), spatial-perceptual abnormalities (Behar et al., 1984), impaired performance in the Wisconsin Card Sorting Test (Grant and Berg, 1948; Lezak, 1983; Hymas et al., 1991) and deficits on tasks involving attention (Insel et al., 1983) have also been reported.

The Wechsler Memory Scale (WMS; Wechsler, 1945) is a useful and well-standardized instrument for global memory assessment. Poor performance on the WMS has been described in normal volunteers with checking behavior (Sher et al., 1984), in a subgroup of OCD patients (Sher et al., 1989), and in OCD children (Behar et al., 1984).

In this study we evaluate the WMS performances in 43 OCD patients compared with 39 sex, age, handedness and education matched normal subjects. We used a factorial WMS profile rather than individual performance in terms of raw subtest scores or the global memory level (Quotient of Memory). This evaluation of the WMS scores was proposed as a ‘more rational scale of measurement’ (Kear-Colwell, 1973) to emphasize the relationship between single factorial score and selective mnesic performance.

SUBJECTS AND METHODS

Eighty-two subjects (43 OCD patients and 39 normal subjects matched for age, sex, educational level and handedness) participated in the investigation. Table I shows the clinical and demographic characteristics of the sample. The 43 OCD patients were all recruited from the Anxiety
TABLE I. Clinical and demographic characteristics of the sample

<table>
<thead>
<tr>
<th>Factor</th>
<th>OCD (n = 43)</th>
<th>Controls (n = 39)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>Age (years)</td>
<td>30.8 (9.9)</td>
<td>30.4 (6.7)*</td>
</tr>
<tr>
<td>Education (years)</td>
<td>10.3 (3.5)</td>
<td>11.7 (4.1)**</td>
</tr>
<tr>
<td>Onset</td>
<td>20.6 (7.4)</td>
<td></td>
</tr>
<tr>
<td>Duration of illness</td>
<td>10.4 (8.5)</td>
<td></td>
</tr>
</tbody>
</table>

Sex
M = 20 F = 23 M = 23 F = 16***

* ANOVA: F(1,80) = 0.05, p = N.S.
** ANOVA: F(1,80) = 2.90, p = N.S.
*** Chi-square = 0.83 with 2 df, p = 0.36 (Yates corrected).

TABLE II. Clinical characteristics of the OCD sample

<table>
<thead>
<tr>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Washers</td>
<td>5 7</td>
</tr>
<tr>
<td>Checkers</td>
<td>9 3</td>
</tr>
<tr>
<td>Mental checkers</td>
<td>2 6</td>
</tr>
<tr>
<td>Mixed</td>
<td>4 7</td>
</tr>
</tbody>
</table>

Chi-square = 5.97 with 3 df, p = 0.12

<table>
<thead>
<tr>
<th>BOCS scores</th>
<th>Mean</th>
<th>S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obsessions</td>
<td>13.5</td>
<td>3.4</td>
</tr>
<tr>
<td>Compulsions</td>
<td>13.2</td>
<td>3.4</td>
</tr>
<tr>
<td>Total score</td>
<td>26.7</td>
<td>5.7</td>
</tr>
</tbody>
</table>

In-patients Unit at the Psychiatric Branch of S. Raffaele Hospital. Two psychiatrists assessed the diagnosis by means of the computerized version of DIS-R (Robins et al., 1989) according to the DSM-III-R criteria (APA, 1987). In addition, OCD symptomatology was assessed using the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) (Goodman et al., 1989a,b). At the time of their neuropsychological evaluation, all the OCD patients had been treated with fluvoxamine maleate (mean dosage of 234 ± 35 mg/day) for at least 2 months. No patients had taken benzodiazepines in the 2 weeks preceding the tests. Table II shows the phenomenology of OCD patients.

The 39 normal subjects were recruited from hospital employees and nursing staff; the inclusion criteria for recruitment were a personal negative history of neurological and psychiatric illnesses, alcohol abuse, history of documented head injuries, loss of consciousness, neurosurgical treatment or perinatal trauma. Before the tests all subjects were submitted to a complete physical and neurological examination in order to exclude any somatic illness. All subjects were right handed as judged by a handedness questionnaire (Raczkowski et al., 1974). Informed consent about the purposes of the study was obtained from each subject before testing.

