RESTING EEG ASYMMETRY
AND SPIDER PHOBIA

HARALD MERCKELBACH*, PETER MURIS*,
KARIN POOL* and PETER J. DE JONG*

*Department of Psychology, **Department of Experimental Abnormal
Psychology, Maastricht University, P.O. Box 616, 6200 MD,
Maastricht, The Netherlands

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This study examined whether resting EEG asymmetries are related to symptom
severity and treatment outcome in spider phobia. Prior to treatment, EEG was
recorded in a sample of spider phobic patients (N = 16). Correlations between frontal
and parietal asymmetries in alpha power, on the one hand, and pre- and post-treat-
ment symptom measures, on the other hand, were then computed. Only relative right
parietal hyperactivation was found to be related to higher pre-treatment spider phobia
scores. No convincing correlations between EEG asymmetry and post-treatment
outcome measures were found. The findings suggest that cognitive processes mediated
by the right hemisphere may modulate pre-treatment phobic symptoms.

Keywords: Spider phobia; Treatment effects; EEG asymmetry; Alpha power

There is growing consensus among researchers that the right hemi-
sphere plays a predominant role in negative emotions. Evidence that
supports this idea comes from three sources. To begin with, neuro-
psychological studies on the behavioral sequelae of unilateral lesions
show that left-hemisphere damage is often followed by catastrophic
and depressive reactions that can be attributed to the intact right
hemisphere (Robinson & Downhill, 1995). Secondly, using visual

* Corresponding author. Fax: +31-43-3615735.
half-field techniques, a number of studies have found that the right hemisphere of healthy subjects is more sensitive to negative emotional material (e.g., Wittling, 1995) and aversive conditioning (e.g., Johnsen & Hugdahl, 1993) than their left hemisphere. Thirdly, EEG studies by Davidson, Fox, and co-workers (see, for reviews, Davidson, 1992a,b; Fox, 1991) indicate that in normal subjects, negative emotions such as disgust, fear, and depression are accompanied by a stronger right-hemisphere than left-hemisphere activation. In general, these studies suggest that asymmetrical EEG patterns during fear and dysphoria reflect a state-like involvement of the right hemisphere in negative emotions. Meanwhile, there are also good reasons to believe that right-hemisphere hyperactivation, especially in the frontal areas, is a trait-like characteristic that reflects susceptibility to avoidance-related behavior. Germaine to this issue is a study by Davidson and Fox (1989) which demonstrated that young infants with a strongly activated right anterior hemisphere tend to react with crying to subsequent maternal separation. Likewise, Tomarken, Davidson, and Henriques (1990) found that increased relative right-hemisphere activation during rest predicts the extent to which healthy adults react with fear and disgust to emotional film clips. The idea that resting EEG asymmetries tap a stable temperamental factor is further supported by the finding that such EEG parameters show good internal consistency (Lund, Sponheim, Iacono, & Clemen, 1995) and test–retest stability (Tomarken, Davidson, Wheeler, & Kinney, 1992).

Given the prominent role of fear, avoidance, and disgust (Mulkins, de Jong, & Merckelbach, 1996) in spider phobia, the question arises whether resting EEG asymmetries would predict severity and treatment outcome of this type of phobia. How would resting EEG asymmetry be linked to phobic symptomatology? At least two possibilities suggest themselves. The first can be inferred from the work of Davidson (1992a,b) and Fox (1991) who argue that the behavioral dimension of avoidance versus approach is mediated by the right and the left hemisphere, respectively. More specifically, these authors propose that the right anterior areas mediate avoidance-related behavior, while the left anterior areas sustain approach behavior. Between-subject variations in right-hemisphere resting EEG activation would reflect individual differences in thresholds for
avoidance behavior (e.g., Tomarken et al., 1990). There is, indeed, strong evidence that especially frontal asymmetries in resting EEG predict individual differences in emotional reactivity, avoidance-related behavior, and temperament (Davidson, 1992a,b). Following this line of reasoning, one would expect that the greater the habitual right frontal as compared to left frontal activation of phobic subjects, the stronger their avoidance reactions during subsequent exposure treatment and the poorer their treatment outcome. In this context, it is important to emphasize that behavioral exposure treatment consists mainly of confrontation with the feared object. Consequently, on the basis of the findings of Davidson and colleagues (e.g., Davidson & Fox, 1989; Tomarken et al., 1992), one would anticipate that in phobics with a greater relative activation of the right anterior areas, such emotional provocation elicits intense avoidance tendencies that might undermine treatment effects.

