Cardiac defense in response to imminent threat in women with multiple trauma and severe PTSD

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Abstract

Posttraumatic stress disorder (PTSD) arises as a long-term result of exposure to trauma and brings with it an altered autonomic response to potentially threatening stimuli. The present study investigates the dynamic sequence of cardiac defense in women with and without PTSD. An acoustic noise of 0.5-s duration and 105 dB was used to elicit the cardiac defense reaction. The stimulus was repeated three times. Within the PTSD sample, respondents who suffered from more severe PTSD showed a higher heart rate at rest, a higher baseline, and a greater response. Compared to the healthy subjects, the PTSD group showed an elevated heart rate from 6 s to 25 s following the presentation of the first stimulus. There was evidence of habituation in the PTSD group and hints of differential effects on the cardiac defense of traumatic experiences with a high proximity of danger.

Descriptors: PTSD, Cardiac defense, Heart rate, Shutdown dissociation, Sympathetic, Parasympathetic

Larger startle responses are a robust physiological correlate of posttraumatic stress disorder (PTSD; Pole, 2007). Many of the initial studies in this area examined male survivors of trauma related to military service (e.g., Blanchard, Kolb, Pallmeyer, & Gerard, 1982). Exaggerated startle has also been found in samples of female nurse veterans and survivors of sexual assault (Carson et al., 2007; Metzger et al., 1999; Rothbaum, Kozak, Foa, & Whitaker, 2001). The etiology of the elevated level of startle responses is still uncertain. Increased physiological reactivity may reflect a pretraumatic tendency to higher responses (Guthrie & Bryant, 2005) or may develop as a kind of posttraumatic neuronal sensitization in parallel with PTSD (Shalev et al., 2000). Another theory states that the anticipatory stressful challenges that the subject underwent trigger the fear network and drive the organism to stronger reactivity (Elbert, Rockstroh, Kolassa, Schauer, & Neuner, 2006). In contrast to the literature that shows increased physiological reactivity, there is also conflicting evidence of attenuated psychophysiological response in female respondents who suffer from PTSD. Griffin, Resick, and Mechanic (1997) examined a group of sexual assault survivors and found that those subjects with high peritraumatic dissociation exhibited lower heart rate and skin conductance when they were talking about their traumatic experiences. Further studies found evidence that ongoing traumatic stressors such as family violence were correlated with reduced startle reactivity in adult women (Medina, Mejia, Schell, Dawson, & Margolin, 2001). Other authors argued that these subjects show functionally appropriate behavior associated with a different psychophysiology (Gola et al., 2012; Lang, Davis, & Öhman, 2000; Lang, McTeague, & Cuthbert, 2007). Thus, although there is strong evidence for cardiac hyperreactivity in PTSD, a significant number of studies also shows an opposite hyporeactivity.

It is clear that we need a different explanatory framework for the physiology of PTSD in order to reconcile these apparently contradictory findings. Recently, Schauer and Elbert (2010) suggested that an etiological model of the defense cascade progression “freeze-flight-fight-flag-faint” points to sympathetic and parasympathetic nervous system adaptations during a life threat that enhance survival (Lang, Bradley, & Cuthbert, 1998). The stages “fright-flag-faint” present further progression on the defense cascade. Shutdown dissociation consists of transient functional sensory deafferentation, motor paralysis, alternations of consciousness and loss of speech perception and production. To shut down the bodily system, the parasympathetic system takes over dominance, resulting in bradycardia, a decrease in blood pressure and vasodilatation (Scaer, 2001; Schauer & Elbert, 2010). Theories about physiological reactivity state that the physiological responses recapitulate the peritraumatic physiological reactivity (Elbert et al., 2006; Lang et al., 1998). Patients who show active defense behavior such as flight or fight would thus show increased startle responses, whereas patients who show shutdown dissociation responses would respond with reduced reactivity. Traumatic events with a high proximity of danger thereby favor a further progression of the defense cascade. Different physiological reactivity might be influenced by the proximity of danger of past traumatic events, which is higher in cases where an individual was threatened and...
lower where an individual witnessed how another person was threatened. Investigating the dynamics of psychophysiological responding as well as the influence of the sympathetic and parasympathetic system could advance psychophysiological studies in PTSD.

