Lifespan-Developmental Differences in Physiologic Startle in Trauma Victims

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Introduction

The age at which a person experiences a traumatic event has been identified as an important predictor for the development and severity of traumatic sequelae and other posttraumatic spectrum disorders (Maercker et al. 1999) including increased physiological reactivity to reminders of the traumatic event and hyperarousal. One key symptom of PTSD is increased startle response to both trauma-related, conditioned stimuli (e.g., idiosyncratic trauma cues; Orr et al. 1993; Pitman et al. 1990; Shalev et al. 1993) and unconditioned startle stimuli (e.g., loud tones; Butler et al. 1990; Morgan et al. 1995; Orr et al. 1995; Shalev et al. 1992; Flor et al. 1998).

Childhood, adolescence and young adulthood are sensitive developmental stages during which a person is especially prone to respond to a traumatic event with severe PTSD symptoms (see Maercker 1999 for a review). This has been attributed to the psychological (Cole and Putnam 1992; Harter 1993), social (Elder et al. 1994) and biological (Perry et al. 1994; Teicher et al. 1997) specifics of these developmental stages.

The biological alterations relevant to trauma processing and PTSD include dysfunctions in several brain structures (hippocampal, amygdalar, prefrontal cortical and hemispheric integration abnormalities; Teicher et al. 1997) and in neurochemical
systems such as the noradrenergic (McFall et al. 1990; Blanchard et al. 1991; Southwick et al. 1997), serotonergic (Southwick et al. 1997), opiate (Williams et al. 1984) and corticotropin-releasing (Yehuda et al. 1990) transmitter systems. The profound impact of traumatic stress on these areas and functions of the adult brain has been demonstrated in several animal (Sapolsky 1984; LeDoux 1995) and human studies (e.g., Gurvits et al. 1996; Stein et al. 1997; Freemann et al. 1998; Shin et al. 1999). In the developing brain, which is more plastic than the adult brain, stress and trauma occurring during critical developmental stages may lead to irreversible neurobiological alterations (Perry et al. 1994, Teicher et al. 1993, 1996). During adolescence, frontocortical dominance in information processing and arousal regulation (Dampster 1992) is established in the course of brain maturation. Arousal is a multicentric process mediated by brain-stem and thalamic mechanisms and modulated by frontal cortical activity. The frontal lobe is the ontogenetically youngest area of the brain. Major projections to the frontal and prefrontal cortex do not begin to myelinate until adolescence. This development continues well into the third decade (Benes et al. 1994; Sowell et al. 1999). Increased impulsive behaviour in adolescence is often explained in terms of incomplete frontal brain development, and indicates premature arousal regulation (Marcia 1983).

Dopamine and other monoamine projections to the prefrontal cortex are especially activated by stress. This excessive activity may alter the development of the prefrontal cortex (Deutch & Roth 1990). Indeed, development may be arrested, and the prefrontal cortex prevented from reaching its full adult capacity (Teicher et al. 1996). We therefore hypothesize that people who experience traumatic stress in adolescence and early adulthood will show more severe PTSD symptoms, including higher levels of arousal, than people who experience traumatic stress in later adulthood. One indicator
of arousal is the startle response – a physiological reaction to a sudden loud tone or noise. It is mediated by polysynaptic brain-stem mechanisms (Davis et al. 1982) and modulated by cortically mediated attention (Silverstein et al. 1981). It can be measured using eye blink, cardiac and electrodermal response maximum, and response slope as a measure of habituation.

In a previous study on former political prisoners (Maercker, 1998), we compared clinical data from a group of people imprisoned in late adolescence/early adulthood (17-21 years) with that from groups of people imprisoned in young (22-34 years) and middle adulthood (35-50 years). Background data to political imprisonment in former communist East Germany are presented in other publications of our group (Maercker, 1998, 1999; Maercker & Schützwohl, 1997; Maercker, Beauducel & Schützwohl, 2000). Age-at-imprisonment groups differed in the extent of their PTSD symptoms and reported trauma severity, with adolescent victims showing the highest values.

The aim of the present study was to investigate physiological parameters (electrodermal and cardiac reactivity to startle tones) in participants exposed to trauma either in adolescence or in adulthood, and to explore whether adolescent victims show higher levels of physiological hyperarousal.

**Methods and Materials**

**Subjects:**

Former political prisoners were recruited from a sample of participants in a previous study on traumatization by political imprisonment in communist East Germany. Of the 46 participants contacted, N = 9 from the trauma-in-adolescence (ADL) group and N = 6 from the trauma-in-adulthood (ADU) group replied. Others
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declined to participate because of time constraints or for health reasons.

The ADL group consisted of 9 subjects (8 male, 1 female) with a mean age of 19.0 (±1.5) years at the time of imprisonment. The ADU group consisted of 6 subjects (5 male, 1 female) with a mean age of 37.7 (±4.4) years at the time of imprisonment. All subjects gave written informed consent for the study.