Another possible link between phobic responding and hemisphere asymmetry is suggested by regional Cerebral Blood Flow (rCBF) studies. These studies have consistently shown that phobic patients are characterized by elevated blood flow in the secondary visual areas (e.g., Wik, Fredrikson, Erickson, Eriksson, Stone-Elander, & Greitz, 1993). This finding has been related to visual scanning for threat. It should be noted, though, that individual differences in left-right asymmetries were not the primary object of these rCBF studies. Also, the increased rCBF in secondary visual areas has been observed during acute fear states. More relevant to the issue under consideration is a recent study by Heller, Etienne, and Miller (1995; see also Heller, Nitschke, & Lindsay, 1997). These authors found that high trait-anxious students display a left hemispatial bias, suggesting that trait anxiety is accompanied by relatively higher levels of right parietotemporal activity. Heller et al. (1995) proposed that the right parietotemporal system "is differentially involved in an arousal component of emotion, specifically with regard to self-reported arousal and the regulation of autonomic functions" (p. 331). In light of this, it would be interesting to examine whether within a sample of phobic subjects, differences in resting posterior (i.e., parietal) EEG activation are associated with symptom severity and treatment outcome (see also Merkelbach, de Jong, Muris, & van den Hout, 1996).
In the present study, resting midfrontal and parietal EEG of spider phobic subjects was recorded under baseline conditions (i.e., in the absence of the phobic object). Pre-treatment data on fear and avoidance of spiders were also collected. Following this, the patients underwent behavioral treatment and post-treatment fear and avoidance data were obtained. The major aim of the study was to explore whether within a sample of phobic patients, midfrontal and parietal asymmetries are related to pre-treatment and/or post-treatment anxiety measures.

**METHOD**

**Participants**

Participants were 16 spider phobic women who applied for treatment at the Spider Phobia Project because their phobia interfered with daily life. Mean age was 27.3 years (range: 19–41 years). All patients met DSM-IV criteria for specific phobia (animal type). Subjects participated in the experiment in return for free treatment.

**Procedure**

Several weeks before treatment, subjects underwent an EEG recording session. They were explicitly told that they would not be exposed to spiders. Subjects were instructed to relax and close their eyes for a 45-sec period. During this period, background EEG activity was recorded using a stretchable cap (ECI Electro-cap) in which electrodes were imbedded. Electrodes were arranged in keeping with the international 10–20 system. EEG was recorded from the left and right midfrontal (F3, F4) and parietal (P3, P4) sites. Recording sites were prepared by scratching the skin surface with a sterile, blunted needle. Following this, electrodes were filled with gel (ECI Electro-Gel) and impedance of each electrode was checked. The left earlobe (A1) was used as a reference site. Beckman miniature Ag-AgCl electrodes (2mm) attached above and below the left eye were employed to monitor eye movements and blinks (EOG). All electrode impedances were well below 10 K ohms. A Picker-Schwarzer ED 14 coupler was used for recording EEG and EOG. The signals were passed through a low pass (70 Hz) and a high pass filter (0.26 Hz) and
sampled at 1000 Hz by a Compaq computer. A 50 Hz notch filter was active while recording.

After approximately 40 days, subjects returned to the lab and received behavioral treatment. More specifically, subjects were treated with a one-session exposure program lasting for 2.5 h. This treatment technique has been extensively described by Öst (1989). Basically, it consists of a hierarchically structured and prolonged confrontation with the phobic object as well as modelling by the therapist. This method yields good immediate and long-term results in the case of specific phobias (Öst, Salkovskis, & Hellström, 1991). Immediately before and after treatment, subjects completed the Spider Phobia Questionnaire (SPQ; Klorman, Weerts, Hastings, Melamed, & Lang, 1974) and engaged in a Behavioral Approach Task (BAT). The SPQ is a widely used 31-item self-report instrument measuring subjective fear of spiders. A typical SPQ item is: "I dislike looking at pictures of spiders in a magazine". The SPQ has adequate psychometric properties. For example, in their study on the SPQ, Muris and Merckelbach (1996) reported a Cronbach alpha of 0.91 and a 3-week test–retest stability of 0.94. High scores on this instrument indicate a strong fear of spiders. The BAT is carried out as follows. Subjects are asked to approach a live spider in a glass jar in a step-wise fashion. There are 8 steps, ranging from 1 (walk towards the spider as near as you can) to 8 (let the spider walk over your hand). The steps are introduced by a research assistant and the phobic patient is free to refuse each step (see for details, de Jong, Visser, & Merckelbach, 1996).

**EEG Analysis and Quantification**

EEG recordings were scored off-line with the help of a specially developed software program. First, using the EOG information, the EEG was visually inspected and edited for eye-movement artifacts. The average length of artifact-free EEG was 23.5 s (range: 20–28 s). The artifact-free EEG was then filtered with a 65 Hz low pass FIR filter to remove additional high frequency artifacts. Following this, 4 s chunks were extracted with overlapping Hamming Windows. For each chunk, a Fast Fourier Transform was used to assess power density ($\mu$V$^2$/Hz) in the alpha band (8–13 Hz). The resulting values were averaged to obtain a grand mean of alpha power density for each recording.
site (F3, F4, P3, P4). Alpha power is known to be inversely related to hemisphere activation, i.e., higher alpha power indicates lower activation (e.g., Davidson, 1992a,b). Finally, to obtain an index of asymmetry, difference scores were computed for midfrontal and parietal recording sites (i.e., F3 minus F4 and P3 minus P4, respectively; see Tomarken et al., 1992). Note that higher values of these asymmetry measures indicate greater relative right-hemisphere activation. Correlations between asymmetry measures and pre- and post-treatment SPQ and BAT scores were then computed.