Cardiac Defense and the Autonomic Nervous System

Heart rate change seems to be the most reliable parameter for examining elevated psychophysiological reactivity (Pole, 2007). Both the sympathetic and parasympathetic branch of the autonomic nervous system regulates heart rate: heart rate acceleration could be due to sympathetic activation, parasympathetic withdrawal, or both. Contemporary findings suggest that the relationship between these two branches of the autonomic nervous system could be reciprocal (while one branch increases, the other decreases its activity), independent (one branch is active without systematically changing the activity of the other branch), or coactive (both increase together) (Berntson, Cacioppo, & Quigley, 1993). The cardiac defense reaction is a specified pattern of heart rate changes that follows the presentation of a startling noise. Usually, an intensive noise of 109 dB is presented for 500 ms with an instantaneous rise time (Fernández & Vila, 1989). The pattern of cardiac defense consists of two accelerations and two decelerations in heart rate (see Figure 1). The first accelerative/decelerative component reaches its maximum about 3 s to 4 s after the presentation of the startling noise. Subsequent to the initial increase in heart rate, it then drops to baseline level or even lower. This decrease is followed by a more progressive increase in heart rate that peaks at around 35 s following the presentation (Vila et al., 2007). The cardiac defense response habituates itself to repeated presentations of the acoustic stimulus. The second accelerative/decelerative component in particular vanishes after the first presentation, whereas the first accelerative/decelerative component shows less reduction over trial repetition (Mata, Rodríguez-Ruiz, Ruiz-Padial, Turpin, & Vila, 2009; Vila et al., 2007). Sympathetic and parasympathetic influences on the cardiac defense response were systematically examined in studies that used a pharmacological blockade of one branch of the autonomic system or assessed indirect, biological measures such as pulse transit time or respiratory sinus arrhythmia. The results of these studies argue for vagal and sympathetic involvement of the cardiac defense pattern (Fernández & Vila, 1989; Vila et al., 2007). A withdrawal of the parasympathetic branch was associated with the first increase in heart rate. The decrease in the first component was accompanied with a dominance of the parasympathetic branch. Changes in dominance in the sympathetic branch play a minor role in the first component, whereas the second component coincides with an activation and deactivation of the sympathetic branch (Reyes del Paso, Godoy, & Vila, 1993; Reyes del Paso, Vila, & García, 1994).

Theoretical Approaches to Cardiac Defense

The theoretical background of the cardiac defense reaction includes cognitive and motivational approaches. Graham and Clifton (1966) proposed that the cardiac response reflects the disposition to attentive augmentation or a reduction in environmental stimuli. The deceleration of the heart rate facilitates attention and perception, whereas acceleration is associated with a reduction in attention and perception. From a motivational perspective, the cardiac response reflects a bodily preparation for fight-or-flight responses. The defense cascade model “freeze-flight-fight-fright-flag-faint” attempts to unite both theories. In life-threatening situations, attentive and motivational processes play an important role in ensuring survival of the organism (Bracha, 2004; Lang et al., 1998). The first accelerative/decelerative component reflects an attentional process. The immediate increase in heart rate interrupts the ongoing perceptual processes as well as physical movements; the subsequent decrease in cardiac response facilitates or is linked with the organism’s heightened perceptual attention to threatening cues. The second long latency heart rate acceleration and deceleration prepares the body for active defense behavior such as fight or flight. Apart from those active responses to a threat, Schauer and Elbert (2010) proposed that a shutdown of perceptual and emotional information processing as well as a shutdown of the bodily system could be adaptive in situations where the organism is physically overwhelmed by a predator, such as sexual assault (Marx, Forsyth, & Lexington, 2008). This shutdown is reflected at a physiological level by a parasympathetic dominance.

The Rationale of the Present Study

The cardiac defense paradigm and the physiological background provide a good framework for noninvasively estimating parasympathetic and sympathetic functions as well as the regulation of the dynamic physiological response in patients with PTSD. A sample of severely traumatized refugees was chosen because event types with a high proximity to danger such as sexual assaults or torture were common and would favor ongoing shutdown dissociation. The present study investigates the following questions: (1) Are
there different heart rates at resting state, baseline, as well as response level across the trial repetition in the PTSD compared to the non-PTSD group? (2) Is there a different cardiac defense pattern for the PTSD versus the non-PTSD group? (3) Are there differences in the magnitude of the first and second accelerative components across the trial repetition in the PTSD and non-PTSD groups? (4) Exploratory analysis of correlates: does PTSD symptom severity, the number of traumatizing event types that were experienced (with a high proximity of danger) or witnessed (lower proximity to danger), or the tendency to shutdown dissociation play a role in the modulation of the heart rate or the heart rate changes?

