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Skin conductance and memory fragmentation after exposure to an emotional film clip in depersonalization disorder

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ABSTRACT

It is often assumed that when confronted with an emotional event, patients with DPD inhibit information processing. It is also thought that this fosters memory fragmentation. This hypothesis has not been tested in chronic depersonalization. The aim of this study was to investigate the temporal pattern of autonomic responding to emotional material in depersonalization disorder, along with concomitant deficits in subjective and objective memory formation (i.e., difficulties to form a coherent narrative consisting of an ordered sequence of events). Participants with depersonalization disorder ($n=14$) and healthy control participants ($n=14$) viewed an emotional video clip while their skin conductance (SC) levels were measured. Peritraumatic dissociation was measured before and after the clip, and memory performance was measured 35 min after viewing. Compared to controls, depersonalized participants exhibited a distinctly different temporal pattern of autonomic responding, characterized by an earlier peak and subsequent flattening of SCLs. Maximum SCLs did not differ between the two groups. Moreover, unlike the control group, depersonalized participants showed no SC recovery after clip offset. In terms of memory performance, patients exhibited objective memory fragmentation, which they also reported subjectively. However, they did not differ from controls in free recall performance. Apparently, emotional responding in DPD is characterized by a shortened latency to peak with subsequent flattening and is accompanied by memory fragmentation in the light of otherwise unremarkable memory functioning.

1. Introduction

Depersonalization disorder (DPD) is characterized by persistent or recurrent episodes of "detachment or estrangement from one's self" (p. 530; American Psychiatric Association, 2000). The Diagnostic and Statistical Manual, Fourth Edition, Text Revision (DSM-IV-TR) (American Psychiatric Association, 2000) classifies DPD as one of the dissociative disorders, but some scholars have argued that it is an anxiety or mood disorder (Baker et al., 2003; World Health Organization, 1992). However that may be, there is evidence that patients with DPD exhibit deficits in emotion processing. For example, Sierra et al. (2002) reported that DPD patients have reduced magnitudes and increased latencies of skin conductance responses to static aversive stimuli, as compared to both healthy controls and patients with anxiety disorder. It has also been suggested that the chronic state of depersonalization in DPD hampers the formation of emotional memories, thereby promoting memory fragmentation (van der Kolk and Fisler, 1995). Thus, when confronted with an emotional event, patients with DPD are thought to inhibit information processing (Ladwig et al., 2002), which in turn leads to difficulties to form a coherent narrative consisting of an ordered sequence of events. In line with this assumption, some trauma victims say they experience difficulties in recalling the temporal order of events (van der Kolk and Fisler, 1995) and patient with DPD report temporal disintegration of autobiographical memories (Simeon et al., 2007). Germane to this is also a PET study by Simeon et al. (2000) that is suggestive of deficient sensory integration in DPD. However, no study has directly looked at emotional memories in DPD using an objective measure of memory fragmentation (for a review, see Giesbrecht et al., 2008a).

With these considerations in mind, the aim of the present study was twofold. First, we wanted to investigate the time course of autonomic responding to emotional material in DPD. Specifically, we were interested in the dynamics of emotional responding during an emotional video clip of 12:30 min. An emotional stimulus with a relatively long duration as employed in the current study provides an opportunity to test the hypothesis that an initial and brief increase in arousal would be followed by shutdown and blunting in those who dissociate (Simeon, 2004). Such a process cannot be captured, and therefore has not been addressed, by the studies which have either used brief stimuli (Sierra et al., 2002; Lemche et al., 2007) or

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peripheral neurohormonal measures obtained pre- and post-stress which only capture the "net" response to the stressor (Giesbrecht et al., 2007).

Second, we hypothesized that patients with DPD might exhibit memory deviations related to overall recall and temporal sequencing (i.e., fragmentation). In addition, we were interested in whether such changes would be associated with autonomic responsivity.

2. Methods

2.1. Participants

Participants were 14 patients with Depersonalization Disorder (DPD) and 14 healthy controls (HC). The study was approved by the Institutional Review Board of Mount Sinai School of Medicine, New York and was conducted at this institution. All participants gave written informed consent prior to participation in the study. Participants received 60$ for participation. The diagnosis of DPD was established by Daphne Simeon, MD using the Structured Clinical Interview for Dissociative Disorders (SCID-D-R; Steinberg, 1994), a well-validated interview of dissociative symptoms and disorders. Participants diagnosed with DPD, as defined by the DSM-IV-TR (American Psychiatric Association, 2000) experienced persistent or recurrent episodes of depersonalization associated with significant distress and/or dysfunction and these episodes did not occur in the context of another psychiatric or medical condition, including another dissociative disorder. As per SCID-D-R guidelines, participants with elevated amnesia or identity alteration scores did not receive a DPD diagnosis, notably these patients would receive the diagnosis of dissociative identity disorder or dissociative disorder not otherwise specified. Participants with a lifetime history of psychotic disorders, current substance use disorders, and/or major medical or neurological disorders were excluded from the study. DPD participants taking psychotropic medications were not excluded ($n=6$). Medications and daily doses were sertraline 50 mg, venlafaxine 50 mg (two patients), quetiapine 50 mg in combination with ramelteon 8 mg, tranylcypromine 70 mg, and donepezil 5 mg in combination lamotrigine 50 mg. The remaining DPD patients did not take any psychotropic medication ($n=8$). Current comorbidity in the DPD groups were generalized anxiety disorder ($n=4$), obsessive compulsive disorder ($n=2$), major depressive disorder ($n=2$), panic disorder ($n=2$), dysphoria ($n=1$), claustrophobia ($n=1$), dysthymia ($n=1$), social phobia ($n=1$), anxiety disorder NOS ($n=1$), and seasonal affective disorder ($n=1$).

