Brain Electrical Asymmetries during Cognitive Task Performance in Depressed and Nondepressed Subjects

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Background: *Studies using electroencephalogram (EEG) measures of activation asymmetry have reported differences in anterior asymmetry between depressed and nondepressed subjects. Several studies have suggested reciprocal relations between measures of anterior and posterior activation asymmetries. We hypothesized that depressed subjects would fail to show the normal activation of posterior right hemisphere regions in response to an appropriate cognitive challenge.* ***Methods:*** *EEG activity was recorded from 11 depressed and 19 nondepressed subjects during the performance of psychometrically matched verbal (word finding) and spatial (dot localization) tasks. Band power was extracted from all epochs of artifact-free data and averaged within each condition. Task performance was also assessed.* ***Results:*** *Depressed subjects showed a specific deficit in the performance of the spatial task, whereas no group differences were evident on verbal performance. In posterior scalp regions, nondepressed controls had a pattern of relative left-sided activation during the verbal task and relative right-sided activation during the spatial task. In contrast, depressed subjects failed to show activation in posterior right hemisphere regions during spatial task performance.* ***Conclusions:*** *These findings suggest that deficits in right posterior functioning underlie the observed impairments in spatial functioning among depressed subjects.* © *1997 Society of Biological Psychiatry*

Key Words: Depression, laterality, asymmetry, cognition, electroencephalography, electrophysiology

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Introduction

In previous work in our laboratory on brain function in depression, we have focused mostly on resting electroencephalographic (EEG) asymmetries in depressed and nondepressed subjects (e.g., Henriques and Davidson 1990, 1991; Schaffer et al 1983). In these studies, we have used alpha power to inversely reflect activation (see, e.g., Shagass et al 1972). These studies have found that depressed subjects differed from nondepressed controls in the patterning of EEG asymmetries. Specifically, depressed subjects showed a pattern of left frontal hypoactivation both when they were in a current depressed state (Henriques and Davidson 1991; Schaffer et al 1983) and when they were examined following remission in a normothymic state (Henriques and Davidson 1990). It should be noted that this pattern of decreased left frontal activa-

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tion has been found in both depressed patients meeting formal diagnostic criteria for major depressive disorder, as well as among depressed students selected on the basis of stable and extreme scores on the Beck Depression Inventory. This pattern of decreased left anterior functioning has been replicated in other labs (e.g., Allen et al 1993) and using positron emission tomography (PET) measures of regional cerebral blood flow (rCBF) (e.g., Bench et al 1992, 1993) and glucose metabolism (e.g., Baxter et al 1989; Martinot et al 1990), though there have been some inconsistencies in the PET findings (Drevets et al 1992).

Less consistent have been findings of activation asymmetries in more posterior regions during resting conditions. Schaffer et al (1983) reported that depressed subjects showed less relative right-sided activation in the parietal region in contrast to nondepressed controls who showed greater relative right-sided activation in this region, but this difference did not reach statistical significance. In a study examining patterns of EEG activation in response to lateralized facial stimuli, Davidson et al (1985) found that depressed subjects had an inverse relation between frontal and parietal asymmetries that was not seen in control subjects. Among depressed subjects, larger decreases in left frontal activation were associated with greater decreases in right parietal activation. A subsequent study of remitted depressives found that there were significant group differences in posterior asymmetry such that previously depressed subjects had a pattern of decreased right posterior activation compared to controls (Henriques and Davidson 1990). Allen et al (1993) also reported that depressed subjects had more right parietal alpha power (i.e., less activation) as compared to controls. Another study examining differences between currently depressed subjects and normal controls, however, did not find any difference in posterior asymmetries (Henriques and Davidson 1991). Uytdenhoef et al (1983) found that depressed subjects had decreased cerebral flow in the right posterior region compared to controls. Both Post et al (1987) and Drevets et al (1992) also found decreased blood flow in the right midtemporal cortex (see review by Drevets and Raichle 1995).

An indirect approach to the assessment of patterns of hemispheric activation is through the use of cognitive tasks. Although no task relies solely upon the activation of a single hemisphere, some tasks rely more on the function of one hemisphere or another. Studies that have examined the performance of depressed and nondepressed subjects on cognitive tasks have consistently found deficits suggestive of an impairment in right parietotemporal functioning. Heller et al (1995) found that students classified as high-depressed had a decreased left hemispatial (i.e., right hemisphere) bias on a chimeric faces task in comparison to low-depressed students. In contrast, students classified as high-anxious had an increased left hemispatial bias in comparison to low-anxious students. Decreased left hemispatial bias among depressed subjects on this task has also been found by Jaeger et al (1987). A decrease in left hemispatial bias is interpreted as reflecting lower levels of right posterior activation. A large body of work by Bruder and his associates using dichotic listening tasks has found that depressed subjects have a lack of the normally observed left ear (right hemisphere) advantage on a complex tone task (see Bruder 1995 for a review).

Using a dot localization task that had been validated with patients having relatively restricted unilateral damage as requiring the integrity of the right temporoparietal region, Miller et al (1995a) found that depressed subjects had a selective impairment on this task, but they did not differ from controls on a verbal word finding task. We have previously used these two tasks with normal subjects and demonstrated that the verbal task was associated with electrophysiological activation at left posterior electrode sites, whereas the spatial task was associated with right-sided posterior activation (Davidson et al 1990).

