Regional Brain Electrical Activity in Posttraumatic Stress Disorder After Motor Vehicle Accident

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This study examined whether patients with posttraumatic stress disorder (PTSD) related to motor vehicle accidents (MVAs) would show an abnormal pattern of electroencephalographic (EEG) alpha asymmetries, which has been proposed for particular types of anxiety. Patients with PTSD ($n = 22$) or subsyndromal PTSD ($n = 21$), traumatized controls without PTSD (non-PTSD with MVA; $n = 21$), and healthy controls without MVA ($n = 23$) underwent measurement of EEG activity during baseline and exposure to a neutral, a positive, a negative, and an accident-related picture. Differences in brain asymmetry between groups were observed only during exposure to trauma-related material. PTSD and subsyndromal PTSD patients showed a pattern of enhanced right anterior and posterior activation, whereas non-PTSD with MVA participants showed the opposite pattern. Furthermore, posterior asymmetry in nontraumatized healthy controls varied with gender, with female participants showing a pattern of higher right posterior activation. The results support the hypothesis that symptomatic MVA survivors are characterized by a pattern of right hemisphere activation that is associated with anxious arousal and symptoms of PTSD during processing of trauma-specific information.

Keywords: electroencephalograph, alpha asymmetry, posttraumatic stress disorder, emotion

Electroencephalograph (EEG) asymmetries have been associated with motivational and affective traits and states (Coan & Allen, 2003). Davidson's (1995) model of anterior asymmetry and emotion assumes that certain regions within the left hemisphere are involved in approach-related behavior and emotion, whereas right prefrontal and anterior temporal regions have been proposed to be related to withdrawal-related behavior and emotion. Evidence for the model has come from research investigating frontal EEG asymmetry in relation to emotional–motivational traits (Tomarken, Davidson, Wheeler, & Doss, 1992) and emotional responding (Wheeler, Davidson, & Tomarken, 1993), although Hagemann, Naumann, Becker, Maier, and Bartussek (1998) failed to replicate these relationships. Frontal baseline EEG asymmetry has also been associated with psychopathology such as depression (Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1991). For pathologic anxiety, Wiedemann et al. (1999) found relative right frontal activation during rest and confrontation with anxiety-related pictures in patients with panic disorders. Comparable findings of relative right-sided activation in anterior temporal, lateral frontal, and parietal regions were reported by Davidson, Marshall, Tomarken, and Henriques (2000) in a sample of participants with social phobias during the anticipation of a public speech.

However, a number of studies investigating brain asymmetries in relation to psychopathology have shown results inconsistent with Davidson's model (Bruder et al., 1997; Reid, Duke, & Allen, 1998). Heller and colleagues (e.g., Heller & Nitschke, 1998) proposed an alternative model in which anterior cortical regions are involved in the modulation of valence, whereas the right parietotemporal region is involved in the modulation of emotional arousal. This model suggests that depression is associated with right parietotemporal hypoactivation, whereas emotional arousal is related to hyperactivation of the same region. Evidence for the model has come from studies investigating depressed patients

(Keller et al., 2000) and students with high and low levels of depression and anxiety (Heller, Etienne, & Miller, 1995; Keller et al., 2000).

In a refinement of this theory, Heller, Nitschke, Etienne, and Miller (1997) proposed a distinction between subtypes of anxiety related to different patterns of brain activation. Anxious arousal should be associated with greater right parietotemporal activation, whereas anxious apprehension (e.g., worry and rumination) should be associated with greater left anterior activity. Support for these assumptions has come from studies examining regional EEG asymmetry in self-reported anxious arousal (Nitschke, Heller, Palmieri, & Miller, 1999) and during experimentally manipulated anxious arousal (Heller et al., 1997).

To our knowledge, only two studies have directly investigated EEG asymmetries in posttraumatic stress disorder (PTSD). One study reported right-sided activation in response to trauma-related olfactory stimuli in a small sample ($n = 5$) of Vietnam War veterans with PTSD (McCaffrey, Lorig, Pendrey, McCutcheon, & Garrett, 1993). Recently, Metzger et al. (2004) reported that PTSD arousal symptoms were associated with increased relative right parietal baseline activity in a sample of female Vietnam War nurse veterans. However, it remained unclear whether the observed relations could be explained by differences in emotional state during baseline, because this was not assessed in the study. It is interesting to note that the amount of depression symptoms did not attenuate posterior asymmetry but, rather, was associated with increased relative right parietal activity.

In the current study, we examined brain electrical activity during rest and during the presentation of emotional (neutral, positive, negative, and trauma-related) pictures in survivors of motor vehicle accidents (MVAs) with PTSD, with subsyndromal PTSD, and without PTSD as well as in healthy controls without severe accidents. Patients with PTSD and subsyndromal PTSD are characterized by symptoms of anxiety, avoidance, and hyperarousal, and they display psychological distress to trauma-related cues. On the basis of research highlighting the role of right anterior regions in withdrawal-related emotions and right posterior regions in anxious arousal, we expected that patients with PTSD and subsyndromal PTSD would exhibit increased activation (EEG alpha reduction) of right hemisphere anterior and posterior regions during exposure to a trauma-related picture. We did not have specific hypotheses about other emotion conditions. On the basis of research highlighting the role of resting brain activity as a trait marker for psychopathology, we assumed that PTSD and subsyndromal PTSD patients would show increased relative right anterior and posterior baseline activity. It has been proposed that depression and anxious arousal may produce opposing effects on posterior asymmetry. Because a large number of PTSD patients have comorbid depression (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), it was important to rule out the possibility that comorbid depressive symptoms attenuate right posterior activation.

Method

Participants and Assessments

Survivors of MVAs and controls without MVAs were recruited for a treatment study through self-referral local media coverage and advertising. Participants were included when the accident dated at least 6 months prior to testing. Exclusion criteria were a history of neurological problems (e.g., epilepsy), brain surgery, brain damage, and/or severe head injury during the accident. Furthermore, we excluded participants with current alcohol and/or substance abuse or dependence and current or past schizophrenic, bipolar, or psychotic disorder. All participants were required to be off all psychotropic medication for at least 1 month before testing. All participants were right-handed, as assessed with the Edinburgh Handedness Inventory (Oldfield, 1971).

Diagnostic procedure. All participants received a comprehensive description of the study and provided written informed consent at the initial diagnostic assessment. Participants were assessed by advanced doctoral and diploma students in clinical psychology who had received extensive training in the assessment procedures. Each diagnostic session, including an accident interview and clinical interviews, was tape-recorded and lasted 2–3 hr.

Injury severity. Injury severity was assessed with the Injury Severity Score (ISS), which was abstracted from medical records using the Abbreviated Injury Scale (AIS 90; Association for the Advancement of Automotive Medicine, 1990). The ISS is defined as the sum of the squares of the highest scores on the AIS 90 for each of the three most severely injured body regions.

Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995). This scale represents a standardized method that allows for the generation of categorical diagnoses of current and lifetime PTSD as well as a total score, obtained by summing the ratings of frequency and severity of each of the 17 PTSD symptoms defined in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [*DSM–IV*]; American Psychiatric Association, 1994). For analytic purposes, we averaged the scale items corresponding to four PTSD dimensions: *reexperiencing* (5 items), *avoidance* (2 items), *numbing* (5 items), and *hyperarousal* (5 items). Splitting of the avoidance–numbing cluster was performed on the basis of theoretical considerations regarding our hypotheses and empirical work showing that these two dimensions diverge as differentiable dimensions (Asmundson et al., 2000). In this study, we used a German version of the CAPS (Schnyder & Moergeli, 2002), which has comparable reliability and validity to the English version. In this sample, Cronbach's coefficient alphas were .89, .74, .61, .77, and .74 for the CAPS total score and the dimensions of reexperiencing, avoidance, numbing, and hyperarousal, respectively. A good diagnostic agreement ($\kappa = .82$, $p < .001$) was established by rescoring of the CAPS proportion of a randomly selected subsample ($n = 18$) of interviews (performed by psychologists blinded to the diagnosis).

Structured Clinical Interview for the DSM–IV (SCID; First, Spitzer, Gibbon, & Williams, 1996; Wittchen, Wunderlich, Gruschwitz, & Zaudig, 1997 [German version]). We used the SCID to evaluate the presence of concurrent and lifetime *DSM–IV* Axis I disorders. We administered all Axis I modules of the standard SCID except the PTSD module.

Self-report measures. We used a German version (Hautzinger, Bailer, Worall, & Keller, 1994) of the Beck Depression Inventory (BDI; Beck & Steer, 1987). The BDI is a 21-item self-rating scale designed to measure the presence and severity depressive symptoms. It had good reliability (Cronbach's $\alpha = .90$) in this sample.

We used the German version (Laux, Glanzmann, Schaffner, & Spielberger, 1981) of the State–Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970). The STAI is a 20-item self-report measure scoring for cognitive and affective components of both state anxiety and trait anxiety (Cronbach's $\alpha = .94$ in this sample).

Self-reported state and trait positive affect (PA) and negative affect (NA) were assessed with an extended (24-item) German version (Krohne, Egloff, Kohlmann, & Tausch, 1996) of the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). Cronbach's alpha values for PANAS state ranged from .84 to .92 for positive affect and from .80 to .92 for negative affect.

Participant characteristics. We classified the MVA participants into three groups on the basis of the CAPS: full PTSD, subsyndromal PTSD,

and non-PTSD. Patients with full PTSD were required to meet all three symptom clusters (B–D) for PTSD according to *DSM–IV* criteria. We also classified individuals as subsyndromal PTSD if they met the *DSM–IV* Cluster B (reexperiencing) and either Cluster C (avoidance–numbing) or Cluster D (hyperarousal), following the most prominent definition of subsyndromal PTSD—that proposed by Blanchard and colleagues (e.g., Blanchard, Hickling, Taylor, et al., 1996). Similar to PTSD patients, patients with subsyndromal PTSD were also required to meet Criterion F (experience distress because of their PTSD symptoms). All of the 21 subsyndromal PTSD patients met the Cluster B (reexperiencing) criteria. Twenty patients additionally met the Cluster D (hyperarousal) criteria, and only 1 met the Cluster C (avoidance–numbing) criteria. Subsyndromal or partial PTSD has been shown to characterize a significant proportion of MVA survivors that is clinically meaningful and associated with significant distress (Marshall et al., 2001; Schuetzwohl & Maercker, 1999). The non-PTSD group was required to meet either no cluster or one (but not Criterion F).

Seventy MVA survivors were included in the current study. Twenty-four met the *DSM–IV* criteria for full PTSD, 23 met the criteria for subsyndromal PTSD, and 23 were characterized as non-PTSD. Twenty-eight nontraumatized healthy controls without a history of any severe accident or other trauma also participated. Healthy controls were required to have an absence of current or past *DSM–IV* Axis I psychopathology, as assessed with the SCID. The data sets of 2 patients with full PTSD, 2 patients with subsyndromal PTSD, 2 non-PTSD controls, and 5 healthy controls were excluded because of an insufficient amount of artifact-free EEG data in one of the recording conditions. The resulting final sample comprised patients with full PTSD ($n = 22$), patients with subsyndromal PTSD ($n = 21$), non-PTSD controls ($n = 21$), and healthy controls ($n = 23$).

Per the SCID interview, 9 PTSD patients fulfilled criteria for current major depressive disorder (MDD), and 4 PTSD patients fulfilled criteria for lifetime but not current MDD. Additional diagnoses in the PTSD group were current dysthymia ($n = 2$), panic disorder without agoraphobia ($n =$

2), panic disorder with agoraphobia ($n = 1$), agoraphobia without panic disorder ($n = 2$), social phobia ($n = 2$), specific phobia ($n = 5$), lifetime generalized anxiety disorder ($n = 1$), and obsessive compulsive disorder ($n = 1$). Additional diagnoses in the subsyndromal PTSD group were MDD ($n = 4$), agoraphobia without panic disorder ($n = 1$), social phobia ($n = 2$), and specific phobia ($n = 1$). Lifetime but not current diagnoses in the non-PTSD group were MDD ($n = 6$), panic disorder without agoraphobia ($n = 1$), and social phobia ($n = 1$). Two of the non-PTSD participants met the criteria for mild current specific phobia.

As can be seen in Table 1, there were no significant differences between the diagnostic groups in age, degree of right-handedness, time since MVA, concussion, loss of consciousness, injury severity, or gender, although (on a descriptive level) the non-PTSD controls had a larger percentage of men. On the basis of CAPS values, the subsyndromal PTSD group would be characterized as having high levels of reexperiencing and avoidance symptoms, typical hyperarousal symptoms, and essentially no numbing symptoms.

Procedure

EEG was recorded in an electrically shielded room with dim light while participants were resting in a comfortable arm chair. First, EEG baseline data were recorded. The recording procedure was kept similar to that in Tomarken, Davidson, Wheeler, and Kinney (1992). Participants were informed that there would be eight 1-min resting baselines—four with eyes open, four with eyes closed—and that they should try to minimize eye blinks and movements. Two randomly assigned, counterbalanced orders were used for eyes-open (O) and eyes-closed (C) trials of the resting baselines (COOCOCCO, OCCOCOOC). Participants heard a tone indicating the beginning and a double tone indicating the end of each 1-min recording. During the 20-s breaks between baselines, participants were informed via PC monitor whether the following trial was eyes open or eyes closed. Immediately after the final resting period, baseline mood was