2.2. Self-report questionnaires

2.2.1. Dissociative Experiences Scale (DES; Cronbach's alpha = 0.95; Bernstein-Carlson and Putnam, 1993)

The DES is a 28-item self-report scale which asks respondents to indicate the frequency of various dissociative experiences, rated on a 0% to 100% scale scored in 10% increments. In a meta-analytic study, van IJzendoorn and Schuengel (1996) provided evidence for the sound psychometric properties of the DES. In addition to the DES total score and following the three-factor solution proposed by Carlson et al. (1991), we calculated separate subscale scores for amnesia (Cronbach's alpha = .67), absorption (Cronbach's alpha = .91), and depersonalization/derealization (Cronbach's alpha = .88).

2.2.2. Cambridge Depersonalization Scale (CDS; Cronbach's alpha = 0.97; Sierra and Berrios, 2000)

The CDS consists of 29 items which ask the respondent to rate depersonalization symptoms over the "last 6 months" on a 5-point frequency scale (anchors: 0 = never; 4 = all the time) and a 6-point duration scale (anchors: 1 = few seconds; 6 = more than a week). All CDS frequency and duration scores are summed to obtain a total score. The scale is able to differentiate patients with DPD from other patient groups and from healthy controls. Sierra and Berrios (2000) report sound psychometric properties for the CDS.

2.2.3. Childhood Trauma Questionnaire (CTQ; Cronbach's alpha = 0.93; Bernstein et al., 2003)

The CTQ is a widely used self-report scale of childhood interpersonal trauma, rated on a 5-point scale. In the present study, we employed the short form, which consists of 25 items, for which Bernstein et al. (2003) reported satisfactory psychometric qualities. Factor analysis has revealed 5 factors, accounting for 48% of the total variance, with each factor consisting of 5 items. These 5 factors are emotional abuse, physical abuse, emotional neglect, sexual abuse, and physical neglect.

2.2.4. Beck Depression Inventory (BDI; Cronbach's alpha = 0.96; Beck et al., 1961)

The BDI is a 21-item multiple-choice self-report inventory which measures the presence and degree of depression in adolescents and adults. Its items pertain to depression symptoms such as hopelessness and irritability, cognitions such as guilt or feelings of being punished, as well as physical symptoms such as fatigue, weight loss, and lack of interest in sex. The BDI is one of the most widely used measures of depression.

2.2.5. Beck Anxiety Inventory (BAI; Cronbach's alpha = 0.96; Beck and Steer, 1990)

The BAI assesses anxiety, and was specifically designed to minimize the overlap between depression and anxiety. Both physiological and cognitive components of anxiety are addressed in the 21 items describing subjective, somatic, or panic-related symptoms. The BAI differentiates well between anxious and non-anxious groups in a variety of clinical settings and is appropriate for various adult mental health samples.

2.2.6. Peritraumatic Dissociative Experiences Questionnaire (PDEQ; Cronbach's alpha's = 0.72–0.78; Marshall et al., 2002)

The PDEQ is the most widely used self-report measure of peritraumatic dissociative reactions and consists of 8 items. These items quantify the amount of acute dissociation. Respondents are asked to indicate on a 5-point scale (anchors: 1 = not at all true, 5 = extremely true) to what extent they experienced particular dissociative symptoms during a specific event (e.g., "I felt confused or couldn't make sense of what was happening"). Items were summed to obtain a PDEQ total score. The PDEQ was administered twice, immediately before (Cronbach's alpha = 0.78) and after (Cronbach's alpha = 0.72) participants had been exposed to the video clip (i.e., 5 min post-stimulus offset, see below).

2.2.7. Profile of Mood States (POMS; Cronbach's alpha = 0.81–0.90; McNair et al., 1992)

The POMS is a commonly used questionnaire designed to assess transient, distinct mood states. The original version consists of 65 items rated on a 5-point scale ranging from "not at all" to "extremely." In the present study, we employed the POMS tension-anxiety subscale as a measure of state anxiety. The POMS referred to how participants "feel at the moment," and was administered two times, immediately before (Cronbach's alpha = 0.90) and after (Cronbach's alpha = 0.81) the video clip (i.e., 5 min post-stimulus offset, see below).

