Heart Period and Variability Findings in Preschool Children with Posttraumatic Stress Symptoms

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Background: Traumatic experiences for young children might result in profound neurodevelopmental changes, compared with adults. Our aim was to examine autonomic control of heart rate in traumatized young children.

Methods: Sixty-two children who had suffered traumas and 62 nontraumatized control children, aged 20 months to 6 years, were assessed for posttraumatic stress disorder (PTSD) symptoms, interbeat interval, respiratory sinus arrhythmia (RSA), family rehearsal of the trauma, and parent-child relationship quality.

Results: Traumatized children with PTSD and traumatized children without PTSD both had decreased heart period in response to a trauma stimulus relative to the nontraumatized group (both p < .0167). There was no main effect for RSA change scores; however, there was a significant interaction effect between parental positive discipline with PTSD symptoms and RSA. The most symptomatic children had decreased RSA during the trauma stimulus when they had caregivers with less positive discipline during a clean-up task (p < .0125). There were marginally significant interactions for interbeat interval change between positive discipline and family rehearsal with PTSD symptoms.

Conclusions: These findings underscore that psychopathology in young children ought to be assessed in the context of psychophysiology and parent-child relationships to optimally understand the mechanisms of maladaptation during this complex developmental period.

Key Words: Preschool children, posttraumatic, heart period variability

Research has shown that neuroanatomic and psychophysiological variables are significantly altered in subjects with posttraumatic stress disorder (PTSD) (Vermotten and Bremner 2002). On the basis of this research, it has been speculated that the experience of life-threatening trauma has the capacity to alter normative development and physiologic function in the neural, neurochemical, and hormonal systems related to memory and the processing of fear. Furthermore, because the first 3 or 4 years of life is the period within which most of the progressive processes of postnatal brain growth occur (proliferation of glia and myelination of nerve fibers) (Nowakowski and Hayes 1999), and most of the regressive processes of brain development occur (neuronal cell death and synapse pruning), and consolidation of use-dependent neural networks overall occurs (Kandel et al 2000), it has been theorized that chronic fear responses, typical of PTSD re-experiencing symptoms, can disrupt, perhaps permanently, the development of the central nervous system (Heim and Nemeroff 2002; Kaufman and Charney 2001; Sapolsky 2000). This disruption could plausibly lead to abnormal development of regulatory processes, maladaptive fear response traits (Perry et al 1995), and/or enduring personality and relational characteristics (Schore 2002). Despite these intriguing speculations, research on brain development in young children is quite limited. This study represents the first examination of psychophysiologic associations with concurrent assessments of PTSD symptoms in children less than 7 years of age.

The sympathetic and parasympathetic branches of the autonomic nervous system are components of the complex of stress responses. These two branches generally produce coordinated and opposite physiologic effects from one another, with sympathetic activity increased and parasympathetic activity decreased during stress (Patton 1989) and in PTSD (Heim and Nemeroff 2002). During the past 20 years, increased heart rate (decreased heart period) with trauma stimuli has been one of the most robust autonomic findings from studies of numerous types of traumas, including combat (Keane et al 1998), motor vehicle collisions (Blanchard et al 1996), and sexual abuse in female subjects (Orr et al 1998). We attempted to replicate this finding from adults with PTSD for the first time in young children.

Preliminary evidence exists for relative sympathetic overactivity in traumatized children and adolescents. Perry (1994), in a non-peer-reviewed publication, found evidence for resting tachycardia plus two patterns of abnormal response to an orthostatic challenge in prepubertal children with PTSD. One pattern was a greater initial increase in heart rate upon standing and a second pattern was a slow return to baseline. Bonnano et al (2003) studied female adolescent sexual abuse survivors and found increased heart rate in one subgroup but decreased heart rate or a no-change pattern in a subgroup that also scored higher on dissociation measures. DeBellis et al (1999) studied maltreated children (aged 8–13 years) with PTSD and demonstrated elevated 24-hour urinary catecholamine levels compared with two non-PTSD groups.

