EEG Asymmetry in Schizophrenic Patients before and during Neuroleptic Treatment

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Lateral asymmetry of electroencephalographic (EEG) spectra was assessed in schizophrenic patients compared to normal controls. Ten predominantly unmedicated schizophrenic inpatients and nine normal controls performed monitored cognitive tasks during bilateral recording of EEG from parietal and temporal sites. Lateralization of EEG power in five frequency bands was compared between the groups; separate analyses were performed for linked ears and vertex references. A subsample of schizophrenic patients was restudied after a period of neuroleptic treatment. All significant group differences were obtained with the linked ears reference only. Pretreatment schizophrenics manifested relatively less alpha power over the right hemisphere during all conditions than controls, particularly in the parietal leads. After treatment, there was a significant shift in alpha lateralization toward the control values. These latter effects were also present in the theta frequency band to a lesser extent.

Introduction

Various authors have reported unusual, but inconsistent, findings when measuring patterns of electroencephalographic (EEG) lateralization in schizophrenic patients. Results have included relatively higher left-sided alpha power in schizophrenic patients than in normals, particularly in posterior brain regions (Goldstein et al. 1973; d’Elia et al. 1977; Etevenon et al. 1979; Kemali 1981), anomalous lateralized alpha attenuation during performance of verbal and spatial tasks (Flor-Henry 1976; Alpert and Martz 1977; Stevens and Livermore 1982), and left-sided prominence of high-frequency, low-amplitude (high beta) activity (Flor-Henry 1976; Coger et al. 1979; Serafetinides et al. 1981; Morihisa et al. 1983; Morstyn et al. 1983a). Additional claims include higher right-sided alpha anteriorly (Etevenon et al. 1979; Giannatrapani and Kayton 1974), relative normalization of anomalous alpha asymmetry after administration of neuroleptic medication (d’Elia et al. 1977; Etevenon et al. 1979), differences in alpha asymmetry between paranoid and hebephrenic...
schizophrenics (Etevenon et al. 1979), and in high beta asymmetry between schizophrenics with verbal versus those with emotional symptoms (Serafetinides et al. 1981). However, some reports have described either relatively more right-sided alpha in schizophrenics (Matousek et al. 1981; Shagass et al. 1983) or no differences in alpha asymmetry from controls at all (Fenton et al. 1980; Iacono 1982; Guenther and Breitling 1985).

Making sense of these disparate findings is difficult. Differences in sample selection, recording techniques, and data analysis are considerable. Other methodological issues include lack of variance stabilizing transformations (e.g., log or square root) (Kemali et al. 1981), medication (Goldstein et al. 1973; Giannitrapani and Kayton 1974; Matousek et al. 1981; Iacono 1982; Morstyn et al. 1983a; Guenther and Breitling 1985), the use of grossly unequal amounts of EEG from individual subjects (Etevenon et al. 1979), and artifact detection and removal (Volavka et al. 1981). In addition, little attention has been paid to the possible effects of differences in ongoing mental state between schizophrenics and normals. In studies examining task related asymmetries, there has apparently been no monitoring of task performance.

Lateralized findings in schizophrenics have emerged from studies using other neurophysiological techniques, including cerebral blood flow (Gur et al. 1985), cerebral computed tomographic scan (Golden et al. 1981; Luchins et al. 1982; Largen et al. 1983), cortical evoked potentials (Connolly et al. 1983; Morstyn et al. 1983b), positron emission tomography (Sheppard et al. 1983; Wolkin et al. 1985), and postmortem dopamine assay (Reynolds 1983). Thus, there are indications from converging paradigms that schizophrenic illness may be related to lateralized brain disorder.

In the current study, we examined EEG asymmetry in a group of newly admitted schizophrenics. We explored the effects of medication, clinical status, and verbal versus spatial cognitive activity. We intended to elucidate (1) whether or not schizophrenics differed in alpha lateralization from controls, and if so, if this was pervasive or limited to resting conditions; (2) given adequate engagement in cognitive tasks, would schizophrenics show anomalous patterns of lateralized alpha attenuation during verbal versus spatial information processing; and (3) whether or not neuroleptic drug treatment affects alpha asymmetry in schizophrenics.