Table 1

Means, Standard Deviations, and Significance Tests for Diagnostic and Demographic Characteristics of the Diagnostic Groups

| | Group | | | | | |
Characteristic	Healthy control	Non-PTSD with MVA	Subsyndromal PTSD	PTSD	Test	p
n	23	21	21	22		
Gender						
Male/female: n	7/16	10/11	7/14	3/19	$\chi^2(3) = 5.89$	.12
Female: %	70	52	67	86		
Age in years: M (SD)	37.65$_x$ (10.72)	43.05$_x$ (16.63)	35.76$_x$ (9.82)	42.50$_x$ (11.64)	$F(3, 83) = 1.79$	.16
Handedness: M (SD)	74.92$_x$ (29.39)	70.66$_x$ (31.82)	83.22$_x$ (23.69)	77.93$_x$ (31.33)	$F(3, 83) = 0.90$	.41
Months since MVA: M (SD)		66.86$_x$ (92.05)	39.76$_x$ (36.81)	77.95$_x$ (104.51)	$F(2, 61) = 1.18$	.31
Injury Severity Score: M (SD)		13.33$_x$ (12.67)	6.62$_x$ (5.99)	13.18$_x$ (11.17)	$F(2, 61) = 2.90$	.06
Concussion (without/with): n		18/3	17/4	14/8	$\chi^2(2) = 3.25$	.20
Loss of consciousness (without/with): n		11/10	13/8	12/10	$\chi^2(2) = 0.43$	.81
PTSD symptoms (CAPS)						
Total score: M (SD)		6.10$_x$ (4.53)	33.05$_y$ (10.92)	56.45$_z$ (11.26)	$F(2, 61) = 152.37$	<.001
Reexperiencing: M (SD)		1.81$_x$ (2.36)	12.62$_y$ (6.00)	14.86$_y$ (5.48)	$F(2, 61) = 43.20$	<.001
Avoidance: M (SD)		0.57$_x$ (1.33)	5.81$_y$ (3.40)	6.55$_y$ (3.80)	$F(2, 61) = 24.12$	<.001
Numbing: M (SD)		1.57$_x$ (2.94)	2.57$_x$ (3.60)	15.86$_y$ (5.97)	$F(2, 61) = 71.21$	<.001
Hyperarousal: M (SD)		2.14$_x$ (2.26)	12.05$_y$ (6.59)	19.18$_z$ (4.00)	$F(2, 61) = 71.21$	<.001
BDI: M (SD)	3.61$_x$ (4.64)	5.50$_x$ (5.42)[a]	11.45$_y$ (7.76)[a]	22.82$_z$ (9.69)	$F(2, 61) = 73.27$	<.001
STAI–T: M (SD)	35.26$_x$ (7.42)	34.85$_x$ (9.30)[a]	44.45$_y$ (7.82)[a]	54.27$_z$ (10.77)	$F(2, 61) = 23.00$	<.001

Note. Means within columns with the same subscript are not significantly different at $p = .05$ by Tukey's honestly significant difference test. Handedness was measured by the Edinburgh Handedness Inventory, with scores ranging from -100 (strong left-hand preference) to 100 (strong right-hand preference). PTSD = posttraumatic stress disorder; MVA = motor vehicle accident; CAPS = Clinician-Administered PTSD Scale; BDI = Beck Depression Inventory, STAI–T = State–Trait Anxiety Inventory—Trait Scale.
[a] $n = 20$.

assessed with the PANAS state questionnaire. After the resting baselines, four phases of mood induction via picture presentation were conducted. The positive (two bunnies), negative (a barking dog), and neutral (spoon) pictures were taken from the International Affective Picture System (Lang, Öhmann, & Vaitl, 1988). The MVA picture was an actual photograph of a crashed car lying on its roof. The sequence of the picture-presentation phases was counterbalanced between participants. The picture-presentation phases lasted 1 min and were separated by 2-min intervals. At the end of these conditions, participants rated their actual mood using the PANAS state questionnaire. Following this procedure, an event-related potential experiment (not reported here) was administered.

Electrophysiological Recording and Analysis

Electroencephalograms were recorded from 28 scalp placements (Fp1, Fp2, F7, F3, Fz, F4, F8, Fc5, Fc1, Fc2, Fc6, T7, C3, Cz, C4, T8, Cp5, Cp1, Cp2, Cp6, P7, P3, Pz, P4, P8, POz, O1, O2) according to the extended 10/20 system (Pivik et al., 1993) using a stretchable electro cap (Falk Minow Services, Munich, Germany). Moreover, we recorded EEG activity at linked mastoid positions (A1, A2). All sites were referenced to a computer-averaged F3/F4 reference and grounded at AFz. Impedances were maintained below 5 kΩ and within 500 Ω at homologous sites. The EEG signal was recorded by a Nihon Kohden (Tokyo, Japan) amplifier (NeuroFileII system), filtered with a time constant (10 s) and a high-frequency cutoff (300 Hz), digitized online at 1024 Hz, and stored at 256 Hz. Additionally, we recorded electromyographic activity, electrodermal activity, and the electrocardiogram. The data of these measures are not presented here.

A linked-mastoids reference was rederived offline. Physical linking of mastoids has been theoretically criticized for producing a low resistive shunt between the two sides of the head (for discussion, see Hagemann, 2004). However, empirical evidence suggests that there is no difference between physically and computer-averaged ears (Senulis & Davidson, 1989). EEG artifacts (eye blinks and muscle artifacts) were removed by applying an independent components analysis (Jung et al., 2000) to the EEG segments of interest. Prior to artifact screening, an offline bandpass filter (1–30 Hz) was applied. Continuous EEG data were divided offline in 4-s epochs (50% overlap) and again visually inspected for artifacts. The mean percentages of accepted epochs in this study were 93.06 (SD = 10.01) for the baseline eyes-closed condition, 81.01 (SD = 21.20) for the baseline eyes-open condition, 70.21 (SD = 26.53) for the neutral picture, 75.54 (SD = 23.81) for the positive picture, 73.44 (SD = 25.22) for the trauma-related picture, and 76.36 (SD = 25.12) for the negative picture. The percentages of accepted epochs did not differ between diagnostic groups (p > .11). All epochs free of artifacts were subjected to a fast Fourier transformation using a Hamming window over the distal 50% of each epoch. By averaging segments, we derived estimates of spectral power (μV^2) for 0.25-Hz bins, averaged between 8 and 13 Hz, and normalized (natural log) to obtain ln power density (ln μV^2/Hz) in the alpha band (Gasser, Bächer, & Möcks, 1982).

Statistical Analysis

Our analytic strategy to assess alpha asymmetry was to quantify activity recorded over four brain regions: left anterior, right anterior, left posterior, and right posterior. We therefore averaged electrode sites within anterior (left: F3, F7, T7; right: F4, F8, T8) and posterior (left: Cp5, P3, P7; right: Cp6, P4, P8) regions. This approach has the advantage of reducing the amount of data and, according to the Spearman–Brown prophecy formula, increasing reliability of anterior and posterior brain asymmetry measures. The four quadrants of the scalp were included in statistical analyses. To examine the hypothesized asymmetries, we computed repeated measures multivariate analyses of variance (MANOVAs) using Pillai's correction with the within-subject factors hemisphere (left, right) and region (anterior, posterior) and the between-subjects factors group (healthy control, non-

PTSD, subsyndromal PTSD, PTSD) and gender (female, male). For the baseline, the additional within-subject factor baseline condition (eyes open, eyes closed) was used.

To assess brain asymmetry during the emotion conditions, we adopted an approach similar to that of Davidson et al. (2000). To examine emotion-induced activation relative to the neutral condition, we computed neutral-minus-emotion-condition change scores by subtracting alpha activity during the three emotion conditions (positive, negative, trauma-related) from that of the neutral condition. A decrease in alpha power is assumed to reflect increased activity. Thus, positive change scores indicate greater activation during an emotion condition compared with the neutral condition. All effects reported here are based on these change scores. This approach has the advantage of controlling individual differences (e.g., produced by individual differences in skull thickness) in the total amount of recorded alpha power. We investigated differences in alpha-power change scores using MANOVAs that included emotion condition (positive, negative, trauma-related) as an additional within-subject factor. On the basis of our prediction that groups would differ in regional alpha asymmetry during certain conditions, significant interactions involving group, hemisphere, and condition were followed by separate MANOVAs. Furthermore, we conducted simple-effects MANOVAs for each group separately to test our specific predictions of hemispheric (left vs. right) differences between homologous regions.

