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Frontal and Parietal Electroencephalogram Asymmetry in Depressed and Nondepressed Subjects[1]

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High vs. *low scorers on the Beck Depression Inventory (BDI) were compared on measures of resting EEG activation asymmetry from frontal and parietal brain regions. Depressed subjects showed greater relative right frontal activation compared with nondepressed subjects. Parietal asymmetry did not distinguish between the groups. These data support the hypothesis of right hemisphere hyperactivation in the frontal region of depressed individuals and are consistent with the growing body of literature which suggests that the left and right frontal regions may be differentially specialized for particular positive and negative affects.*

A variety of data from both normal and clinical populations has begun to challenge the hypothesis that the right hemisphere is specialized for the processing of affect in general. These data are derived from both behavioral and electrophysiological studies and suggest that the two hemispheres are differentially specialized for the processing and experience of certain positive *vs.* negative emotions. The data are based upon studies with (i) brain-damaged populations (Gainotti, 1972; Bear and Fedio, 1977; LeDoux *et al.*, 1977; Robinson and Benson. 1981; Sackeim *et al.*, 1982); (ii) psychiatric populations (Hommes and Panhuysen, 1971; Flor-Henry *et al.*, 1979; Perris and Monakhov, 1979;

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Wexler and Heninger, 1979; Yozawitz *et al.*, 1979), and (iii) normal populations (Dimond *et al.*, 1976; Reuter-Lorenz and Davidson, 1981; Tucker *et al.*, 1981; Davidson and Fox, 1982; Bennett *et al.*, 1982), and all converge upon the claim that specific regions of the left hemisphere are specialized for the processing of certain forms of positive affect and specific regions of the right hemisphere are specialized for the processing of certain forms of negative affect.

Although relatively little work exists on hemispheric asymmetries in individuals with depressive disorders, the available literature appears consistent with the formulation introduced above. A number of studies using behavioral procedures have found that depressed subjects do not show normal patterns of left hemisphere specialization for language (e.g., Wexler and Heninger 1979; Yozawitz *et al.*, 1979).

A variety of studies using EEG asymmetry as a dependent measure have found that depressed subjects exhibit greater relative right-sided activation during resting baseline in certain scalp regions (Flor-Henry *et al.*, 1979; Perris and Monakhov, 1979) relative to controls. With a large patient series, Matousek *et al.* (1981) found that endogenously depressed subjects showed slightly greater right hemisphere activation in the fronto-temporal region but less right-sided activation in the parieto-occipital region compared with normal controls. This pattern of reversed asymmetry in anterior *vs.* posterior scalp regions is consistent with other data (e.g., Davidson, 1983; Flor-Henry and Koles, 1980).

The present study compared baseline EEG from individuals who differ in their characteristic affective style. In previous research, we have found baseline EEG recorded from posterior scalp leads to be highly correlated with certain features of cognitive style (Davidson *et al.*, 1982). We reasoned that baseline frontal EEG should be associated with certain features of affective style based upon previous research suggesting that frontal lobe asymmetry discriminates between positive and negative affect (e.g., Davidson *et al.*, 1979; Davidson and Fox, 1982; Tucker *et al.*, 1981). We predicted that depressed subjects would show greater relative right frontal activation compared with nondepressed subjects and that parietal asymmetry would not distinguish between groups.

METHODS

Subjects

In order to select groups of depressed and nondepressed subjects, 415 undergraduates were administered the Beck Depression Inventory (BDI) (Beck *et al.*, 1961). Those scoring in the top 9% (20 or above; $n = 38$) were considered eligible as potential subjects. After elimination of those whom we were unable