2.3. Stimulus material

Stimulus material consisted of a clip from the Hollywood movie "The Silence of the Lambs" with a duration of 12:30 min.[2] The clip starts with alternating scenes from within the house where a male holds a women captive and a group of police officers preparing to raid another house. Next, a female police officer rings the bell at the house where the victim is held captive, while the other police officers raid the other house and realize that they are at the wrong place. After a short chat with the hostage-taker, the female police officer draws her gun and chases the man through the cellar. She finds the victim who is being held captive and eventually kills the man. Movie clips tend to elicit strong emotional arousal in participants (Jansen and Frijda, 1994). Previous work has also shown that participants evaluate this particular video clip as emotionally provocative. Accordingly, this clip is known to elicit an increase in anxiety (Rottenberg et al., 2007). This makes this particular clip particularly suitable in the context of the present study. Specifically, it is thought that patient with DPD dissociate as a response to increases in anxiety. Thus, one would expect the present clip to provoke dissociative symptoms. These heightened levels of depersonalization and derealization are thought to go along with an inhibition of information processing and physiological responding, which are the subject of investigation in the present study.

Furthermore, we selected this specific clip as it contains a relatively long scene which builds up tension in a gradual fashion. This makes this video clip suitable to investigate emotional processing in patients with DPD (Simeon and Abugel, 2006) and its autonomic concomitants over time.

2.4. Psychophysiological measure

In the present study, skin conductance was measured with two BioPac EL507 Disposable Electrodermal Electrodes that were filled with isotonic gel and were placed on the middle phalanx of middle and ring finger of the non-dominant hand. Before electrodes were attached, participants rinsed their hands with distilled water (Fowles et al., 1981). Skin conductance was recorded using a BioPac GSR100C with gain of 5 μS/V and a low-pass filter at 10 Hz. The signal was sampled at 200 Hz by a BIOPAC MP150 (Biopac Systems, Goleta, CA) system connected to a data-acquisition computer running the Acknowledge v3.8.2 software package. Skin conductance data for one HC participant was lost due to equipment malfunctioning.

2.5. Measures of memory performance

The following three measures were administered, in the order below, from 35 to 60 min after stimulus offset. One DPD patient was unable to complete measures of memory performance.

2.5.1. Subjective memory fragmentation (Kindt and van den Hout, 2003)

Subjective memory fragmentation refers to the subjective (i.e., meta-memory) experience of fragmentation and does not necessarily reflect actual (i.e., objective) fragmentation. Subjective fragmentation was quantified using three 100-mm visual analogue scale items. Participants had to indicate the "snap-shot" character of their recollections of the video clip. The items were as follows: "How much does your memory of the video exist of fragmented pieces as opposed to a whole entity?", "How much does your memory of the video exist of visual images?", and "How intense are emotions in your memory of the video?". Items were summed to obtain a measure of subjective memory fragmentation.

[2] The film clip is available from the first author.

2.5.2. Free-recall of video clip

Participants were asked to write down everything they could remember of the clip. Their accounts were scored in terms of hits and commission errors (i.e., fabrications) by two independent raters (TG, KA) who were blind to the diagnostic status of the participants. For the present study, a customized scoring form was developed containing 195 details. Hits and commission errors were averaged across raters. Agreement between raters was excellent for hits ($r = .95$) and sufficient for commission errors ($r = .78$).

2.5.3. Objective memory fragmentation

Objective memory fragmentation was measured along the lines of Wegner et al. (1996). These authors were interested in the effect of thought suppression on memories. To this end, they developed a method which allowed them to investigate disruptions in the temporal organization of memories for a movie clip. More specifically, the objective memory fragmentation task required participants to sort 5 different scenes of 4 fragments , each lasting 5 s, into the correct order (Kindt and van den Hout, 2003). Fragments were selected from easily distinguishable parts of the clip. Participants received one point for correct identification of the first scene and for every scene for which they could indicate correctly the preceding scene. Scores for the 5 fragments were summed to obtain a measure of objective memory fragmentation.[3]

2.6. Procedure

After their baseline psychiatric evaluation and interviews, participants completed the CDS, DES, BAI, BDI, and CTQ. Thirty minutes later, they were connected to the psychophysiological equipment, completed the PDEQ, and POMS and were instructed as follows: "We will start this procedure with 10 minutes of relaxation and baseline measurements. After 10 minutes, you will see a video clip which will last about 12:30 minutes. This video clip is followed by another 5 minutes of relaxation. Please sit quietly because movement can affect the physiological recording. Also, please keep your eyes open during the procedure. Do you have any questions?"

Immediately after completing the psychophysiological measurements (i.e., 5 min post stimulus offset), the PDEQ and POMS were re-administered. Next, participants completed unrelated filler-questionnaires for about 35 min. Then, the subjective memory fragmentation measure was administered, followed by a free-recall, and the memory fragmentation task. Finally, participants were thanked for participating and debriefed.

2.7. Data reduction

Skin conductance (SC) levels in the 10 min preceding the video clip were averaged to obtain the average SC resting level. SC levels during the video were averaged into twenty-five 30-s bins. These bins were then transformed by subtracting the average SC resting level. Moreover, a maximum SC level amplitude (i.e., average baseline resting level to highest SC level at any point during viewing) and rise time to peak (i.e., latency from clip onset to the point of maximum amplitude) were extracted from the continuous SC data. To quantify recovery after the offset of the clip, SC levels were averaged in 10 30-s bins, starting immediately after the offset of the clip. Again, these bins were then transformed by subtracting the average SC resting level. SC recovery was analyzed by comparing SC levels after clip offset with SC levels at the end of the recovery period (4:30 min later). Following Orr et al. (2003), square-root transformations were performed to reduce potential heteroscedasticity of SC levels.