Similar to Wiedemann et al. (1999), we computed Pearson product–moment correlations between the anterior or posterior (right minus left hemisphere) alpha asymmetry scores and measures of PTSD symptom severity (CAPS total score; PTSD dimensions: reexperiencing, avoidance, numbing, and hyperarousal), self-reported depression (BDI), and PANAS state negative affect and positive affect ratings. These correlations were conducted on an exploratory basis, so two-tailed significances are reported in Tables 4 and Table 6 (presented later). Because the sample size was rather small for correlational analyses, we did not perform corrections for multiple testing so as to avoid the analyses being substantially underpowered. Demographic and state affect variables were assessed using analysis of variance (ANOVA) with Tukey post hoc testing and chi-square tests.

Results

Baseline

Affective ratings. Group × Gender ANOVAs were computed for negative (PANAS–NA) and positive (PANAS–PA) affect scores separately. For PANAS–NA scores, this analysis revealed a significant effect of group, $F(3, 79)$ = 4.18, p < .01, η^2 = .137. Tukey multiple comparison tests indicated that PTSD patients reported significantly more negative affect than did non-PTSD participants (p < .05) and healthy controls (p < .001) but did not differ from patients with subsyndromal PTSD (see Table 2). There were no significant differences between the non-PTSD, healthy control, and subsyndromal PTSD groups (all ps > .27).

For PANAS–PA scores, there was a significant effect of group, $F(3, 79)$ = 4.44, p < .01, η^2 = .144. PTSD patients reported significantly less positive affect than non-PTSD participants (p < .05) and healthy controls (p < .03) but did not differ from subsyndromal PTSD patients (p = .90). The two control groups did not differ in baseline positive affect. There was a tendency for participants with subsyndromal PTSD to show less positive affect than did the non-PTSD participants (p = .08), but the positive affect of subsyndromal PTSD participants was not lower than that of healthy controls (p = .14). No main effects or interactions with gender were revealed for baseline affect.

Electrophysiology. A MANOVA conducted on baseline EEG alpha power (see Table 3 for EEG values) revealed a main

Table 2
Means, Standard Deviations, and Significance Tests for PANAS State Scales of the Diagnostic Groups

| | Group | | | | | | | | One-way ANOVA | |
| | Healthy control | | Non-PTSD with MVA | | Subsyndromal PTSD | | PTSD | | | |
Condition	M	SD	M	SD	M	SD	M	SD	F	p
	PANAS state negative affect									
Baseline	12.74	0.86	13.43	2.29	14.33	2.24	16.23	4.79	5.99	<.001
Neutral picture	12.26	0.86	13.52	3.68	13.62	2.44	13.00	6.18	1.54	.21
Positive picture	12.17	0.83	12.24	0.77	13.19	2.52	12.68	1.73	1.82	.15
Negative picture	21.17	8.85	19.76	7.41	20.19	7.65	22.95	9.14	0.63	.60
Trauma-related picture	23.48	8.47	17.95	4.98	26.57	8.51	32.91	8.41	13.91	<.001
	PANAS state positive affect									
Baseline	36.52	9.26	37.24	7.01	31.43	8.15	29.77	6.31	4.88	.01
Neutral picture	30.30	8.80	30.00	10.52	27.43	9.13	23.68	10.40	1.44	.24
Positive picture	41.52	9.39	38.81	10.31	39.57	9.19	35.27	9.96	1.61	.20
Negative picture	31.35	6.49	26.81	7.59	27.29	8.15	25.68	6.30	2.70	.06
Trauma-related picture	29.48	4.80	26.62	6.14	24.10	3.82	22.95	7.80	10.19	<.001

Note. PANAS = Positive and Negative Affect Schedule; PTSD = posttraumatic stress disorder; MVA = motor vehicle accident; ANOVA = analysis of variance.

effect of baseline condition, $F(1, 79) = 153.47$, $p < .001$, $\eta^2 = .660$, reflecting higher alpha power during the eyes-closed condition. There was a significant main effect of region, $F(1, 79) = 176.65$, $p < .001$, $\eta^2 = .660$, and a significant Region × Baseline Condition interaction, $F(1, 79) = 11.76$, $p < .01$, $\eta^2 = .130$. Alpha power was higher at posterior sites. Furthermore, alpha activity was more reduced at posterior sites in the eyes-open condition than the eyes-closed condition. There was also a main effect of hemisphere, $F(1, 79) = 7.05$, $p < .05$, $\eta^2 = .082$, and a Hemisphere × Region × Baseline Condition interaction, $F(1, 79) = 7.39$, $p < .01$, $\eta^2 = .086$. This reflects lower alpha power (greater activity) over left than over right anterior regions in the eyes-open condition compared with the eyes-closed condition. However, there was no main effect of group or interactions with group. Furthermore, there were no significant interactions with gender of relevance for our hypotheses (i.e., interactions with group or hemisphere).

Correlational analysis. We computed alpha asymmetry scores (right minus left hemisphere) for the anterior and posterior regions on the basis of baseline EEG alpha power (collapsed for eyes-open and -closed conditions). Positive correlations reflect relative increased left hemisphere activation. As can be seen in Table 4, there were no significant associations between trait and state measures and asymmetry scores for the baseline. BDI and PTSD severity (CAPS total score) were highly correlated ($n = 62$; $r = .72$, $p < .001$). It has been hypothesized that depression and anxious arousal may have opposite effects on brain asymmetry (Heller & Nitschke, 1998). We therefore computed partial correlations between anterior–posterior asymmetry scores and PTSD symptoms using the BDI as covariate. Partial correlations between baseline asymmetry and symptoms of PTSD (ranging from −.15 to +.22) were not significant (see Table 4, bottom).

Stimulus Phases

Affective ratings. Repeated measures Group × Gender × Emotion Condition (neutral, positive, negative, trauma-related) ANOVAs were computed for PANAS–NA and PANAS–PA scores separately. For PANAS–NA scores, this analysis revealed significant main effects of group, $F(3, 79) = 4.63$, $p < .01$, $\eta^2 =$

Table 3
Electroencephalograph ln Alpha Power Values for Left and Right Anterior and Posterior Regions During Baseline

| | Healthy control | | | | Non-PTSD with MVA | | | | Subsyndromal PTSD | | | | PTSD | | | |
| | Left | | Right | | Left | | Right | | Left | | Right | | Left | | Right | |
Region	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Anterior	−1.174	0.788	−1.166	0.748	−1.676	1.063	−1.618	1.033	−1.732	0.853	−1.664	0.779	−1.458	0.689	−1.417	0.720
Posterior	−0.685	0.869	−0.616	0.818	−1.185	1.228	−1.091	1.208	−1.252	0.954	−1.209	0.871	−0.935	0.859	−0.875	0.859

Note. Lower alpha scores indicate greater activity. PTSD = posttraumatic stress disorder; MVA = motor vehicle accident; Left = left hemisphere; Right = right hemisphere.