Heart period variability (HPV) is an additional metric for analyzing the electrocardiogram (ECG) signal that provides more precise information about parasympathetic versus sympathetic activity. Respiratory sinus arrhythmia (RSA), a subset of total HPV, is the normal cyclic fluctuation of heart period with respiration. Heart rate increases during inspiration and decreases during expiration. This cyclic coupling of respiratory phase and heart rate is centrally driven, with the primary efferent being the vagus nerve (parasympathetic). In pharmacologic-blockade studies, RSA has been demonstrated to be a valid measure of tonic parasympathetic activity under controlled respiratory conditions and a valid measure of phasic parasympathetic changes (e.g., Grossman et al 1991). Consequently, parasympathetic...
activity can be estimated from power spectral analysis for the frequency band that coincides with the age-appropriate respiratory rate (Mezzacappa et al 1994). Cohen and colleagues have studied low (sympathetic) and high (parasympathetic) frequency power spectrum density in adult subjects with PTSD, as reported in several publications. They found that adults with PTSD demonstrated higher sympathetic tone at rest (Cohen et al 1997), a significant failure to marshal either a sympathetic or parasympathetic response to trauma stimuli (Cohen et al 1998), and normalization of abnormal HPV parameters in patients who responded to pharmacotherapy (Cohen et al 2000).

In contrast, RSA has been used commonly in the child-development literature. It has been demonstrated to index the maturing central nervous system, to vary in expected ways in a variety of at-risk populations, and to serve as a psychophysiological correlate of emotion regulation (Porges 1995). Emotion regulation, the process that influences the intensity, timing, and quality of discrete emotions (Thompson 1994), has been cast as critical in emotional development, relevant to adaptive behavioral strategies, and concerned with individual differences. Theoretical models have postulated that autonomic regulation of the heart is one of the basic sources of emotion regulation (Calkins 1994). In general, increased HPV occurs with development, and stress responses that involve relatively more HPV correlate positively with more adaptive outcomes. In this direction, RSA has correlated to many variables of clinical and developmental interest, such as temperament (e.g., Porges et al 1994; Snidman et al 1995), attention (Suess et al 1994), premature newborn status (DiPietro et al 1992), and insecure attachment classifications (e.g., Calkins and Fox 1992; Izard et al 1991). In this study, we assessed RSA as an index of parasympathetic control for the first time in children with PTSD symptoms.

Because of the unique salience of the caregiving context for both adaptive development (Crockenberg and Leerkes 2000) and the developmental psychopathology of young children (Zeanah et al 1997), it is important to consider the predictive value of the parent-child relationship in the exploration of emotion regulation and psychopathology as child outcome variables. Maternal depression was a significant mediator of treatment outcome in sexually abused preschool children (Cohen and Mannarino 1996). Mother-child concordance of change in vagal tone (parasympathetic), but not baseline vagal tone, has been empirically demonstrated, and suggests a role for the regulatory capacity of mothers on their children’s parasympathetic regulatory capacities (Bornstein and Suess 2000). Therefore, parent-child relationship quality might serve as an explanatory variable to predict children’s psychophysiolologic reactions.

We formulated two hypotheses: 1) traumatized subjects with PTSD would manifest relative sympathetic overactivity (i.e., decreased heart period) in response to trauma-related stimuli compared with traumatized subjects without the full PTSD diagnosis and compared with nontraumatized control subjects; and 2) subjects with PTSD would manifest decreased parasympathetic activity (i.e., decreased RSA [less variability]) in response to trauma-related stimuli compared with the other two groups.