2.8. Statistical analysis

Using independent *t*-tests, groups were compared with regard to their scores on self-report questionnaires. PDEQ scores were log-transformed to reduce skewness. PDEQ and POMS scores before and after the clip were evaluated using analyses of variance (ANOVA) with the respective measure over time as the repeated measure. Group differences in SC level bins during the video were evaluated with ANOVA's, with the SC level at the 25 time points as the repeated measure; Greenhouse–Geisser corrections were applied to *P*-values associated with multiple DF repeated measures. Group differences in average resting SC levels, SC maximum response amplitude, and rise time to peak were compared with independent sample *t*-tests. SC recovery was analyzed by comparing SC levels after clip offset with those at the end of the recovery period (4:30 min later) using an ANOVA with SC level at 10 time points as repeated measure. Group differences in memory performance measures were evaluated with independent sample *t*-tests. P's < 0.05 were considered statistically significant; all comparisons were two-tailed, unless otherwise noted.

[3] The scenes that were used during the memory fragmentation task are available from the first author.

3. Results

3.1. Self-report questionnaires

Mean age and gender distribution for the groups are presented in Table 1. As can be seen, the groups did not differ with respect to gender distribution or age. Individuals with DPD differed significantly from HC's on measures of dissociation and depersonalization (i.e., CDS, DES), all P's < 0.01 (see Table 1). This underscores the diagnostic integrity of the DPD group. Moreover, compared to HC's, individuals with DPD scored significantly higher on the BAI, BDI, and CTQ emotional abuse subscale (all P's < 0.05), but not on any other subscale of the CTQ or its total score.

Mean POMS Tension-Anxiety subscale scores were 2.42 (S.D. = 0.81) before and 2.04 (S.D. = 0.79) after the video clip for DPD patients and 0.49 (S.D. = 0.40) before and 1.00 (S.D. = 0.66) after for HC's. An ANOVA on POMS scores with time as repeated measure and group as between-subjects factor yielded a significant main effect of group ($F(1,26) = 48.08$, $P < 0.01$), which was due to the DPD group exhibiting more anxiety in general. Moreover, a significant time × group interaction ($F(1,26) = 9.63$, $P < 0.01$) emerged. Post-hoc *t*-tests showed that this was due to the DPD group's anxiety levels not changing significantly ($t(13) = 1.60$, $P > 0.05$), while the HC group reacted with heightened anxiety to the clip ($t(13) = 3.14$, $P < 0.01$).

Mean PDEQ scores before and after the clip were 15.93 (S.D. = 6.37) and 13.86 (S.D. = 5.70), respectively for the DPD group and 8.00 (S.D. = 0.00) and 9.50 (S.D. = 2.77), respectively for HC's. An ANOVA on log-transformed PDEQ scores at the timepoints as repeated measure and group as between subjects factor yielded a significant main effect of group ($F(1,26) = 28.64$, $P < 0.01$), which was due to the DPD group exhibiting heightened levels of acute dissociation in general. Moreover, a borderline significant time × group interaction ($F(1,26) = 4.20$, $P = 0.05$) emerged. Post-hoc *t*-tests showed that the DPD group's dissociation levels did not change significantly ($t(13) = 1.16$, $P > 0.05$), while the HC group reacted with heightened levels of dissociation to the video clip ($t(13) = 2.27$, $P < 0.05$).

3.2. Psychophysiological measure[4]

There was a significant difference in mean resting baseline levels of SC between the DPD (2.35 sqrt(microsiemens); S.D. = 0.80) and the HC (1.68 sqrt(microsiemens); S.D. = 0.58) groups, $t(25) = 2.49$, $P < 0.05$, Cohen's $d = 1.00$). Using forward stepwise linear regression analyses, we examined possible predictors of SC resting baseline, notably age, gender, CDS, DES, BAI, BDI, and CTQ emotional abuse. The CTQ emotional abuse subscale rather than the total CTQ was included as groups did only differ significantly with respect to this subscale and not with respect to the CTQ total score (for a similar finding, see Simeon et al., 2001).BAI scores accounted for 28% of the variance in SC baseline resting levels ($B = 0.03$, SE = 0.08, $\beta = 0.53$, $t(26) = 3.15$, $P < 0.01$). None of the other factors could significantly improve the prediction.

Mean SC levels for the groups at different times during the video are presented in Fig. 1. To investigate the temporal dynamics of SC levels, we conducted a repeated-measures ANOVA on SC levels with group as between-subject factor and SC levels at the timepoints as repeated measure. This analysis yielded a significant time × group interaction ($F(1.67, 41.74) = 4.19$, $P < 0.05$, Greenhouse–Geisser Epsilon = 0.07). Contrast analysis showed that this interaction was best

[4] Excluding DPD patients who were on medication did not alter the general pattern of results (see Figs. 1 and 2). Effect sizes for differences in SC resting baseline and latency to peak between the HC group and DPD patients without medication were similar to the ones obtained with analyses including all DPD patients, with Cohen's *d*'s being 1.33 and 0.72, respectively.

Table 1
Psychometric and demographic data of patients with Depersonalization Disorder and healthy controls.