One possible explanation for the greater consistency seen in the literature on differences between depressed and nondepressed subjects in cognitive functioning, compared to the studies of baseline electrophysiological abnormalities, is that differences in right posterior activation between depressed and nondepressed subjects are more likely to appear when subjects are challenged with tasks that normally engage these cortical zones; however, little has been done to directly examine brain activation during cognitive task performance among depressed subjects, with the exception of work done by Bruder and associates (e.g., Bruder et al 1995; Tenke et al 1993). They have recorded evoked potentials during a complex tone task, and have found that control subjects who had a strong left ear (right hemisphere) advantage had right greater than left P3 amplitudes. This asymmetry was seen in different regions within the hemispheres. Depressed subjects had an overall lower P3 amplitude compared to controls. Most relevant to the subject of the present report, they failed to show the normal right greater than left P3 asymmetry during this task. Thus, this work suggests that impairments in cognitive tasks designed to tap the function of the right posteriotemporal region are associated with differences in the activation of those regions when the tasks are performed.

The purpose of this study was to add to the scant extant literature and directly examine patterns of brain activation among a group of depressed and nondepressed subjects during the performance of cognitive tasks chosen to differentially activate posterior regions of the left and right hemispheres. EEG was recorded from a group of depressed and nondepressed students during the performance

of psychometrically matched verbal and spatial cognitive tasks. We chose to test depressed and nondepressed students to eliminate any possible confounds from concurrent or previous medication history that would be likely with more severely depressed patients. Moreover, we wished to ascertain the extent to which a selective spatial cognitive deficit would be present in a group whose depression severity is typically more mild than clinically depressed patients. Consistent with our prediction that such deficits would indeed be present in such a group are the data from Heller et al (1995) cited above that were derived from depressed students selected in a manner similar to that used in the present report. We predicted that depressed subjects would exhibit a specific impairment in performance of the spatial task, with no group differences expected in performance on the verbal task, consistent with previous findings (e.g., Miller et al 1995a). Furthermore, we predicted that EEG recorded during the performance of these two tasks would show task-appropriate patterns of activation in the controls, with the verbal task producing relative left-sided posterior activation, and the spatial task producing relative right-sided posterior activation. We predicted that in response to the spatial task, depressed subjects would fail to show the normal pattern of relative right-sided posterior activation compared to brain activity recorded during the verbal task. No group differences in the frontal region were expected during cognitive task performance.

Methods and Materials

Subjects

Twenty-nine depressed and 30 nondepressed female subjects were recruited from the Introductory Psychology pool at the University of Wisconsin–Madison. Subjects were selected on the basis of their scores on the Beck Depression Inventory (BDI) (Beck et al 1961). Nondepressed control subjects were required to have a BDI score of six or less, and depressed subjects were required to have a BDI of 18 or above. (Although a BDI score of 12 is the cutoff point for mild depression, we used a more stringent cutoff point at Time 1 to increase the likelihood that depressed subjects would still be in a depressed mood at Time 2.) At the time of testing approximately 6 weeks later, controls were required to have a score of eight or less on both the state and trait forms of the BDI, and depressed subjects were required to have scores of 12 or above on the two forms. All subjects were right-handed as assessed with the Chapman Handedness Inventory (Chapman and Chapman 1987). Data from 12 subjects (10 depressed, 2 control) were not used because they failed to meet criteria at Time 2. An additional 17 subjects (8 depressed, 9 control) had an absence of sufficient artifact-free data in one or both conditions or had data lost because of equipment malfunctions. This resulted in a final sample of 19 control and 11 depressed subjects. The two groups did not differ in age, $t(21.3) = -.50, p > .50$. (The degrees of freedom reported here have been corrected for unequal variances.) Relevant subject variables are listed in Table 1. All subjects received course credit for their participation.

Table 1. Subject Characteristics [Mean (SD)]

	Control	Depressed
Age (years)	22.16 (6.91)	21.09 (1.64)
BDI–Trait, Time 1	2.22 (2.16)	26.00 (5.33)[a]
BDI–Trait, Time 2	2.05 (2.12)	23.09 (7.26)[a]
BDI–State, Time 2	0.95 (1.18)	16.64 (4.95)[a]
STAI–Trait	29.05 (4.70)	54.09 (8.15)[a]
STAI–State	27.47 (5.49)	49.00 (9.62)[a]
Verbal performance	.89 (.10)	.91 (.07)
Spatial performance	.79 (.20)	.65 (.23)

For controls $n = 19$, for depressed $n = 11$. Standard deviations are in parentheses.

[a] $p < .0001$.

Procedure

Subjects were tested individually. They were told that the experiment was concerned with the relation between brain activity and cognitive processing. After signing a consent form, subjects completed the state and trait versions of both the BDI and the State–Trait Anxiety Inventory (STAI) (Spielberger et al 1983). Then an experimenter applied electrodes to measure EEG and electro-oculogram. The subject was given general instructions about the verbal and spatial tasks and told which task was to be presented first. The order of task presentation was randomized across subjects.

Subjects were seated in a straight-backed chair 54 cm from the screen on which the tasks were presented. They were seated in front of a small desk on which response sheets were placed, and instructed to keep their hands flat on the table until they made their response.

Tasks

Two tasks designed to differentially engage the hemispheres were used. The Dot Localization task was chosen as a measure of visuospatial right hemisphere functioning, and the Word Finding task as a measure of left hemisphere functioning. These two tasks were adapted by Miller et al (1995b) as paper-and-pencil versions of two neuropsychological measures, and have been shown to be matched on their distributions of item difficulty and on coefficient alpha (see Davidson et al 1990).

