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Autonomic response in the perception of disgust and happiness in depersonalization disorder

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Abstract

Patients with depersonalization disorder have shown attenuated responses to emotional unpleasant stimuli, hence supporting the view that depersonalization is characterised by a selective inhibition on the processing of unpleasant emotions. It was the purpose of this study to establish if autonomic responses to facial emotional expressions also show the same blunting effect. The skin conductance responses (SCRs) of 16 patients with chronic DSM-IV depersonalization disorder, 15 normal controls and 15 clinical controls with DSM-IV anxiety disorders were recorded in response to facial expressions of happiness and disgust. Patients with anxiety disorders were found to have greater autonomic responses than patients with depersonalization, in spite of the fact that both groups had similarly high levels of subjective anxiety as measured by anxiety scales. SCR to happy faces did not vary across groups. The findings of this study provide further support to the idea that patients with depersonalization have a selective impairment in the processing of threatening or unpleasant emotional stimuli.

Keywords: Depersonalization; Dissociative disorders; Disgust; Emotion perception

1. Introduction

Depersonalization disorder is characterised by persistent or recurrent episodes of 'detachment or estrangement from one's self.' The individual may feel like an automaton or there may be the sensation of being an outside observer of one's own mental processes (American Psychiatric Association, 1994). Depersonalization has been shown to correlate with anxiety measures, and most patients with a diagnosis of depersonalization disorder (DPD) have been shown to have significant levels of anxiety or comorbid anxiety disorders (Simeon et al., 2003a; Baker et al., 2003). This, together with the high prevalence of depersonalization at times of life threatening situations, has been interpreted as suggesting that depersonalization represents an anxiety-triggered 'hard wired' inhibitory response intended to ensure the preservation of adaptative behaviour during situations normally associated with overwhelming and potentially disorganizing anxiety (Sierra and Berrios, 1998). In such circumstances, it has been suggested that depersonalization will result in the *inhibition* of non-functional emotional and autonomic responses whilst maintaining vigilant attention. In patients with DPD this response would become abnormally persistent and dysfunctional (Sierra and Berrios, 1998).

Recent fMRI (Phillips et al., 2001) and psychophysiological studies (Sierra et al., 2002) support the above

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model and have indicated that patients with DPD show lack of activation in limbic areas, and marked autonomic attenuation in response to pictures depicting disgusting or distressing situations. In the same vein Lanius et al. (2002) recently studied patients with sexual-abuse-related posttraumatic stress disorder and found that, while 70% of patients had increased heart rate during a traumatic script-driven symptom provocation, those patients who had a dissociative response (30%) during the experiment did not show any concomitant increase in heart rate. Also supporting an anxiety-suppressing mechanism in depersonalization, is the finding by Simeon et al. (2003b) of a striking negative correlation ($r = -0.8$) between intensity of depersonalization and urine norepinephrine levels.

In view of the fact that some facial emotional expressions can signal threatening situations to others (e.g. fear or disgust), it was hypothesised that patients with DPD would have selectively attenuated SCR to facial expressions of negative emotions as compared to facial expressions of positive emotions (e.g. happiness). In fact, given that perception of positive emotions is usually a safety signal to others, SCR responses to happy expressions was predicted to be normal in the DPD group. To test these predictions we compared event related SCRs to visual presentations of genuine (as opposed to posed) facial expressions of disgust and happiness, in patients with DPD and two control groups: normal controls, and patients with a diagnosis of an anxiety disorder.

2. Methods

2.1. Subjects

16 patients with a DSM-IV diagnosis of DPD were recruited from the Depersonalization Disorder Clinic at the Maudsley Hospital, London (Baker et al., 2003). The diagnosis of depersonalization disorder was ascertained by means of a semistructured interview using the Present State Examination (PSE; Wing et al., 1974), and scores above cut-off point (score of 70) on the Cambridge Depersonalization Scale (CDS; Sierra and Berrios, 2000). All subjects had chronic and continuous (as opposed to intermittent) depersonalization of durations ranging from 1 to 10 years. None of them was taking any medication at the time of the study and had been medication free for two weeks or more. Exclusion criteria included lifetime incidence of psychotic disorder, current substance abuse disorder, current major depression, other dissociative disorder, and history of neurological disorder.

An anxiety control group was composed by 15 patients meeting DSM-IV criteria for panic or generalised anxiety disorder were recruited from the behavioural psychotherapy unit of the Maudsley Hospital. Patients were diagnosed by experienced clinicians by means of a thorough standard clinical interview. In order to make sure that the anxiety patients did not suffer from significant depersonalization, patients providing a history of depersonalization and scores above 70 on the CDS were excluded.

