

Parasympathetic Reactivity to Recalled Traumatic and Pleasant Events in Trauma-Exposed Individuals

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Parasympathetic and heart rate (HR) reactivity to memories of traumatic and pleasant events were investigated in trauma-exposed individuals (n = 40). Vagal tone and HR were recorded before, during, and following recall of traumatic and pleasant events. Posttraumatic stress (PTS) was related to blunted parasympathetic reactivity among trauma-exposed individuals. Specifically, PTS severity was related to lower parasympathetic activation and lower recovery following trauma recall, and to lower parasympathetic activation (but not recovery) in response to a pleasant event recall. No association was observed between PTS severity and initial parasympathetic tone. However, PTS severity was associated with initial HR. Assessment of parasympathetic reactivity may enhance our understanding of the normative and pathological reactions to trauma and stress.

High sympathetic arousal to trauma recall has been widely documented in posttraumatic stress disorder (PTSD; see Pole, 2007, for meta-analysis). Moreover, sympathetic arousal is consistently found to be related to symptom severity (e.g., Veazey, Blanchard, Hickling, & Buckley, 2004). Whereas sympathetic reactivity in PTSD has been a subject of significant scrutiny, parasympathetic reactivity in traumatic stress is a relatively new area of investigation (e.g., Cohen et al., 1997, 1998, 2000; Sack, Hopper, & Lamprecht, 2004; Sahar, Shalev, & Porges, 2001), and is the primary focus of the present study.

Our main aim was to examine the relations between severity of posttraumatic stress (PTS) and parasympathetic reactivity to trauma recall among trauma-exposed (nondiagnosed) individuals. Parasympathetic reactivity to unpleasant environmental events is considered adaptive, and conversely, the lack of such reactivity is considered maladaptive. Cohen and colleagues (1997, 1998, 2000) found that individuals with PTSD exhibited virtually no parasympathetic reactivity (but also no sympathetic or heart rate reactivity) to 15 minutes of trauma recall. In contrast, they found parasympathetic (as well as sympathetic and heart rate) reactivity among normal controls and panic-disordered patients in response to recall of stressful events and panic attacks, respectively. Moreover, depressed individuals who had decreased vagal tone in response to a sad film had greater likelihood of recovery as compared to those who did not demonstrate such vagal reactivity (Rottenberg, Salmon, Gross, & Gotlib, 2005). Based on these findings, we hypothesized that higher severity of PTS in trauma-exposed individuals will be related to lower parasympathetic reactivity to trauma recall, even in the absence of a full PTSD diagnosis.

Patients with PTSD were found to differ from controls not only in their reactivity to trauma reminders, but also in their responses to pleasant stimuli. For instance, Bosnian refugees suffering from PTSD, rated pleasant pictures as nonarousing (i.e., as similar to neutral stimuli), while non-PTSD Bosnian refugees (as well as controls) rated such stimuli as arousing (Spanish-Mihajlovic et al., 2005). Indeed, it was argued that vagal reactivity deficiency in PTSD patients may not be restricted to trauma recall (Sahar et al., 2001), but may extend to pleasant event recall as well. Examining this proposition was the second aim of our study. Specifically, we hypothesized that PTS severity will be related to low vagal reactivity to a pleasant event recall. Thus, we postulated that PTS severity would be associated with blunted parasympathetic reactivity to reminders of both traumatic and pleasant events (blunted parasympathetic reactivity hypothesis).

Additionally, we sought to examine the relations between PTS severity and heart rate (HR) in response to trauma recall. Based on research cited above (Pole, 2007), we hypothesized that PTS severity will be related to greater HR increase in response to trauma recall (enhanced HR activation to trauma hypothesis).

METHOD

Participants

Individuals who experienced a traumatic event were recruited through advertisements placed in a university campus in Israel. Forty Hebrew-speaking individuals (26 women) took part in the study in return for course credit or monetary compensation (an

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equivalent of \$12). All participants were exposed to a traumatic event that met Criterion A of the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)*; American Psychiatric Association, 1994) PTSD definition (e.g., assaults, accidents, terror attacks, sexual abuse, and unexpected or violent death of a relative). Mean age was 25 ($SD = 4$; range = 19–42); mean years of education was 14 ($SD = 2$; range = 12–19). The study was approved by the local ethics committee.

Measures

Three symptom measures were included. A Hebrew translation of the 17-item Posttraumatic Diagnostic Scale (PDS; Foa, Cashman, Jaycox, & Perry, 1997), widely used to assess the severity of posttraumatic distress, was used (Helpman, Aderka, Daie-Gabai, Schindel, & Gilboa-Schechtman, 2008). A validated Hebrew translation of the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) was used to assess severity of depression (Stein, Apter, Ratzoni, Har-Even, & Avidan, 1998). The Hebrew translation of the 20-item State Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970) was used (Stein et al., 1998).

For cardiac output, electrocardiogram (ECG) was monitored via three Ag/AgCL electrodes. The ECG signal was amplified and input to the Biolog (UFI, Morro Bay, CA), a portable device that detects the peak of the R-wave to the nearest millisecond and time-sequential heart period. MXedit software (Delta-Biometrics, Inc., 1988) was used to visually display the heart period data, to edit outliers, and to quantify heart period and the cardiac vagal tone index using Porges method (Porges, 1985). The vagal tone index was determined using a sampling interval of 500 ms for heart period data and a bandpass filter to extract the variability within the frequency of respiration (0.12–

0.40 Hz). Heart period and respiratory sinus arrhythmia were calculated and averaged across sequential 30-s epochs for each 5-min session. Heart period was converted into a more intuitive HR metric.