Method

Structured Clinical Interviews

Participants were recruited through the outpatient clinic for refugees at the University of Konstanz. They were referred to the clinic by a human rights organization, medical doctors, or lawyers for diagnostic clarification or potential treatment. Prior to the testing, subjects underwent a structured clinical interview. Experienced psychologists carried out the interviews with the support of trained translators. We conducted 49 interviews between April 2010 and February 2012 (inclusion criteria were female refugee with multiple traumatic experiences). Thirty-nine of the interviewed women were included in the PTSD group. Five subjects didn’t participate for the following reasons: three for technical reasons, one subject was afraid of the laboratory, and one subject gave informed consent but showed severe dissociation after the beginning so the procedure was aborted. Thirty-four subjects who presented with PTSD symptoms participated in the present study and were compared to 17 healthy control subjects with similar ethnic backgrounds who were recruited from the general community. The study was approved by the Ethics Committee of the University of Konstanz. After providing informed consent, each subject was interviewed. First, demographic data as well as medication were recorded. Next, the number of traumatic experiences was assessed with the sum of the event checklist of the Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995). We furthermore differentiated between the number of traumatic event types that were self-experienced (high proximity to danger) and the number of different traumatic event types that were witnessed (low proximity to danger). A traumatic event type was judged as self-experienced if the participant was the victim or as witnessed if the participant had observed someone else being seriously threatened. For the PTSD diagnosis, we used the CAPS and the sum score for the symptom severity. Current comorbid DSM-IV disorders (such as depression, dysthymia, abuse, or dependency on alcohol/illegal substances, suicidality, and psychotic disorder) were assessed with the Mini-International Neuropsychiatric Interviews (M.I.N.I.; Sheehan et al., 1998). The score on the Hamilton Rating Scale for Depression (HAM-D; Williams, 1988) estimated the degree of depression. To assess dissociation symptoms, we used the 13-item Shutdown Dissociation Scale (Schauer, Schalinski, & Elbert, 2010; Schalinski, Elbert, & Schauer, 2011), which we developed based upon our concept of shutdown dissociation (Schauer & Elbert, 2010). Responses to all items were given on a scale of 0 (not at all), 1 (once a week or less), 2 (2 to 4 times a week) and 3 (5 or more times a week). Sum scores could range from 0 to 39.

Subjects and Demographic Data

The demographic data are presented in Table 1. On average, the subjects in the PTSD group were significantly less educated when compared to the control group, t(45.22) = 10.67, p < .001. On average, the participants in the PTSD group had experienced 5.3 (SD = 2.3; range 2–11) and witnessed 2.9 (SD = 2.0; range 0–7) different types of traumatic stressors. The following self-experienced events were reported most frequently: physical assault (76%), assault with a weapon (62%), and sexual assault with penetration (52%). The following events were witnessed most frequently: physical assaults (64%), homicide (40%), and a serious traffic accident (33%). The non-PTSD group was significantly less exposed to traumatic stress (see Table 1). They had experienced on average 1.2 traumatic event types (SD = 1.4; range 0–4) and witnessed on average 1.2 traumatic event types (SD = 1.2; range 0–3). Four subjects in the non-PTSD group reported that they had never experienced any traumatic stressor. On average, the time elapsed since the worst traumatic event did not differ between the PTSD and non-PTSD group. Of the 34 subjects in the PTSD sample, 32 fulfilled all criteria of the diagnosis. Two fulfilled the DSM-IV criteria of PTSD (A, B and D, E, F), but met only two of three avoidance symptoms (criteria C). All respondents in the PTSD

Table 1. Demographic and Clinical Data

<table>
<thead>
<tr>
<th></th>
<th>PTSD M/n</th>
<th>SD/SD%</th>
<th>Non-PTSD M/n</th>
<th>SD/%</th>
<th>Statistics for group differences</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>36.45</td>
<td>10.8</td>
<td>31.88</td>
<td>6.83</td>
<td>t(45.22) = -1.82, p = .075</td>
</tr>
<tr>
<td>Education (years)</td>
<td>5.33</td>
<td>3.2</td>
<td>18.68</td>
<td>3.46</td>
<td>t(44.99) = 10.67, p &lt; .001</td>
</tr>
<tr>
<td>Regions of origin. n%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>χ²(3, 50) = .12, p = .990</td>
</tr>
<tr>
<td>Middle and Far East</td>
<td>17</td>
<td>51.5%</td>
<td>9</td>
<td>53%</td>
<td></td>
</tr>
<tr>
<td>The Balkans</td>
<td>5</td>
<td>15.2%</td>
<td>2</td>
<td>11.8%</td>
<td></td>
</tr>
<tr>
<td>Africa</td>
<td>9</td>
<td>27.3%</td>
<td>5</td>
<td>29.4%</td>
<td></td>
</tr>
<tr>
<td>India</td>
<td>2</td>
<td>6%</td>
<td>1</td>
<td>5.9%</td>
<td></td>
</tr>
<tr>
<td>Number of traumatic event types (self-experienced)</td>
<td>5.27</td>
<td>2.30</td>
<td>1.24</td>
<td>1.40</td>
<td>t(46.53) = -7.72, p &lt; .001</td>
</tr>
<tr>
<td>Number of traumatic event types (witnessed)</td>
<td>2.94</td>
<td>2.00</td>
<td>1.24</td>
<td>1.15</td>
<td>t(47.32) = -3.83, p = .002</td>
</tr>
<tr>
<td>Time elapsed since the worst traumatic event (years)</td>
<td>11.33</td>
<td>10.63</td>
<td>8.72</td>
<td>6.37</td>
<td>t(44.2) = -0.77, p = .449</td>
</tr>
<tr>
<td>CAPS Symptom Severity</td>
<td>79.06</td>
<td>18.61</td>
<td>0.94</td>
<td>2.66</td>
<td>t(34.47) = -23.65, p &lt; .001</td>
</tr>
<tr>
<td>Hamilton Depression Severity</td>
<td>19.84</td>
<td>7.89</td>
<td>2.24</td>
<td>2.51</td>
<td>t(41.79) = -11.41, p &lt; .001</td>
</tr>
<tr>
<td>Shutdown Dissociation Score</td>
<td>20.09</td>
<td>8.28</td>
<td>0.94</td>
<td>1.25</td>
<td>t(34.55) = -10.33, p &lt; .001</td>
</tr>
</tbody>
</table>