In the Dot Localization task, the subject is shown a

drawing of two open rectangles, one above the other. The top rectangle contains two dots and the bottom rectangle contains an array of numbers. The bottom rectangle is slightly offset to the right or left of the top rectangle. The side to which it was offset was randomized from trial to trial with the constraint that half the trials be offset to each side. The subject is asked to indicate the numbers that the two dots would cover if the two rectangles were superimposed. The difficulty of the task was manipulated by using five different size arrays of numbers with the smallest containing eight numbers and the largest containing 50 numbers.

In the Word Finding task, the subject is presented with definitions of words and is asked to produce the word that is defined by the phrase. An example is: "a box or house for bees to live in." The correct answer is "hive."

Each task consisted of 24 items, and there were an additional five practice items for each task. The tasks were presented in slide format, with each trial presented on a single slide (see Davidson et al 1990 for further details regarding task standardization and presentation).

During the actual experiment, subjects were instructed to attend to each slide and decide on the appropriate response. For the Word Finding task, this consisted of the word that corresponded to the definition presented. For the Dot Localization task, it consisted of the two numbers whose spatial location corresponded to the position of the dots. When the subject arrived at her response, she was instructed to press a button and then write her response in the appropriate position on an answer sheet. After making her response, the subject then pressed the button once more to initiate the next trial.

Coincident with onset of the slide was a trigger sent to a computer, which started digitization. Data were continuously digitized until the subject pressed the button to indicate that a response had been chosen. The slide was terminated by the subject's button press. Thus, EEG was collected only during the time the slide was present on the screen, prior to the subject's written response.

Apparatus and Recording Procedure

EEG was recorded from the left and right midfrontal (F3, F4), central (C3, C4), and parietal (P3, P4) regions referred to a common vertex (Cz). Two additional channels were recorded to rederive the EEG off-line using an averaged-ears montage. These channels were Cz-left ear (A1) and Cz-right ear (A2). All scalp recordings were performed using a stretchable lycra electrode cap (Electro-Cap Inc.). The electrode cap was positioned on the subject's head using known anatomical landmarks. Elastic straps from the cap attach to a strap that traverses the subject's torso, and this enabled the subject to move comfortably without altering the placement. This procedure results in accurate electrode placements (Blom and Anneveldt 1982). The ear recordings were made with modified Grass ear clip electrodes. Eye movements were recorded with Beckman miniature electrodes from the external canthus to the supra-orbit of the left eye. The impedances of all electrodes were below 5K Ω, and less than 500 Ω separated the impedances of homologous electrode pairs.

The EEG was amplified with a Grass Model 12 Neurodata system using Model 12A5 amplifiers with the bandpass set to 1 and 100 Hz. The EEG was then low-pass filtered (48 dB/octave) at 85 Hz to prevent aliasing. The filtered signals were digitized at 250 samples per second. The EEG was calibrated by inputting before and after each subject a series of 25- and 50-μV sine waves.

Data Analysis

ARTIFACT EDITING OF EEG. All EEG records were visually scored for artifact. All eye movement and muscle artifacts were removed from the data prior to analysis. In addition, the 1-sec period just prior to the subject's response was edited out at this stage to eliminate response-related activity. If artifact was present on any channel, data from all channels were removed so that the EEG data across channels were always from coincident points in time.

EEG ANALYSIS. EEG data from all artifact-free periods during the two tasks constituted the data that were analyzed for this report. The data were rederived off-line using the Cz-A1 and Cz-A2 data channels to obtain a computer-derived linked ears reference. Data were extracted for chunks of continuous EEG 1.02 sec in duration. Chunks were extracted using a Hamming window. Chunks were overlapped by 75% to capture data at the tails of the chunks that would otherwise be attenuated if contiguous windows were applied. A Fast Fourier Transform (FFT) was applied to each chunk of EEG. Power values from all chunks within a trial were averaged, and all trials within a task type were averaged together weighted by the number of artifact-free chunks. The two groups did not differ in the number of artifact free chunks, $F(1,28) = 0.50$, ns, nor was there an interaction between group and condition, $F(2,56) = 1.43$, ns (for controls, verbal mean = 26.72, SD = 4.26, spatial mean = 32.13, SD = 8.26; for depressives, verbal mean = 37.77, SD = 4.92, spatial mean = 27.82, SD = 2.69).

The dependent measures that were obtained from this analysis were power density (in $\mu V^2/Hz$) in the delta (1–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), and beta (13–20 Hz) frequency bands. These data were log transformed to

normalize their distributions because power values are positively skewed (see Davidson 1988). Alpha power is generally considered to be inversely related to activation in the awake adult, with decreases in alpha power reflecting increases in activation (e.g., Lindsley and Wicke 1974).

Results

Performance Data

Repeated-measures analyses of variance (ANOVAs) (SAS Institute Inc 1989) were computed on the accuracy data with task (verbal/spatial) as the within-subject variable and group (depressed/control) as the between-subjects variable. There was a significant group × task interaction, $F(1,28) = 4.92$, $p < .04$. This interaction was a result of depressed and control subjects performing similarly on the Word Finding task, $t(28) = .48$, $p > .5$, and depressed subjects performing worse than controls on the Dot Localization task $t(28) = -1.93$, $p < .05$ (one tailed; see Table 1).