Methods and Materials

Sample

Sixty-two traumatized children and 62 nontraumatized control children who were 20 months through 6 years of age were recruited (see Scheeringa et al 2003 for details). The traumatized children were recruited from an inpatient unit of a level I trauma center (n = 21), three battered women’s shelters (n = 19), an outpatient mental health program that specializes in treating violence-exposed children (n = 9), a cancer program (n = 6), and by word of mouth (n = 7). The trauma center subjects experienced automobile collisions and other acute accidental injuries. The battered women’s shelter subjects witnessed domestic violence against their mothers. The pediatric cancer patients experienced highly invasive medical procedures while awake during their initial cancer work-up (spinal tap and bone marrow extraction) and repeated spinal taps during the course of treatment. Inclusion criteria were that the child 1) was between 20 and 83 months of age at the time of the event and assessment; 2) experienced an event that was life-threatening or witnessed a life-threatening event to their parent or older sibling; 3) spoke English; and 4) showed at least one PTSD symptom more than 2 months after the event. At least one symptom was required to ensure that only symptomatic subjects were enrolled. Because of the preliminary nature of trauma studies in young children, it is not yet entirely clear what constitutes a traumatic stressor. By requiring at least one symptom, we sought to make sure we were not too liberal in the types of traumas that were included. Exclusion criteria were 1) disabling preexisting medical disorders; 2) severe developmental disorders; and 3) for the hospitalized injury victims, a Glasgow Coma Scale score of ≤6 in the emergency room. No potential subjects met the exclusion criteria.

Three families refused to participate in the study when they were approached.

The mean duration between the most severe trauma and assessment was 11.3 months, and the median was 7.5 months (SD 11.1, range 2–52 months). Sixteen of the subjects met the criteria for the diagnosis of PTSD by the alternative criteria and algorithm (Scheeringa et al 2003), and 46 did not (no subjects met the diagnosis by the DSM-IV algorithm). The control children were recruited from neighbors of the trauma subjects (n = 25) and from a Head Start center (n = 37). No control subjects had experienced a life-threatening trauma.

Measures

Posttraumatic Stress Disorder Semi-Structured Interview and Observational Record for Infants and Young Children. This is a semi-structured interview of the primary caregiver (Scheeringa and Zeanah 1994). This measure contains modified wording for five PTSD symptoms, to make them more developmentally appropriate for this age group and is based on the studies of Scheeringa et al (1995, 2001, 2003). A diagnosis can be made either by the DSM-IV algorithm or by the alternative algorithm for young children, which has been empirically validated. Symptoms must have been present over the preceding month. A random sample of 18% of the interviews (n = 11) of the trauma subjects from this study were coded by the principal investigator from videotape for interrater reliability. Cohen’s k for all of the 18 PTSD symptoms was .75. Cohen’s k for scoring enough criteria to meet the alternative diagnosis was .79.

Stimuli. First, ECG was recorded for a 3-min baseline. The children were allowed to draw to keep them occupied. Second, for the control stimulus, the examiner asked the children if they remembered a unique and pleasant event. The caregiver had supplied the details of this event to the examiner beforehand during a telephone call. We tried to use events that were single occurrences (e.g., trip to Disneyland) within the last 2–6 months and could not be confused with other events. The investigator began with an open-ended question about the event to encourage free recall and then followed a semi-structured script to ask about six component
features of the event: who was present, how they were transported, what the child did, how the child felt, what the child ate, and what they brought home. As a validity check, five misleading questions were inserted to verify that the children were not suggestive and answering yes to everything. The children's answers were coded from videotape for yes/no accuracy. One point was given for an accurate response, thus the range of possible scores was 0–11. This memory interview lasted approximately 3 min. Third, the children were allowed to draw for another 3-min baseline epoch. Fourth, for the trauma stimulus, the investigator followed the same procedure as for the control stimulus. The range of possible memory accuracy scores was 0–12, based on seven component and five misleading items. A random sample of 20% (n = 12) of the trauma memory interviews were coded from videotape by a second coder. The percent agreement for interrater reliability for all items was 85%.

Because the control subjects had not experienced a traumatic event, a separate method was used for experimental induction of a stress reaction in this epoch. The investigator verbalized and enacted with props a car crash in which mother and child dolls were injured. The investigator then asked the children, “What happens next?” to make the children actively participate in the story to simulate the experience of the trauma subjects. A toy ambulance, doctor, and nurse figures were in reach for the children to use. This lasted 3 min.

Family Rehearsal. This was a frequency count of how often children had been exposed to any discussions about the traumatic events since they occurred. This was used as an index of repetitive exposure to memories of the trauma. The scores could range from 0 to no upper limit.