Procedure

Upon arrival at the laboratory, participants were informed that the experiment concerns physiological reactions to reminders of traumatic and pleasant events. Informed consent was obtained following this introduction. Next, participants provided idiosyncratic reminders of two autobiographical events: a traumatic event (five words) and a pleasant event (five words). Participants were requested to relive the events they described as fully as possible. To facilitate engagement with the memories, the event-related words were displayed on a computer screen in front of the participants.

The procedure began with 5 minutes of rest during which baseline levels of vagal tone and HR were collected. Each event was recalled for 5 minutes followed by 5 minutes of rest. The order of events recall was counterbalanced across participants (i.e., pleasant followed by traumatic or vice versa). Physiological measures were recorded continuously through the experimental procedure. Questionnaires were filled following the completion of the experimental sessions. At the end of the session, the participants were debriefed and thanked for their participation.

RESULTS

Mean PDS for the sample was 10.45 (range = 0–27, $SD = 6.94$). Mean BDI was 8.77 (range = 0–28, $SD = 7.15$), and mean STAI trait was 39.9 (range = 26–58, $SD = 8.85$). Mean time elapsed since trauma was 5.37 years (range = 0.25–16, $SD = 3.96$). No

Table 1. Correlations Between Study Variables

	1	2	3	4	5	6	7	8	9	10	11
1 PDS	–	.57****	.39**	–.07	–.36**	–.41***	–.32**	.22	.29*	.28*	–.02
2 BDI		–	.52****	.10	–.00	.11	–.11	–.01	–.05	.22	–.10
3 STAI-Trait			–	–.02	.04	.04	–.05	–.00	.10	.05	–.03
4 VT Baseline				–	.22	–.01	.37**	–.35**	–.17	–.06	–.05
5 VT Activation Trauma					–	–.83****	.44***	–.45***	–.06	.10**	–.36**
6 VT Recovery Trauma						–	–.31**	.25	–.03	–.04	–.37**
7 VT Activation Pleasant							–	.61****	.00	–.39**	.08
8 VT Recovery Pleasant								–	.00	.26	.01
9 Peak HR Baseline									–	–.14	–.01
10 Peak HR Activation Trauma										–	–.61****
11 Peak HR Recovery Trauma											–

Note. PDS = Posttraumatic Diagnostic Scale; BDI = Beck Depression Inventory; STAI = State Trait Anxiety Inventory; VT = Vagal Tone; HR = Heart Rate.
* $p < .10$. ** $p < .05$. *** $p < .01$. **** $p < .001$.

correlation was found between PDS and time elapsed since trauma $r(40) = .17, ns$. The PDS was highly correlated with BDI and STAI trait (Table 1).

Reactivity to events was measured using two parameters—activation and recovery. Activation was defined as the difference between initial baseline activity and activity during recall. Recovery was defined as the difference between activity during recall and activity during the baseline following recall. To examine our hypotheses, we correlated PDS with baseline, activation, and recovery indices of vagal tone and HR. These correlations are presented in Table 1.

Consistent with our blunted reactivity hypothesis, higher PDS was related with lower vagal tone activation for both trauma and pleasant recall. Moreover, higher PDS was also related to lower recovery following trauma. However, this relationship was not significant for recovery following pleasant event. Importantly, no correlation between PDS and initial baseline level of vagal tone (above $-.07$) was observed.

In the direction of our enhanced HR activation hypothesis, higher PDS was marginally related to a greater increase in peak HR from initial baseline to trauma recall. However, PDS was not related to increase in average HR, $r(40) = -.16, ns$, possibly because average HR masked short-term HR responses. Unexpectedly, high PDS was also related to greater initial baseline levels of average HR, $r(40) = .33, p < .05$. The relationship to peak baseline was indeterminate.

DISCUSSION

Posttraumatic stress severity was related to less parasympathetic activation and recovery following trauma recall, supporting our blunted parasympathetic reactivity hypothesis. Importantly, the relations between PTS severity and parasympathetic reactivity cannot be attributed to PTS association with baseline parasympathetic tone. Posttraumatic stress severity was also related to low parasympathetic activation, but not recovery, following pleasant event recall. This finding may be taken to support the generality of parasympathetic reactivity deficiency associated with PTS severity.

Consistent with our enhanced heart rate activation hypothesis, an association was found between PTS severity and HR increase in response to trauma recall. Unexpectedly, PTS severity was also related to greater HR at baseline. Prins, Kaloupek, and Keane (1995) suggested that baseline differences in arousal between PTSD patients and controls may be confounded by anticipatory anxiety. This suggestion is especially relevant to the present study, as baseline HR was recorded after participants generated a list of trauma-related words (but before they engaged with the memory of the event). It is possible that generating trauma-related words elevated HR among highly distressed individuals.

In closing, we would like to note several limitations of the present study. First, our participants were not diagnosed for PTSD

or other mental disorders, and no data was collected regarding medications or substances use. This information is highly relevant as certain disorders as well as certain medications and substances can affect HR and vagal tone. Second, we have used a novel method of recall, in which the reliving of the traumatic and pleasant events was augmented by the presentation of idiosyncratic reminders (e.g., Gilboa-Schechtman, Revella, & Godlib, 2000). More data may be collected to validate the efficacy of this method as compared to other schema-driven procedures. Third, participants were requested to generate a list of trauma-related words immediately prior to their engagement with the memories. This might have troubled individuals with high PTS and impacted the findings.

In summary, the present findings indicate that PTS severity is related to blunted parasympathetic reactivity. Assessment of parasympathetic reactivity may enhance our understanding of the normative and pathological reactions to trauma and stress.

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