

# Psychophysiological ambulatory assessment of affective dysregulation in borderline personality disorder

Ulrich W. Ebner-Priemer<sup>a,\*</sup>, Stacy S. Welch<sup>b</sup>, Paul Grossman<sup>c</sup>,  
Thomas Reisch<sup>d</sup>, Marsha M. Linehan<sup>e</sup>, Martin Bohus<sup>a</sup>

<sup>a</sup> Department of Psychosomatic Medicine and Psychotherapy, Central Institute of Mental Health, Postfach 122120, 68072 Mannheim, Germany

<sup>b</sup> Evidence Based Treatment Centers of Seattle, Seattle WA, USA

<sup>c</sup> Division of Psychosomatic Medicine, Department of Internal Medicine, University of Basel Hospital, Basel Switzerland

<sup>d</sup> University Hospital of Social and Community Psychiatry, Bern, Switzerland

<sup>e</sup> Department of Psychology, University of Washington, Seattle WA, USA

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## Abstract

Many experts now believe that pervasive problems in affect regulation constitute the central area of dysfunction in borderline personality disorder (BPD). However, data is sparse and inconclusive. We hypothesized that patients with BPD, in contrast to healthy gender and nationality-matched controls, show a higher frequency and intensity of self-reported emotions, altered physiological indices of emotions, more complex emotions and greater problems in identifying specific emotions. We took a 24-hour psychophysiological ambulatory monitoring approach to investigate affect regulation during everyday life in 50 patients with BPD and in 50 healthy controls. To provide a typical and unmanipulated sample, we included only patients who were currently in treatment and did not alter their medication schedule. BPD patients reported more negative emotions, fewer positive emotions, and a greater intensity of negative emotions. A subgroup of non-medicated BPD patients manifested higher values of additional heart rate. Additional heart rate is that part of a heart rate increase that does not directly result from metabolic activity, and is used as an indicator of emotional reactivity. Borderline participants were more likely to report the concurrent presence of more than one emotion, and those patients who just started treatment in particular had greater problems in identifying specific emotions. Our findings during naturalistic ambulatory assessment support emotional dysregulation in BPD as defined by the biosocial theory of [Linehan, M.M., 1993. *Cognitive–Behavioral Treatment of Borderline Personality Disorder*. The Guildford Press, New York.] and suggest the potential utility for evaluating treatment outcome.

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*Keywords:* Ambulatory monitoring; Heart rate variability; Additional heart rate; Biosocial theory; Emotion; Ecological validity

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## 1. Introduction

Borderline personality disorder (BPD) is characterized by severe deficits in interpersonal, cognitive, and emotional functioning (Lieb et al., 2004). Many experts now believe that the pervasive problems in affect regulation (often termed “emotion dysregulation”) represent

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\* Corresponding author. Tel.: +49 621 17034404; fax: +49 621 17034405.

E-mail address: [ulrich.ebner-priemer@zi-mannheim.de](mailto:ulrich.ebner-priemer@zi-mannheim.de) (U.W. Ebner-Priemer).

the central area of dysfunction (Linehan, 1993; Coid, 1993; Corrigan et al., 2000; Skodol et al., 2002; Sanislow et al., 2002). According to the biosocial theory of Linehan (1993), emotion dysregulation in BPD comprises increased sensitivity to emotional stimuli, unusually strong reactions, the occurrence of complex emotions (more than one emotion simultaneously), and problems in identifying emotions. However, data actually demonstrating emotion dysregulation is sparse and inconclusive.

Support for the theory of emotion dysregulation is primarily based on subjective measures of emotion and experimental studies. A few studies that used multiple self-rating (diaries) over time reported a higher level of unpleasant affect in BPD patients, compared to a clinical control group (Stein, 1996) and psychologically healthy controls (Cowdry et al., 1991; Stiglmayr et al., 2001). Using self-rating questionnaires, Koenigsberg et al. (2002) found however no evidence of elevated affect intensity in BPD patients when compared with other personality-disordered individuals. Experimental studies that applied emotion-inducing techniques also supported greater emotional dysregulation in BPD: Levine et al. (1997) elicited a heightened intensity of negative emotions in BPD patients, and Herpertz et al. (1997) provided evidence of elevated baseline emotional activation among patients with self-injuries, as compared to healthy controls.

Studies using psychophysiological indicators of emotion have thus far failed to find a consistent pattern of affective dysregulation in BPD. Herpertz et al. (1999) exposed BPD patients and healthy controls to affect-inducing pictures but did not find any hyperreactivity among patients, either with respect to physiological indicators of emotion (heart rate, electromyogram or skin conductance) or to subjective ratings of emotion. In a similar study using functional MRI, Herpertz and colleagues (2001) found no differences in subjective emotions. However, BPD participants did show elevated blood flow in the amygdala. Likewise, Donegan et al. (2003) revealed greater left amygdala activation to emotional facial expression among patients with BPD, and Ebner-Priemer et al. (2005) revealed significantly higher startle response in BPD; enhanced startle response is caused by increased amygdala activation (e.g. by electrical stimulation), at least in animals (Davis et al., 1999). Unfortunately, neither Donegan et al. (2003) nor Ebner-Priemer et al. (2005) reported subjective emotional ratings.