Table 4

Correlations and Partial Correlations of Baseline EEG Alpha Asymmetry Scores (Right Minus Left Hemisphere) for Anterior and Posterior Regions With Symptom Severity Measures and State Affect

Measure	Anterior asymmetry	Posterior asymmetry
	Corellations	
CAPS total score[a]	.07	−.07
Reexperiencing[a]	.02	−.18
Avoidance[a]	−.01	−.01
Numbing[a]	.07	.01
Hyperarousal[a]	.12	−.03
BDI[b]	−.04	−.07
PANAS state NA[c]	.13	.11
PANAS state PA[c]	.08	.08
	Partial correlations (BDI as covariate)	
CAPS total Score[d]	.22	.01
Reexperiencing[d]	.09	−.15
Avoidance[d]	.03	.01
Numbing[d]	.18	.10
Hyperarousal[d]	.21	.07

Note. EEG = electroencephalograph; CAPS = Clinician-Administered PTSD Scale; BDI = Beck Depression Inventory; PANAS = Positive and Negative Affect Schedule; NA = negative affect; PA = positive affect.
[a] $n = 64$. [b] $n = 85$. [c] $n = 87$. [d] $n = 62$.

.149, and emotion condition, $F(3, 79) = 98.43$, $p < .001$, $\eta^2 = .555$, and a significant Group × Emotion Condition interaction, $F(9, 237) = 6.27$, $p < .001$, $\eta^2 = .192$. Figure 1 shows an increase in the self-reported negative affect in response to the trauma-related picture compared with neutral and positive pictures. PTSD patients showed the highest increase in negative affect in response

to the traumatic picture. The group differences in the PANAS–NA ratings were not significant in post hoc one-way ANOVAs for the neutral, positive, and negative pictures (see Table 2). However, for the trauma-related picture, there was a large group difference, $F(3, 79) = 13.91$, $p < .001$, $\eta^2 = .334$, with PTSD patients reporting significantly more negative affect than non-PTSD participants ($p < .001$), healthy controls ($p < .001$), and subsyndromal PTSD patients ($p < .05$). Furthermore, patients in the subsyndromal PTSD group rated their mood after the trauma-related picture significantly more negatively than did non-PTSD participants with MVA ($p < .01$) but not healthy controls ($p = .56$), who showed a tendency to display more negative affect than did non-PTSD participants ($p = .09$).

For PANAS–PA scores, this analysis revealed significant main effects of group, $F(3, 79) = 3.88$, $p < .05$, $\eta^2 = .124$, and emotion condition, $F(3, 79) = 70.31$, $p < .001$, $\eta^2 = .462$, but no significant Group × Emotion Condition interaction ($p = .57$). The positive picture elicited more positive affect than did the other stimulus types. Nontraumatized healthy controls showed the most positive affect, whereas PTSD patients showed the least. No main effects or interactions with gender occurred for PANAS–NA or PANAS–PA.

Electrophysiology. A Group × Gender × Hemisphere × Region × Emotion Condition MANOVA using EEG alpha power change scores (neutral minus emotion condition) as dependent variables (see Table 5 for EEG values) revealed a significant Group × Hemisphere × Emotion Condition interaction, $F(6, 158) = 2.19$, $p < .05$, $\eta^2 = .077$, and a Group × Gender × Hemisphere × Region × Emotion Condition interaction, $F(6, 158) = 2.27$, $p < .05$, $\eta^2 = .079$. To further explore the group differences in alpha activity, we computed separate Group × Gender × Hemisphere × Region MANOVAs for each condition. For the positive condition, there were no significant main effects or interactions with group (all ps $> .22$). For the negative condition,
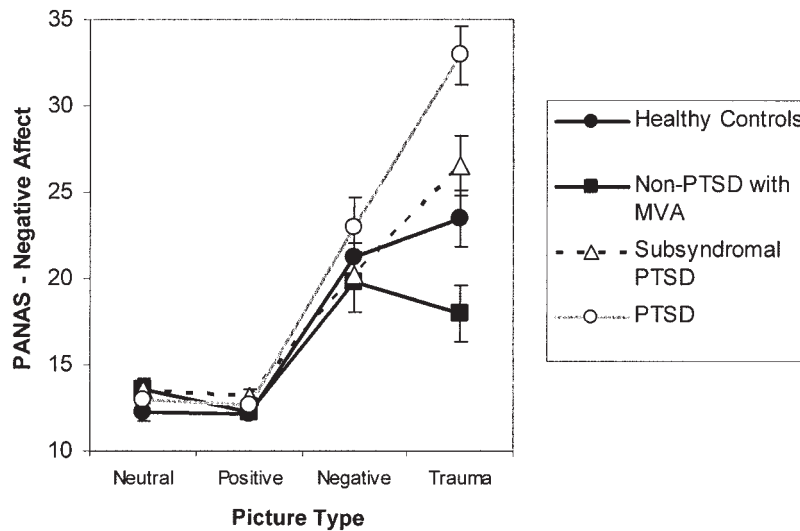

Figure 1. Mean state negative affect as assessed by the Positive and Negative Affect Schedule (PANAS; Krohne et al., 1996) during picture presentation in healthy controls, non-PTSD controls with MVA, patients with subsyndromal PTSD, and patients with full PTSD. Error bars represent standard errors of the mean. PTSD = posttraumatic stress disorder; MVA = motor vehicle accident.

Table 5

Electroencephalograph (EEG) Alpha Power Change Scores (Neutral Minus Emotion Condition) for Left and Right Anterior and Posterior Regions

	Healthy control				Non-PTSD with MVA				Subsyndromal PTSD				PTSD			
	Left		Right		Left		Right		Left		Right		Left		Right	
Picture type and region	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Positive																
Anterior	.234	.373	.238	.408	.090	.697	.092	.790	−.198	.371	−.182	.502	.121	.553	.059	.444
Posterior	.145	.344	.123	.354	.132	.799	.075	.691	−.098	.297	−.087	.455	.073	.467	.081	.354
Negative																
Anterior	.091	.378	.095	.414	.159	.531	.073	.431	.083	.510	−.013	.386	.023	.460	.047	.711
Posterior	−.007	.527	.053	.485	.190	.548	.174	.456	−.018	.418	.045	.347	−.021	.431	−.099	.352
Trauma-related																
Anterior	.208	.350	.128	.302	.181	.402	.015	.334	.192	.450	.293	.469	.272	.617	.403	.430
Posterior	.184	.326	.183	.352	.198	.368	.101	.256	.041	.412	.215	.355	.197	.499	.315	.369

Note. Because EEG alpha power is inversely related to activity, positive change-score values (neutral minus emotion condition) denote increased activation during exposure to the positive, negative, or trauma-related picture. PTSD = posttraumatic stress disorder; MVA = motor vehicle accident; Left = left hemisphere; Right = right hemisphere.