EEG Data

The major sets of analyses consisted of repeated-measures ANOVAs (SAS Institute Inc 1989) on data collected from the central and parietal regions during the two tasks. The between-groups variable was group (depressed/control), and the within-groups variables were task (verbal/spatial), hemisphere (left/right), and region (central/parietal). A four-way (group × task × region × hemisphere) ANOVA was computed. Our specific a priori prediction was that depressed subjects would fail to show differences in the asymmetry of alpha power during the two tasks in contrast to control subjects, who would have a pattern of relative left-sided activation during the verbal task and relativeright-sided activation during the spatial task. This hypothesis was tested by computing three-way (task × region × hemisphere) ANOVAs separately on the data for the control and depressed subjects. Exploratory analyses were conducted on data from the frontal regions and on data from the other frequency bands recorded.

ALPHA POWER. The four-way ANOVA revealed a nonsignificant group × task × hemisphere interaction, $F(1,28) = 2.30$, $p < .15$. The analyses designed to test our specific a priori hypotheses revealed a significant task × hemisphere interaction, $F(1,18) = 6.97$, $p < .02$, for the control subjects, such that the verbal task was associated with left-sided activation, whereas the spatial task was associated with right-sided activation. The task × hemisphere interaction was significant in both the central and parietal regions, $F(1,18) = 6.44$ and 6.13, respectively, both $ps < .03$ (see Figure 1). In contrast, the depressed subjects did not have any significant differences in asymmetry, i.e., there was no main effect or interaction with hemisphere, all $ps > .40$ (see Figure 2). Depressed subjects showed left-sided activation (i.e., less alpha power in the left versus right hemisphere) during both the verbal and spatial task. [We also examined the data referenced to the vertex montage. These results were similar to those observed with the computer-averaged ears montage. The task × hemisphere interaction was significant for control subjects, $F(1,18) = 5.12$, but not for the depressed subjects, $F(1,10) = 0.04$.] Group differences in asymmetry are displayed in Figure 3, which presents the data as asymmetry scores (log right minus log left alpha power), split by task and group, separately for each region.

FRONTAL REGION. Examination of data from the frontal region revealed no significant difference in asymmetry between the two tasks for control subjects, $F(1,18) = 1.22$, $p > .20$. There was a significant task × hemisphere interaction in the frontal region for depressed subjects, $F(1,18) = 12.02$, $p < .01$. This was a result of depressed subjects showing left-sided activation during the verbal task and right-sided activation during the spatial task (see Figure 4).

OTHER BANDS. Since we had no specific hypotheses about activity in other frequency bands, four-way repeated-measures ANOVAs were computed for each band with group (control/depressed) as the between-subjects variable and task (verbal/spatial), hemisphere (left/right), and region (central/parietal) as within-subject variables on band power in each of the three other bands (delta, theta, and beta). To reduce the likelihood of a type I error, an alpha of $p < .0167$ (.05/3) was adopted.

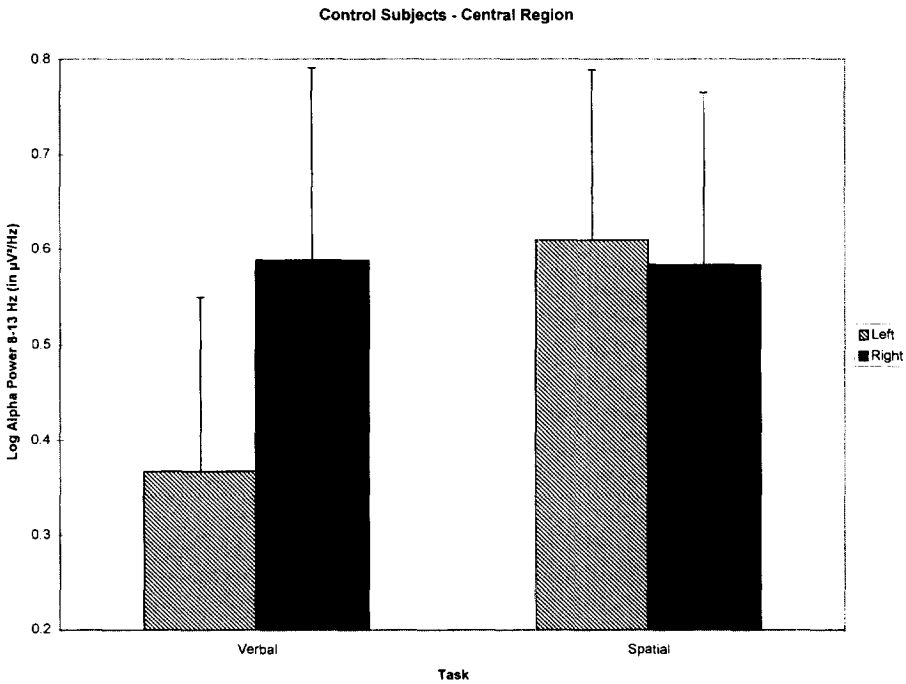
Delta. There were no significant group differences in the patterning of delta power, all uncorrected $ps > .10$.

Theta. Analysis of theta power revealed no significant group differences or interactions with group, all uncorrected $ps > .05$.

Beta. All main effects or interactions with group were nonsignificant, all uncorrected $ps > .20$.