RESULTS

The average pre-treatment SPQ score of this sample of spider phobics was 23.6 (SD = 3.0), which is somewhat higher than the mean SPQ score that Öst et al. (1991) report for their sample of clinical spider phobics. The pre-treatment BAT score was 3.5 (SD = 1.7), which comes close to step 4, i.e., “take the jar in your hands”. Overall, the one-session exposure was effective in that SPQ scores dropped from pre- to post-treatment [t(15) = 8.2, p < 0.01], while BAT scores increased [t(15) = −11.5, p < 0.01]. Mean post-treatment scores of SPQ and BAT were 9.9 (SD = 6.6) and 7.8 (SD = 0.5), respectively.

Mean alpha power density estimates (μV^2) for F3, F4, P3, and P4 were 0.31 (SD = 0.23), 0.35 (SD = 0.24), 0.91 (SD = 0.58), and 1.17 (SD = 0.68), respectively. Paired t tests showed that alpha power values were significantly lower for left frontal and left parietal than for right frontal and right parietal sites [t(15) = −3.8, p < 0.01 and t(15) = −3.1, p < 0.01, respectively].^1

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^1 While the present study focused on the within-subjects connections between resting EEG asymmetries and phobic symptomatology, resting EEG data were also obtained from a control group as a check on the quality of the power density values. Thus, among the phobic patients, a small sample of non-phobic women (N = 7; mean age = 24.1 years) was tested. Mean alpha power density values (μV^2 in this control sample for F3, F4, P3, and P4 were 0.40 (SD = 0.21), 0.46 (SD = 0.26), 1.29 (SD = 1.07), and 1.18 (SD = 1.23), respectively. Separate 2 (group: phobics versus controls) × 2 (asymmetry: left versus right) Analyses of Variance (ANOVAs) for frontal and parietal recording sites revealed no significant main effects of group: all F(1, 21) < 1.0. The only effect reaching significance was the main effect of asymmetry for the frontal recording sites [(F(1, 21) = 16.1, p < 0.01), due to overall lower alpha power at the left than at the right frontal areas.
Table I shows Pearson product–moment correlations between EEG asymmetry measures and pre- and post-treatment SPQ and BAT. As can be seen, there were no significant correlations between frontal asymmetry, on the one hand, and SPQ or BAT measures, on the other hand. It should be noted, though, that the correlation between frontal asymmetry and pre-treatment BAT just fell short of the conventional level of significance \( r = -0.31, p = 0.06, \) one-tailed). That is, there was a tendency for higher right frontal activation to be associated with lower pre-treatment BAT score (i.e., stronger avoidance behavior). Parietal asymmetry was linked to pre-treatment SPQ \( r = 0.58, p < 0.01, \) one-tailed). That is, the stronger the right parietal activation, the higher pre-treatment self-reported fear.

Two additional analyses were carried out. First, in most studies on hemisphere EEG asymmetries, mean alpha power is subjected to log transformations in order to normalize the data (e.g., Tomarken et al., 1990). Therefore, power density values for F3, F4, P3, and P4 were log-transformed and the scores thus obtained were entered into the hemisphere asymmetry subtractions (i.e., \( F3–F4 \) and \( P3–P4 \)). Next, correlations were computed with pre- and post-treatment SPQ and BAT. By and large, this yielded a similar correlational pattern, with the only robust relationship being that between parietal asymmetry and pre-treatment SPQ \( r = 0.54, p < 0.02, \) one-tailed). Second, one could argue that the absence of significant correlations between hemisphere asymmetry and post-treatment scores is due to ceiling effects for SPQ and particularly BAT. In order to circumvent this potential problem, correlations were computed between hemisphere asymmetries and treatment effects in terms of pre- minus post-treatment scores (i.e., pre-treatment SPQ minus post-treatment SPQ and pre-treatment BAT minus post-treatment BAT). None of these correlations attained significance (all \( p \)'s > 0.26).

