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Clinical correlates of word recognition memory in obsessive–compulsive disorder: An event-related potential study

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Abstract

Memory disturbances found in obsessive–compulsive disorder (OCD) may partially be related to dysfunction of cortico–subcortical circuits. However, it is still unknown how OCD symptomatology is related to memory processing. To explore this question, event-related potentials (ERPs) were recorded in a continuous word-recognition paradigm in OCD patients with either severe or moderate scores on the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) (group S and group M, $n=8$ each) and in normal healthy controls ($n=16$). Typically ERPs to repeated items are characterized by more positive waveforms beginning approximately 250 ms post-stimulus. This "old/new effect" has been shown to be relevant for memory processing. The early old/new effect (*ca.* 300–500 ms) with a frontal distribution is proposed to be a neural correlate of familiarity-based recognition. The late old/new effect (post-500 ms) is assumed to reflect conscious memory retrieval processes. The OCD group S showed a normal early old/new effect and a reduced late old/new effect compared with group M and the control group, but no difference was found between group M and the control group. Source analyses for the late old/new effect showed statistically reduced cerebral activation in the anterior cingulate for OCD group S in contrast to the control group. Additionally, the early old/new effect in OCD group S was negatively correlated with the Y-BOCS total scores, and the late old/new effect was negatively correlated with obsession sub-scores. The severely, not moderately, ill OCD patients showed an impaired conscious recollection of the word-to-be-remembered, which suggested an impairment of working memory capacity in these patients due to a dysfunction in the frontal and cingulate cortex.

Keywords: Obsessive–compulsive disorder; Y-BOCS; Event-related potential; Word recognition; Old/new effect; Source analysis

1. Introduction

Obsessive–compulsive disorder (OCD) is characterized by recurrent obsessions and compulsions that are time-consuming, interfere with daily living, and are distressing to the patient (American Psychiatric Association, 1994). A disturbance of fronto–striato–thalamic regulatory loops has been repeatedly demonstrated by brain-imaging studies (Swedo et al., 1989, 1992; Baxter et al., 1992; Rauch and Jenike, 1997; Rauch et al., 1997, 2001; Schmidtke et al., 1998). Additionally, the characteristic symptoms of OCD, such as chronic doubts, repetitive checking, ruminations, decreased behavioral flexibility and ritual behavior (Savage and Rauch, 2000),

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have been associated with neuropsychological deficits, that have been found in the areas of learning and memory (Christensen et al., 1992; Dirson et al., 1995; Cohen et al., 1996; Savage et al., 1999), visuospatial processing (Cohen et al., 1996) and executive functions (Abbruzzese et al., 1997; Purcell et al., 1998a,b).

A strategic memory deficit has been shown by Savage et al. (1999) in a non-verbal memory task and by others using verbal materials (Deckersbach et al., 2000; Savage et al., 2000; Cabrera et al., 2001; Henin et al., 2001). These studies required the organization of encoded material. Other authors have pointed out that memory decisions in OCD patients are usually made with reduced confidence (Radomsky and Rachman, 1999; Radomsky et al., 2001; Tolin et al., 2001). This phenomenon, known as memory bias, is thought to be responsible for the peculiar insecurity of OCD patients.

Neurophysiological studies on auditory and visual event-related potentials (ERPs) in patients with OCD have suggested the existence of abnormal sensory and cognitive information processing compared with processing in normal controls (Towey et al., 1994; de Groot et al., 1997; Miyata et al., 1998; Johannes et al., 2001; Dietrich et al., 2005). Additionally, these abnormalities in OCD have been related to clinical severity. In an auditory "oddball" paradigm, Towey et al. (1990, 1993) found a shorter latency of the N200 and P300 wave and a greater negativity in the N200. In particular, the later study (Towey et al., 1993) found that higher N200 amplitude correlated with less severe obsessions, better response to subsequent treatment with serotonin reuptake inhibitors, and fewer neurological soft signs in OCD. Such correlations support the view that OCD is a heterogeneous diagnostic syndrome and exists along a continuum with different clinical and neurological features. Also, such relationships suggest the potential value of ERP measures for identifying a subtype of treatment-responsive OCD patients with distinctive clinical and psychobiological features (Towey et al., 1993). Another relevant investigation from de Groot et al. (1997) revealed negative correlations between the severity of OCD symptomatology and the mean amplitude of infrequent waveforms in the region of the slow waves, which suggested that clinical symptomatology may be reflected in the ERP waveforms.

The present investigation deals with the neural correlates of memory problems of OCD patients that may be modulated by clinical severity. In a continuous word-recognition paradigm, the subject is exposed to a list of words that appear successively on a video monitor. Some of the words are repeated after several intervening items, and the subject's task is to classify each word as "new" (i.e. first appearance in the word list) or "old" (repeated appearance). In this explicit memory test, correctly classified old items elicit more positive-going waveforms than new items, beginning approximately 250 ms after word presentation (Rugg and Nagy, 1989; Rugg and Doyle, 1992, 1994). This "old/new" effect is thought to be caused by a modulation of several ERP components, i.e. a reduction of the negative N400 component and an enhancement of the late positive component (LPC or P3b) (Rugg, 1995; Rugg et al., 1996, 1997, 1998a,b; Friedman and Johnson, 2000; Mecklinger, 2000). The early old/new effect between approximately 300 and 500 ms after stimulus onset, bilateral shift with a frontal distribution, differed according to whether items were old or new but was insensitive to the depth of processing manipulation. This is a form of recognition memory assumed by some authors to be independent of recollection and to underlie recognition judgments associated with 'Know' rather than 'Remember' responses (Tulving, 1985). According to these functional properties, Rugg et al. (1998a,b) identified the effect as a correlate of familiarity or implicit memory. The late old/new effect (post-500 ms) is divided into two parts. One onsets around 400–500 ms post-stimulus and is maximal over the left parietal scalp, which appears to be a neural correlate of the episodic recollection of study items (Wilding and Rugg, 1996). Another onsets quite late (*ca.* 700 ms), persists for a second or more, and is distributed over the right frontal scalp (so-called right-frontal old/new effect) (Wilding and Rugg, 1996), a component that tends to be small or non-existent in a simple word-recognition test (Allan et al., 1998). This effect is held to reflect the retrieval of contextual information from the study episode and is assumed to reflect various strategic processes that operate on the products of memory recollection or retrieval (Allan et al., 1998; Rugg et al., 1998a,b; Friedman and Johnson, 2000; Mecklinger, 2000). Using this continuous word-recognition paradigm, one of our recent studies (Zhang et al., 2005) demonstrated that OCD patients showed a normal early old/new effect and reduced late old/new effect at the left fronto–temporal electrode sites compared with the controls. Impaired word- recollection processing in OCD appears to be interpreted as consistent with a dysfunction in the frontal and cingulate cortex that underlies the etiology of this disorder. Nevertheless, few studies have correlated these changes of memory processing with clinical severity measures.

To this end, the current study was undertaken to investigate the correlation of the neural substrate of word-memory processing in the continuous word-recognition paradigm with the clinical severity of OC symptoms in