Correlations between EEG Asymmetry and Task Performance

To further explore the relations between asymmetry and task performance, alpha asymmetry difference scores (log right minus log left power) during each task were correlated with task performance for that task. In general,



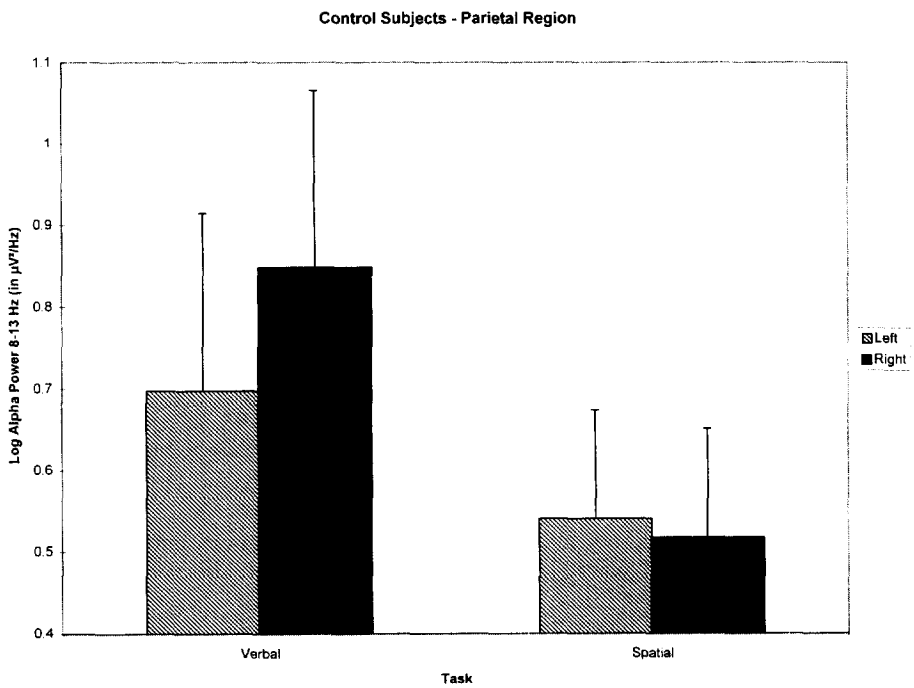


Figure 1. Averaged-ears referenced alpha power density for control subjects (n = 19) in the central and parietal regions during the verbal and spatial tasks.

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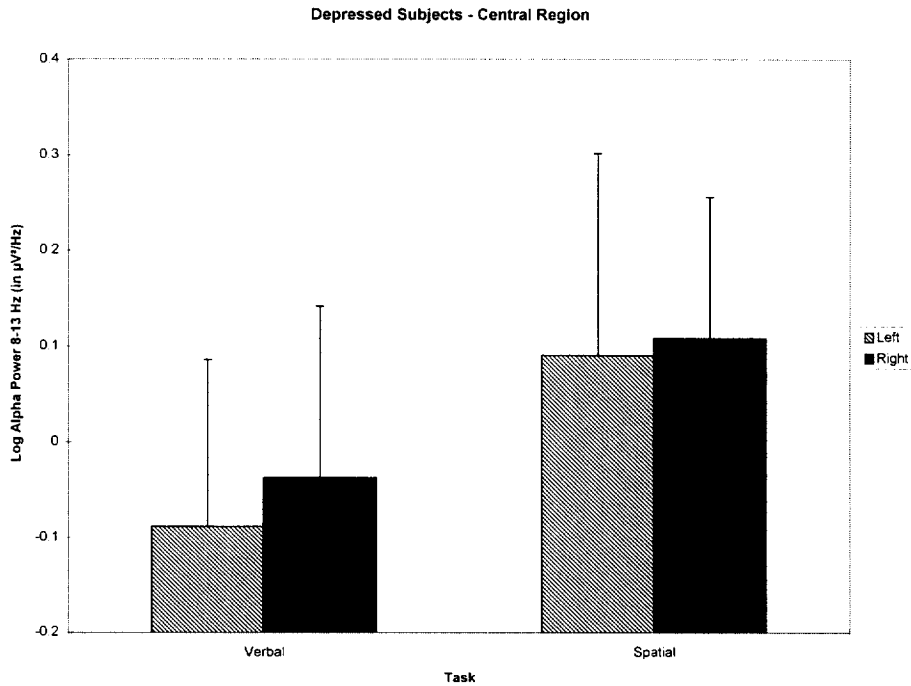