There are a number of possible explanations for these conflicting results. First, BPD patients without medication, who are often recruited for psychophysiological studies, are extremely rare (Zanarini et al., 2004) and may represent a healthier subgroup of BPD patients than

medicated individuals. The same argument may apply to patients with BPD who are assessed after a long treatment program. Secondly, it is possible that the affect-induction methods used were insufficient to evoke affective dysregulation and that more personally relevant stimuli are preferable. Thirdly, most studies have been performed in a laboratory environment, and it is possible that the somewhat artificial conditions of the laboratory (e.g. insufficient time for adaptation, the psychological consequences of being observed and contrived experimental protocols) influenced the measurement of affective dysregulation.

One difficulty encountered in the investigation of physiological indices of emotions in ambulatory studies has been the teasing apart of emotional and physical influences. In order to address this problem, Myrtek (2004) developed an algorithm to partition that part of heart rate (HR) increase that does not directly result from physical or metabolic activity; he has termed this measure “additional heart rate” (aHr). The algorithm is based on two experimental findings: namely, that HR reactivity, elicited by a combination of mental, emotional and physical stressors, closely approximates the additive combination of the HR responses evoked by the individual factors (Myrtek and Spital, 1986; Roth et al., 1990), and that metabolic demand can be estimated with sensitive motion detectors (Myrtek, 2004).

This non-metabolic heart rate increase (aHr) appears to be a valid measure of emotional response and has now been validated in multiple studies including more than 1300 participants. For example, aHr in male students was found to be greater when watching erotic films compared to comedies and this applied not only during rest but also under varied levels of physical exercise, and that these physiological differences corresponded to self-ratings of excitement (Myrtek and Bruegner, 1996). This demonstrates that the algorithm is not only able to detect emotional events under basal conditions but also under conditions of activity. Effects on aHr have also been demonstrated for children watching television at home: both school-age boys (Myrtek et al., 1996b) and preschool boys and girls (Wilhelm et al., 1997) manifested greater aHr during films with action scenes. In the same study, girls showed increased aHr while watching commercials that targeted girls (e.g. Barbie), as compared to gender-neutral commercials (Wilhelm et al., 1997). Additionally, one investigation found that train drivers showed higher aHr during situations associated with heightened risk for accidents (Myrtek et al., 1994). aHr is increased during leisure-time activities compared to periods of monotonous work (Myrtek et al., 1999), and in social interactions as compared to being alone (Myrtek et al., 1995). This is

consistent with the use of aHR as an emotional indicator. There are several indications that aHR is primarily under sympathetic control: beta-adrenergic blockade studies of HR (Langer, 1985), evidence that metabolically induced HR responses are mainly vagally mediated (Grossman, 1983; Grossman and Svebak, 1987; Watkins et al., 1998) and the missing correlation between aHR and heart rate variability (Myrtek et al., 1996a).

A large and growing literature has linked indices of reduced baseline vagal activity to disorders with dysregulated affective styles, like depression, anxiety and panic (Beauchaine, 2001) and estimates of high vagal activity to relaxation (Houtveen et al., 2002).

In order to address the question of emotional dysregulation in BPD, we employed psychophysiological ambulatory monitoring, which is also called ecological momentary assessment (Stone and Shiffman, 1994), to investigate both self-report measures (frequency and intensity of discrete emotions) and physiological indices of emotions (additional heart rate, spectral analysis indices of cardiac vagal activity) during 24 h of normal daily life. This approach circumvents several methodological limitations. Investigating naturally occurring symptoms in everyday life may render affect-inducing methods unnecessary and eliminates many of the problems associated with laboratory studies. We included patients who were currently in our treatment programs and did not alter their medication schedules; this was in order to provide a typical and experimentally unmanipulated sample of BPD patients in treatment, and therefore increase the generalizability of our findings.

According to the biosocial theory (Linehan, 1993), we specifically hypothesized that patients with BPD, in contrast to matched healthy controls, would 1) show a higher frequency and intensity of self-reports of emotion, 2) manifest greater levels of additional heart rate and lower levels of heart rate variability indices of autonomic control, 3) report more complex emotions, and 4) show greater difficulty in identifying discrete emotions during periods of reported emotional arousal.

## 2. Method

### 2.1. Subjects

139 subjects were initially recruited for this study. Ten controls were excluded due to psychiatric disorders. Twenty-three data sets had to be eliminated because of technical problems such as defect electrocardiogram leads, damage to accelerative sensors, or data loss in the palm top. Six data sets were aborted early on account of compliance problems and were therefore also excluded.