Testing procedures
The WMS is a widely used task for assessing global memory impairment. The WMS-R was administered by a trained neuropsychologist in a quiet laboratory according to standard procedures (Wechsler, 1945). The time to administer the complete task never exceeded 30 min. The examiner was not blind to diagnosis. The WMS battery is based on seven subtests: Orientation, Information, Mental Control, Logical Memory, Digit Span, Visual Reproduction and Associate Learning. According to the model proposed by Kear-Colwell (Kear-Colwell, 1973), a more rational WMS interpretation can be made by means of factor scores rather than the raw subtest scores or the Quotient of Memory. This model is based on the following formulae for converting subtest scores to factor scores:

Factor I = -0.27 + (0.25 × Logical Memory) + (0.19 × Visual Reproduction) + (0.21 × Associate Learning)
Factor II = -1.80 + (0.28 × Information) + (-0.34 × Orientation) + (0.43 × Mental Control) + (0.45 × Digit Span)
Factor III = -9.68 + (0.78 × Information) + (2.34 × Orientation)

Factor I concerns immediate learning and recall abilities, Factor II includes attention and concentration and Factor III orientation and long-term information recall (Skilbeck and Woods, 1980).

Our group has recently demonstrated the validity of this approach in the neuropsychological evaluation of mental disorders (Colombo et al., 1993).

Statistical analysis
Analysis of variance (ANOVA) was performed in order to assess the groups’ differences on the three WMS factors. Pearson’s product-moment correlations between WMS and BOCS scores were produced to evaluate the relationship between test performances and symptom severity. Kruskal-Wallis one-way ANOVA was performed to compare the differences between the OCD subtypes.

RESULTS
Table III summarizes the WMS results in the OCD and control groups. Among the WMS factor scores, only Fac-

TABLE III. WMS performances in OCD patients and normal subjects

<table>
<thead>
<tr>
<th>Factor</th>
<th>OCD Mean (S.D.)</th>
<th>Normal subjects Mean (S.D.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor I</td>
<td>6.78 ± 1.74</td>
<td>7.34 ± 1.76</td>
</tr>
<tr>
<td>Factor II</td>
<td>6.06 ± 1.45</td>
<td>6.31 ± 1.58</td>
</tr>
<tr>
<td>Factor III</td>
<td>5.66 ± 1.31</td>
<td>6.50 ± 0.40**</td>
</tr>
</tbody>
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* p < 0.01.
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tor III was significantly lower in the OCD patients \( [F(1,80) = 14.75, p < 0.002] \). No significant differences were found in OCD subtypes with regard to the other two WMS factors. No sex differences were found in the three WMS factor scores. No significant correlations at the \( p = 0.01 \) level were found between OCD symptomatology (BOCS scores) and WMS factor scores.

DISCUSSION

The results of this investigation reveal a WMS profile in which the OCD patients show selective mnemonic deficits when compared with normal subjects. Age, gender, educational level and handedness differences between the groups were not statistically significant, so it is reasonable to suppose that these variables do not play a critical role in the WMS performances. The OCD patients showed poorer performances when compared with normal subjects in the WMS Factor III, which has been related to orientation and long-term information recall (Skilbeck and Woods, 1980).

Even though we have utilized a standardized interpretation of the WMS by means of a factorial model, separate analysis of the two subtests (Information and Orientation) subsumed under Factor III may be useful in order to clarify their neuropsychological meaning. The Information subtest can be considered as a measure of verbal skills and remote memory; in brain injured populations, information is the least affected of the WMS and WMS subtests (O'Brien and Lezak, 1981) and has been considered a poor predictor of left hemisphere involvement (Smith, 1966; Spreen and Benton, 1965). Therefore, these findings cannot support the hypothesis of localized cerebral malfunctioning. As far as the Orientation subitem is concerned, some authors consider impairment an expression of frontal dysfunction (Benton, 1968; Joslyn and Hutchell, 1979), and marked impairment in Orientation can be found in cases of bilateral lesions of frontal lobes. Orientation impairment is a frequent symptom of brain disease involving the cortical areas and is closely related to attention deficits (McGhie, 1969; Eis dorfer and Cohen, 1980). OCD deficits on the orientation subitem may be related therefore to mild disturbances in attention, resulting from frontal lobe dysfunction (Posner and Petersen, 1990). This finding appears to be in agreement with a hypothesis of frontal lobe involvement in OCD (Flor-Henry et al., 1979; Modell et al., 1989). It is also possible that the everyday problems reported by OCD patients may be an expression of attention and orientation problems.

We have not found differences in WMS performances between checkers or washers, as reported elsewhere (Sher et al., 1984), or a significant correlation between memory abilities and clinical symptomatology. These findings suggest that in terms of memory it is difficult to differentiate OCD patients into distinct clinical subgroups. We have also failed to find significant correlations between symptom severity (i.e. time spent in compulsions or obsessions) or the duration of illness and WMS performances.

The results obtained suggest that memory deficits may occur in OCD and that frontal rather than temporal dysfunction seems to be principally involved. This finding appears to be consistent with the recent biological models which attribute a fundamental role to pathways connecting the frontal lobe to subcortical regions in OCD psychophysiology (Wise and Rapoport, 1989).

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