Interbeat Interval. We collected heart period (interbeat interval [IBI]) from ECG data during the stimului epochs while children were seated in a child’s chair at a play table. The mothers sat 3 ft to the side. The examiner sat across the table from the children. Electrodes were placed axially on the left and right rib cage and centrally on the stomach. Data were collected with equipment and software from the James Long Company (Cargoga Lake, New York). The bioamplifier was set for bandpass filtering at frequencies of .1 and 1000 Hz. Data were digitized at a sampling rate of 512 Hz with a 12-bit A-D board in a laboratory computer. Electrocardiogram was resampled off-line at 1000 Hz. The rising edges of the R waves were automatically identified with a multiple-pass, self-scaling algorithm. An interactive graphic program enabled manual correction of missed or misidentified R waves. The IBIs between R waves were prorated in to equal time intervals of 125 msec. The IBI change scores for the memory epochs were calculated by subtracting the mean IBI for the immediately preceding 3-min baseline from the mean IBI for the 3-min memory interviews. A negative IBI change score reflects that IBI decreased during the stimulus epoch (heart rate increased). A positive IBI change score reflects that IBI increased during the stimulus epoch (heart rate decreased).

Respiratory Sinus Arrhythmia. Prorated IBIs were detrended with a high-pass filter with a period of 10 sec (roughly .1 Hz) to minimize variability from aperiodic shifts in the tonic heart period. Fourier analyses were applied to the residual IBI data for each epoch. Respiratory sinus arrhythmia was estimated from power spectral analysis in the empirically validated respiratory frequency for the child's age. Frequency bands for respiratory rate were chosen based on previous empirical work: 3–75 Hz for 1–3-year-old children and 2–65 Hz for 4–6-year-old children (Bar-Haim et al 2000). Mean RSA values for each epoch were not normally distributed, as is typical of HPV measures; however, the RSA change scores, calculated in an identical fashion as the IBI change scores, were normally distributed and did not require natural log transformation. A negative RSA change score reflects that RSA decreased during the stimulus epoch (withdrawal of parasympathetic activity, or loss of variability). A positive RSA change score reflects that RSA increased during the stimulus epoch (increased parasympathetic activity, or gain of variability).

Parent–Child Interaction. Relational qualities were assessed with the method used by Crowell and colleagues (Crowell and Feldman 1989; Crowell et al 1988), which has discriminated between clinic-referred and nonclinical dyads. The dyad was videotaped during 5 min of free play, 2 min of clean-up, and 20 min during four structured puzzle tasks. Subsequent puzzle tasks increased in difficulty, so that the third and fourth tasks were too difficult for the child to complete alone, thus increasing the demand for dyadic collaboration. Of the eight caregiver variables, five were rated on 1–7 point scales: behavioral responsiveness, emotional responsiveness, positive affect, withdrawal/ depressed, and irritability/anger. The other three scales were rated on 1–3 point scales: verbal aggression, positive discipline during clean-up, and negative discipline during clean-up. Two raters were trained to reliability with an expert rater. When a rater’s score was within one point of the expert, they were considered in agreement. The percent agreements for rater 1 with the expert on 21 training tapes ranged from .71 to 1.0 (median .91). The percent agreements for rater 2 with the expert ranged from .67 to 1.0 (median .91). The percent agreements between rater 1 and rater 2 on the 21 reliability tapes ranged from .48 to 1.0 (median .95). Rater 1 scored 104 of the tapes. Rater 2 scored 10 tapes. Raters were blind to trauma status.

Procedure. The Tulane University Health Sciences Center Committee on Use of Human Subjects approved this study. When subjects arrived at the lab, caregivers received a complete description of the study, and written informed consent was obtained. Voluntary assent was obtained from children. The first procedure was the ECG recording during the baseline and memory epochs. Then the parent–child interaction procedure was completed. Next, the caregiver was interviewed with the diagnostic interviews.