to contact, were uninterested, left-handed, recently pregnant, or used medication, 17 students came to the laboratory and prior to the experiment filled out the BDI in both trait and state forms. Eleven subjects met the requirement of scoring 14 or above on the trait form and completed the experimental tasks. Baseline data were not recorded from the initial four depressed subjects since it was not part of the original protocol[4] and one record was excessively confounded. The remaining six subjects (n = 4 females) had a mean score of 29.7 (SD = 7.84) on the initial BDI, 25.7 (SD = 8.26) on the trait scale, and 18.0 (SD = 5.9) on the state scale, the latter two administered just prior to the experimental session. The nondepressed subjects were selected to be matched to the depressed subjects on sex, age, and marital status. Individuals were required to score 6 or below (n = 211) on the BDI. In addition, the Marlowe-Crowne Scale of Social Desirability (MC) (Crowne and Marlowe, 1964) was administered along with the BDI, for the purpose of assessing defensiveness in self-reports of individuals reporting low ratings of depression. Subjects scoring 11 or below (n = 177) on the MC met the criterion for nonrepression. To be eligible for inclusion in the control group, scores of 6 or below on the BDI and 11 or below on the MC were necessary as well as meeting criteria such as age, sex, marital status, medication, etc. Fifteen subjects meeting these criteria came to the laboratory and prior to the experiment, again filled out the BDI in trait and state forms. In order to participate further, subjects were required to score 8 or below on the trait scale. One individual was excluded on this basis. The remaining 14 subjects completed the experimental procedures. After exclusion of subjects with confounded records or no baseline and those not matched to the final depressed group on age and sex, nine subjects (n = 6 females) with a mean score of 4.2 (SD = 1.39) on the initial BDI, 4.8 (SD = 2.11) on the trait form, and 4.3 (SD = 2.35) on the state form, were analyzed for the present study.[5] On the MC the control group had a mean score of 8.4 (SD = 2.65) while the depressed group had a mean score of 12.8 (SD = 0.84). All subjects were right-handed as assessed by the Edinburgh Handedness Inventory (Oldfield, 1971). To rule out any transient mood fluctuations that could result from phasic neuroendocrine activity (Wiener and Elmadjian, 1962; Janowsky

[4] The major goals of this research were to compare depressed and nondepressed subjects on a variety of behavioral and electrophysiological indices of hemispheric function. The baseline condition was added to the protocol after the experiment began. Consequently, data from this condition were not obtained from the first four depressed subjects and the first nondepressed subject.

[5] In order to examine "vegetative signs" in the depressed and nondepressed groups, the scores on the final six items of the BDI (items 16-21) were separately tabulated. These items specifically related to vegetative symptomatology. The depressed group had a mean of 5 (SD = 2.19) on these items while the nondepressed group had a mean of 1.67 (SD = 1.19), $t(13) = 3.91$, $p = 0.002$. Thus, subjects in the depressed group presented with unambiguous vegetative signs.

et al., 1966) women participating in the study were scheduled during the middle 2 weeks of their menstrual cycle.

Procedure

Resting EEG was recorded for 30 sec in both eyes-open and eyes-closed periods before the administration of various cognitive and affective tasks and again after completing the tasks. The order in which eyes-open *vs.* eyes-closed baseline periods were presented was counterbalanced within and across subjects. Subjects were instructed to "Just sit and relax, thinking of nothing in particular."

EEG Recording

EEG was recorded with a Lycra electrode cap (Electro-cap Inc.). The electroencephalogram was monitored with a Grass Model 7B, 8-channel polygraph. EEG was recorded from left and right frontal sites (F3 and F4) and left and right parietal sites (P3 and P4). All placements were referenced to Cz (Jasper, 1958). Electrode impedances were all under 5000 ohms. EEG was filtered for the presence of narrow-band alpha activity (9-11 Hz) with Rockland band-pass filters whose roll-off was 48 dB octave. The use of narrow-band filtering helps to reduce the contributions of low frequency activity to the alpha measure. In addition, the peak alpha frequency for most adults falls between 9 and 11 Hz (Doyle *et al.*, 1974). The signal was then passed into four Coulbourn contour-following integrators. The outputs of the integrators were multiplexed and sent into a voltage-controlled oscillator whose output was quantified using a 16-digit printing counter. The recording system was calibrated to yield microvolt seconds of alpha activity. This was accomplished by inputting a 25-μV 10-Hz signal prior to every subject and then scaling the raw data to this known voltage. Alpha values were printed every 3 sec for the duration of each 30-sec epoch.

RESULTS

Alpha values from all electrode leads from all artifact-free 3-sec epochs were averaged together, separately for each subject, separately by condition. The analyses were conducted on these within-subject means.

Alpha data were first examined by computing the ratio score R-L/R + L. Higher numbers on this ratio are indicative of greater relative left-sided activation. ANOVAs comparing the two groups on eyes-open and eyes-closed resting asymmetry for both periods from both the parietal and frontal leads were performed. The ANOVAs on the eyes-open data revealed no significant Group

effects for either frontal or parietal asymmetry for either the beginning or final resting baseline periods, $F < 1$ for all comparisons.