Variable		Depersonalization Disorder ($n=14$) Mean (S.D.)	Healthy Controls ($n=14$) Mean (S.D.)	t	P
Age		30.50 (6.70)	28.36 (6.43)	−0.86	ns
Sex, F/M no. (%)		7/7 (50%)	9/5 (64%)	$x^2(1)=0.58$	ns
CDS		139.21 (58.32)	7.79 (12.10)	−8.26	<0.01
DES	Total	28.19 (13.66)	4.06 (4.64)	−6.26	<0.01
	Amnesia	8.48 (8.42)	2.14 (3.38)	−2.61	<0.01
	Absorption	35.71 (18.88)	7.06 (7.97)	−5.23	<0.01
	Depersonalization	43.81 (23.13)	0.83 (1.93)	−6.93	<0.01
BAI		28.99 (11.28)	2.71 (4.27)	−8.15	<0.01
BDI		24.71 (10.15)	2.36 (2.98)	−7.91	<0.01
CTQ	Total	48.89 (14.91)	43.71 (11.49)	−1.03	ns
	Emotional abuse	11.61 (6.14)	7.07 (3.25)	−2.44	<0.05
	Physical abuse	9.07 (2.53)	10.29 (2.49)	1.28	ns
	Sexual abuse	6.71 (3.27)	6.79 (5.41)	0.04	ns
	Emotional neglect	14.07 (2.87)	12.07 (2.64)	−1.92	ns
	Physical neglect	7.43 (2.38)	7.50 (3.08)	0.07	ns

Note: CDS, Cambridge Depersonalization Scale; DES, Dissociative Experiences Scale; BAI, Beck Anxiety Inventory; BDI, Beck Depression Inventory; CTQ, Childhood Trauma Questionnaire; ns, non-significant.

represented by a linear ($F(1,25)=5.53$, $P<0.05$) rather than a quadratic ($F(1,25)=0.01$, $P>0.05$), or cubic ($F(1,25)=1.87$, $P>0.05$) contrast. We carried out an additional repeated-measures ANOVA comparing average SC levels during early (bin 1–8), middle (epoch 9–18), and late bins (epoch 19–25) with group as between-subject factor and SC levels at the timepoints as repeated measure. This analysis yielded a significant time×group interaction ($F(1.08, 26.93)=5.29$, $P<0.05$, Greenhouse–Geisser Epsilon = 0.54). A follow-up repeated-measures ANOVA with SC levels at the timepoints as repeated measure, but conducted for each group separately indicated that this interaction was due to SC levels increasing significantly over time in the HC group ($F(1.07, 12.84)=5.10$, $P<0.05$, Greenhouse–Geisser Epsilon = 0.54), but not in patients with DPD ($F(1.10, 14.24)=0.35$, $P=0.58$, Greenhouse–Geisser Epsilon = 0.55).

To further investigate group differences in time course of affective responding, we compared SC maximum response amplitude and rise time to peak. Groups did not differ in terms of mean maximum response amplitude (DPD: 0.64 sqrt (microsiemens); S.D. = 0.41, HC: 0.61 sqrt (microsiemens); S.D. = 0.52), $t(25)=0.18$, $P=0.86$, Cohen's $d=0.06$. Mean rise time to peak differed significantly between the two groups ($t(25)=2.9$, $P<0.05$, Cohen's $d=0.96$), with the DPD group (276.75 s; S.D. = 280.47) exhibiting a substantially shorter rise time than the HC's (511.53 s; S.D. = 250.89).

Using forward stepwise linear regression analyses, we examined possible predictors of SC rise time to peak, notably age, gender, CDS, DES, BAI, BDI, and CTQ emotional abuse. Only CDS scores were significant and accounted for 29% of the variance in SC rise time ($B=-1.97$, SE = 0.61, $\beta=-0.54$, $t(26)=3.31$, $P<0.01$). None of the other factors could significantly improve the prediction. Using a similar regression approach, none of the factors significantly predicted maximum SC response amplitude.

Recovery was investigated with a repeated-measures ANOVA on SC levels after clip offset, with SC levels at the time points after offset as repeated measure and group as between subject factor. Using this approach, we found a time×group interaction ($F(1.44, 35.87)=3.91$, $P<0.05$, Greenhouse–Geisser Epsilon = 0.16). Subsequent contrast analysis showed that this interaction was best represented by a linear ($F(1,25)=4.39$, $P<0.05$) rather than a quadratic ($F(1,25)=0.05$, $P>0.05$), or cubic ($F(1,25)=2.68$, $P>0.05$) contrast. Thus, it seems that individuals with DPD do not show any sign of recovery after clip offset, while HC's do exhibit this tendency (see Fig. 2). We also carried out a repeated-measures ANOVA comparing average SC levels during