Data Analysis

Electrocardiogram data were missing for one of the 16 Trauma/PTSD subjects, for three of the 46 Trauma/No PTSD subjects, and for two of the 62 control subjects. Visual inspection of the distributions revealed one outlier in the Trauma/PTSD group. The IBI and RSA dependent variables were transformed into z scores for a statistical outlier test. The z scores for all four dependent variables of this subject were more than 2 SDs outside the means (z scores > 3), so this subject was dropped. The final three groups, Trauma/PTSD (n = 14), Trauma/No PTSD (n = 43), and the control subjects (n = 60), were compared for differences in IBI change scores for each stimulus epoch with a mixed-design two-way repeated-measures analysis of variance (ANOVA). Similar analyses were conducted for RSA change scores. Significant effects were followed by pairwise testing with the multiple range Newman-Keuls test. Post hoc exploratory analyses for interaction effects were examined with two-way ANOVAs.

Results

Heart Period

First, it was established that the three groups did not significantly differ on mean IBI at baseline (p = .24). There were no differences in the heart variables by age or gender.
Next, the two-way repeated-measures ANOVA showed that the group $\times$ time interaction was significant [$F(2,114) = 5.75, p = .03$]. The pairwise comparisons of interest for this study were between groups. None of the pairwise comparisons were significant in the pleasant-recall condition. In the trauma-recall condition, the Trauma/PTSD group had decreased heart period ($-6.5$ msec, SD 19.3) whereas the control group had increased heart period ($9.5$ msec, SD 16.6) ($p = .006$, Newman-Keuls). The Trauma/No PTSD group also had decreased heart period ($-1.4$ msec, SD 25.0) and significantly differed from the control group ($p = .005$, Newman-Keuls); however, the Trauma/PTSD group did not differ from the Trauma/No PTSD group, contrary to expectations. The IBI change scores by group and by memory condition are displayed in Figure 1. These results were partially but not entirely supportive of our hypotheses.

Visual inspection of the distribution of IBI change scores during the trauma recall task showed that within both of the trauma groups, even though the group means showed a decrease in heart period, there were a substantial number of subjects who had increased heart period. This suggested the possibility of a hidden interaction effect due to some other variable that was affecting the group means analyses above. Stepwise exploratory analyses were undertaken to determine whether an interaction effect might explain these different responses. The predictor variables chosen for these analyses included the parent variables from the parent–child interaction assessment, on the basis of the theoretic reasoning that the caregiving context has the most proximal effect on the emotion regulation capacities of young children. In addition, the memory accuracy and the family rehearsal variables were chosen as potential predictors, on the basis of the reasoning that the salience of the trauma recall task (i.e., how clearly a child remembered the event and/or how often they had been exposed to reminders of the trauma before the lab session) might be one of the most direct influences on their psychophysiological responsivity. Four of the eight parent variables (withdrawn/depressed, irritability/anger, aggression, and negative discipline during clean-up) did not have a sufficient range of scores to permit cleaving of the group into equitably sized subgroups, and they were dropped from analysis. Of the remaining four parent variables, three of them—behavioral responsiveness, emotional responsiveness, and positive affect—were conceptually similar and were all significantly intercorrelated with each other (pairwise Spearman correlations ranged from .49 to .74; all $p < .0001$). To reduce the number of comparisons and the risk of a type I error, we chose to use only one of these variables. Emotional responsiveness was chosen on conceptual grounds as being the most potentially salient for the emotional regulation of the child. This left four predictor variables: emotional responsiveness, positive discipline, memory accuracy, and family rehearsal. For these analyses, each predictor was dichotomously recoded along a median split, so that approximately half of the traumatized subjects were in the “high” group for that variable, and half were in the “low” group. We chose this strategy for conceptual clarity, to emphasize the difference between high- and low-scoring groups on each of the predictors. Similarly, the group was dichotomized by a median split on the number of PTSD symptoms, rather than by PTSD/No PTSD diagnostic status, to more evenly balance subgroup size and maximize power. Four two-way ANOVA exploratory tests were run with High PTSD/Low PTSD as one of the predictors, plus one of the other predictors, and an interaction term, with IBI change for the trauma memory epoch as the outcome variable. Contrasts were conducted with an $\alpha$ level of significance corrected by the Bonferroni method for the number of contrasts ($\alpha = .05/4 = .0125$).