During eyes-closed resting baseline a significant Group effect was obtained on the frontal ratio score for the initial period, $F(1, 13) = 4.51, p = 0.05$. The relevant means and standard deviations are presented in Table I and indicate that depressed subjects show greater relative right frontal activation compared with nondepressed subjects. Importantly, no significant Group effects were obtained on the parietal ratio score for this period, $F(1, 9) = 2.27$, indicating that the Group difference was specific to the frontal recordings (see Table I). Data from the final baseline period revealed no significant group effects on either frontal or parietal asymmetry, $F < 1$ for both comparisons. However, the frontal ratio scores during the two eyes-closed periods were significantly correlated, $r = 0.56$, $df = 13, p = 0.05$.

Figure 1 presents the individual subject means for the frontal ratio score derived from the initial eyes-closed resting baseline, separately for each group. As Fig. 1 indicates, all nondepressed subjects showed frontal ratio scores indicative of relative left-sided activation, while five of six depressed subjects showed the opposite pattern, $\chi^2 = 7.81$, $df = 1, p < 0.01$ with Yates' correction for continuity. The raw alpha values of each subject are presented in Table II.

We next examined the individual contributions of each hemisphere to the ratio score effect described above. ANOVAs with Group and Hemisphere (i.e., left *vs.* right) as factors were computed for the eyes-closed initial baseline period for the frontal data. The results revealed a significant Group × Hemisphere interaction, $F(1, 13) = 10.14$, $p = 0.007$. Depressed subjects showed less alpha activity in the right *vs.* left frontal region ($p = 0.004$) while nondepressed subjects showed slightly less alpha in the left *vs.* right frontal region

Table I. Means and Standard Deviations for EEG Laterality Ratio Scores for the Eyes-Closed Resting Period, Split by Group and by Scalp Region[a]

	Depressed		Nondepressed	
	F ratio	*P* ratio	*F* ratio	*P* ratio
$\bar{x}$	−0.015[b]	0.158	0.034[b]	0.072
SD	0.059	0.110	0.031	0.080

[a] The ratio scores were computed with the following formula: R-L/R+L alpha power. Higher numbers on this ratio are indicative of greater relative left-sided activation. The *F* ratio was derived from F3 and F4 leads and the *P* ratio was derived from P3 and P4 leads.

[b] $p = 0.05$.

Table II. Mean Alpha Activity (in μV sec) for Left and Right Frontal Leads, Separately by Subject and Across Subjects for the Initial Eyes-Closed Baseline Period[a]

Subject no.	F3	F4
Depressed		
1	4.72	4.26
2	6.11	5.89
3	20.47	19.45
4	2.06	2.50
5	16.41	14.12
6	16.59	15.96
$\bar{x}$	11.06	10.36
Nondepressed		
7	5.16	5.39
8	5.29	5.30
9	3.48	3.80
10	3.08	3.10
11	3.89	4.44
12	11.99	12.26
13	2.62	3.17
14	4.96	5.27
15	6.05	6.43
$\bar{x}$	5.17	5.46

[a] Data are normalized to a 3-sec epoch.

($p < 0.02$). The groups also differed in overall alpha levels with depressed subjects showing greater alpha abundance compared with nondepressed subjects, $F(1, 13) = 4.11$, $p = 0.06$. The mean left and right hemisphere frontal alpha values for each group are presented in Table II.

In order to evaluate the relations between overall alpha amplitude and the absolute magnitude of the laterality ratio score, we correlated these two variables across subjects. The correlation was -0.23 and clearly not significant. This suggests that the small group difference in overall amplitude is not responsible for the asymmetry difference between groups.

