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Emotional memory in depersonalization disorder: A functional MRI study

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Abstract

This study examines emotional memory effects in primary depersonalization disorder (DPD). A core complaint of DPD sufferers is the dulling of emotional responses, and previous work has shown that, in response to aversive stimuli, DPD patients do not show activation of brain regions involved in normal emotional processing. We hypothesized that DPD sufferers would not show the normal emotional enhancement of memory, and that they would not show activation of brain regions concerned with emotional processing during encoding and recognition of emotional verbal material. Using fMRI, 10 DPD patients were compared with an age-matched healthy control group while performing a test of emotional verbal memory, comprising one encoding and two recognition memory tasks. DPD patients showed significantly enhanced recognition for overtly emotive words, but did not show enhancement of memory for neutral words encoded in an emotive context. In addition, patients did not show activation of emotional processing areas during encoding, and exhibited no substantial difference in their neural responses to emotional and neutral material in the encoding and emotional word recognition tasks. This study provides further evidence that patients with DPD do not process emotionally salient material in the same way as healthy controls, in accordance with their subjective descriptions of reduced or absent emotional responses.

Keywords: Depersonalization; Emotional memory; Contextual processing

1. Introduction

Depersonalization (DPD) is defined in the DSM-IV (American Psychiatric Association 1994) as an 'alteration in the perception or experience of the self so that one feels detached from and as if one is an outside observer of ones mental processes or body'. The subjective DPD experience is one of disturbing unreality in the experience of one's physical and emotional state, and often occurs with derealisation (DR), the experience of a similarly strange and unreal quality to one's surroundings. DPD is a relatively common phenomenon, with a prevalence rate variously estimated at 2.4% to 20% in the general population (Simeon et al., 1997). It frequently occurs as a transient phenomenon in

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healthy individuals under certain conditions e.g. when sleep-deprived, or while under the influence (or recovering from the effects) of alcohol or drugs. It may also occur in a range of psychiatric contexts such as panic disorder (Segui et al., 2000) and major depression (Sedman, 1966), and in neuropsychiatric conditions such as temporal lobe epilepsy (Lambert et al., 2002). In a minority of patients, it occurs as a persistent and disabling phenomenon in the absence of other psychopathology, and is then considered to be a primary disorder.

One of the key complaints of many patients with DPD is that they feel their emotional responses are numbed or even non-existent. Davidson (1966) coined the term "de-affectualization" to describe this phenomenon. De-affectualization is not usually accompanied by an objectively blunted affect such as that seen in chronic schizophrenia (Ackner, 1954; Torch, 1978; Sierra and Berrios, 1998). Striking first-person accounts of de-affectualization abound in the literature (Mayer-Gross, 1935; Shorvon et al., 1946; Sedman, 1970), and similar descriptions are common amongst patients attending a specialist DPD clinic (Phillips et al., 2001a; Baker et al., 2003). A recent analysis of responses of DPD patients to a detailed phenomenological questionnaire identified de-affectualization as a key part of the depersonalization syndrome (Sierra et al., 2005). Furthermore, our group have shown that psychophysiological measures of autonomic arousal are reduced in response to aversive stimuli in DPD patients (Sierra et al., 2002). Functional neuroimaging provides an opportunity to examine the effects of emotion on cognition in the brain in DPD.

In a previous fMRI study of patients with primary DPD (Phillips et al., 2001b), we examined the neural correlates of viewing aversive and neutral scenes. A markedly different pattern of brain activation was seen in DPD subjects compared to healthy controls and a patient control group, providing some evidence for abnormal emotional processing in patients with DPD. In particular, DPD patients showed reduced neural responses in emotion-sensitive brain regions (such as posterior occipital–temporal cortex and insula), and increased responses in regions associated with emotion regulation (inferior and lateral frontal cortex), when viewing aversive stimuli. In addition there was evidence that the neural responses to emotive and neutral stimuli were not as distinct in patients with DPD as they were in normals. The current study aimed to build on these findings by examining emotional memory in patients with DPD. Since emotional processing appears to be abnormal in DPD, we hypothesized that performance (and the neural correlates of that performance) of an emotional memory task would also be abnormal in this patient group.

A number of studies have examined abnormalities of emotional memory in clinical groups e.g. patients with amygdala damage (Phelps et al., 1998), patients with Alzheimer's disease (Mori et al., 1999; Kensinger et al., 2002) but this is the first such study in patients with depersonalization. There is strong evidence that emotion normally enhances episodic memory (Burke et al., 1992; Hamann, 2001). The neural and cognitive mechanisms of this effect have been the subject of much study, and functional neuroimaging has been an important tool in this work. One landmark study (Cahill et al., 1996) examined the neural substrates of the encoding of emotionally salient information and, by correlating this data with the results of memory tests conducted after the scanning session, implicated the activity of the right amygdala at encoding as being crucial in modulating the strength of the subsequent memory trace. Other studies have examined neural correlates of encoding (Hamann et al., 1999; Canli et al., 2000) and recall (Fink et al., 1996) of emotional material, though emphases and methods vary widely. A number of more recent studies have addressed the issue of contextual emotional memory i.e. memory for emotionally neutral stimuli encoded in an emotional context (Maratos et al., 2001; Erk et al., 2003; Smith et al., 2004). This work is discussed in detail in our previous paper presenting the normal control data from this study (Medford et al., 2005), and this discussion is not repeated here. Overall, however, the data suggest a key role for amygdala–hippocampal interactions underlying memory for emotional contextual information. A more recent study (Smith et al., 2006) enlarged on these findings by examining connectivity between amygdala and hippocampus during testing of recognition memory for contextual information encoded in either emotional or neutral contexts. It was found that this connectivity showed a bidirectional increase during explicit retrieval of emotionally salient information, and (as in Medford et al., 2005) that the left amygdala was particularly implicated in retrieval of emotional contextual material compared to neutral contextual material. Fenker et al. (2005) have argued that the concerted action of amygdala and hippocampus co-ordinates a cortical recapitulation of emotionally relevant stored contextual information, thus permitting successful memory retrieval.

A key methodological issue, not addressed in many of these studies, is the differentiation of memory for emotional content from that for surrounding context (Burke et al., 1992). We have previously attempted to