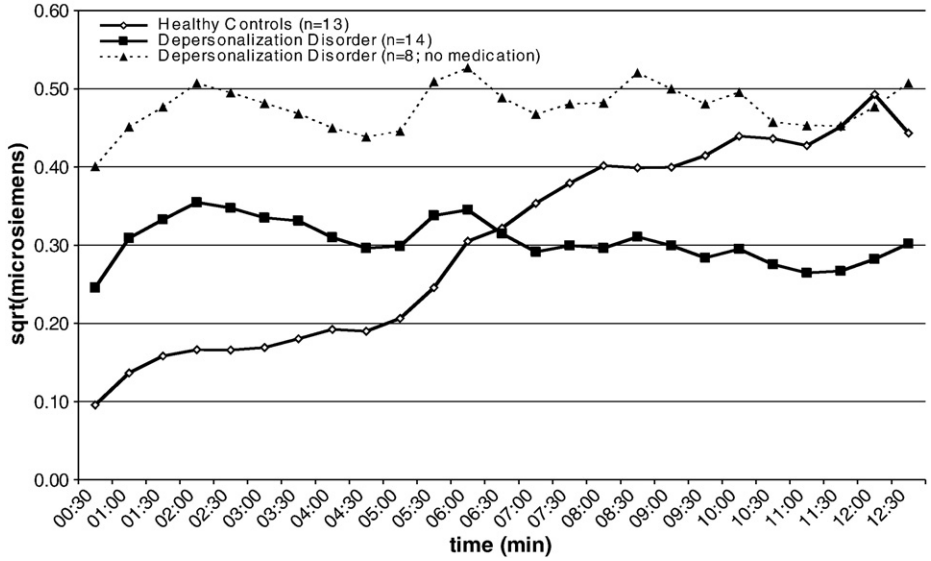

Fig. 1. Temporal dynamics of skin conductance levels to an emotional clip for patients with Depersonalization Disorder (DPD), a subgroup of DPD patients without medication, and Healthy Controls.

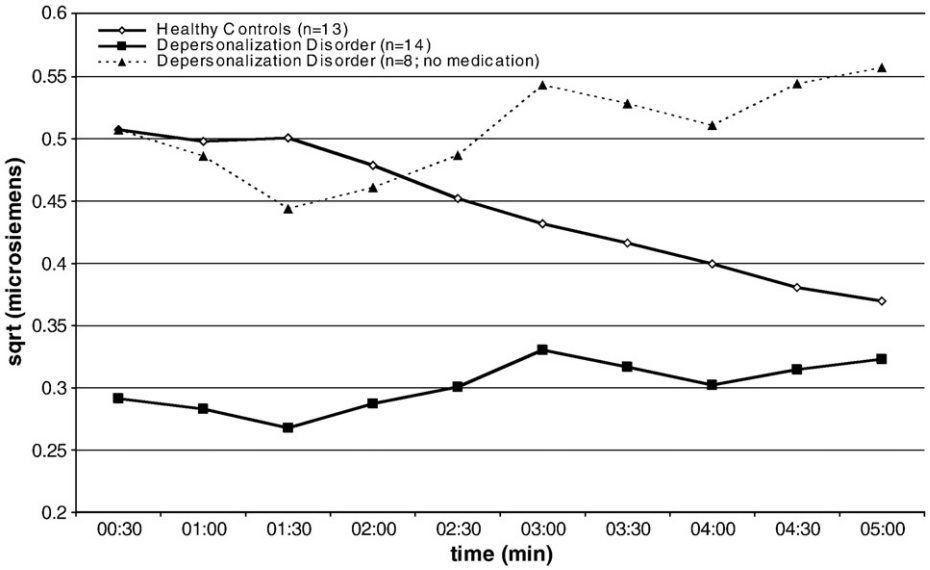

Fig. 2. Recovery of skin conductance levels after offset of an emotional clip for patients with Depersonalization Disorder (DPD), a subgroup of DPD patients without medication, and Healthy Controls.

early (bin 1–4), middle (epoch 5–7), and late recovery bins (epoch 8–10) with group as between-subject factor and SC levels at the timepoints as repeated measure. This analysis yielded a significant time × group interaction ($F(1.11, 27.66) = 4.67$, $P<0.05$, Greenhouse–Geisser Epsilon = 0.55). A follow-up repeated-measures ANOVA with SC levels at the timepoints as repeated measure, but conducted for each group separately indicated that this interaction was due to SC levels decreasing significantly over time in the HC group ($F(1.07, 12.88) = 4.78$, $P<0.01$, Greenhouse–Geisser Epsilon = 0.54), but not in patients with DPD ($F(1.19, 15.50) = .0.15$, $P = 0.20$, Greenhouse–Geisser Epsilon = 0.60).

To investigate the contribution of possible mediators, we conducted a forward stepwise regression analysis trying to predict SC levels at the end of the recovery period. In a first step, we entered SC levels in the first 30 s after clip offset. This accounted for 92% of the variance in SC levels at the end (last 30 s) of the recovery period. Next, we proceeded in a stepwise fashion. At this point, besides early recovery SC levels ($B = 1.01$, SE = 0.06, $\beta = 0.95$, $t(24) = 18.45$, $P<0.01$), only CDS scores were significant and accounted for another 1.3% of the variance ($B = 0.001$, SE = 0.001, $\beta = 0.11$, $t(24) = 2.20$, $P<0.05$). Thus, CDS scores predicted higher SC levels at the end of the recovery period (i.e., slower recovery). None of the other factors could significantly improve the prediction.