Correlations were computed between frontal and parietal ratio scores derived from the eyes-closed initial baseline period and scores on the BDI. As we would predict, all correlations of the frontal ratio with the BDI were negative, indicating that greater relative right frontal activation (i.e., lower ratio scores) was associated with greater self-reports of depression (i.e., higher BDI scores). The highest correlation and the only one approaching significance was that between the frontal ratio score and the BDI state measure administered just prior to the experimental session, $r = 0.46$, df = 13, $p = 0.08$. The correla-

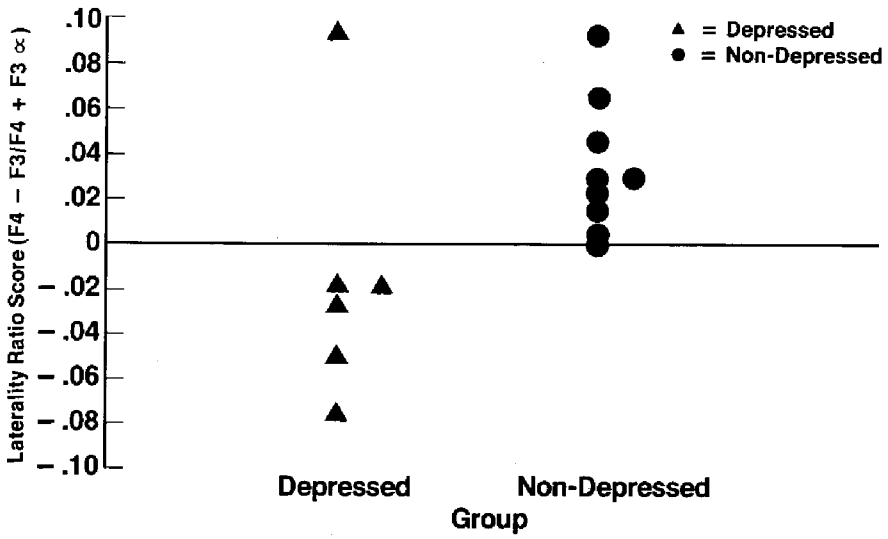

Fig. 1. Frontal laterality ratio (R-L/R + L alpha) scores for individual subjects split by group. Positive numbers on this score are indicative of left-sided frontal activation while negative numbers are indicative of right-sided frontal activation.

tion between the parietal ratio score from this identical period and the BDI state score was 0.10, again indicating rostral/caudal specificity in the relation between asymmetrical activation and affect.

DISCUSSION

The data from this experiment support the hypothesis of relative hyperactivation in the right frontal region among individuals reporting depression. Importantly, parietal EEG asymmetry did not discriminate between the depressed and nondepressed groups. These findings highlight the specificity of frontal asymmetry for affective processes.

The connections between findings of hemispheric dysfunction and neurochemical abnormalities in depression are just beginning to be elucidated. It has recently been demonstrated that right frontal lesions in the rat produce a decrease in norepinephrine level bilaterally compared with comparable left-sided lesions (Pearlson and Robinson, 1981). These investigators concluded that "Unilateral dysfunctions in the right cerebral cortex could lead to bilateral chemical changes. Hence, mood changes which may result from bilateral catecholamine depletion could be initiated by a unilateral process" (p. 240).

In addition to finding a lateral difference between depressed and nondepressed subjects, we also observed that the depressed subjects had greater overall alpha levels in the frontal leads compared with nondepressed subjects. These findings could possibly reflect depressive symptomatology such as psychomotor retardation, loss of interest, fatigue, and slowed thinking.

We found that frontal asymmetry discriminated between depressed and nondepressed groups only for the baseline recorded prior to task administration. It may be that the tasks produce phasic effects which minimized more tonic group differences.

It was also found that only the eyes-closed baseline produced reliable group differences. It is possible that the reduction of visual information in the eyes-closed condition allows individual differences in mental state to be more prominently expressed.

This study found a reliable difference in frontal activation asymmetry between subclinically depressed and normal subjects. Care must be exercised when generalizing these findings to clinical depression. DePue and Monroe (1978) have criticized studies which have used the BDI to screen depressed subjects since it was developed to estimate the severity of a previously diagnosed illness. When the BDI is used to identify depressed individuals, some might score highly because of normal unhappiness or loneliness. Since it is unclear whether subclinical depression differs quantitatively or qualitatively from clinical depression, it is important to assess somatic symptoms since these have been found to discriminate most reliably between these two forms (Hogarty and Katz, 1971; Zung, 1972; Weissman *et al.*, 1975). In the present study, we separately assessed group differences in somatic symptom items and verified that our depressed group did indeed show more vegetative signs compared with the normals. We are currently examining the degree to which this finding obtains in a group of psychotically depressed patients. If this result is replicable across groups, it may prove to be a useful diagnostic procedure and will also contribute to our understanding of the neural dysfunction associated with depression.

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