Two of these analyses yielded marginally significant interaction effects that were below the $\alpha = .05$ level but not below the Bonferroni corrected level of .0125. There was a marginally significant interaction effect between family rehearsal and PTSD symptoms ($n = 52$) [$F(1,48) = 5.70, p = .02$]. The direction of the effect was such that the High PTSD group that had lower family rehearsal scores was the only subgroup that had substantially decreased IBI (mean $-23.1$ msec, SD 22.9). The High PTSD group that had higher family rehearsal had minimally decreased IBI ($-1.2$ msec, SD 22.0). The Low PTSD group with lower family rehearsal had increased IBI (8.2 msec, SD 18.2), and the Low PTSD group with higher family rehearsal showed essentially no change (.7 msec, SD 24.8).

There was a marginally significant interaction effect between parental positive discipline (PD) during clean-up and PTSD symptoms ($n = 55$) [$F(1,51) = 5.6, p = .02$]. The High PTSD group that had lower PD during the clean-up task showed the largest decrease in IBI ($-18.6$ msec, SD 24.6). The High PTSD group that had higher PD had decreased IBI ($-5.2$ msec, SD 22.5). The Low PTSD group with lower PD had increased IBI (7.8 msec, SD 19.5), and the Low PTSD group with higher PD had decreased IBI ($-8.9$ msec, SD 20.8). Parental emotional responsiveness and child memory accuracy did not produce significant interaction effects with PTSD symptoms.

As an additional control, these same interaction analyses were run for the IBI change scores for the pleasant memory recall epoch. These produced no marginally significant interactions.

**Respiratory Sinus Arrhythmia**

First, it was established that the three groups (Trauma/PTSD, Trauma/No PTSD, and control subjects) did not significantly differ at baseline ($p = .55$).

Next, the two-way repeated-measures ANOVA showed that the group $\times$ time interaction was not significant [$F(2,114) = .48, p = .62$], contrary to expectations. The main effects for group [$F(2,114) = .89, p = .41$] and for time [$F(1,114) = 1.0, p = .32$] were also not significant. The mean RSA change scores by group and by memory condition are displayed in Figure 2.

Because of similar concerns about the distribution of RSA change scores as were described above for IBI change scores, the same post hoc exploratory analyses for interaction effects were
during each memory condition. PTSD, posttraumatic stress disorder.

conducted. There was a significant interaction between PD and PTSD symptoms \( (n = 55) \) \( F(1,51) = 10.2, p = .003 \). The direction of the effect was the same as for IBI (i.e., the High PTSD group with lower PD showed the largest decrease in RSA \( -9.9 \text{ msec}, \text{SD 14.6} \)). This interaction effect is best viewed graphically in Figure 3. There were no significant interactions between emotional responsiveness, memory accuracy, or family rehearsal, and PTSD symptoms.

When these same interaction analyses were run for the pleasant memory recall epoch as an additional control condition, none of the predictors yielded marginally significant interactions. To visually show the pattern of RSA change scores after the traumatized group had been split into four subgroups by High/Low PTSD and High/Low PD, the means are graphed in Figure 4 for each memory condition. It is evident that the subgroup with the double impact of High PTSD symptoms and Low PD is the only subgroup that showed a trend for relatively decreased RSA between the pleasant memory and the trauma memory epochs.

Discussion

This is the first study to explore psychophysiological correlates of posttraumatic stress symptoms in very young children, and it produced two important findings. First, both the Trauma/PTSD and the Trauma/No PTSD groups differed from the nontraumatized control subjects on RSA change when probed with a trauma-related stimulus. Because each traumatized subject was required to manifest at least one symptom of PTSD for entry into the study, this is suggestive evidence that even minimally symptomatic young children have altered psychophysiological reactivity compared with nontraumatized children. Although there are acknowledged limitations in the methodology (the control group did not have a prior traumatic experience as the basis for a true trauma-related stimulus), this finding is not easily dismissed as an artifact of the methodology, because the groups did not differ when probed with a nontrauma stimulus (the pleasant memory recall). This supports the notion that a traumatic experience has an effect on psychophysiological reactivity regardless of PTSD diagnostic status. This finding is similar to those in older traumatized (sexually abused) children, who demonstrated abnormal catecholamine (DeBellis et al 1994b) and cortisol (DeBellis et al 1994a; Hart et al 1996) levels, regardless of PTSD status.