3.3. Memory performance

Subjective memory fragmentation and objective memory performance for both groups are presented in Table 2. Responses of one HC participant on the memory fragmentation task were identified as outliers and were omitted from all subsequent analyses. Individuals with DPD reported significantly higher levels of subjective memory fragmentation than HC, and performed significantly worse on the memory fragmentation task. On the free recall task, the two groups performed similarly in terms of both hits and commission errors. Using the regression approach outlined previously, only the CTQ emotional abuse subscale predicted subjective memory fragmentation ($R^2 = .45$, $B = 0.37$, SE = 0.08, $\beta = 0.67$, $t(25) = 4.55$, $P<0.01$), while none of the symptom scales, age, gender, or SC parameters could significantly predict objective memory fragmentation task performance. Patients with DPD did not differ from HC in whether or not they had seen the clip before ($\chi^2(2) = 1.93$, $P>0.05$). Moreover, as in prior studies from our lab (Smeets et al., 2005), prior exposure to the clip was unrelated to memory performance in terms of hits, commission errors, or sorting performance (all P's >0.1).

4. Discussion

The main findings of the present study can be summarized as follows. First, during the video clip, both peritraumatic dissociation and anxiety increased in the HC group, while remaining constant in the DPD group. Second, DPD patients exhibited heightened resting baseline SC levels which seemed to be primarily mediated by comorbid anxiety (i.e., anxiety produces the effect; Baron and Kenny, 1986). Third, DPD patients' pattern of affective responding over time differed markedly from HC's, in that they showed an overall flatter rather than rising pattern during the course of the entire clip and had

Table 2
Performance of patients with Depersonalization Disorder and healthy controls on subjective memory fragmentation and the objective memory task.

Variable		Depersonalization Disorder ($n = 13$) Mean (S.D.)	Healthy Controls ($n = 13$) Mean (S.D.)	t	P
Subjective memory fragmentation		12.69 (2.42)	9.62 (2.79)	3.00	<0.01
Objective memory performance	Hits	30.38 (14.96)	31.14 (13.54)	0.14	ns
	Commission errors	1.27 (1.17)	1.39 (1.36)	0.25	ns
	Sort performance	6.31 (2.59)	8.77 (3.17)	2.17	<0.05

Note: ns, non-significant.

a shortened rise time to peak, while not differing in terms of peak amplitude. The DPD group also demonstrated an overall lack of recovery after clip offset. Group differences in these temporal patterns were related to severity of depersonalization symptoms. Fourth, DPD patients had a specific memory impairment, namely, fragmentation, in light of otherwise intact recall.

Thus our findings, in combination with prior findings showing selective reduction in responses to brief emotionally negative stimuli (Sierra et al., 2002, 2006), support Sierra et al.'s (2002) hypothesis that patients with DPD simultaneously show evidence of "an excitatory mechanism leading to a state of heightened alertness" and "an inhibitory mechanism on emotional responses" (p. 837). The interplay of these two mechanisms may explain why DPD patients display specific deviations in the time course of emotional responding. Specifically, their heightened alertness at baseline may drive fast initial responses to the video fragment. However, a phasic selective inhibitory mechanism may prevent further physiological escalation during ongoing emotional stimulation.

The finding that anxiety and peritraumatic dissociation increased during the video clip in our HC group concords with prior studies relying on healthy volunteers (Giesbrecht et al., 2008b; Sterlini and Bryant, 2002). It is in line with the idea that peritraumatic dissociation serves to regulate the impact of emotions. Germane to this issue are also recent studies showing that peritraumatic dissociative symptoms are a common response to acute stress. For example, Morgan et al. (2001) found that during U.S. Army survival training, 96% of healthy military personnel experienced peritraumatic dissociation. In another study, Sterlini and Bryant (2002) examined peritraumatic dissociative responses in individuals who made their first skydive. As was the case in the Morgan et al. (2001) study, the large majority of these individuals reported peritraumatic dissociation. Surprisingly, patients with DPD failed to show this increase, as their anxiety and peritraumatic dissociation levels remained constant. This may have to do with the chronically high levels of trait, as opposed to state, dissociation in DPD. Indeed, Sierra and Berrios (1998) have proposed that in DPD, peritraumatic dissociation is a 'hard-wired' response to stress which becomes abnormally persistent and hardly subject to fluctuation in intensity.

Patients with DPD had higher resting baseline SC levels compared to HC's. This finding is in contrast with Sierra et al. (2002)who found that patients with DPD did not differ in resting SC from HC's, but did exhibit lower resting SC levels than patients with an anxiety disorder. This discrepancy is most likely due to the higher levels of anxiety in our DPD patients as compared to those in Sierra et al.'s (2002) DPD group (BAI scores were 28.9 vs. 20.5, respectively). As sympathetic activation is considered a cardinal biological manifestation of anxiety (Friedman and Thayer, 1998), we assume that the raised anxiety levels in our DPD patients is reflected in their relatively high SC resting levels. Indeed, a substantial part of the variability in SC resting levels was explained by anxiety rather than depersonalization symptoms per se, which did not explain any additional variance. This pattern is very much in line with Simeon et al.'s (2003) finding of elevated resting 24-hour urine norepinephrine in DPD compared to HC's, which was apparent only before controlling for anxiety scores, while dissociation scores were inversely associated with norepinephrine levels.