Ultimately, 50 female patients meeting criteria for BPD and a comparison group of 50 female psychologically healthy control subjects (HC) participated in this study. 42% of the participants was investigated at the University of Washington, Seattle, USA, and the remaining 58% was assessed at the University of Freiburg, Germany. All patients met DSM-IV criteria for BPD, assessed according to the appropriate section of the International Personality Disorder Examination: IPDE (Loranger, 1999). Axis-I comorbidity was assessed with the Structured Clinical Interview for DSM-IV Axis-I Disorders: SCID-I (First et al., 1997). Participants with a lifetime history of schizophrenia, bipolar disorder, or current alcohol/drug abuse were excluded. Trained psychologists (Freiburg) and Masters-level clinical assessors (Seattle) administered all diagnostic instruments. HCs were randomly selected from the national resident register of the City of Freiburg or by means of advertisement in Seattle. Exclusion criteria for the control group included the diagnosis of BPD (IPDE), any current or lifetime Axis-I disorder (SCID-I), current psychotherapy, current medication (based on verbal interview) and other Axis-II disorders (SCID-II). However, the last criterion applies only to the German sample. 20% of the patients was free of psychotropic medication. Of the 80% of patients on drugs, 65% received antidepressants, 32% antipsychotics, and 30% hypnotics. Comorbid Axis-I disorders of patients from the BPD group included major depressive disorder (current: 36%), anxiety disorders without PTSD (current: 60%), PTSD (current: 60%), eating disorders (lifetime: 50%), and substance abuse (lifetime: 60%). All patients participated in the psychotherapy programs. The German group was investigated before the start of dialectical behavior therapy (DBT), whereas the US group was examined during an ongoing DBT treatment. The age of BPD patients (Mean=31.3, S.D.=8.1) and HC (Mean=27.7, S.D.=6.8) differed significantly ( $t=-2.44$ ,  $df=98$ ,  $P=0.016$ ). Age did not differ between centers (total group  $t=0.619$ ,  $df=67$ ,  $P=0.538$ ; controls  $t=-0.394$ ,  $df=33.6$ ,  $P=0.696$ ; patients:  $t=1.496$ ,  $df=33.7$ ,  $P=0.144$ ). All subjects were paid for participating in the study. After complete description of the study to the subjects, written informed consent was obtained. The study was approved separately by the respective ethical review committees of the University of Freiburg and the University of Washington.

### 2.2. Online physiological measurements

All participants underwent 24-hour ambulatory monitoring of physical activity and ECG. Subjects carried in a leather case the portable physiological digital recorder and

analysis system Vitaport II (Becker Engineering, Karlsruhe, Germany) and a palmtop computer (Psion 3 a, London, UK) for psychological assessment of emotions. ECG was sampled at 256 Hz from standard limb electrocardiogram leads (Blue Sensor, Medicotest, Germany). Physical activity was sampled at 32 Hz by two accelerative, three-dimensional sensors placed on the chest below the clavicle and on the thigh above the knee. Heart rate and physical activity (PA) were calculated online for each minute and compared to the moving average of the previous 3 min in order to detect an episode of additional heart rate (aHr). An “emotional” event was defined as having occurred when the heart rate of a given minute was at least 3 beats/min greater than the average of the previous minutes, with no (or only a small) increase in physical activity ( $\leq 10$  units). For greater details: Activity values [Mean (S.D.)] during different postures are as follows: lying 5.05 (1.81), sitting 16.0 (4.8), standing 31.2 (8.7), and walking 76.4 (7.4). If physical activity increased, the threshold for an aHr episode increased as well:  $\text{Threshold} = (\text{Physical activity} + 90)/30$ . The mean aHr values were divided by 3 to yield the factor by which the minimal heart rate was exceeded, and then multiplied by 10. The total amount of aHr for a segment of the record was determined by the number of aHr and the amount of aHr for every event. A more detailed description of the methodology and algorithm can be found in Myrtek (2004).

### 2.3. Offline physiological data transformation

Physical activity was separated offline into AC and DC components by a FIR digital filter with a cut-off frequency at 0.5 Hz. Raw signal, DC-values, and rectified AC-values were averaged across data points for each condition and monitoring segment. Data segments were classified hierarchically by referring to an individual standard protocol, resulting in eight different posture patterns (3 subtypes of sitting, standing, 3 subtypes of lying, walking): for details see Foerster and Fahrenberg (2000). Because heart rate is highly dependent on an individual’s physical condition, a heart rate baseline was calculated by identifying the hour of the night at which the heart rate reaches a minimum. High-frequency heart rate variability (HF-HRV) was calculated from the high-frequency band (0.12–0.5 Hz) of spectral power according to published specifications (Berntson et al., 1997). ECG was transformed in inter-beat-intervals, resampled at 4 Hz, and Short-Time Fourier Transform (STFT) was performed for every data point (time window  $\pm 50$  s). Spectral density was averaged every minute. All online and offline analyses and artifact checks were performed by the interactive software package “Freiburg Monitoring System”

(Myrtek et al., 2001) according to a published procedure (Myrtek, 2004).