Note. For group differences, t tests were used for continuous variables and χ² tests were applied for nominal variables. M = Mean; SD = standard deviation; n = absolute number of respondents; PTSD = posttraumatic stress disorder; CAPS = Clinician-Administered PTSD Scale.
sample met the DSM-IV criteria for depression. None of the respondents in the non-PTSD group met the criteria for depressive disorder. None of the study participants fulfilled the criteria for current or past psychotic disorder or alcohol or substance abuse/dependency. Part of the PTSD sample ($n = 8$, 24%) was being treated with psychoactive medication. These medications were antidepressants ($n = 7$, 21%), neuroleptics ($n = 1$, 3%), and anxiolytics ($n = 1$, 3%). The subjects in the non-PTSD group reported no experience in either psychotherapy or psychiatric treatment. The medication was not considered as a covariate in the present study due to different medication and doses. None of the subjects took medication targeting the cardiovascular system or directly altering heart rate response, such as digitalis, beta blockers, or anticholinergics.

**Procedure**

For testing, subjects were placed in a separate room in a supine position. The following sequence was followed: (a) at least 10 min of resting/adaptation; (b) the instructions were given while the subjects stayed in the supine position; subjects were requested to remain still and breathe as normally and evenly as possible; (c) each subject underwent the first presentation of a binaural auditory stimulus following a baseline of 20 s; (d) at least 80 s poststimulus, the heart rate was recorded. Step (c) and (d) were repeated, and the subjects underwent three presentations of the stimulus. The intertrial interval was set to 10 s or 15 s on top of the baseline of 15 s. The testing for all subjects was carried out between 10:00 a.m. and noon.

**Stimulus Properties**

The auditory stimuli were binaurally presented using an air tube system attached to a sound amplifier (ASG, Version 0.90.1, Biomagnetic Technologies). The air tube system delivered the tone to the subject’s ear via flexible plastic tubes that were 7.5 m long and had a diameter of 20 mm. With the help of an artificial ear, the sound level was set to 105 dB. The white noise fed into the system contained frequencies between 20 Hz and 20 kHz, the power spectrum at the ear still contained this whole frequency range, but displayed a peak at around 1000 Hz. The stimulus was presented for 0.5 s with an instantaneous rise time.

**Apparatus and Physiological Measure**

Two Ag/AgCl electrodes were positioned above the right collarbone and the left lower costal arch. An electrode attached above the right zygomatic bone provided grounding. The electrocardiogram (ECG) was amplified with a SynAmps (Neuroscan Laboratories, Sterling, VA) and digitized online at a rate of 678.17 Hz. The R wave was semiautomatically detected using BESA TM software, and R-R intervals were converted to the beat-by-beat heart rate. Prior to testing, resting heart rate was assessed over an interval of 6 min. The parameters (heart rate at resting level, baseline, and response level) used the beat-by-beat heart rate. With respect to the individual beat-by-beat heart rate, the median heart rate change was assessed based on twelve intervals within the 80 s following stimulus onset. The first two intervals lasted 3 s ($0 < 3$, (3 < 6), the following interval 5 s ($6 < 11$), and the remaining intervals 7 s ($11 < 18$, (18 < 25), (25 < 32), (32 < 39), (39 < 46), (46 < 53), (53 < 60), (60 < 67), (67 < 74) (compare Fernández & Vila, 1989).

**Habituation Measures of the First and Second Accelerative Components**

Maximal heart rate changes were evaluated using an in-house MATLAB algorithm. The algorithm interpolated the heart rate change over 80 s following the acoustic stimulus in half-second steps using a 15-s prestimulus baseline. The maximum heart rate acceleration within seconds 1 and 6 was determined using the interpolated heart rate data of each subject. To assess the maximum acceleration response of the second accelerative component, heart rate change was calculated as the maximum of the mean of five consecutive data points in the interval between 25 s and 45 s. Additionally, the latency of the first and second accelerative component was assessed. The measure of habituation was based on statistical reductions in the maximum acceleration heart rate response across the three trials.

**Statistical Analysis**

Analyses were performed using SPSS 20.0; alpha level was set at .05. To compare the demographic data between the PTSD and non-PTSD group, $t$ tests were used for continuous variables and $\chi^2$ tests were applied for nominal variables. We assessed the effects of trial repetition on heart rate at baseline and response level as well as on heart rate changes and the latency of the first and second accelerative components using a repeated measures analysis of variance (ANOVA). In case of sphericity violations, the Greenhouse-Geisser estimates were used to correct the degrees of freedom (Greenhouse & Geisser, 1959). Post hoc analysis consisted of $t$ tests with Bonferroni correction; $t$ tests were applied to analyze group differences at the intervals of each trial. The data of one respondent had to be removed from further analysis due to extra sysstoles followed by compensatory pauses. Two patients started to hyperventilate during the first trial and showed heart rate increases of over 50 beats per minutes (bpm). These trials were excluded from further analysis. Due to acquisition problems, the third trial of one PTSD subject had to be excluded. Another two patients rejected further participation after the first trial. Altogether, we collected 30 from each trial (trial 1: 31, trial 2: 31, trial 3: 30). Scores with missing data were partially excluded from further analysis ($n = 3$).