Even though both groups did not differ in peak amplitude, DPD patients showed a shorter time to reach maximum amplitude as compared to HC's. Moreover, DPD patients had a faster initial rise and their SC remained flat thereafter. Shorter latencies were related to severity of depersonalization symptoms. That is, patients with more severe depersonalization tended to reach their maximum amplitude faster. In contrast, HC's SC seemed to steadily rise over the entire clip exposure period. These findings are in line with Sierra et al. (2002) who investigated autonomic (i.e., skin conductance) responses of DPD patients and controls to emotional slides and found that DPD patients manifested reduced amplitudes, but shorter skin conductance response latencies to emotionally neutral stimuli. Moreover, Sierra et al. (2006) demonstrated that DPD patients exhibited smaller SCRs to facial expressions of happiness and disgust than patients with an anxiety disorder, while both groups exhibited comparable levels of anxiety. Reminiscent of our findings is also an fMRI study by Lemche et al. (2007, 2008) who found that modulations of haemodynamic responses in brain areas projecting to the autonomic nervous system occur earlier in DPD as compared to HC and that DPD patients exhibit anomalous limbic activation during the presentation of neutral stimuli.

Depersonalization symptoms were related to a failure to recover physiologically after offset of the video clip. Quantifying the temporal pattern of affective responding after stimulus offset in the absence of explicit instructions has been used as a measure of automatic emotion regulation (Jackson et al., 2003; Thompson, 1991; for a review of the neural underpinnings of emotion regulation, see Phillips et al., 2008). Disruptions in spontaneous automatic emotion regulation rather than emotional responsivity per se, appear to be particularly important in the onset and maintenance of anxiety and depression (Davidson et al., 2000) and bipolar disorder (Phillips et al., 2008). Perhaps, then, heightened alertness in DPD prevents effective automatic emotion regulation.

With respect to cognitive functioning, one might speculate that a chronic state of dissociation may hamper information processing and consequently memory. However, in line with Montagne et al. (2007), memory performance of our DPD group was unremarkable. That is, DPD patients and HC's did not differ in objective memory performance, i.e., in their number of omission and commission errors. Thus, we found no support for the idea that when confronted with emotional stimuli, DPD patients react with an avoidant information processing style.

While neither avoidant information processing nor a heightened tendency to commit commission errors in memory was evident in DPD patients, they did report more subjective memory fragmentation. Subjective fragmentation refers to the feeling that one's memories lack temporal structure. Importantly, patients with DPD often describe a lack temporal integration (Simeon et al., 2007). Reports of temporal disintegration in depersonalization date back to Lewis (1931; for a review, see Sierra and Berrios, 2001), who proposed that this phenomenological feature is of central importance to depersonalization. This view was reiterated by Freeman and Melges (1977, 1978), who argued that a distorted experience of time leads to a distorted experience of the self. Two recent factor analytic studies seem to underline this point. These studies were conducted to quantify the phenomenological complexity of DPD (Sierra et al., 2005; Simeon et al., 2008) and employed the CDS. They identified dimensions pertaining to subjective anomalies of recall (Sierra et al., 2005) and time distortion (Simeon et al., 2008).

The current study showed that patients with DPD not only performed worse on the subjective, but also on the objective memory fragmentation task. Our finding of fragmented memory in the context of a normal pattern of hits and commission errors is redolent of a study by Medford et al. (2006). These authors devised stimulus sentences containing either an emotional or neutral target word together with the same embedded context word (for more information on the stimulus sentences, see Brierley et al., 2007; Medford et al., 2005). Using this methodology, they found that both in patients with DPD and healthy controls, emotionality of stimuli did enhance recognition memory for target stimuli (Medford et al., 2006). However, relative to controls, patients with DPD exhibited an absence of enhanced learning of contextual stimuli in the emotional condition. This was accompanied by a lack of differential cortical activation in patients with DPD when encoding emotional stimuli as compared to neutral ones. Thus, assuming that contextual information is essential for the temporal reconstruction of memory, one may hypothesize that a deficient encoding of contextual cues in DPD patients may underlie their poor performance on memory fragmentation tasks. Clearly, the precise causal links between, psychophysiological responding, encoding of contextual information, and memory fragmentation in DPD is an important issue for future research.

Important strengths of the present study include strict selection and diagnostic criteria, a rigorous experimental paradigm, use of a broad range of symptom scales, and the inclusion of both psychophysiological and cognitive measures. The most important limitation of the study is its relatively small sample size and the fact that not all participants were medication free. Moreover, although the present study obtained baseline and post offset measures, it did not include a neutral clip condition. This makes the interpretation of our findings as being specifically related to negative emotions less compelling. In addition, we employed an excerpt from a well-known video. Therefore, prior knowledge could have affected semantic and episodic memory. In conclusion, the present findings indicate that DPD is characterized by a deviant time course of emotional responding and subsequent memory fragmentation, in light of otherwise unremarkable memory functioning. Future work should a) try to disentangle the interplay of the two presupposed mechanisms, heightened alertness and selective inhibition, that may be responsible for differences in physiological responding in DPD and b) investigate the role of early information processing deviations (e.g., encoding deficits) in DPD patients'memory fragmentation.

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