### 2.4. Subjective measurements of emotion

A feedback signal was emitted by the minicomputer every 10 to 20 min. The software-program MONITOR (Fahrenberg et al., 2001) displayed questions regarding the subject’s current emotions (How did you feel just before the beep?), with a list of possible answers: happy, interest, anxious, angry, sad, shame, disgust, emotion but can’t name it, and no emotion. This list was derived from studies defining basic emotions (Linehan, 1993). Participants were further asked to score the intensity of their feeling on an 11-point Likert scale. They were also queried about the occurrence of any second emotion (same list as previous, without the first emotion mentioned) and its intensity (11-point Likert scale). Response was automatically time-stamped by the software program. Prompting consisted of 3 signals (duration 5 s) with an intersignal interval of 40 s. If the participant did not respond at all (within 340 s), the trial was recorded as missed. Participants were instructed in the use of the equipment and trained how to respond to questions presented on the pocket PC minicomputer’s display, and were told how to turn the device off prior to sleep or nap (suspension of prompting) and how to turn it on again upon waking. The physiological data recorder was not shut down during the night.

### 2.5. Data analysis

For independent frequency data, relative frequencies (of occurrences of specific emotions) were calculated and tested non-parametrically with a Wilcoxon-Test (rank-sum, two-tailed). Effect sizes were calculated with the formula,  $r = Z/\sqrt{N}$  (Clark-Carter, 1998); p. 455). For independent interval data (including physiological data and intensity of specific emotions), independent sample *t*-tests (two-tailed) were used and pooled effect sizes were calculated. Statistical analysis was performed with SAS 6.12 (SAS Institute Inc, Cary, NC) and SPSS 9.0 (SPSS Inc, Chicago, IL). A *P* value of 0.05 was considered to be a statistically significant difference.

### 2.6. Data acquisition

To ensure that physical activity reflects metabolic demand, there must be a strong correlation between physical activity, as measured by the movement sensors, and heart rate. To make sure that additional heart rate is not caused by metabolic demand, there should be no



Table 1

Criteria of the psychophysiological ambulatory monitoring: data acquisition, missing data, posture, and motion pattern in BPD and HC

N	BPD	HC	$\chi^2$	df	P
	50	50			
Hours of data acquisition	23.5(0.88)	23.3(1.45)	0.32	1	0.569
Hours of shut-down during night	9.65(1.34)	8.44(1.91)	17.17	1	<0.001
Number of ratings	52.3(7.25)	55.9(9.14)	5.97	1	0.015
	BPD	HC			P
% of ignored requests*	5.36 (8.67)	2.71 (4.16)			0.196
MD heart rate (%)*	0.13 (0.29)	0.09 (0.41)			0.408
MD physical activity (%)*	0.04 (0.16)	0.08 (0.41)			0.355
MD additional heart rate (%)*	0.16 (0.33)	0.13 (0.43)			0.702
% of time sitting*	33.6 (9.4)	37.8 (9.2)			0.058
% of time standing*	18.4 (7.6)	19.3 (8.8)			0.801
% of time lying*	40.9 (10.4)	37.8 (10.1)			0.096
% of time walking*	7.1 (4.2)	5.1 (2.8)			0.009

MD = missing data; \* = Wilcoxon-Test.

correlation between aHr and physical activity (Myrtek et al., 1996a). As expected, the (pooled) within-subject correlation between activity and heart rate was high (all  $r=0.761$ ; BPD  $r=0.747$ ; HC  $r=0.776$ ), and low between aHr and activity (all  $r=0.056$ ; BPD  $r=0.046$ ; HC  $r=0.065$ ). Additionally, the correlation of HF-HRV, our vagal index, and aHr was low ( $r=-0.134$ ). This is also to be expected if metabolic HR change is predominantly vagally mediated. Altogether, these correlations indicate a proper operation of the algorithm.

Table 1 indicates that the duration of data acquisition did not differ between groups. Patients did however shut down the minicomputer for a longer time at night, and their mean number of ratings was consequently lower, although other values related to ratings in the table are fairly similar, such as ignored requests (range: BPD: 0.0–37.2; HC: 0.0–16.6). Furthermore, missing data in physiological parameters were low and did not differ between groups. Behavior differences were quantitatively relatively small, although the patients did tend to spend slightly more time walking and lying and less time sitting than controls. To verify ecological validity, we asked participants after conclusion of the whole procedure if the device and the monitoring procedure altered their behavior (e.g. altered attention to emotions, altered attention to body sensations, extraordinary events during the day, unpleasantness of the device etc.). Reported distress and reactivity to monitoring was very minimal and did not differ between groups (data available upon request).