Methods

Subject Selection

Patients were new admissions to the Psychiatric Inpatient Unit at the Veterans Administration Medical Center, San Francisco, for whom a Research Diagnostic Criteria (RDC) (Spitzer et al. 1978) diagnosis of schizophrenia was made, using the Schedule for Affective Disorders and Schizophrenia (SADS) (Spitzer and Endicott 1978). Ten male subjects were selected who were aged 26–38 years (mean 32 ± 4). RDC diagnoses included schizophrenia, paranoid type, chronic (6); schizophrenia, disorganized type, chronic (1); schizophrenia, undifferentiated type, chronic (2); and schizoaffective disorder, depressed type, subchronic (1). All subjects met DSM-III criteria for schizophrenia; the patient with a schizoaffective diagnosis developed his affective symptoms after schizophrenic symptomatology was well established. All patients had been free of regular neuroleptic medication for at least 1 month before admission. However, one patient had taken 100 mg of chlorpromazine single doses of 1 were rated on the 1 Six patients were 1 when their clinical posttreatment, 28.

Control subject nine right-handed or neurological his

Experimental Pr.

Subjects were seat all of which were recorded between Scalp potentials w Quincy, MA) with filter was on-line recorder at 3.75 in Data were digit eliminate epochs cof referenced data we net–linked ears (VX (Cooley and Tukey (0.5–3.5 cycle/sec (13.5–20.5 cycle/s power ratios (R/L) f within conditions. 1 leads.

Each session cor during which subj reported here. The to read a passage a if the record seeme four designs of the motor components, left hand (BLH).

Data Analysis

Two-way groups-by frequency band–lead of epochs analyzed, ANOVA, we compa
of chlorpromazine each of the three nights prior to admission, and two others were given single doses of a neuroleptic drug 16–17 hr before their first recording session. Patients were rated on the Brief Psychiatric Rating Scale (BPRS) on the day EEGs were obtained. Six patients were restudied from 8 days to 5 weeks after the initiation of drug treatment, when their clinical status had significantly improved (mean BPRS pretreatment, 44.0 ± 5.9; posttreatment, 28.2 ± 4.3; paired differences, t = -6.345, df = 5, p = 0.0016).

Control subjects were selected from the psychiatric and ward staff and consisted of nine right-handed men, aged 25–39 years (mean 32 ± 5). All were free of psychiatric or neurological history, and none were taking psychotropic medication.

**Experimental Procedure**

Subjects were seated in a comfortable chair and electrodes affixed to T3, T4, P3, P4, C, all of which were referenced to linked ears (A1, A2). An electrooculogram (EOG) was recorded between Fp2 and the left outer canthus. Impedances were under 10 Kohms.

Scalp potentials were amplified by a Grass Model 7B polygraph (Grass Instrument Co., Quincy, MA) with half-amplitude cut-offs at 1 and 35 cycle/sec.; a 60-cycle/sec notch filter was on-line. Calibration signals and data were recorded a Vetter Model A FM recorder at 3.75 inch/sec (ips), with flutter compensation.

Data were digitized at 64 samples/sec and edited on a second-by-second basis to eliminate epochs contaminated by EOG or muscle artifact (see Doyle et al. 1974). Vertex referenced data were computed off-line, resulting in temporal–linked ears (T-LE), parietal–linked ears (P-LE), temporal–vertex (T-VX), parietal–vertex (P-VX), and vertex–linked ears (VX-LE) data sets. The artifact-free epochs were Fast Fourier Transformed (Cooley and Tukey 1965); averaged over all 1-sec artifact-free epochs, banded into delta (0.5–3.5 cycle/sec), theta (3.5–7.5 cycle/sec), alpha (7.5–13.5 cycle/sec), low beta (13.5–20.5 cycle/sec), and high beta (20.5–32.5 cycle/sec), and log transformed. Log power ratios (WL) for each band were computed and averaged over all artifact-free seconds within conditions. Due to oversight, two schizophrenics had no recordings from vertex leads.