Fifteen normal controls were volunteers selected from staff members and students of the Institute of Psychiatry and King's College London. All of the normal controls denied personal history of mental illness and scored below cut-off points on the administered scales.

The three groups were similar for sex and age as these two variables have been shown to affect electrodermal activity (Venables and Mitchell, 1996). All subjects were paid for their participation in the study and were asked to provide informed written consent. The study was approved by the ethics committee of the institute of psychiatry.

2.2. Stimuli

A set of stimuli previously developed and validated by one of the authors (CS) as part of his PhD dissertation (Senior, 1999), consisted of clips (or static pictures thereof) of subjects displaying spontaneous happy (laughing or smiling) or disgust expressions. Stimuli were presented in both colour and black and white versions. The rationale for using different presentation parameters, was based on recent findings, which suggest that recognition of emotional expression is affected by visual parameters of stimuli presentation such as movement and colour (Kemp et al., 1996; Lee and Perrett, 1997; Simons et al., 1999; Lander et al., 1999; Kamachi et al., 2001).

Preparation of stimuli: In order to make sure that the stimuli showed genuine, spontaneous emotional expressions, 53 volunteers were covertly recorded whilst they were shown short video excerpts previously known to elicit intense disgust (e.g. clips depicting cannibalism, urodipsia and coprophagia), or laughter (excerpts from two comedy films) (Gross and Levenson, 1995). Nineteen subjects who showed definite and distinct emotional expressions were retained. These stimuli were then piloted on a sample of normal volunteers and those identities obtaining interrater agreement >90% on emotion recognition were used for the study (Senior, 1999).

Stimuli were presented in 24 blocks, each containing the same emotional expressions from 12 different subjects (12 blocks contained disgust expressions, and the other half happy), who alternated in gender. All faces in each block had the same presentation parameters (i.e. black and white or colour shown as moving images or static photographs). Each face in the block was presented for 3 s. Each block was followed by a 30 s blank screen gap, during which subjects gave verbal ratings (see below). All stimuli were presented to subjects through two video cassettes (VHS PAL) each with an alternating order of presentation. Each video cassette was allocated randomly to approximately half of each group of subjects.

Four black and white pictures of happiness, disgust from the Ekman and Friesen series (1976) were shown at the beginning to habituate subjects to the experimental conditions (see Ekman and Friesen, 1976).

Stimuli were presented on a colour TV monitor placed 1.5 m from the subject. All faces were shown in a presentation window in the middle of the monitor screen that measured 6 by 9 in.

During the 30-s interval between the blocks subjects were presented with a multiple choice format listing six emotion labels: surprise, happiness, anger, disgust, sadness and fearfulness and were instructed to name the emotion portrayed by the block of faces just seen. In addition to identifying the emotion, they were also asked to rate the intensity of the emotion on a 9-point rating scale (9 represented maximum intensity and 1 minimal expression).

2.3. Scales

All the following scales were administered just before the start of the SCRs measurements: (1) Cambridge Depersonalization Scale (Sierra and Berrios, 2000). This is a recently validated scale which has been shown to discriminate patients with depersonalization disorder against patients with anxiety disorders or temporal lobe epilepsy. (2) Spielberger's trait and state anxiety scales (Spielberger et al., 1977). Beck's depression inventory (BDI; Beck et al., 1961).

2.4. Procedure

Skin conductance was recorded using standard Ag–AgCl electrodes 0.5 cm in diameter. Electrodes were attached to the distal phalanges of the 1st and 2nd digits of the non-dominant hand. Skin conductance was measured using a constant voltage (0.6 V) skin conductance module (Contact Precision Instruments SC4) attached to a PC. K-Y Jelly (Johnson and Johnson) was used as an electrolyte. The skin conductance signal was sampled at 100 ms intervals. Only deflections equal to or greater than 0.04 μS were computed. The timing of the stimuli presentation was synchronised to the skin conductance recording program.

To standardise the dermo-gel–electrode interface, subjects were requested to wash their hands using a non-abrasive soap (ivory soap) as recommended by Cacioppo and Tassinary (1991). The subject was then led into the testing room (adjacent) and sat on a comfortable chair. After the electrodes had been placed, there was a 5-min habituation period during which the subject was requested to sit quietly, relax, and move as little as possible.

2.5. Data analysis

Amplitude was defined as the highest deflection (phasic increase in conductance) initiated 1–4 s after stimulus onset (first face of each block) and exceeding 0.04 μS. For stimulus presentation SCR magnitude (mean value of amplitude computed across all stimulus presentations including those without a measurable response) was obtained. A log transformation (log[SCR+1]) was used to normalise the magnitude data.