again no significant main effects or interactions with group were revealed (all $ps > .27$). However, as predicted, for the trauma-related condition there was a significant Group × Hemisphere interaction, $F(3, 56) = 9.55$, $p < .001$, $\eta^2 = .338$, and a Group × Gender × Hemisphere × Region interaction, $F(3, 79) = 3.97$, $p < .05$, $\eta^2 = .131$. The Group × Hemisphere interaction was significant for both anterior, $F(3, 79) = 3.46$, $p < .05$, $\eta^2 = .116$, and posterior, $F(3, 79) = 4.25$, $p < .01$, $\eta^2 = .116$, regions. Post hoc simple-effects MANOVAs conducted for each group separately revealed for the anterior region marginally significant hemisphere effects for the PTSD group, $F(1, 21) = 3.23$, $p < .09$, $\eta^2 = .133$, and the subsyndromal PTSD group, $F(1, 20) = 3.45$, $p < .08$, $\eta^2 = .147$, indicating greater relative right-sided activation (see Figure 2). In contrast, the non-PTSD group showed the opposite pattern of increased relative left anterior activation, $F(1, 20) = 11.18$, $p < .01$, $\eta^2 = .359$, which was not significant for the healthy control group, $F(1, 22) = 2.37$, $p = .14$. For the posterior region, post hoc simple-effects MANOVAs showed greater right than left activation in the PTSD group (simple hemisphere effect), $F(1, 21) = 4.82$, $p < .05$, $\eta^2 = .187$, and the subsyndromal PTSD group, $F(1, 20) = 6.15$, $p < .05$, $\eta^2 = .235$, and no significant hemispheric difference in the non-PTSD group, $F(1, 20) = 2.42$, $p = .14$, or the healthy control group, $F(1, 22) = 0.00$, $p = .99$.

To explore gender differences in brain electrical asymmetry during the trauma-related condition, we computed separate MANOVAs for anterior and posterior regions. There was no Group × Gender × Hemisphere interaction for the anterior region ($p = .70$) but there was for the posterior region, $F(3, 79) = 3.37$, $p < .03$, $\eta^2 = .113$. Follow-up Gender × Hemisphere MANOVAs conducted for each diagnostic group separately revealed that there was a significant Gender × Hemisphere interaction for healthy controls, $F(1, 21) = 12.92$, $p < .01$, $\eta^2 = .381$, but not for the three MVA groups (all $ps > .19$). Healthy women showed a pattern of right-sided posterior activation, whereas healthy men showed the opposite pattern.[1]

Correlational analysis. We computed alpha asymmetry scores (right minus left hemisphere) for the anterior and posterior regions on the basis of EEG alpha power change scores (neutral minus emotion condition). Positive correlations reflect relative increased right hemisphere activation. As can be seen in Table 6, significant correlations were found for only the trauma-related condition between anterior asymmetry and overall PTSD severity (CAPS total score) and the three PTSD dimensions of reexperiencing, avoidance, and hyperarousal as well as the BDI. Furthermore, we observed positive associations of posterior asymmetry for the trauma-related condition with PTSD severity (CAPS total score), reexperiencing, and hyperarousal. In addition, for the trauma-related condition only, there were significant positive correlations between increased negative affect (PANAS–NA) and anterior asymmetry as well as posterior asymmetry. Higher negative affect was associated with greater relative right hemisphere activation during the trauma-related condition. Finally, PANAS–PA was negatively related to posterior asymmetry.

To assess whether the relationship of PTSD symptoms with relative right hemisphere activation during the trauma-related condition was specific to the PTSD symptoms, we computed partial correlation coefficients, partialing out BDI depression (see Table 6, bottom). Again, for the trauma-related condition, there were significant partial correlations between anterior asymmetry and

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[1] To rule out the possibility that gender was confounding our results, we repeated the analyses for the trauma-related condition using female participants only ($n = 60$). Again, there was a significant Group × Hemisphere interaction, $F(3, 56) = 9.55$, $p < .001$, $\eta^2 = .338$, whereas the Group × Hemisphere × Region interaction failed to reach significance, $F(3, 56) = 2.15$, $p = .11$. For the anterior region, there was a significant Group × Hemisphere interaction, $F(3, 56) = 6.25$, $p < .001$, $\eta^2 = .251$. Post hoc simple-effects MANOVAs conducted for each group separately revealed greater right than left anterior activation for women with full PTSD or subsyndromal PTSD (both $ps < .05$) and greater left than right anterior activation for the non-PTSD ($p < .05$) and healthy control groups ($p < .10$). For the posterior region, there was a significant Group × Hemisphere interaction, $F(3, 56) = 6.32$, $p < .001$, $\eta^2 = .253$, indicating greater right than left hemisphere activation for the PTSD, subsyndromal PTSD, and healthy control groups (simple hemisphere effects; all $ps < .05$) and greater relative left hemisphere activation in the non-PTSD group ($p < .05$).
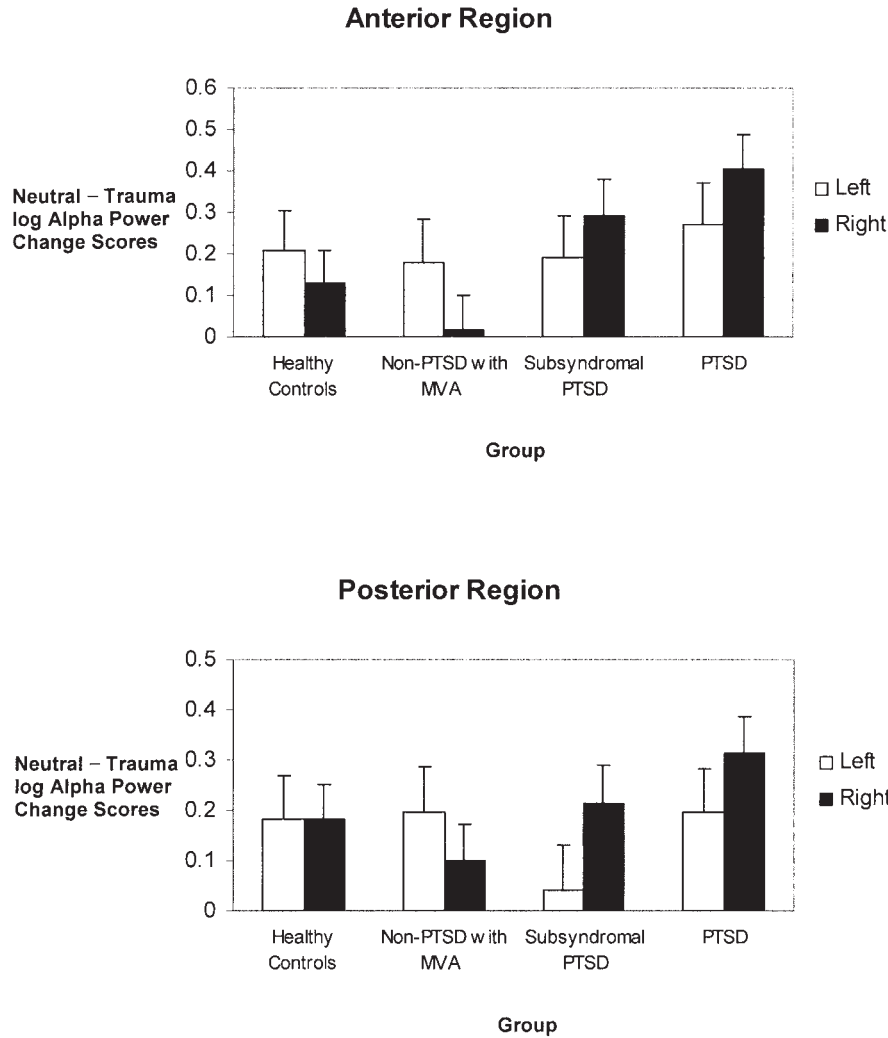
Anterior Region

Posterior Region

Figure 2. Electroencephalograph (EEG) alpha power change scores (neutral minus trauma condition) for the left and right anterior and posterior regions in healthy controls, non-PTSD controls with MVA, patients with subsyndromal PTSD, and patients with full PTSD. Because EEG alpha power is inversely related to activity, positive change score values denote increased activation during exposure to the trauma-related picture. Error bars represent standard errors of the mean. PTSD = posttraumatic stress disorder; MVA = motor vehicle accident; Left = left hemisphere; Right = right hemisphere.

overall PTSD severity as well as the PTSD dimension reexperiencing. Posterior asymmetry was associated with overall PTSD severity, reexperiencing, and hyperarousal even when BDI depression was controlled for statistically.