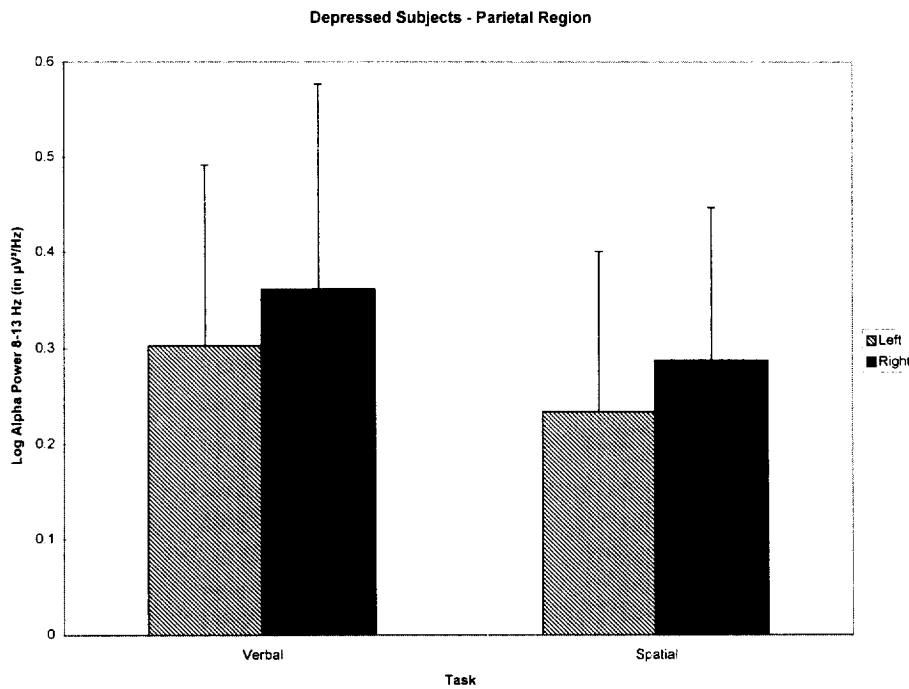


Figure 2. Averaged-ears referenced alpha power density for depressed subjects (n = 11) in the central and parietal regions during the verbal and spatial tasks.

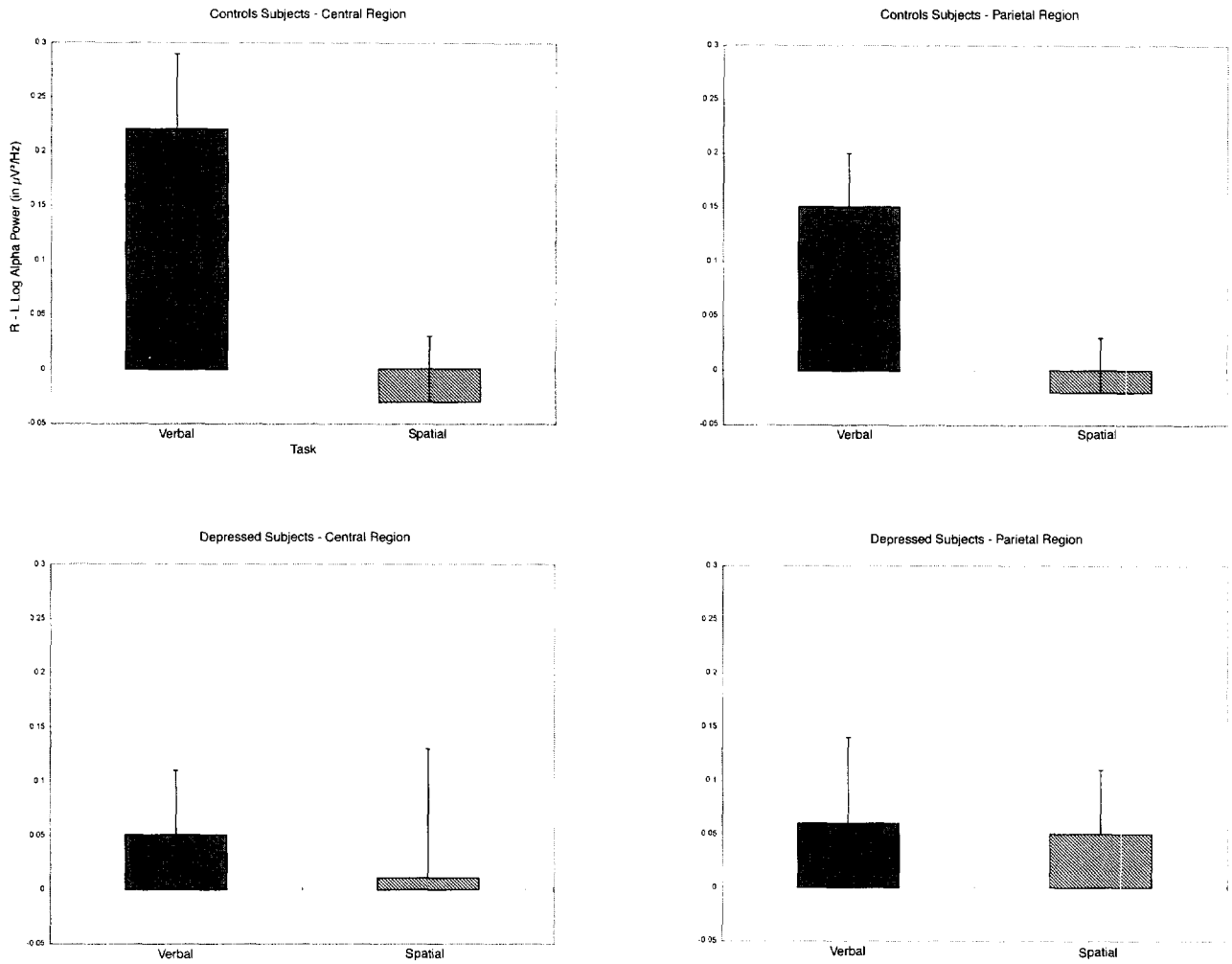
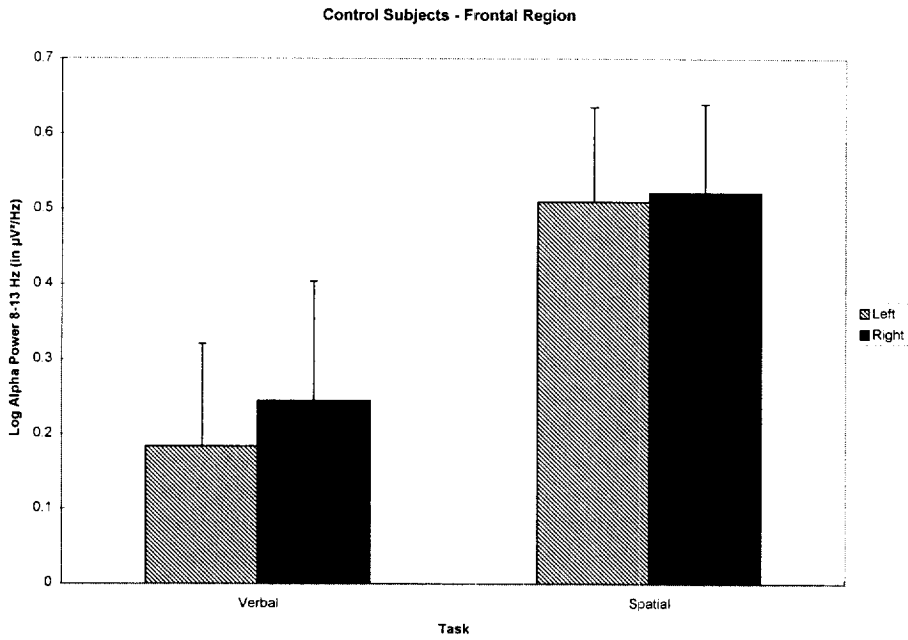