### 3. Results

#### 3.1. Subjective emotions

The relative frequency of the first reported emotion (overall and for specific emotions) is shown in Fig. 1. BPD patients reported specific negative emotions (anxious, angry, sad, shame, and disgust) significantly more often than the HC group. Probabilities and effect sizes are shown in Table 2. Furthermore BPD individuals also reported significantly fewer positive emotions (happy and interest). Effect sizes for group differences of all specific positive and negative emotions range between medium and large.

Because teaching patients to identify and name emotions is a major focus in DBT and patients in Germany/US differed in regard of duration of treatment participation (the German group was investigated before start of DBT treatment, whereas the US group was examined during ongoing DBT treatment), we analyzed the non-specific emotion for both countries separately, resulting in significant differences. German BPD participants had significantly more non-specific emotions than the German HC ( $\chi^2=4.18$ ,  $df=1$ ,  $P=0.041$ ), whereas American patients reported fewer non-specific emotions than American HC ( $\chi^2=5.59$ ,  $df=1$ ,  $P=0.018$ ).

The overall intensity of subjective emotions (see Fig. 1) was significantly increased in BPD patients compared to the HC group (see Table 2 for probabilities and effect sizes). Examination of the specific emotions revealed significantly elevated intensities of all negative emotions for BPD participants, with large effect sizes. The intensity of positive emotions, however, was not significantly higher in the BPD group.

Complex emotional responses consist of more than one simultaneous emotion. In analyses of these responses, examination of the second reported emotions revealed results similar to those in the analyses of the first reported emotions (probabilities and effect sizes are listed in Table 2). The frequency of complex emotional responses (overall) was significantly higher in the BPD group (see Fig. 1), as was the experience of all negative emotions. There were no group differences in the specific positive emotions reported. Effect sizes for the frequency of negative emotions ranged between medium and large.

The intensity of the second specific negative emotion was significantly higher in BPD patients compared to HC, and this applied for all specific negative emotions (see Fig. 1 and Table 2). All effect sizes for the intensity of negative emotions were large. Positive emotions (second emotion) were not significantly increased in the BPD group.

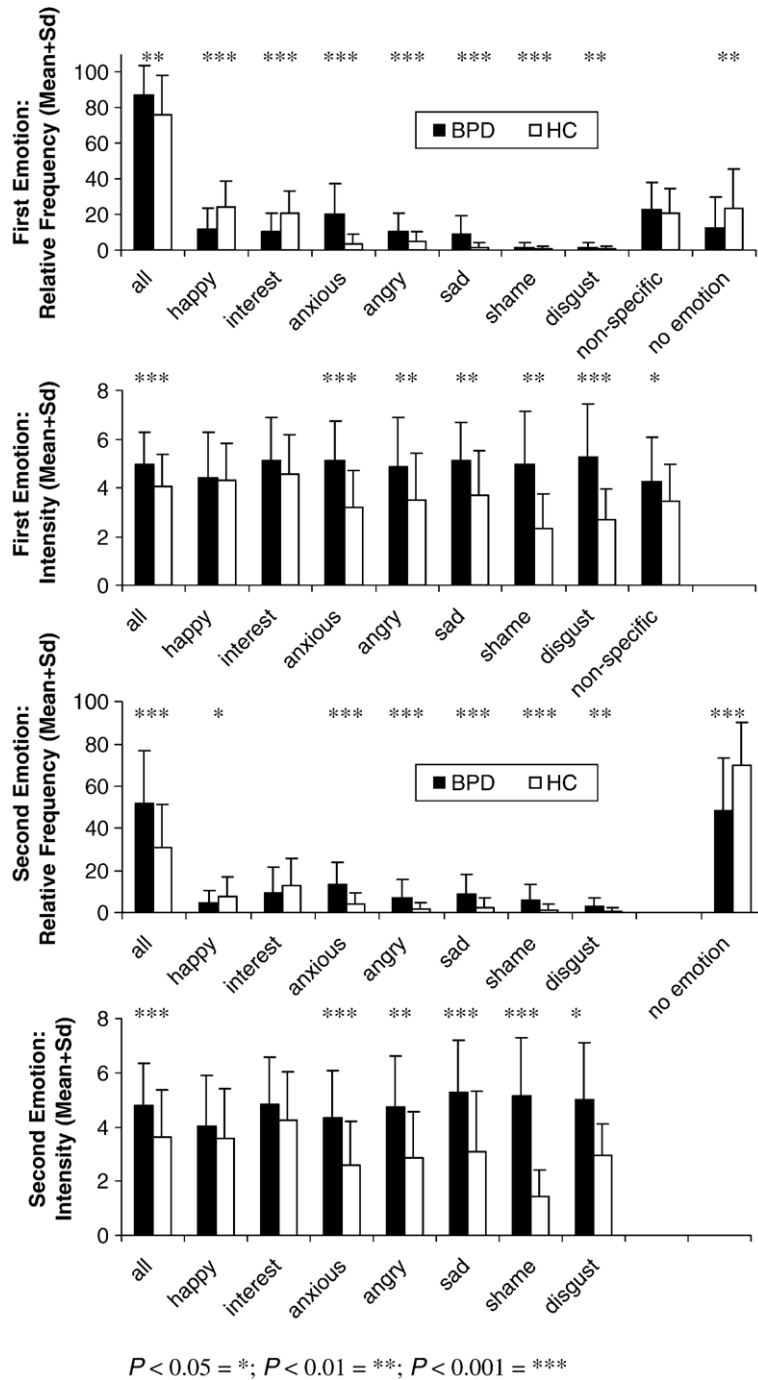


Fig. 1. Frequency and intensity of self-reported emotions for BPD (*n*=50) and HC (*n*=50) during 24-h ambulatory monitoring.