**Results**

**Group Comparison and Correlates of Heart Rate at Resting State**

The resting heart rate was assessed prior to testing. The average heart rate was $M = 67.67$ bpm ($SD = 7.08$) in the PTSD group and $M = 66.86$ bpm ($SD = 7.74$) in the non-PTSD group. The difference in resting heart rate was not significant, $t(48) = -0.37$, $p = .712$. Within the PTSD group, the correlation between the resting-state heart rate and the clinical data was calculated. Those participants with a more severe PTSD symptom score exhibited an increased heart rate at a resting state of 6 min ($r = .39$, $p = .023$). In contrast, the shutdown dissociation score was not associated with heart rate at a resting state ($r = .11$, $p = .528$). Furthermore, the number of different self-experienced traumatic stressors and those that were witnessed was not related to the heart rate at rest ($r = .09$, $p = .628$; $r = .15$, $p = .405$).
Table 2. Heart Rate at Baseline and Response Level

<table>
<thead>
<tr>
<th></th>
<th>PTSD</th>
<th></th>
<th>Non-PTSD</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Trial 1</td>
<td>Baseline</td>
<td>65.84</td>
<td>6.60</td>
<td>67.32</td>
</tr>
<tr>
<td></td>
<td>Response</td>
<td>68.76</td>
<td>8.56</td>
<td>67.71</td>
</tr>
<tr>
<td>Trial 2</td>
<td>Baseline</td>
<td>66.20</td>
<td>6.36</td>
<td>65.49</td>
</tr>
<tr>
<td></td>
<td>Response</td>
<td>67.66</td>
<td>6.88</td>
<td>66.04</td>
</tr>
<tr>
<td>Trial 3</td>
<td>Baseline</td>
<td>67.59</td>
<td>6.82</td>
<td>65.57</td>
</tr>
<tr>
<td></td>
<td>Response</td>
<td>67.79</td>
<td>6.24</td>
<td>66.68</td>
</tr>
</tbody>
</table>

Note. Group means (M) and standard deviations (SD) for the heart rate at baseline level (15 s prior to the acoustic trigger) and response level (80 s after the presentation of the acoustic trigger).

Group Comparison of Heart Rate at Baseline and Response Level

Table 2 presents the mean and standard deviation of the heart rate at baseline (15 s prior to the stimulus) and response level (80 s after stimulus presentation). Trial repetition effects and group differences for the baseline (15 s prior to the stimulus) and heart rate at response level (80 s after the stimulus) were analyzed with repeated measures ANOVA. Mauchly’s test indicated that the assumption of sphericity had been violated for the main effect of trial repetition for the baseline heart rate, $\chi^2(2) = 9.24, p = .010$. Degrees of freedom were therefore corrected using Greenhouse-Geisser estimates of sphericity, $\varepsilon = .84$. Neither the trial repetition, $F(1.69, 77.60) = 1.57, p = .216, \eta^2 = .03$, nor the group effect, $F(1.46) = 0.01, p = .942, \eta^2 < .01$, was significant. The interaction effect of group and trial did not reach significance, $F(1.69, 77.60) = 2.66, p = .085, \eta^2 < .01$. Statistical analysis of the heart rate at response level (80 s after the presentation of the acoustic stimulus) yielded a significant violation of the sphericity assumption for the trial repetition effect, $\chi^2(2) = 48.63, p < .001$. The degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity, $\varepsilon = .60$. The analysis showed no significant change as a function of trial repetition, $F(1.19, 52.47) = 3.15, p = .075, \eta^2 = .07$. The analysis indicated no significant interaction, $F(1.19, 52.47) = 0.37, p = .586, \eta^2 < .01$, and also no group effect, $F(1.44) = 0.56, p = .457, \eta^2 = .01$.

Correlates of Heart Rate at Baseline and Response Level

Correlates were assessed within the PTSD group. The correlations of the heart rate at baseline and response level with the PTSD symptom severity are presented in Figure 2. Those respondents with a higher PTSD symptom severity also had a higher baseline heart rate ($r_{\text{Trial 1}} = .41, p = .018; r_{\text{Trial 2}} = .36, p = .046; r_{\text{Trial 3}} = .44, p = .014$). The PTSD symptom severity accounted for 17% of the baseline heart rate variance of the first trial, 13% of the second trial, and 19% of the third trial. Additionally, PTSD symptom severity was correlated with heart rate at response level ($r_{\text{Trial 1}} = .45, p = .010; r_{\text{Trial 2}} = .48, p = .006; r_{\text{Trial 3}} = .43, p = .018$). However, the degree of shutdown dissociation was neither associated with heart rate.