Figure 3. Alpha power asymmetry (log right minus log left averaged-ears referenced alpha power density), separately by group and region for each task. Positive numbers on this index denote left-sided activation (i.e., more alpha power in the right than left hemisphere), whereas negative numbers denote right-sided activation.

central and parietal asymmetries were more highly correlated with task performance than was frontal asymmetry, and none of the correlations with frontal asymmetry approached statistical significance. Table 2 presents the correlations for central and parietal asymmetries with verbal and spatial task performance separately for both control and depressed subjects. For control subjects, the correlations between asymmetry during the verbal task and performance are positive, indicating that greater relative left-sided activation during the verbal task is associated with better performance on that task. The correlations between asymmetry and performance during the spatial task are negative, indicating that greater relative right-sided activation during this task is associated with better performance. In contrast, depressed subjects show the opposite pattern of relations between asymmetry and task performance. The difference in these asymmetry/performance correlations between the two groups is significant for the spatial task for both central and parietal asymmetry, $z = 1.96$ and 2.11, respectively, both $ps < .05$.

Discussion

As predicted, depressed subjects showed a task-specific deficit in performance of the Dot Localization task. These subjects did not differ from controls in their performance during the verbal task. This finding of a specific deficit in spatial functioning is consistent with extant literature (e.g., Bruder 1995; Heller et al 1995; Miller et al 1995a) and extends this work by revealing a specific deficit in a sample of sub-clinically depressed subjects. Furthermore, we found that group differences in the patterning of EEG activation underlies these group differences in performance. Normal control subjects showed relatively greater



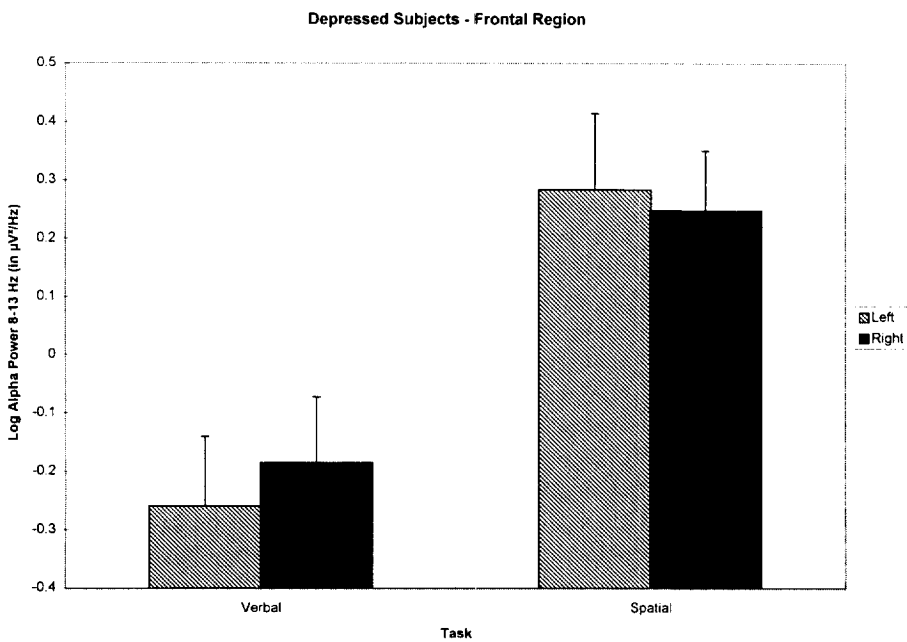


Figure 4. Averaged-ears referenced alpha power data for depressed subjects (n = 11) and control subjects (n = 19) in the frontal region during the verbal and spatial tasks.