Each session consisted of active tasks, separated by rest periods with eyes open (EO), during which subjects fixated on a central target. The results for two active tasks are reported here. The verbal task used was Reading Aloud (RA); subjects were instructed to read a passage aloud from a popular magazine. At least 3 min were recorded, more if the record seemed unusually high in artifact. For the spatial task, the most difficult four designs of the WAIS Block Design subtest were used. To control for lateralized motor components, the task was performed separately with the right hand (BRH) and the left hand (BLH).

**Data Analysis**

Two-way groups-by-tasks (EO, RA, BRL, BRH) ANOVAs were performed on each frequency band–lead–reference combination for log power, log power ratios, the number of epochs analyzed, and the number of epochs edited out. Using repeated-measures ANOVA, we compared schizophrenics before and after medication.
Results

Amount of Artifact and Artifact-Free Record

The artifact-free epoch length analyzed in schizophrenic subjects was lower than that in controls \((F = 6.878, \text{df} = 1,17; p = 0.018)\). Schizophrenics had more epochs edited out \((34.8 \pm 22.2 \text{ for RA}; 47.9 \pm 46.1 \text{ for BLH})\) than controls \((32.8 \pm 20.0 \text{ for RA}; 24.4 \pm 19.0 \text{ for BLH})\), but the difference was not significant. These data are tabulated in Table 1.

Spectral Asymmetry

Linked ears data for alpha activity, including significance test results, are presented in Table 1. Pretreatment, schizophrenics showed a consistent pattern of negative log alpha ratios \((L > R)\), whereas normals exhibited positive ratio values. This difference was significant at the parietal leads, both as a main effect \((F = 8.999, \text{df} = 1,17; p = 0.008)\) and for each task. There was no significant group-by-task interaction \((F = 0.366, \text{df} = 3,51; p = 0.78)\). This difference was corroborated when frequencies of subjects with positive versus negative alpha ratios were examined \((p = 0.02 \text{ and } p = 0.05 \text{ for EO and RA, respectively, by Fischer's exact test})\). In addition, two of the three schizophrenics who had taken some neuroleptic before admission to the study had negative alpha ratios. We measured the reliability of log power and log power ratios for the alpha band between the first and last repetition of the EO condition \((\text{Winer 1971})\). The reliabilities obtained were quite high, above 0.90 for both schizophrenic and control parietal linked ears referenced power and above 0.84 for the corresponding parietal ratios. Underlying the ratio differences, all significant differences between groups in absolute spectral power occurred on the right side. The temporal leads were similar to the parietals, although there were no significant group differences. The same pattern of significant asymmetry differences between groups was present in the theta band, with a main effect for group in parietal theta ratios \((F = 8.207, \text{df} = 1,17; p = 0.011)\) and significant group differences for each task. There were no group differences in log theta power.

To assess group differences in number of epochs and second-to-second variability, we compared the variance across the second-to-second spectral estimates between the groups. Computing the sum of the variance for bins centered at 6–16 cycle/sec for the parietal leads revealed no significant difference between groups \((p > 0.4)\), and in fact, the schizophrenic group tended to have lower second-to-second variation than the controls \((107.8 \text{ versus } 144.6 \text{ for } P_3; 94.0 \text{ versus } 170.9 \text{ for } P_4)\). If peak alpha power occurred at a different frequency in schizophrenics, standard banding would not be valid for intergroup comparison. To rule out this potential problem, we examined the average spectra in 1-cycle/sec bins from 6 to 16 cycle/sec in the EO linked ears data, manually locating alpha peak power and recording its frequency and amplitude for each subject. If a subject had more than one peak, the one with the largest amplitude was used. One schizophrenic subject did not have a discernable peak at \(P_4\). Peak frequencies were essentially identical groups for both \(P_3\) (schizophrenic, \(9.7 \pm 1.5\); control, \(9.9 \pm 1.3\); \(t = -0.309, p > 0.38\)) and \(P_4\) (schizophrenic, \(10.2 \pm 1.6\); control, \(10.4 \pm 1.6\); \(t = -0.252, p > 0.40\)).