Figure 2. The correlation between the averaged beat-by-beat heart rate in beats per minute (bpm) at baseline (15 s prior to stimulus presentation) and response level (80 s poststimulus presentation) as well as PTSD symptom severity as a function of trial and group (filled circles = PTSD group; open circles = non-PTSD group member). CAPS = Clinician Administered PTSD Scale.
rate at baseline level ($r_{\text{Trial1}} = .18$, $p = .315$; $r_{\text{Trial2}} = .21$, $p = .270$; $r_{\text{Trial3}} = .23$, $p = .207$) nor at response level ($r_{\text{Trial1}} = .13$, $p = .476$; $r_{\text{Trial2}} = .27$, $p = .141$; $r_{\text{Trial3}} = .32$, $p = .089$). Due to intercorrelation, we explored partial relationships of the heart rate average at response level with PTSD severity and the number of traumatizing event types. The correlation between heart rate at response level and PTSD symptom severity was also significant upon partialling out the number of traumatic event types ($p_r = .49$, $p = .009$). Independent of this relationship, the positive relationship between the number of event types and the PTSD severity remained when controlling for heart rate ($p_r = .40$, $p = .037$).

Cardiac Defense Response and Group Differences

The characteristic pattern of cardiac defense response was found in the non-PTSD group. After 3 to 4 s, the first accelerative component reached the maximum, and a second accelerative component was observed at around 35 s. Figure 3 shows the cardiac defense response as a function of trial and group. The PTSD group showed a prolonged higher heart rate change compared to the non-PTSD group. Differences were significant in the first trial in the interval of 6 < 11 s, 11 < 18 s, and 18 < 25 s ($t_{\text{df1}(30,18)} = -2.77$, $p = .009$; $t_{\text{df1}(35,55)} = -2.41$, $p = .020$; $t_{\text{df1}(37,26)} = -2.02$, $p = .050$). There were no group differences in the second trial, but a tendency for a difference was observed in the interval from 3 to 6 s ($t_{\text{df1}(43,06)} = -1.97$, $p = .056$). In the third trial, there was no difference between the two groups for the cardiac defense pattern. Marginal differences could be found in the interval 3 < 6 s, 6 < 11 s ($t_{\text{df1}(36,07)} = -1.98$, $p = .055$; $t_{\text{df1}(37,50)} = -1.95$, $p = .058$).

Habitation and Latency of the First and Second Accelerative Components

Table 3 presents the mean and standard deviations of the maximum heart rate changes and the latency during the first (1–6 s following the presentation of the acoustic stimulus) and second acceleration (25–45 s following the presentation of the acoustic stimulus) components. Group and repetition effects were separately assessed with repeated measures ANOVA for both components. Descriptive data indicated a habituation of the cardiac defense response across the trials for both groups. The results showed that trial repetition significantly affected the magnitude of the first component, $F(2,90) = 12.93$, $p < .001$, $\eta^2 = .22$. However, there was no significant difference between the PTSD and the non-PTSD group, $F(1,45) = 0.04$, $p = .950$, $\eta^2 < .01$, and no effect of interaction (Trial × Group), $F(2,90) = .75$, $p = .477$, $\eta^2 = .02$. A post hoc paired $t$ test indicated a significant decrease in magnitude from the first to the second and from the first to the third trial repetitions (all $p < .017$) in both groups. However, no decrease could be observed between the second and third trials in both groups. The magnitude of the second accelerative component seemed to decrease over the trial repetition. First, Mauchly’s test indicated that the assumption of sphericity had been violated for the main effect of trial repetition, $\chi^2(2) = 18.67$, $p < .001$. Degrees of freedom were therefore corrected using Greenhouse-Geisser estimates of sphericity, $\varepsilon = .74$. The main effect of trial repetition indicated a significant reduction in the second accelerative component, $F(1,48.65,08) = 3.71$, $p = .042$, $\eta^2 = .08$. There was no significant difference between the PTSD and the non-PTSD group, $F(1,44) = 1.27$. 

Figure 3. Cardiac defense as a function of trials averaged across the groups. Each point corresponds to the median of the respective interval and represents the group mean heart rate change with respect to baseline (15 s prior to stimulus onset). *$p < .05$; • $1 > p > .05$. 

rate at baseline level ($r_{\text{Trial1}} = .18$, $p = .315$; $r_{\text{Trial2}} = .21$, $p = .270$; $r_{\text{Trial3}} = .23$, $p = .207$) nor at response level ($r_{\text{Trial1}} = .13$, $p = .476$; $r_{\text{Trial2}} = .27$, $p = .141$; $r_{\text{Trial3}} = .32$, $p = .089$). Due to intercorrelation, we explored partial relationships of the heart rate average at response level with PTSD severity and the number of traumatizing event types. The correlation between heart rate at response level and PTSD symptom severity was also significant upon partialling out the number of traumatic event types ($p_r = .49$, $p = .009$). Independent of this relationship, the positive relationship between the number of event types and the PTSD severity remained when controlling for heart rate ($p_r = .40$, $p = .037$).
The second accelerative component and PTSD symptom severity