Second, the value of RSA reactivity as an index of parasympathetic neural control that is correlated with posttraumatic symptomatology has been demonstrated for the first time in children in a preliminary fashion. As shown in the Figure 3, it is suggestive that the child’s autonomic regulation (RSA change) is impacted differentially, depending on the interaction between the caregiving context (parental positive discipline [PD]) and the child’s level of PTSD symptomatology. Although the meaning of such an interaction is difficult to interpret definitively, the most noteworthy feature is that the children who were most symptomatic and had the least PD showed the largest decrease in RSA. More specifically, it suggests that PD manifests its primary impact on the parasympathetic regulatory system of the child (i.e., greater PD increases parasympathetic activity in children) (Bornstein and Suess 2000). This makes theoretic sense and agrees with the previous body of literature on at-risk young children that has demonstrated higher parasympathetic tone with better child outcomes (Porges 1995).

This type of interaction effect is consistent with other preliminary explorations of psychophysiological outcomes in samples of at-risk young children. For example, Nachmias et al (1996) found elevated cortisol levels after the stress of a mother-separation stimulus in 18-month-old children with high levels of behavioral inhibition only when they had an insecure attachment relationship with their mothers. Fox and Davidson (1987) demonstrated

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a relative decrease in right frontal electroencephalogram power in infants during the stress of a separation from their mothers only when the infants also cried during the separation, compared with those who did not cry.

The direction of the (marginally significant) interaction effect for family rehearsal on IBI change also makes sense in the context of what we currently know of traumatized young children and their caregivers. The treatment literature for trauma survivors has demonstrated that re-exposure to the trauma reminders with emotional engagement is absolutely crucial to psychotherapy treatment for PTSD in adults (Rothbaum and Foa 1996) and in young children (Cohen and Mannarino 1996). Families who avoid discussion of the trauma with their young children might inadvertently have an adverse impact on the children’s adaptation after stress. Also, case studies with very young children anecdotally have demonstrated the importance of caregivers discussing the trauma with their children for treatment success (MacLean 1980; Pruitt 1979).

There are acknowledged limitations of this preliminary investigation. The methodology for the trauma stimulus had to be, by the nature of the developmental limitations of this age group, different from the method used in adult studies. One cannot ask very young children to perform mental imagery quietly inside their heads and know with certainty that they have done what you asked. Therefore, we opted to question them about their trauma, thereby knowing for certain that they were thinking about the traumatic event. One direction for future studies would be to develop a standardized stimulus that could be applied to all subjects. In addition, research on adults has shown that the manipulation of social support (i.e., the presence of a friend or family member in the testing room) has an attenuating effect on cardiovascular reactivity (Thorsteinsson and James 1999). Although that research did not reflect parent-child relationships, it raises the possibility that the reactivity seen in our subjects might be greater if the mothers were not in the room. Also, two studies with adults (Bernardi et al. 2000; Sloan et al. 1991) have found that the act of speaking alters HPV. Although it is not clear how salient this factor is to the findings of this study, the development of standardized stimuli for the future ought to take this finding into account. Last, because IBI and RSA reactivity were not measured in a prospective fashion before the traumatic experiences of these subjects, we cannot know whether these psychophysologic patterns were present beforehand and serve as vulnerability factors for the development of PTSD symptoms, as opposed to being changes that were caused by the trauma. This is a study design limitation of the majority of psychophysologic studies in traumatized adults and should be viewed with the same degrees of caution and value.

In conclusion, the significant interaction effect from this study indicates a more complex set of relationships between automatic reactivity, the development of PTSD symptoms, and the caregiving context. Another potentially complicating factor might be that we studied a group that was heterogeneous in regard to type of trauma. Rather than being a limitation, this heterogeneity suggests a generalizability of these findings across trauma types; nevertheless, a reasonable strategy for future studies would be to study more homogenous trauma groups. Overall, the assessment of psychopathology in young children ought to include measurements for psychophysologic processes and parent-child relationship qualities to understand more comprehensively the mechanisms of maladaptation during this complex developmental period.

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