Table 2. Correlations between Alpha Asymmetry and Performance

	Central	Parietal
Word Finding task		
Control	.32	.56[a]
Depressed	−.35	−.13
Dot Localization task		
Control	−.25	−.12
Depressed	.53	.66[a]

For controls $n = 19$, depressed $n = 11$.
[a] $p < .03$.

right-sided central and parietal activation during the spatial task compared to the verbal task. In contrast, the depressed subjects failed to show any differences in posterior asymmetry between the two tasks. The failure of the spatial task to elicit relative right-sided posterior activation is consistent with other studies that have found deficits in right hemisphere functioning accompanying the performance decrements in depressed subjects (e.g., Bruder et al 1995; Tenke et al 1993). The correlations between asymmetry and performance highlight these group differences during the spatial task. Whereas control subjects had a negative correlation between asymmetry and performance, depressed subjects had positive correlations in both the central and parietal regions. These correlations indicate that not only did the spatial task fail to elicit relative right-sided activation, but depressed subjects performed better the greater the relative left-sided asymmetry during this task. These results suggest that depressed subjects who performed well on this task were able to overcome their deficits in right posterior function by utilizing a different processing approach to solve this task.

The only region where depressed subjects showed task-related differences in asymmetry was in the frontal region, where they had a pattern of activation asymmetry comparable to that seen in control subjects at posterior sites. It may be that the asymmetric task-related activation in the frontal region, which is intact in depressives, reflects components of task processing different from those associated with processing in posterior cortical regions. The left-sided frontal activation during the verbal task may be associated with the generation of the correct word in the word-finding task. The right-sided frontal activation may be associated with certain types of attentional processes such as vigilance (e.g., Posner 1995) which may be accentuated in depressives, particularly those with accompanying anxiety symptoms. A variety of studies suggest highlighted vigilance among subjects with anxiety-related symptomatology (e.g., Mathews 1993); however, the fact that the depressed subjects show a pattern of right-sided frontal activation, but deficient activation in right-sided posterior regions further underscores the specificity of this effect to the posterior cortical regions.

The results from this study provide further evidence supporting a role for deficits in right posterior function among depressed subjects. We should note that consistent with previous work in our laboratory (i.e., Henriques and Davidson 1990, 1991), these group differences were only found in the alpha band. Examining activation in the other frequency bands failed to differentiate between depressed and nondepressed subjects.

In contrast to findings by Heller et al (1995), our depressed subjects did not have increases in right-sided activation associated with their high levels of anxiety. It may be that the high levels of anxiety among the depressed subjects diminished the magnitude of the observed asymmetry differences between the depressed and control subjects. Likewise, it may be that the presence of depression results in a decrease in right-sided activation that attenuates any anxiety-related increases. To more fully explore the relation between anxiety and posterior function, it would be necessary to recruit samples of subjects in whom high levels of depression and anxiety are dissociated, e.g., a group of nonanxious depressed individuals and a group of anxious nondepressed subjects.

There are several limitations with the current study that must be acknowledged. First, our depressed subjects were selected on the basis of extreme and stable BDI scores, and they cannot be considered a clinical sample; however, these subjects were individuals for whom traitlike symptoms of depression were present across a 6-week period. Clearly these subjects were not just in a bad mood at the time of testing, but could more aptly be described as dysthymic. Furthermore, we believe that our findings do provide valuable information about depression, and expect that they will replicate in a sample of clinically diagnosed depressives. We make this prediction because our BDI depressives had a task-specific deficit in performance comparable to that seen in a sample of clinically depressed subjects (i.e., Miller et al 1995a). Furthermore, we have previously found that differences in EEG asymmetries among BDI depressives and nondepressed controls (Schaffer et al 1983) have replicated in a population of clinically diagnosed depressed subjects (Henriques and Davidson 1991). Concerns may also be raised about our choice to test only female subjects. This decision was based upon the greater incidence of depression among women (Nolen-Hoeksema 1990), and upon the body of work this laboratory has performed with this population (e.g., Henriques et al 1994; Schaffer et al 1983). Given the findings of Miller et al (1995a), we believe that these results will replicate in a sample of male subjects. Another limitation with the current study is the paucity of scalp sites from which data were recorded. Any subsequent

replication of this study should sample from a greater number of scalp sites to more precisely specify the region(s) involved in this task-related impairment in functioning. At present, we can only say that there are differences in right posterior activation; whether these regions include portions of the temporal or occipital lobes is unclear at this point in time. Another possible concern with this study is the relatively small sample size. We would argue that this limitation should have impaired our ability to find our predicted differences, and in fact some of our marginally significant differences might become statistically significant with a larger sample size. The small sample size does limit our ability to look for differences within our group of depressed subjects. For instance, a larger sample size would enable the examination of asymmetry and performance differences among depressed subjects who differ in their level of anxiety.

Heller et al (1995) have postulated that decreases in right parietotemporal functioning underlie the decreases in autonomic responsiveness seen in depression. It may also be the case that these decreases in right posterior functioning may manifest in the impaired social skills of depressed subjects, where the decoding of nonverbal expressive behavior is required. One possible direction for future research would be to examine the relation between deficits in right posterior activation and competence in social skills that require accurate decoding of facial expression. Another area for future investigation is a comparison between depressed and anxious subjects. Given the comorbidity of these two disorders, it would be useful to disentangle the unique contributions of anxiety and depression to the difference in right posterior function that has been implicated in these disorders.

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