\( r = .471 \) for the PTSD group (Pole, 2007). It may, therefore, be possible that the autonomic imbalance tended to appear in those patients with a strong PTSD symptom severity. On the one hand, the elevated heart rates at resting state could be attributed to the anticipatory stressful stimuli that were presented after the sequence of resting. It is believed that PTSD patients with a stronger symptom severity also have a well-established fear network with intensive association between its representations. On the other hand, elevated long-term changes in basal heart rate could play a role in this finding (Lovallo & Gerin, 2003). A stronger symptom severity means more chronic stress in daily life. Repeated cardiovascular reactivity to stress may accompany altered functioning of the autonomic system (Fredrikson & Matthews, 1990). The within-variation could explain the inconsistent findings of elevated (Carson et al., 2007) and nonelevated heart rate reactivity in PTSD (Wolfe et al., 2000), suggesting that the likelihood of finding differences between PTSD patients would increase when patients with a higher symptom severity are tested. This further assumes that the probability of finding differences should be greater in samples of persons seeking treatment when compared to samples drawn from the general community. The analysis of partial correlations gives further evidence that, besides the positive relationship between the number of different event types and PTSD severity (e.g., Neuner et al., 2004), heart rate also seems to play a crucial role in elevated PTSD symptom severity. Of course, this

### Table 3. Peak Amplitude and Latency of the Accelerative Components

<table>
<thead>
<tr>
<th></th>
<th>PTSD amplitude (bpm)</th>
<th>Latency (s)</th>
<th>Non-PTSD amplitude (bpm)</th>
<th>Latency (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
</tr>
<tr>
<td>Trial 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First</td>
<td>13.42 (9.08)</td>
<td>3.17 (1.55)</td>
<td>14.37 (10.36)</td>
<td>3.06 (1.38)</td>
</tr>
<tr>
<td>Second</td>
<td>7.55 (8.05)</td>
<td>3.21 (6.54)</td>
<td>5.33 (7.83)</td>
<td>3.38 (7.66)</td>
</tr>
<tr>
<td>Trial 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First</td>
<td>10.65 (7.20)</td>
<td>3.07 (1.45)</td>
<td>9.23 (7.36)</td>
<td>2.82 (1.20)</td>
</tr>
<tr>
<td>Second</td>
<td>4.32 (3.58)</td>
<td>3.52 (6.44)</td>
<td>2.87 (6.06)</td>
<td>3.09 (7.70)</td>
</tr>
<tr>
<td>Trial 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First</td>
<td>9.42 (6.08)</td>
<td>3.18 (1.46)</td>
<td>9.32 (7.16)</td>
<td>3.32 (1.44)</td>
</tr>
<tr>
<td>Second</td>
<td>3.30 (6.14)</td>
<td>3.50 (7.08)</td>
<td>2.85 (8.54)</td>
<td>3.26 (6.74)</td>
</tr>
</tbody>
</table>

Note. The peak amplitude is measured in changes in beats per minute (bpm) with respect to baseline. The latency is presented in seconds (s) after the stimulus onset. M = mean; SD = standard deviation.

The present study examined cardiac defense reaction in a sample of severely traumatized female PTSD patients in comparison to a non-PTSD group. Neither age nor ethnic background differed between the groups. However, PTSD patients were less educated compared to the control group. As expected, the PTSD group had significantly higher scores of exposure to traumatic stressors and clinical symptoms than the control group (PTSD symptom severity, depression severity, and shutdown dissociation score).
relationship is correlative. Given the lack of a group difference, reverse causality seems more plausible: individuals with a greater cardiovascular responsiveness might develop more intense symptoms when exposed to traumatic stressors.

Cardiac Defense Pattern in Women With and Without PTSD

To assess the physiological dynamics of heart rate, the pattern of cardiac defense was analyzed. The analyses yielded significant changes in the pattern between the PTSD and the non-PTSD group. After the initial increase in heart rate, the PTSD group showed an elevated heart rate change from 6 s to 25 s poststimulus presentation in the first trial. Similar findings have been reported in animal and blood phobia patients when they were simultaneously confronted with their phobic object and the acoustic noise (Vila et al., 2007). The withdrawal of the parasympathetic branch of the autonomic system contributes to the pattern of the first acceleration (Vila et al., 2007), whereas the decrease in the first component is associated with parasympathetic dominance of the autonomie nervous system. From this perspective, there is evidence of ongoing parasympathetic withdrawal and parasympathetic dysfunction congruent with the prolonged higher heart rate levels. This is in line with other findings that hyperarousal in PTSD is associated with a low vagal tone and not just with higher activity of the sympathetic branch (Hopper, Spinazzola, Simpson, & van der Kolk, 2006; Sack, Hopper, & Lamprecht, 2004). According to the defense cascade model, the first increase in heart rate is associated with an interruption of information processing. The following decrease in heart rate, as evoked in the non-PTSD group, is associated with the organism turning all sensory channels to the source of threat and evaluating the stimulus. The immediate and prolonged increase in heart rate in PTSD could be associated with poor information processing and a more rapid preparation to fight or flight. This motivational preparedness could be adaptive in an environment with a high probability of traumatic stressors but maladaptive in a safe environment. Adenauer, Catani, Keil, Aichinger, and Neuner (2009) also found evidence of almost immediate increases in heart rate when PTSD patients were exposed to threatening pictures. The second accelerative/decelerative component was found in both groups in the first trial. Across trial repetition, the magnitude of the first accelerative/decelerative component showed a significant reduction in PTSD and non-PTSD subjects. In the first trial, an intensive, unknown noise was likely to be from a nearby threat and elicits a defense response in everyone. If the threat of the stimulus was not confirmed after the first presentation, subsequent preparation of active response was attenuated (Mata et al., 2009).