Finally, we examined the possible differences between the subjective ratings of emotion of patients with and without medication. The 36 comparisons (single and global frequency and intensity of all primary and secondary emotions) revealed only one significant difference

(not adjusted;  $\alpha < 0.05$ ). This is below the expected probability doing 36 comparisons. Furthermore, there are no overt systematic differences such as heightened intensity or frequency of negative or positive emotions in one of the groups.

Table 2

Probabilities and effect sizes of group differences (BPD vs. HC) for frequency and intensity of all specific positive and negative emotions

	Frequency of first emotion*		Intensity of first emotion				Frequency of second emotion*		Intensity of second emotion			
	<i>P</i>	ES ( <i>r</i> )	<i>t</i>	<i>df</i>	<i>P</i>	ES ( <i>d</i> )	<i>P</i>	ES ( <i>r</i> )	<i>t</i>	<i>df</i>	<i>P</i>	ES ( <i>d</i> )
Overall	0.009	0.26	3.30	98	0.001	0.67	<0.001	0.43	3.49	94	<0.001	0.71
Happy	<0.001	0.47	0.33	87	0.739	0.07	0.048	0.20	0.90	61	0.369	0.23
Interest	<0.001	0.42	1.61	92	0.110	0.33	0.123	0.15	1.48	74	0.143	0.34
Anxious	<0.001	0.55	4.90	69	<0.001	1.20	<0.001	0.48	4.25	66	<0.001	1.06
Angry	<0.001	0.33	3.02	80	0.003	0.67	<0.001	0.34	3.25	42	0.002	1.05
Sad	<0.001	0.55	2.91	55	0.005	0.81	<0.001	0.44	3.56	50	<0.001	1.04
Shame	<0.001	0.43	2.98	25	0.006	1.44	<0.001	0.47	7.26	25.3	<0.001	2.24
Disgust	0.004	0.29	4.24	28.8	<0.001	1.48	0.002	0.31	2.54	30	0.016	1.24
Non-specific	0.705	0.04	2.29	93	0.024	0.47						
No emotion	0.009	0.26					<0.001	0.43				

\* = Wilcoxon-Test; according to Cohen (1988) effect sizes are considered as follows:  $r=0.1$ : small,  $r=0.3$ : medium,  $r=0.5$ : large;  $d=0.2$ : small,  $d=0.5$ : medium,  $d=0.8$ : large.

### 3.2. Physiological data

Physiological data and statistics are presented in Table 3. As previously mentioned, we did not exclude patients on medication in order to improve generalizability of the findings. Unfortunately, problems are encountered in the psychophysiological assessment of patients on medication, due to known cardiovascular and autonomic effects of several psychotropic medications (Yeragani et al., 2002; Ikawa et al., 2001). When we compared BPD patients with and without medication, there were significant differences (with large effect sizes) on nearly every physiological parameter: heart rate, additional heart rate, heart rate baseline, and HF-HRV.

Comparing only non-medicated BPD participants to the HCs revealed significantly elevated aHr in the (non-medicated) BPD group. Because the comparison of groups

with such different sample sizes is statistically problematic, we compared aHr differences between HC ( $n=50$ ) and non-medicated BPD patients ( $n=10$ ), using a non-parametric test (Wilcoxon rank-sum). Again, there was a significant group difference ( $P=0.023$ ). To further validate this, we age-matched a subgroup of HC ( $n=10$ ) with the non-medicated BPD patient and found also a significant difference for aHr ( $t=2.21$ ,  $df=18$ ,  $P=0.040$ ).

Another critical point is that the differences of aHr may be caused by age differences of the groups (BPD vs. HC). We therefore double checked our findings using age as a covariate, resulting in the same finding, a significant effect for aHr and a non-significant effect for age (aHr:  $F=6.47$ ,  $df=1$ ,  $P=0.014$ ; age:  $F=2.58$ ,  $df=1$ ,  $P=0.114$ ).