Measures

Trauma severity: Trauma severity was assessed using the Harvard Trauma Questionnaire (HTQ; Mollica et al. 1992), which consists of 16 specific traumatic events plus three items “drowning”, “suffocating” and “beating on the head” (value range 0-16). Participants are asked whether they have experienced, witnessed or heard about such events.

Symptoms: PTSD symptoms were assessed using the Impact of Event scale-revised (IES-R; Weiss & Marmar, 1996) and a German version of the ADIS-R structured clinical interview (DiNardo and Barlow 1988).

Startle stimulus material: Fifteen acoustic stimuli (95db, 0ms rise and fall time, 1200 Hz square frequency) with a duration of 1300ms were presented (cf. Orr et al. 1995). The intensity of the stimuli was controlled by determining the sound pressure level. The tones were emitted by two loudspeakers placed at a distance of 3m to the subject. The interstimulus interval (ISI) of between 16 and 87 s was randomly generated by computer.

Physiological data recording: An electrocardiogram (ECG) was recorded continuously at a sampling rate of 500 Hz from the manubrium sterni and above the left costal arch in the axillary line. Skin conductance (SC) was recorded continuously at a
sampling rate of 50 Hz from the thenar of the non-dominant hand. A constant voltage of 0.4V was applied for the SC recording. A neutral electrode was attached at the processus xiphoideus. Analog cardiac and electrodermal responses were digitized by an A/D converter implemented in PARON-Biosignal™ recording. Parallel to the raw ECG recording, the QRS complex was detected online, RR distances were determined and transformed into frequency values (heart rate, HR).

Procedure: Participants completed the questionnaires prior to the physiological recording. They were relaxed and seated with their eyes open in a comfortable recliner. Testing consisted of a 5-minute adaptation phase, a 5-minute resting phase, and the presentation of the auditory startle stimuli. The overall duration of recording was about 15 minutes.

Data analysis

The raw data from the physiological assessment were averaged over 2000 ms. Heart rate responses were determined using the protocol by Orr et al. (1995). SC responses were determined by subtracting the mean SC within 0-1s post stimulus from the SC within 1-4s post stimulus (Lykken et al. 1988). To determine the habituation of the HR and SC responses, standardized Beta positive slope coefficients were computed using a linear regression of z-transformed response scores and number of stimuli.

Statistical analyses were conducted using t-tests and ANCOVAs with demographic and clinical variables as covariables for which t-tests revealed significant group differences. Due to the small sample size, the significance level was set to p < .10.
Results

Trauma and symptom severity: As expected, and in line with our previous study (Maercker, 1998), the two groups differed in their reported exposure to trauma, with the ADL group reporting more severe traumatization (Table 1). There were no significant differences in the duration of imprisonment.

However, the two groups did not differ significantly in their PTSD symptom severity. Neither Impact-of-Event-R scores (Table 1) nor number of diagnosed PTSD symptoms differed significantly or substantially: reexperience symptoms ($M_{ADL} = 2.1$, $SD = 2.0$; $M_{ADU} = 2.5$, $SD = 2.0$; $t(13) = .4$, n.s.), avoidance/numbing symptoms ($M_{ADL} = 1.9$, $SD = 2.2$; $M_{ADU} = .8$, $SD = 1.2$; $t(13) = 1.1$, n.s.), and hyperarousal symptoms ($M_{ADL} = 2.4$, $SD = 1.5$; $M_{ADU} = 2.2$, $SD = 2.0$; $t(13) = 0.3$, n.s.).

Skin conductance: ADL participants tended to have higher SC response scores to the auditory stimuli ($t[13] = 1.15$; $p = .13$). An ANCOVA with “number of traumatic events” and “time interval between trauma and study” as covariables for the mean SC response scores to the stimuli revealed a significant main effect for “age at time of imprisonment” ($F[1,14] = 6.36$; $p < .01$) and a significant effect for the covariables ($F[2,13] = 5.35$; $p < .05$). The correlation between SC response scores to the stimuli and “time interval between trauma and study” was $r = -.50$ ($\beta = -.65$; $F[2,13] = 5.15$; $p < .05$).

In both groups, the level of SC response decreased continuously over time (i.e., with habituation), while the overall level of response tended to be higher in participants traumatized in adolescence (see Figure 1). However, there were no significant group
differences in the SC response slopes at conventional significance levels. Furthermore, the ANCOVA did not reveal any significant explanatory effects.

Heart rate: Participants exposed to trauma in adolescence had a higher HR in the resting condition ($t[13] = 1.46; p = .08$). Though an ANCOVA for the resting heart rate did not reveal any significant explanatory effects, a high correlation was observed between the resting heart rate and the time interval between imprisonment and participation in the study ($r=.98, \beta=.65$; $F[1,14] = 3.64; p=.08$). No significant effects were found for the HR response scores to the auditory stimuli, and there were no significant group differences in HR response slopes.

