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Executive function and nonverbal memory in obsessive-compulsive disorder

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Abstract

It has been suggested that memory impairments found in obsessive-compulsive disorder (OCD) are mediated by organizational problems in encoding that are caused by primary executive dysfunction. Performance on different nonverbal memory and executive skills was tested in 68 subjects (35 non-depressed OCD sufferers and 33 healthy controls). Multiple regression models were performed to analyze the role of different cognitive variables, especially organizational encoding strategies in nonverbal memory. OCD patients performed significantly worse than controls in immediate nonverbal memory [Rey–Osterrieth Complex Figure Test (RCFT)] and on all the executive functions such as interference control (Stroop test), mental set shifting (Trail-Making Test), and organizational strategies (copy organization). As no differences were found in the memory of faces, where organizational strategies are minimal, it is possible to speculate that immediate nonverbal memory problems in OCD appear only when organizational strategies mediate the recalling process. Thus, memory deficits appear to have less to do with memory, per se, and more to do with the degree of organization necessary to effectively complete the task. Statistical analyses of mediation models showed the highest explanatory power for the organizational approach and demonstrated the mediation effect of organizational strategies in nonverbal impairment.

Keywords: Obsessive-compulsive disorder; Memory; Executive function; Organizational strategies

1. Introduction

Obsessive-compulsive disorder (OCD) is a psychiatric condition characterized by intrusive unwanted thoughts and/or repetitive and stereotyped behaviours severe enough to interfere with a person's ability to function on a daily basis (American Psychiatric Association, 1994). In recent years, OCD, formerly classified under neuroses, has been reinterpreted as a neuropsychiatric disorder with an organic basis (Stein, 2002). Accumulated data from clinical, electrophysiological, psychosurgical, pharmacological, and neuroimaging investigations point to dysfunction within

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associative frontostriatal circuits, specifically those involving the caudate nucleus and mediobasal areas of the frontal cortex (Khanna, 1988; Baxter et al., 1992; Baxter, 1994; Kwon et al., 2003).

Globally, neuropsychological findings are consistent with the frontostriatal etiologic hypothesis of OCD and include impairments in a variety of domains. However, neuropsychological studies in OCD are still not definitive as there are some inconsistent results (Abruzzese et al., 1995; Boone et al., 1991). It is possible that such inconsistency could depend on factors including clinical state, medication effects, intelligence, or the heterogeneity of symptoms. Executive dysfunction and impairment in nonverbal memory have been the most frequent findings across the studies (for reviews, see Alarcón et al., 1994; Greisberberg and McKay, 2003; Kuelz et al., 2004). Additionally, several impairments ranging from visuospatial skills (Hollander et al., 1993; Aronowitz et al., 1994; Cohen et al., 1996) to selective executive functions (Veale et al., 1996; Purcell et al., 1998a; Schmidtke et al., 1998) have been reported. Nevertheless, it has been suggested that memory impairment plays an important role in OCD as patients frequently express memory complaints, and one of the hallmark symptoms of the condition is a persistent doubt about the adequacy of previous actions despite their repetition (Tallis et al., 1999).

The pioneering work of Sher et al. (1983, 1984, 1989) showed memory impairments in clinical and nonclinical checkers. However, it is only recently that memory has been investigated using neuropsychological tests. Christensen et al. (1992) found that there was a difference with controls, specifically for delayed nonverbal recall but not for verbal memory, either short-term or delayed. Martinot et al. (1990) found deficits in all assessed areas of memory functioning, including visual, verbal, and numerical memory. Purcell et al. (1998b) showed impaired spatial working memory in OCD sufferers relative to other conditions such as panic or unipolar depression and to healthy controls. The accumulated empirical evidence of memory impairment in OCD has led to the formulation of different models in attempts to determine whether there is an underlying cognitive substratum of symptoms.

The most classical model is the ‘mnemonic hypothesis’ based on the assumption that memory deficits would represent the cognitive substrate of doubt-related phenomena such as checking (Tallis, 1995). Unfortunately, the link between memory deficits and symptom severity has not been well established. Some studies reported a significant link (Gross-Isseroff et al., 1996, Tallis, 1997; Tallis et al., 1999), but others could not find any link (Flor-Henry et al., 1979; Okasha et al., 2000). Furthermore, mixed results have also been reported (Christensen et al., 1992; Boone et al., 1991). The absence of such correlations might suggest that memory deficits should not be considered causal with respect to compulsive checking or other related symptoms. In addition, selective memory deficits in OCD have been difficult to link to specific underlying brain systems (Martinot et al., 1990).

The ‘organizational approach’ formulated by Savage (1998) tries to explain memory impairment in OCD from a more comprehensive perspective. This approach is based on four general concepts: (1) the main cerebral dysfunction in OCD is in associative frontostriatal circuitry; (2) frontostriatal dysfunction produces a secondary nonverbal memory impairment as a consequence of primary executive dysfunction; (3) neuropsychological dysfunction influences clinical symptoms; and (4) symptoms interact with cognitive dysfunctions creating a vicious circle. According to the model, memory impairments found in OCD patients could be mediated by impaired strategic processing problems caused by a primary executive dysfunction (Savage et al., 1995, 1999).

To date, some empirical evidence supporting the organizational model has been found in neuroimaging (Savage et al., 2000b) and cognitive studies. Memory problems secondary to executive dysfunction as described in the model could potentially be related to frontostriatal system dysfunction (Baxter et al., 1987, 1988; Rauch et al., 1994; Savage et al., 2000b). However, the relationship between executive dysfunction and clinical symptoms has not been a consistent finding as some positive (Harvey, 1987; Mataix-Cols et al., 1999) and some negative results (Cox et al., 1989; Christensen et al., 1992) have been reported. From a neuropsychological point of view, there is increasing evidence linking executive dysfunction and secondary memory impairments. Thus, verbal and nonverbal memory impairments have been reported in a series of studies as being related to impairments in