Non-medicated BPD participants tended also to have heightened vagal activity (HF-HRV 24 h) in comparison to

Table 3

Physiological statistics for BPD with medication, BPD without medication, and HC as well as probabilities and effect sizes for group differences

	HC	BPD non-med	BPD med	BPD (total)	BPS med. vs. BPS non-med				HC vs. BPD non-med			
	Mean (S.D.)				<i>t</i>	<i>df</i>	<i>P</i>	ES ( <i>d</i> )	<i>t</i>	<i>df</i>	<i>P</i>	ES ( <i>d</i> )
Participants ( <i>n</i> )	50	10	40	50								
Heart rate (24 h)	78.4 (7.4)	76.9 (6.2)	83.6 (10.8)	82.3 (10.3)	-2.59	24.8	0.016	0.76	-0.60	58	0.551	0.22
Activity (24 h)	17.9 (4.2)	17.3 (3.1)	18.1 (4.4)	18.0 (4.1)	-0.55	48	0.582	0.21	-0.472	58	0.638	0.16
Additional heart rate (24 h)	1.84 (0.40)	2.21 (0.52)	1.52 (0.54)	1.66(0.60)	3.64	48	<0.001	1.25	2.61	58	0.011	0.80
Power of HF-HRV (24 h) $\log(\text{ms}^2+1)$	2.91 (0.32)	3.09 (0.27)	2.45 (0.39)	2.58 (0.45)	4.80	48	<0.001	1.91	1.61	58	0.112	0.61
Heart rate baseline at night	61.4 (6.6)	57.0 (5.5)	66.3 (8.9)	64.4 (9.2)	-3.05	17.5	0.001	1.26	-1.96	58	0.054	0.72
Power of HF-HRV (night) $\log(\text{ms}^2+1)$	3.17 (0.36)	3.32 (0.26)	2.89 (0.43)	2.98 (0.43)	3.01	48	0.004	1.21	1.21	58	0.23	0.48

HC = healthy controls; BPD non-med. = borderline patients without medication; BPS med. = borderline patients with medication; HF-HRV = high-frequency heart rate variability  $\log(\text{ms}^2+1)$ ; according to Cohen (1988) effect sizes (*d*) are considered as follows: 0.2=small, 0.5=medium, 0.8=large.

the HC, and reduced concomitant heart rate baseline. Differences were not significant, but both effect sizes were medium to high. Using a non-parametric test (Wilcoxon rank-sum), we compared vagal activity differences between HCs ( $n=50$ ) and non-medicated BPD patients ( $n=10$ ), and found a nearly significant group difference ( $P=0.077$ ). To further validate this, we age-matched a subgroup of HC ( $n=10$ ) with the non-medicated BPD patient, but there was no significant difference for HF-HRV ( $t=1.25$ ,  $df=18$ ,  $P=0.229$ ). There was no group difference for the HF-HRV night-baseline (Wilcoxon  $P=0.234$ ; age-matched sample  $t=1.37$ ,  $df=18$ ,  $P=0.189$ ).

#### 4. Discussion

Our results confirm the first hypothesis that BPD participants show a higher frequency and intensity of self-reported emotions. Patients with BPD reported more emotions overall than HCs; specifically, BPD patients reported more negative emotions and fewer positive emotions. BPD patients also reported greater intensity of negative emotions, but not positive emotions. Our data therefore replicate the findings of increased negative affect reported in diary and questionnaire studies by BPD participants (Cowdry et al., 1991; Stein, 1996; Levine et al., 1997) and at least partially support the unimpaired ability to experience positive emotions (Stein, 1996; Levine et al., 1997). This finding is of some importance, because negative affect (Stanley et al., 2001) and affective instability (Yen et al., 2004) is linked to suicidal behavior in BPD. However, our findings are in conflict with those psychophysiological studies in which self-reports of emotional response did not demonstrate differences.

There are a number of conceivable explanations for these conflicting results. First, we speculated that non-medicated patients may be less dysregulated. But in our data, there were no differences between medicated and non-medicated patients in self-reported emotions. However, the comparison of subgroups with sample size differences of this magnitude is statistically problematic. The same argument (less dysregulated) may be made for studies assessing patients with BPD after a successful treatment program. Because all investigated patients in our study were participants in treatment programs (before and in the middle, respectively) we were not able to investigate the effects of treatment. However, any discussion about symptom severity in BPD has to be based on appropriate questionnaires and not on medication. Secondly, it is possible that the somewhat artificial conditions of the laboratory or the affect-induction methods affected the measurement of affective dysregulation. For example,

the laboratory situation might prevent the occurrence of personally relevant stimuli which otherwise trigger affective dysregulation in normal daily life. At this point, we want to emphasize two advantages of ambulatory monitoring or ecological momentary assessment: first, this approach does not require an emotion-induction method; and second, naturally occurring symptoms are assessed in a setting where patients generally experience their symptoms: in normal daily life. Another advantage of computer-based technology with online rating systems is the prevention of recall bias (Kaepler and Rieder, 2001) and the prevention of subsequent ratings (Stone et al., 2002), which are problematic in questionnaires and paper-pencil diaries. In a paper-pencil diary study, Stone et al. (2002) revealed only 11% of compliant reports, that is complete and according to the time schedule.