Discussion

Based on a literature review the aim of this pilot study was to compare physiological reactivity to loud tones in people exposed to traumatic events at different developmental stages of life (late adolescence/young adulthood vs. later adulthood). We found a higher skin conductance response to auditory stimuli in the group of participants exposed to a trauma at an earlier age. Because of various limitations of this small study (see below), however, this main result should only be interpreted with caution.

Skin conductance is indicative of sympathetic activity, which is regulated by different areas of the brain. Increasing age has previously been reported to be correlated with decreasing SCR (Catania et al. 1980; Porges and Fox 1986). In addition, Raine et al.
(1991) found a positive correlation between the amplitude of the electrodermal orienting response and the size of certain areas in the frontal cortex as assessed with MRI. Tranel and Damasio (1994) identified the ventromedial frontal region, the right inferior parietal region and the anterior cingulate gyrus as important physiological correlates of electrodermal activity in a lesion study in humans. It is possible that functional and structural alterations in the frontal regions are responsible for the increased electrodermal reactivity observed in victims traumatized in adolescence.

In addition to altered electrodermal reactivity to startle stimuli, participants traumatized in adolescence showed an elevated tonic heart rate in the resting condition. Elevated tonic cardiovascular activity has been found in previous studies among combat veterans in particular (Pitman et al. 1990; Muruoka et al. 1998; Keane et al. 1998), and has been interpreted as indicating either a permanent state of sympathetic overdrive (Muruoka et al. 1998; Orr et al. 1998) or that the participants interpret the experimental situation as threatening or ambiguous (Prins et al. 1995). Thus, subjects traumatized in adolescence may be characterized by elevated tonic sympathetic activation. However, our sample did not yield significant results with respect to the most robust finding in startle research to date – i.e., elevated heart rate mean response.

Shalev et al. (1992) and Orr et al. (1995, 1997) found slower skin conductance habituation in adult PTSD patients than in non-PTSD controls. As this phenomenon is also observed in other anxiety disorders, it has been regarded as a constitutional risk factor rather than a consequence of trauma (Lykken et al. 1988). In our study, however, habituation slopes for electrodermal and cardiac responses did not differ significantly between the groups, although continuous differences in time were found for electrodermal activity.
There are several basic limitations to this pilot study: First, the very small sample size reduced the power of the statistical tests. Future studies should follow up the findings with larger numbers of participants. Second, the two groups differed markedly in the time interval since traumatization (14 years on average). It is hard to exclude the possibility that this difference biases the findings using statistical covariance analysis alone. Age differences may effect outcomes in multiple ways (e.g., biological and psychological maturation, social role changes at different ages). One indication for this may be seen in the high correlations between outcomes and time interval between trauma and study variable. Further studies should include other trauma samples to exclude this age bias. Third, the non-significant differences in the posttraumatic symptoms of the two groups contradict a previous finding (Maercker, 1998). This non-significance may also be due to the limited statistical power of the present study. Fourth, it is necessary to investigate whether reduced electrodermal response to startle stimuli is accompanied by a change in eye-blink amplitude, the conventional measure of startle response. Direct comparison of neuroimaging correlates of PTSD in adolescence and adulthood can provide further insight into mechanisms of pathogenesis, the maintenance of posttraumatic symptoms and other trauma-related disorders, and the impact of severe stress on the developing brain.
References

Catania JJ, Thompson LW, Michalewski (1980) Comparisons of sweat gland counts, electrodermal activity, and habituation behavior in young and old groups of subjects. Psychophysiology 17: 146-152
Albany


Mollica RF, Caspi-Yavin Y, Bollini P, Truong T, Tor S, Lavelle J (1992) The Harvard Trauma Questionnaire. Validating a cross-cultural instrument for measuring torture,
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trauma, and posttraumatic stress disorder in Indochinese refugees. J Nerv Mental Dis 180:111-116


Table 1. Comparison of group features and psychological data (t-tests) and covariate group comparison (ANCOVA) of physiological measures

<table>
<thead>
<tr>
<th></th>
<th>Trauma in adolescence (ADL)</th>
<th>Trauma in adulthood (ADU)</th>
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<tr>
<td></td>
<td>(17-22 years)</td>
<td>(35-50 years)</td>
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<td></td>
<td>(N = 9)</td>
<td>(N = 6)</td>
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<tr>
<td></td>
<td><strong>M</strong></td>
<td><strong>SD</strong></td>
<td><strong>M</strong></td>
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<tr>
<td>Age at time of imprisonment (years)</td>
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<td>Current age (years)</td>
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<td>Interval between trauma and study (years)</td>
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<td>9.6</td>
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<tr>
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<tr>
<td>Response slope</td>
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<td>0.3</td>
<td>-0.4</td>
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ANCOVA with control variables number of traumatic events and time interval between trauma and study.
Figure 1:

Mean skin conductance responses (SCR) to the 15 tone trials in a group traumatized in adolescence (ADL; black squares) and in a group traumatized in adulthood (ADU; white squares).