Since magnitude has the potential disadvantage of confounding frequency and amplitude, which do not always covary, an SCR probability was computed as a measure of response frequency above a threshold regardless of amplitude (number of responses above 0.04 μS/total number of presentations) (Roedema and

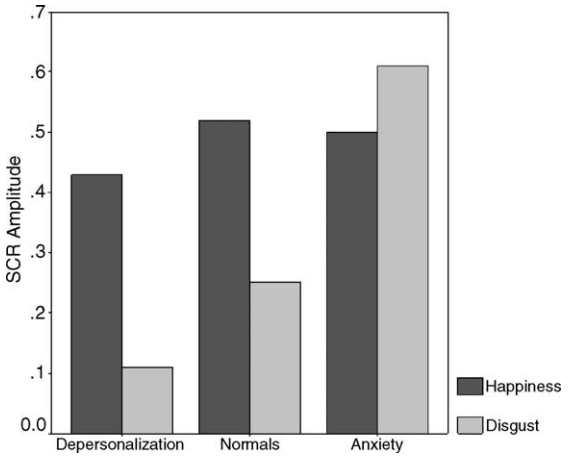

Fig. 1. Mean log-transformed SCRs to disgust and happy facial expressions across the three groups.

Table 1
Mean global scores and standard deviation (in parentheses of administered scales and demographic data

	Depersonalization disorder ($n=16$)	Healthy controls ($n=15$)	Anxiety disorders ($n=15$)	Post hoc (Scheffé $P<0.05$)	One-way ANOVA
Age	32.4 (6.7)	33.2 (7.2)	33.6 (9.2)	NS	$F(2,33)=0.11$ $P=0.89$
Sex (women/men)	7/9	7/8	7/8	NS	$F(2,33)=0.035$ $P=0.96$
CDS	148.7 (41.1)	15 (13.5)	45.21 (32.6)	D vs. C,A	$F(2,31)=29.7$ $P<0.001$
BDI	17.88 (5.3)	3.5 (2.6)	26.4 (12.3)	D,A vs. C	$F(2,30)=25.8$ $P<0.001$
Spielberger (state)	48.6 (13.4)	26.3 (11.4)	52.68 (17.88)	D,A vs. C	$F(2,32)=7.6$ $P<0.001$
Spielberger (trait)	54 (5.2)	33 (2.7)	57 (15.7)	C vs. D,A	$F(2,33)=26$ $P<0.001$

CDS=Cambridge Depersonalization Scale; BDI=Beck's Depression Inventory; BAI=Beck's Anxiety Inventory. Scheffé's post hoc analysis (depersonalization disorder=D, normal controls=C, anxiety disorders=A). NS=Non significant.

Simons, 1999). In addition to the above measurements, the number of non-specific responses (i.e. deflections occurring later than 4 s after stimulus onset) during each 30-s epoch were counted.

Statistical analysis was performed with SPSS version 11 software. Analysis of variance (ANOVA) was used throughout accompanied by post hoc analysis (Scheffé test). An alpha level of significance of 0.05 was used and all significance tests were two tailed.

For the purpose of hypothesis testing, comparison of magnitudes to stimuli across groups (Fig. 1) constituted the primary outcome variables. All other analyses were regarded as exploratory.

3. Results

The three groups did not differ significantly on age [$F(2,33)=0.11$, $P=0.89$] or sex [$F(2,33)=0.035$, $P=0.96$]. As can be seen in Table 1 subjects in the depersonalization and the anxiety groups had very similar scores on both the Spielberger anxiety scales (state and trait) and the BDI. As expected, the normal control group obtained significantly lower scores on these scales. Scores on the CDS differentiated well subjects in the depersonalization group from the two control groups.

3.1. Subjective responses

No difference was found for emotion recognition between the subjects with depersonalization and the two control groups $F(2,30)=1.02$, $P=0.37$. However, intensity rating scores revealed main effects for type of emotion $F(1,28)11.5$, $P=0.002$ (happy>disgust), diagnostic group $F(2,28)10.7$, $P=0.0001$ (depersonalization<control groups), and an interaction of these two $F(2,28)5.7$, $P=0.008$. Analysis of simple effects within groups revealed significantly lower intensity ratings for disgust vs. happiness in the depersonalization group. $F(1,11)$ 20.3, $P=0.001$. Between groups analysis in turn, showed that the depersonalized group had significantly lower disgust intensity ratings than the two control groups $F(2,3)20.4$, $P=0.0001$. There were no differences for happiness ratings between the three groups $F(2,31)2$, $P=0.14$ (Table 2).