In past studies using startling tones, PTSD subjects failed to show habituation or to provide evidence for slower reduction (Rothbaum et al., 1997). The reduction reflects how fast an individual changes the response to repeated exposure to adverse stimuli (Orr, Solomon, Peri, Pitman, & Shapiro, 1997). Thus, the results speak for a functional autonomous adaptation across trial repetition. Again, it is important to consider the within-group variability among PTSD patients. The magnitude of the first accelerative component was correlated with PTSD symptom severity. Across subjects, greater symptom severity correlated with larger heart rate accelerations to the startling noise. The result is consistent with previous findings that patients who suffer more from PTSD are more likely to be physiologically distinguished from their counterparts without PTSD (Pole, 2007).

As mentioned above, it may well be possible that the risk of severe PTSD is greater in those with a high autonomous reactivity. In contrast, an attenuated peak of the first component was found in those who were exposed to more traumatic stressors with high proximity to danger. Furthermore, there is a relationship between the number of traumatizing events to which an individual has been exposed and PTSD symptom severity (Neuner et al., 2004). Despite the positive association, a differentiated effect on the physiological response was found. A less pronounced second accelerative component was negatively correlated with the number of self-experienced traumatic stressors. In contrast, witnessing traumatic stressors (e.g., a serious car accident) showed no significant relationship to the sympathetically mediated component of cardiac defense. Fear responses based on exposure to a self-experienced trauma are more likely to trigger passive defense responses due to a high proximity to danger (Marx et al., 2008). In terms of the defense cascade model, the diminished response could be associated with a suppression of active defense behavior. The reduction of the second component provides evidence for a PTSD subtype that is associated with exposure to trauma such as rape or torture, in which the victim is helplessly overwhelmed by an attacker and must adopt a shutdown defensive response. We failed to find systematic variation of the shutdown dissociation score for the heart rate. This is in contrast to previous studies that found that the degree of dissociation plays a critical role in physiological reactivity (Griffin et al., 1997; Lanius et al., 2002; Medina et al., 2001). The fact that our subjects were tested in a supine position could have prevented dissociative responses and parasympathetic lapse. In addition to this, it is possible that the time resolution was too short (80 s after the tone) to produce evidence of shutdown responses.

Limitations and Conclusions

The limitations of this study include an educational difference between the non-PTSD and PTSD groups that could have lead to a different understanding of the procedure (i.e., predicting the occurrence of the startle). Furthermore, part of the PTSD sample was taking psychoactive medication. However, in a meta-analysis, Pole (2007) found no significant influence on the effect size of the response to startling noise when controlling for medication. Finally, it should be noted that exposure to traumatic experiences not only leads to the core PTSD symptoms, but also to depressive symptoms; groups therefore also differed in depression levels. To conclude, our results suggest that multiple exposures to life threat followed by PTSD can, on the one hand, lead to long-lasting psychophysiological changes to the cardiac defense response and that, on the other hand, subjects with greater autonomous responsivity may respond with more intense symptoms. The results contribute further evidence that PTSD is associated with larger physiological response across different samples and different degrees of exposure to trauma types. Moreover, it is important to consider the large variation within the PTSD sample. Those who suffered from more severe PTSD seemed to show an elevated heart rate at resting state prior to the startle response as well as at response level. Interestingly, an attenuated second accelerative/decelerative component was found in those patients who experienced more serious personal life threats. This result speaks for differential effects on cardiac defense according to the type of trauma suffered. Of course, this relationship is correlative and should not imply that the PTSD severity determines heart rate.
Comparing the heart rate as well as the heart rate reactivity between the PTSD and non-PTSD group has revealed no significant difference. Berntson and Cacioppo (2007) have reported that there is an individual uniqueness in response to psychological stressors and that the response pattern towards stressors is stable over time (individual response consistency). Within the PTSD group, we have demonstrated that the correlation between the heart rate and the PTSD symptom severity remained significant even when considering the amount of exposure to traumatic stress. If the response pattern is indeed stable and unique, the variation of the healthy control group could reflect a premorbid risk for anxiety symptoms when exposed to traumatic stressors.

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