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Cortisol and Imaginal Exposure in Posttraumatic Stress Disorder: a Case Report

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Abstract

Imaginal exposure is closely associated with hippocampal processing of traumatic memory. The hippocampus is a target for glucocorticoids which influence memory retrieval and stress response. Glucocorticoid secretion in response to imaginal exposure has not been investigated. We measured subjective distress and salivary cortisol during the 1st and the 20th exposure session in a patient with PTSD. Despite considerable arousal and anxiety, cortisol did not increase during the first exposure. During the 20th exposure there was a marked reduction of distress, although cortisol values did not differ from exposure 1. The response of glucocorticoids to imaginal exposure and mechanisms of the lacking cortisol response need further research (German J Psychiatry 2002;5:75-77).

Keywords: posttraumatic stress disorder, PTSD, imaginal exposure, cortisol, HPA axis

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Introduction

Core symptoms of posttraumatic stress disorder (PTSD) are disorganized and incomplete trauma memories, often experienced as flashbacks. In addition, PTSD is characterized by hyperactive feedback regulation of glucocorticoids, with lower cortisol secretion and enhanced dexamethasone suppression (Yehuda, 1995).

Importantly, most stressful experiences are mediated by a hippocampus-dependent neuronatomical pathway (LeDoux, 2000) where the hippocampus is a major target for glucocorticoids which, depending on their concentrations, can enhance or impair memory (de Kloet, 1998).

Imaginal exposure is an effective therapeutic technique that consists of systematic recollection of memories of the traumatic event in imagination (Foa, 2000). It is thought to be closely associated with hippocampal processing of information about fear-evoking situations resulting in the laying down of integrated, coherent representations of conscious experience (Brewin, 2001).

This case report examines the glucocorticoid response to imaginal exposure - a trauma-related, fear-evoking, internally generated stimulus - in a patient with PTSD before, during and after the first and twentieth exposure session six weeks later. To the best of our knowledge, this has not been investigated in previous studies.

Case report

Mrs. G, a 45-year-old teacher, had a severe car accident five months prior to admission, without serious physical injury. She was diagnosed with PTSD (DSM-IV) and scored 29 on the Posttraumatic Diagnostic Scale (PDS, Foa, 1997) and 46 on the Impact of Event Scale-revised (IES-R, Weiss, 1996), indicating a severe form of PTSD. She had been medication free prior to and during psychotherapeutic treatment. The imaginal exposure took place from 2.00 p.m. to 3.00 p.m. Salivary cortisol and subjective units of distress (SUD, 100 mm Visual Analogue Scale) were measured every 15 minutes from 1.00 p.m. to 3.30 p.m. Saliva

was taken using a Salivette® (Sarstedt, Nuembrecht, Germany) and cortisol was measured radioimmunometrically using a commercial assay (ICN, Carsson, CA, USA). The detection limit was 0.05 ng/ml; intra- and interassay variability was below 10%. Mrs. G. gave written informed consent, the procedure was approved by the Ethics Committee of the General Medical Council of Hamburg.

In advance of the first exposure session, Mrs. G. experienced extreme anticipatory anxiety up to an SUD score of 85, which increased during the exposure to 95 and dropped at the end of the session to 25. However, these profound symptoms of arousal and distress were not associated with any changes in salivary cortisol concentrations (2.44 ng/ml at 1.00 pm to 2.10 ng/ml at 3.30 p.m, see Figure 1). A similar cortisol pattern was seen in the next two sessions, with almost equivalent SUD ratings (not shown). Cortisol concentrations during exposure sessions did not differ from those of a comparison day when cortisol was collected at the same time and place but without any stressful activities (mean 2.13 ng/ml vs. 2.37).

Six weeks later (session 20), after three sessions of exposure/week, there was a marked reduction of anxiety prior to exposure (mean SUD score 68 vs. 30.75) and during the session (58.7 vs. 25.6). The maximum SUD score fell from 92 to 41. In contrast to this reduction of anxiety and distress, cortisol concentrations did not differ from those of exposure 1.

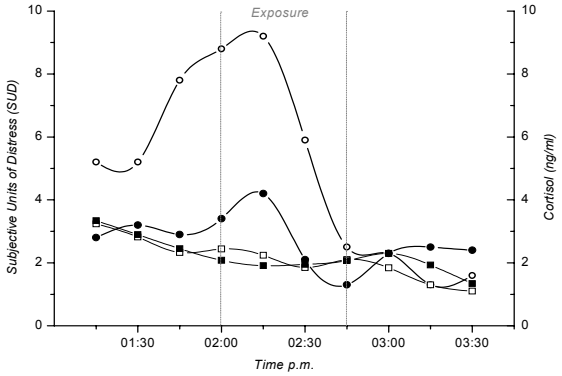

Figure 1. Subjective Units of Distress (SUD, circles) and salivary cortisol (squares) before, during and after the first (open symbols) and twentieth (black symbols) exposure session.

Discussion

In this civilian patient with non-combat related PTSD, the hypothalamus-pituitary-adrenal (HPA) axis is not activated during imaginal exposure. This is in accordance with earlier findings in veterans suffering from PTSD and showing exaggerated responses to combat sound in skin conductance, heart rate, epinephrine and norepinephrine but not ACTH compared to controls (Liberzon et al. 1999).

Further, a lack of ACTH response has also been described in experimentally-induced panic attacks (Kellner, 1998, Otte et al. 2002) as well as during "flooding" in vivo in severe phobias (Curtis, 1976, Woods, 1987). However, Bandelow et al. (2000) found a subtle but significant elevation of salivary cortisol in 25 patients with panic disorder during spontaneous panic attacks compared with levels obtained 24 hours later. Thus, one may speculate that there are differences in the activation of the HPA axis in spontaneously occurring panic attacks compared to exposure-induced arousal. This could at least in part be explained by the reassuring presence of medical staff during the procedure, because Bandelow et al. (2000) measured the cortisol response during naturally occurring panic attacks in the absence of medical staff.

Another possible explanation for the absence of a cortisol response during imaginal exposure is an enhanced negative feedback regulation (Kellner, 1999) and an adrenal hyporesponsiveness (Kanter, 2001) that has been found in PTSD. A low cortisol response during a traumatic event might be a risk factor for the development of PTSD because Yehuda et al. (1998) have demonstrated lower cortisol levels in the immediate aftermath of the traumatic event in those who suffer from PTSD at a follow-up time, compared to those who do not. Thus, it might be possible that the lack of cortisol activation during imaginal exposure is similar to a low cortisol response during the traumatic event itself in those subjects at risk of PTSD. A possible explanation for this phenomenon might be the lack of a "shut off" function of cortisol which - once secreted - leads to a decrease in sympathetic activity (Munck et al. 1984). In fact, an interesting study by Shalev et al. (1998) showed that heart rate levels immediately after a traumatic event predicted the development of PTSD 4 months later.

Other neurotransmitters or neuropeptides which are involved in the stress response, e.g. atrial natriuretic peptide, are known to inhibit the glucocorticoid response and might contribute to the phenomenon of unchanged peripheral cortisol concentrations (Kellner, 1995) during imaginal exposure or the traumatic event itself.

A limitation of this preliminary single case study is that we cannot exclude a response of central glucocorticoids and that we did not measure the catecholamine response.

After this first report of cortisol unresponsiveness during imaginal exposure in PTSD, more systematic studies with more patients should further address the complex interaction between fear reduction, modification of traumatic memories and glucocorticoid regulation in PTSD.

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