Analysis of the effects of presentation parameters on intensity ratings showed main effects for colour of

Table 2
Mean and standard deviation of SCRs, latencies and subjective ratings to administered stimuli

	Depersonalization disorder ($n=16$)	Healthy controls ($n=15$)	Anxiety disorders ($n=15$)	Scheffé $P<0.05$	ANOVA
SCR amplitude to stimuli					
Happiness	0.43 μS (0.027)	0.52 μS (0.062)	0.5 μS (0.042)	NS	$F(2,32)=1.6$ $P=0.201$
Disgust	0.11 μS (0.001)	0.25 μS (0.015)	0.61 μS (0.034)	D,C vs. A	$F(2,28)=9.4$ $P=0.01$
Emotion recognition (% of hits)					
Happiness	90.7 (23.9)	95.3 (11.9)	88.5 (15.7)	NS	$F(2,30)=0.37$ $P=0.69$
Disgust	69.2 (31.2)	78.4 (19)	82.8 (7.5)	NS	$F(2,30)=0.91$ $P=0.41$
Intensity of expression ratings					
Happiness	4.7 (0.68)	5.1 (0.62)	4.7 (0.32)	NS	$F(2,31)=2$ $P=0.144$
Disgust	3.4 (0.44)	5.0 (0.44)	4.8 (0.39)	D vs. C,A	$F(2,30)=20.4$ $P=0.0001$

Significant differences between groups were determined by Scheffé's post hoc analysis. Depersonalization disorder=D, healthy controls=C, anxiety disorders=A. *=Non significant. SC=Skin conductance.

presentation (colour > black and white) [$F(1,31)=13.2$, $P=0.001$] and for diagnostic group (depersonalization < normal and clinical controls) [$F(2,31)=7.6$, $P=0.002$], but no interactions between the two [$F(2,31)=0.52$, $P=0.6$].

There were no main effects of movement [$F(1,31)=1.13$, $P=0.2$], group [$F(1,31)=1.3$, $P=0.27$] or interactions between the two [$F(2,31)=1.3$, $P=0.27$].

3.2. Skin conductance response

Because the prediction made was for an interaction between type of emotion and subject group (i.e. a disproportionately lower response to disgust expressions in the DP patients) a mixed two-factor ANOVA was used. It revealed a main effect for group (Depersonalization < Anxiety Group) [$F(1,28)=13.6$, $P=0.001$], and a main effect for type of emotion (happiness > disgust) [$F(2,56)=4.5$, $P=0.01$]. There was also a significant group × type of emotion interaction [$F(2,32)=4.8$, $P=0.03$]. Simple effect analyses showed that the anxiety group had significantly higher SCR amplitude to expressions of disgust, than the depersonalization and normal control groups ($F=5.3$, $df=2$, $P=0.013$). There was a similar trend between the two control groups, with the DPD group showing smaller SCRs to disgust than the normal controls. In fact the effect size of the difference in SCR to disgust expressions between DPD patients and normal controls was 0.98 which shows that a minimum of 21 subjects in each group should have been necessary for the difference to have been significant (power = 80%, $\alpha=0.05$).

A within group analysis showed that in both the depersonalization group and the normal controls the SCR to disgust was significantly lower than SCRs to happiness expressions (Scheffé $P<0.05$).

Analysis of SC response probability did not reveal significant differences [$F(2,29)=0.713$, $P=0.499$], hence suggesting that the amplitude difference was not confounded by frequency of response.

Comparison of number of deflections during each 30 s epoch did not reveal significant differences for happiness [$F(2,27)0.2$, $P=0.75$] or disgust [$F(2,27)1.2$, $P=0.3$].

Correlations between SCRs and CDS scores in the depersonalization group were as follows: $r=-0.4$ ($P=0.13$) for disgust faces and $r=0.2$ ($P=0.36$) for happy faces. Similarly those between SCRs and scores on the 'state' Spielberger anxiety scale were 0.37 ($P=0.18$) for disgust and 0.13 ($P=0.63$) for happiness.

Analysis of the effects of moving facial expressions on SCR did not reveal significant main effects [$F(1,28)=1.5$, $P=0.21$], group effects [$F(2,28)=1.2$, $P=0.3$] or interactions between the two [$F(2,28)=0.62$, $P=0.54$]. Likewise there were no main effects of colour of presentation [$F(1,28)=0.34$, $P=0.56$], group effects [$F(1,28)=1.5$ $P=0.23$] or interactions between the two [$F(2,28)=0.79$, $P=0.46$].