The Role of Concussion and Loss of Consciousness

To address the question whether reversible neurological trauma in our MVA sample could have affected either baseline EEG or EEG responses to the experimental stimuli, we repeated the above analyses comparing the four groups using only MVA participants without concussion and loss of consciousness. The findings were essentially the same as for the total sample. Again, only for the trauma-related condition was there a significant Group $\times$ Hemisphere interaction, $F(3, 52) = 3.54$, $p < .05$, $\eta^2 = .169$.

The Role of Comorbid Depression

It was important to rule out possible effects related to the presence of comorbid depression. Therefore, we collapsed subsyndromal PTSD patients with comorbid MDD ($n = 4$) and PTSD patients with MDD ($n = 9$) or dysthymia ($n = 2$) as one group (subsyndromal–full PTSD + MDD; $n = 15$). PTSD patients and the subsyndromal PTSD without comorbid depression were also collapsed as one group (subsyndromal–full PTSD − MDD; $n = 28$). Using the group variable (healthy controls, non-PTSD controls with MVA, subsyndromal–full PTSD − MDD, subsyndromal–full PTSD + MDD), we repeated the above analyses for baseline and emotion conditions. Again, only for the trauma-related condition was there a significant Group $\times$ Hemisphere interaction, $F(3, 83) = 7.48$, $p < .001$, $\eta^2 = .213$. To

Table 6

Correlations and Partial Correlations of EEG Asymmetry (Right Minus Left Hemisphere) Based on Alpha Power Change Scores (Neutral Minus Emotion Condition) for Anterior and Posterior Regions With Symptom Severity Measures and State Affect

Measure	Positive picture		Negative picture		Trauma-related picture	
	Anterior asymmetry	Posterior asymmetry	Anterior asymmetry	Posterior asymmetry	Anterior asymmetry	Posterior asymmetry
Corellations						
CAPS total score[a]	.09	.21	.05	.18	.44**	.37**
Reexperiencing[a]	.06	.24	.04	.22	.48**	.47**
Avoidance[a]	.15	.03	.03	.00	.34*	.19
Numbing[a]	.09	.13	.07	.11	.19	.11
Hyperarousal[a]	.02	.21	.03	.16	.41**	.38**
BDI[b]	.10	.14	.07	.16	.29**	.15
PANAS state NA[c]	.16	−.08	−.12	−.01	.35**	.26**
PANAS state PA[c]	−.08	.00	.17	.08	−.15	−.24*
Partial correlations (BDI as covariate)						
CAPS total score[d]	−.08	.04	−.05	.04	.28*	.35**
Reexperiencing[d]	−.03	.13	−.02	.17	.38**	.46**
Avoidance[d]	.12	−.07	−.04	−.11	.22	.15
Numbing[d]	−.05	−.04	−.02	−.04	−.07	−.04
Hyperarousal[d]	−.08	.04	−.08	.02	.25	.35**

Note. EEG = electroencephalograph; CAPS = Clinician-Administered PTSD Scale; BDI = Beck Depression Inventory; PANAS = Positive and Negative Affect Schedule; NA = negative affect; PA = positive affect.
[a] $n = 64$. [b] $n = 85$. [c] $n = 87$. [d] $n = 62$.
* $p < .05$. ** $p < .01$.

directly test the hypotheses that PTSD groups with and without depression are characterized by distinct hemispheric asymmetries, we compared the two groups. We found no statistical difference in brain asymmetry, as revealed by nonsignificant Group × Hemisphere and Group × Hemisphere × Region interactions (all $ps >$ .47). Even when the 4 PTSD patients with lifetime but not current major depression were included in the PTSD + MDD group, the results did not change.

Other Frequency Bands

We computed power for the three other traditional frequency bands: delta (1–4 Hz), theta (4–8 Hz), and beta (13–30 Hz). We repeated analyses for baseline and emotion conditions using the same MANOVAs as for the alpha band. Because we had no specific hypotheses for effects in these three bands, we reduced the likelihood of a Type I error by means of Bonferroni adjustment to an alpha of $p < .017$ (.05/3). There were no significant interactions with or main effects of group (all $ps > .06$). Neither of the key interactions Group × Hemisphere × Condition and Group × Hemisphere × Region × Condition was significant (both $ps >$ .16).

Discussion

This study examined hemispheric asymmetries among MVA survivors with PTSD, with subsyndromal PTSD, and without PTSD as well as nonexposed healthy controls during baseline and in response to neutral, positive, negative, and trauma-related pictures. In accordance with our hypothesis, both PTSD and subsyn-dromal PTSD patients displayed a pattern of increased right-sided activation in anterior and posterior regions during exposure to a trauma-related picture as compared with non-PTSD and healthy controls. This pattern of brain asymmetry in PTSD and subsyn-dromal PTSD was accompanied by increased negative affect. No significant Group × Hemisphere interactions were observed for the other emotion conditions or the resting baseline condition. Correlational analysis revealed that for the trauma-related condition only, relative right hemispheric anterior and posterior activation was associated with greater total PTSD severity and the PTSD dimensions reexperiencing, avoidance (only for anterior asymmetry), and hyperarousal. Furthermore, most of the correlations remained significant after BDI depression was controlled for statistically. The pattern of right hemisphere activation during exposure to the trauma-related picture might in part be explained by increased negative affect given the positive association of brain asymmetry and PANAS–NA ratings. Finally, we ruled out that our results were attributable to reversible accident-related neurological trauma.

Contrary to our hypothesis, we observed no group differences in EEG alpha activity during the baseline condition, although there were group differences in negative affect during this period. However, the absolute differences in baseline affect were rather small. This finding was unexpected, because previous research (Metzger et al., 2004) had led us to anticipate that posterior baseline asymmetry would be associated with PTSD hyperarousal. However, our results are in accord with studies showing that PTSD patients after severe MVA display no heightened baseline psychophysiologic activity (e.g., heart rate) but increased psychophysiologic respon-

siveness to trauma-related stimuli (Blanchard, Hickling, Buckley, et al., 1996; Blanchard, Hickling, Taylor, & Loos, 1994). Our results indicate that MVA-related PTSD is not necessarily related to a trait asymmetry.