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<th>( \text{pre SPQ} )</th>
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<tr>
<td>( F3–F4 )</td>
<td>-0.17</td>
<td>0.27</td>
<td>-0.31</td>
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<td>( P3–P4 )</td>
<td>0.58*</td>
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\textit{Note:} Only relevant correlations are shown. *\( p < 0.01 \). SPQ = Spider phobia questionnaire. BAT = Behavioral approach task.
DISCUSSION

The results of the present study can be summarized as follows. Firstly, the present study found indications that a relative right-hemisphere overactivation is accompanied by more severe phobic symptoms. That is, relative right parietal overactivation was related to higher pre-treatment scores on a self-report measure of spider phobia, while the correlation between relative right frontal overactivation and pre-treatment avoidance behavior approached significance. Secondly, no significant correlations were found between EEG asymmetries and post-treatment measures (i.e., therapy-outcome).

By and large, the pattern of results found in the present study is consistent with the idea that the right frontal areas mediate avoidance behavior (e.g., Tomarken et al., 1990), whereas the right parietal areas are involved in self-reported fear and arousal (e.g., Heller et al., 1995). By this view, one would expect that a predominant right-hemisphere activation predicts symptom severity and this appeared to be case. However, on the basis of this idea, one would also expect that a relative right-hemisphere overactivation is related to treatment resistance and, therefore, post-treatment measures. Clearly, this was not borne out by the current data. Yet, before jumping to the conclusion that right-hemisphere overactivation is a severity marker rather than an outcome predictor, several limitations of the present study should be mentioned.

To begin with, the current study relied on a small sample of patients with a monosymptomatic phobia that is known to react extremely well to one-session exposure treatment (e.g., Öst, 1989; Öst et al., 1991). It may well be the case that under these conditions, relationships between EEG asymmetry and treatment outcome are hard to detect. It will be of interest to examine in future studies the connections between hemisphere asymmetries and treatment outcome in larger samples involving more complex anxiety disorders.

Secondly, in the present study, alpha power estimates were based on only a few EEG chunks. Although Tomarken et al. (1992; see also Tomarken et al., 1990) suggest that even short epochs of artifact-free EEG as the ones used in the present study allow for reliable alpha power estimates, it is possible that with more extensive EEG periods and a greater sample size, correlations between EEG
asymmetry and treatment outcome would have emerged. Another technical issue is that EEG activity was linked to the left ear as a reference site. It is plausible to assume that this site is physiologically silent. Still, as Pivik, Broughton, Coppola, Davidson, Fox, and Nuwer (1993) point out, "the selection of the reference electrode presents a complex problem" (p. 548). Some research groups (e.g., Davidson & Fox, 1989) prefer a vertex reference site (Cz), and it remains possible that with such a reference more sensitive measures of EEG asymmetry can be obtained (cf. Davidson, Chapman, Chapman, & Henriques, 1990).

Thirdly, for the present sample, follow-up data on treatment outcome were not available. Meanwhile, the possibility that frontal asymmetries are related to long-term outcome cannot be ruled out.

Fourthly, the approach of the present study was correlational in nature. A significant correlation between relative right parietal over-activation and self-reported fear might indicate that overactivation of the right posterior areas contributes to self-reported fear and arousal (cf. Heller et al., 1995), but, of course, the opposite (i.e., fear causes this pattern of parietal asymmetry) might also be true. Clearly, this causality issue can only be resolved with longitudinal studies.

The most robust correlation found in the present study was that between parietal asymmetry, on the one hand, and pre-treatment SPQ, on the other hand. Parietal asymmetries have been linked to posterior differences in cognitive processes that are sustained by either the left or the right hemisphere (e.g., Tomarken et al., 1990; Heller et al., 1995). Hemisphere asymmetry in cognitions may be relevant to the regulation of emotions, as is illustrated by the work of Tucker and Newman (1981). These authors found that in normal subjects, analytic and verbal processing inhibits emotions, while holistic and imaginal processing augments emotions. The first type of processing is supposedly linked to the left hemisphere, while the second type is thought to be mediated by the right hemisphere. Whether similar effects occur in clinical conditions such as spider phobia remains to be seen. However, it is a well-documented finding that phobic fear is accompanied by cognitive biases (Williams, Watts, MacLeod, & Mathews, 1988). For example, spider-phobic subjects tend to perceive illusory correlations between phobic cues and negative outcomes (de Jong, van den Hout, & Merckelbach, 1995).
Perhaps, then, a relative right posterior hyperactivation sustains cognitive biases and, in this way, affects symptom severity (cf. Merckelbach, Muris, & de Jong, 1990). While this interpretation remains speculative, the connection between parietal asymmetry and symptom severity observed in the present study is at least consistent with a recent EEG study that found a right parietotemporal hyperactivation in anxious patients (Bruder et al., 1996).

In sum, the present study found indications that asymmetrical resting EEG, in particular parietal asymmetry, is related to symptom severity in spider phobia. Given the small sample size, this finding clearly requires replication. Furthermore, future studies should preferably concentrate on the connections between resting EEG asymmetries, symptomatology, cognitive biases, and treatment outcome in more complex phobias.

References