4. Discussion

To our knowledge this is the first study to explore SCR to emotional facial expressions in depersonalization disorder. One limitation of the study worthwhile mentioning is that the anxiety disorders group was not homogeneous and patients were not diagnosed with standardised diagnostic procedures. However, as we were mainly interested in using clinical controls with high levels of anxiety, rather than testing the autonomic responses of a specific anxiety disorder, it is unlikely that this methodological drawback introduced a significant source of error in the findings.

The main finding of the study is that patients in the anxiety group were found to have heightened autonomic responses to disgust expressions as compared with DPD patients and normal controls. Most interesting however, is the finding that patients with depersonalization did not show this autonomic hyperreactivity, in spite of the fact that both the DPD and anxiety groups reported similar levels of subjective anxiety as measured by the anxiety scales. Indeed, in view of the high anxiety levels in the DPD patients, it is striking that their autonomic responses to disgust resembled those of the non-anxious controls. This finding would seem to suggest that the presence of depersonalization in otherwise anxious patients has a blunting effect on autonomic reactivity. The fact that we found a negative correlation trend between scores on the CDS and SCRs to disgust expressions would seem consistent with this view. As mentioned above, our findings are in agreement with a recent study comparing levels of urinary norepinephrine in patients with depersonalization disorder and normal controls. Although depersonalization accompanied by anxiety was associated with increased noradrenergic tone as compared with normal controls, within the depersonalization group there was a marked basal norepinephrine decline with increasing levels of depersonalization (Simeon et al., 2003b). Similarly, Delahanty et al. (2003) found that amongst survivors of motor vehicle accidents, 15-h urinary norepinephrine was inversely correlated to the severity of peritraumatic dissociation. Furthermore, as mentioned above, in a challenge study on sexual abuse victims with a diagnosis of PTSD it was found that

the presence of a dissociative response during evocation of a traumatic event seemed to prevent a heart rate increase otherwise observed in those patients who did not dissociate during the task (Lanius et al., 2002). Along these lines, it may be speculated that autonomic responses in patients with depersonalization are likely to reflect a balance between two opposing tendencies. One excitatory determined by anxiety levels, and an inhibitory one determined by depersonalization intensity. Further studies are needed to test this idea.

As expected, colour was found to facilitate perceptual recognition in the three groups (movement had a similar trend), but did not seem to have an effect on SCRs, hence suggesting that the differences found were mostly controlled by the emotional processing of the stimuli, rather than by the processing of colour and movement.

Interestingly enough, patients with depersonalization did not differ from the two control groups in their ability to recognise disgust expressions, but rated them as less intense. This finding is in line with results from a different study, which showed that depersonalization patients did not differ from controls in their assessment of how unpleasant aversive visual stimuli were, but rated them as less arousing (Sierra et al., 2002). Our findings of accurate emotion recognition, but underrated intensity (for disgust faces), would seem to suggest that the ability to judge the intensity of an emotional expression depends to some extent on a representation of autonomic arousal. In this regard it is relevant that the insula, which seems involved in the conscious representation of autonomic body states (Critchley et al., 2002), and the recognition of disgust (Phillips et al., 1997), has been found hypoactive in patients with DPD when viewing disgust evoking pictures (Phillips et al., 2001).

Increasing evidence suggests that a bi-directional communication between limbic regions and autonomic activity play a central role in both the expression and experiencing of emotions (Mayer et al., 2000; Katkin et al., 2002). It has been found that emotion-specific patterns of autonomic output are fed back to the brain (Mayer et al., 2000), in particular to the insula and cingulofrontal cortices (Aziz et al., 2000; Critchley et al., 2001). Even though this process unfolds largely without conscious awareness, it may play an important role in emotional perception (Katkin et al., 2002). In this regard, a recent study using structural neuroimaging on 108 subjects with focal brain lesions, found that the perception of intensity in facial emotional expressions appears to require the integrity of the right somatosensory cortices (Adolphs et al., 2000). In fact these authors found correlations between ratings of facial emotion intensity and both the size of the lesion and clinical somatosensory deficit (measured as impaired touch sensation). Similar areas were also found to be important for the non-verbal categorization of emotions. In general the above findings would seem to suggest that an adequate perception of bodily states is relevant for the recognition of emotional states in both self and others or at least the appreciation of their intensity.

In short, the findings of this study provide further support to the idea that patients with depersonalization have a selective impairment in the processing of threatening or unpleasant emotional stimuli. However, given that in the present study we only contrasted disgust and happy faces, it remains to be seen whether our results are specific for disgust, or involve negative emotions in general (i.e. disgust, fear, anger). It is clear that further research is needed to elucidate the nature and mechanisms underlying autonomic responses in DPD.

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