The pattern of increased right anterior activation during the trauma-related condition in patients with PTSD and subsyndromal PTSD was similar to that previously reported during the presentation of anxiety-provoking stimuli in participants with social phobias (Davidson et al., 2000), patients with panic disorders (Wiedemann et al., 1999), and Vietnam War veterans with PTSD (McCaffrey et al., 1993). This pattern is in accord with positron emission tomography findings in PTSD (Rauch et al., 1996) and PTSD among other anxiety disorders (Rauch, Savage, Alpert, Fischman, & Jenike, 1997) reporting patterns of asymmetrical brain activation. The results support Davidson's (1995) model, in which right anterior activation is associated with the activation of an avoidance–withdrawal system. The findings are also in accord with the model of Heller and colleagues (e.g., Heller & Nitschke, 1998), which assumes that negatively valenced emotions should be associated with relative right anterior activation. Our findings of increased relative right posterior activation in PTSD and subsyndromal PTSD is consistent with recent studies reporting right posterior activation during anxiety-provoking situations in anxious participants (Davidson et al., 2000; Heller et al., 1997). It has been hypothesized that right posterior activation is associated with anxious arousal in contrast to anxious apprehension, which should be associated with increased left anterior activation (Heller et al., 1997). This underscores the fact that patients with full and subsyndromal PTSD may be characterized not by anxious apprehension but, rather, by anxious arousal when exposed to trauma-related stimuli. If the present results can be replicated and generalized to other trauma populations, this would indicate that EEG alpha asymmetry may be used to discriminate different types of pathologic anxiety conditions. Recent functional neuroimaging studies have shown that emotionally laden visual stimuli elicit increased activation in posterior cortical regions (Lane, Chua, & Dolan, 1999; Lang et al., 1998). Nitschke, Heller, and Miller (2000) proposed a right hemisphere system that is involved in visual attention, orientation, and response to threat associated with anxiety. This could be an important component of a system mediating symptoms of PTSD-like increased hypervigilance and physiological reactivity to traumatic cues.

The association between the PTSD dimension reexperiencing and greater relative right hemisphere activation during exposure to the trauma-related picture indicates a link to maladaptive information processing and memory for traumatic events. Cognitive theories of PTSD (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000) propose that reexperienced aspects of traumatic events are very vivid, mostly visual and emotional. These trauma memories are very different from recall of ordinary autobiographical memories, in which sensory elements are integrated into a personal narrative and which seems to be primarily dependent on the left hemisphere (Maguire, 2001). However, further research is needed to explore the possible mechanisms underpinning the association of brain asymmetry and specific symptoms of PTSD.

The pattern of brain asymmetry during exposure to trauma-related material was similar between PTSD and subsyndromal PTSD patients. This was expected, because patients with subsyndromal and full PTSD displayed comparable symptom severity in

the PTSD dimensions reexperiencing, avoidance, and (to a lesser degree) hyperarousal. In contrast, psychological numbing, the dimension in which there was a great difference between patients with subsyndromal and full PTSD, was not significantly related to brain asymmetry. However, it is worth noting that the relative right posterior activation in subsyndromal PTSD group was driven by a decrease in left posterior activity. Our results warrant further research on brain mechanisms related to subsyndromal PTSD, because subsyndromal PTSD patients may suffer chronically from PTSD symptoms (e.g., Blanchard, Hickling, Barton et al., 1996).

The opposite pattern of relative left hemisphere activation during exposure to the trauma-related picture (accompanied by the lowest negative affect) was observed in MVA survivors who had recovered from trauma (non-PTSD). This might reflect more adaptive tendencies to process trauma-related information in an approach-related (vs. avoidance-related), low anxious–aroused, and verbally integrated manner. Relative left frontal hemisphere activation has been proposed to be associated with a self-enhancing regulatory style inhibiting negative affective responses (Tomarken & Davidson, 1994). The question of whether the pattern of brain asymmetry in non-PTSD with MVA participants is related to a specific type of coping should be explored in further studies.

Nontraumatized healthy controls displayed a pattern of more symmetrical brain activation. This group reacted with more negative affect to the trauma-related picture than did non-PTSD with MVA controls. Furthermore, only in healthy controls did posterior hemispheric asymmetry in response to the trauma-related picture varied with gender. Healthy women showed a pattern of relative right posterior activation similar to that of patients with PTSD and subsyndromal PTSD, in contrast to healthy men, who showed the opposite pattern in the absence of gender differences in affect ratings. This finding was not predicted, and we do not have a clear explanation for it. However, this gender effect in healthy controls is consistent with a prior report on healthy participants indicating gender differences in posterior asymmetry in response to emotional tasks in the absence of sex differences in subjective ratings (Davidson, Schwartz, Pugash, & Bromfield, 1976). However, it has to be mentioned that our analysis of gender was clearly exploratory given the low proportion of men in our sample (and especially the PTSD group). Recent research has shown that gender might be an important variable for the understanding of associations of brain asymmetry with coping (Kline, Allen, & Schwartz, 1998) and depression (Miller et al., 2002). Therefore, future studies of the potential influence of gender differences on brain asymmetry are clearly warranted.

Post hoc analyses comparing groups with and without comorbid depression showed that EEG alpha asymmetries during baseline and other emotion conditions were not modulated by comorbid depression. When we statistically controlled for symptoms of BDI depression by means of partial correlations, most of the observed relations between PTSD symptoms and brain asymmetry for the trauma-related condition remained significant. This does not support the hypothesis that comorbid depression should attenuate right posterior activation (Heller & Nitschke, 1998). However, our results are in accord with research showing that depression with comorbid anxiety differs from depression without anxiety in measures of EEG asymmetry (Bruder et al., 1997; Kentgen et al., 2000).

Some remarks have to be made concerning our methodology. In the current study, we used pictures. The use of trauma-related pictures has several advantages over other techniques for mood induction in PTSD patients. Unlike personalized scripts, pictures can be presented in an identical fashion to all participants. Furthermore, pictures have a greater resemblance to traumatic triggers that patients encounter in their environment. Even if they are standardized, stimuli like pictures with only vague physical similarity to the traumatic situation can serve as triggers for reexperiencing symptoms (poor stimulus discrimination; Ehlers & Clark, 2000). However, the use of only one stimulus per emotion category may impair generalizability of our results. Furthermore, our assessment of negative affect (PANAS) does not allow us to conclude what discrete emotion or affective state was elicited by the trauma-related picture. Given the relatively low association between negative affect and EEG alpha asymmetry during the trauma-related condition, an increase in general negative affect alone is unlikely to account for increased right-sided activity. Future studies are needed to index what cognitive or affective processes are linked to hemispheric EEG activation in PTSD.

A limitation of the current study is the poor spatial resolution of scalp-recorded electrophysiology. Studies using neuroimaging techniques with better spatial resolution are needed to confirm our findings. However, the EEG can provide useful information about hemispheric differences in broad regions of the cortex, and it has the advantage of being relatively inexpensive and completely noninvasive making it ideally suitable for studies with large samples, especially patient populations.

In the current study, we included only traumatized persons with MVA. Therefore, the results may not be generalizable to other trauma populations. The differences between the two control groups in affective and brain asymmetry responses to trauma-related stimuli underscore the need to include nontraumatized controls in PTSD research.

In summary, persons with PTSD and subsyndromal PTSD demonstrated increased right-sided anterior and posterior activation only during exposure to a trauma-related picture but not during baseline and other emotion conditions. These findings suggest that PTSD may be linked to a context-dependent, trauma-specific alteration in hemispheric processing that is associated with anxious arousal and symptoms of PTSD.

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