Our second hypothesis, that BPD participants have higher values of additional heart rate and lower values of heart rate variability, was only partially confirmed. The non-medicated BPD patients exhibited higher values of additional heart rate compared to HC, as hypothesized. This study therefore revealed a physiological indicator of affective dysregulation in this population, as in the studies of Herpertz et al. (2001), Donegan et al. (2003), and Ebner-Priemer et al. (2005).

Furthermore, vagal activity (HF-HRV 24 h) in non-medicated BPD participants was higher in comparison to the HC, with an at least medium effect size. This finding is surprising because reduced HF-HRV (vagal activity) has been linked to dysregulated affective styles (Beauchaine, 2001), whereas high HF-HRV is linked to relaxation (Houtveen et al., 2002). There are two possible reasons for this: first, HF-HRV is affected by respiration, which, unfortunately, was not recorded in this study. Different breathing patterns between BPD and HC might explain group differences in HF-HRV (Grossman et al., 1991). Secondly, these chronically dysregulated patients could show upregulation of sympathetic (aHR) and parasympathetic (HR-HRV) influence/activity. A simultaneous activation of sympathetic nervous system and parasympathetic nervous system has been reported during conditioned freezing behavior in animals (Iwata and LeDoux, 1988; Nijssen et al., 1998). Some authors use the phenomenon of freezing, or immobility, in animals as a model of dissociation in humans (Scaer, 2001) including increased activity of both systems. Currently, Sack and Lamprecht (2004) reported for the first time increased vagal activity in PTSD patients during dissociation. Dissociation is a DSM-IV criteria in BPD, and the alteration of physiological response, caused by dissociation, has already been demonstrated in BPD (Ebner-Priemer et al., 2005). However, we have not assessed the present state



dissociative experience in this study, so explanations remain speculative. Furthermore, it must be emphasized that the number of non-medicated patients was low and our findings in this regard are preliminary.

The third hypothesis, that BPD patients experience more complex emotions (more than one emotion simultaneously), was also supported by our data. Patients with BPD reported more secondary emotions, and in particular more negative secondary emotions compared to HCs.

Our fourth hypothesis was that patients with BPD will feature the experience of emotions without being able to name them: different results were evident in the two cohorts. German BPD participants had significantly more non-specific emotions than the German HCs, whereas American patients reported fewer non-specific emotions than American HCs. This effect may possibly be explained by differences in participation in psychotherapy: The German group was investigated before start of DBT treatment, whereas the US group was examined during ongoing DBT treatment. However, this suggestion does warrant caution because the possible effects of cohort (nationality) and duration of treatment participation (before and in the middle, respectively) cannot be separated in our design. Furthermore, there were other significant differences between German and US cohorts (not reported here). However, we speculate that this result might be a therapeutic effect, because teaching patients to identify and name emotions is a major focus in DBT, and this should be studied further.

Limitations of this study should be noted. First, while our sample size was large compared with many psychophysiological studies, the necessity of subdividing our borderline patients on the basis of medication reduced the sample and prevented further analysis of the results in physiological variables (e.g. HF-HRV) and further interesting analyses (e.g. correlation between psychological and physiological parameters). Second, respiration is considered important in calculating heart rate variability (Berntson et al., 1997). Since respiration was not recorded in this study, analysis of only HF-HRV may have been insufficient. Third, ambulatory studies also have disadvantages, such as problems on controlling dependent variables (Fahrenberg, 1996). In our study we did not ask participants to report emotionally relevant events or daily life stressors during the monitoring. We only assessed the emotional experience. This is a problem because we do not know if BPD subjects exhibit heightened sensitivity to discrete stimuli and responded more intensively to comparable events (as Linehan's theory of emotional dysregulation postulates), whether they simply encounter more stressful life events, or whether they experience the same life events as more stressful. To clarify

this important question, more studies both in the laboratory and in the field with ambulatory monitoring technology are necessary. Fourth, the process of analysis for identifying specific emotions was complicated by the problem of differentiating cohorts and treatment effects. Information about possible treatment effects must remain speculative. Nevertheless, using patient samples from different countries should increase generalization. Fifth, we only assessed female subjects. A final critical point is whether displaying questions every 10 to 20 min is reconcilable with normal daily life. However, BPD patients as well as HCs reported low distress and reactivity, that is, minimal altered attention to emotions, minimal altered attention to body sensations, minimal extraordinary events during the day, and minimal unpleasantness of the device.

Our findings during naturalistic ambulatory assessment support emotional dysregulation in BPD as defined by the biosocial theory of Linehan (1993) and address discrepancies in previous findings. Results suggest the potential utility of ambulatory assessment for evaluating treatment outcome. Integration of the ambulatory assessment of emotional events, appraisal, behavior (skills), and triggered emotional physiological responses may enable the measurement of treatment response in BPD and other disorders with greater specificity and ecological validity.

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