Finally, there were no significant differences between groups in delta or beta bands for either vertex or linked ears data.
The test results are presented in the pattern of negative log alpha ratios. This difference was significant (F = 8.999, df = 1, 17; p = 0.008) for the three schizophrenics who had negative alpha ratios. When p = 0.05 for EO and RA, and p = 0.05 for EO and RA, the schizophrenics had more epochs edited in parietal ratios. Underlying the pattern of negative log alpha ratios, we found a significant negative association of the positive alpha ratio with a main effect for group (107.8 ± 20.0 for RA, and 52.9 ± 19.7 for EO). The reliability of subjects with positive alpha ratios was lower than that in the controls (32.8 ± 19.7 for RA, and 52.9 ± 19.7 for EO). These data are tabulated in Table 1.

Table 1: Mean Log Power (sd) and Log Power Ratios (R/L) (sd) in Alpha Band for Pretreatment Schizophrenics and Normal Controls, Using Linked Ears Reference. Comparisons by ANOVA and by Independent Groups t-Test

<table>
<thead>
<tr>
<th>Condition</th>
<th>Group</th>
<th>No. of Epochs^a</th>
<th>No. Edited</th>
<th>Left temporal</th>
<th>Right temporal^b</th>
<th>Temporal ratio</th>
<th>Left parietal</th>
<th>Right parietal^b</th>
<th>Parietal ratio^b</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blocks</td>
<td>Schiz.</td>
<td>97.8(84.5)</td>
<td>41.9(31.8)</td>
<td>3.43(0.49)</td>
<td>2.97(0.87)</td>
<td>-0.202(0.304)</td>
<td>3.56(0.51)</td>
<td>3.44(0.48)^b</td>
<td>-0.052(0.060)^f</td>
</tr>
<tr>
<td>(right hand)</td>
<td>Control</td>
<td>124.7(71.8)</td>
<td>26.2(20.2)</td>
<td>3.73(0.95)</td>
<td>4.05(0.70)</td>
<td>+0.142(0.391)</td>
<td>4.03(0.52)</td>
<td>4.19(0.59)</td>
<td>+0.070(0.103)</td>
</tr>
<tr>
<td>Blocks</td>
<td>Schiz.</td>
<td>86.9(59.5)</td>
<td>47.9(46.1)</td>
<td>3.39(0.64)</td>
<td>3.03(0.94)^f</td>
<td>-0.154(0.310)</td>
<td>3.57(0.62)</td>
<td>3.48(0.58)^b</td>
<td>-0.040(0.075)^f</td>
</tr>
<tr>
<td>(left hand)</td>
<td>Control</td>
<td>128.0(50.0)</td>
<td>24.4(19.0)</td>
<td>3.69(0.91)</td>
<td>3.96(0.70)</td>
<td>+0.117(0.373)</td>
<td>3.99(0.56)</td>
<td>4.13(0.58)</td>
<td>+0.061(0.114)</td>
</tr>
<tr>
<td>Eyes open</td>
<td>Schiz.</td>
<td>216.0(118.1)</td>
<td>49.5(28.6)</td>
<td>3.18(0.42)</td>
<td>3.09(0.79)^f</td>
<td>-0.040(0.294)</td>
<td>3.94(0.91)</td>
<td>3.90(0.81)</td>
<td>-0.018(0.080)^f</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>382.6(77.5)</td>
<td>28.3(10.9)</td>
<td>3.67(0.18)</td>
<td>3.95(0.81)</td>
<td>+0.124(0.310)</td>
<td>4.29(0.97)</td>
<td>4.55(0.85)</td>
<td>+0.113(0.118)</td>
</tr>
<tr>
<td>Read aloud</td>
<td>Schiz.</td>
<td>122.7(59.4)</td>
<td>34.8(22.2)</td>
<td>3.67(0.63)</td>
<td>3.60(0.79)</td>
<td>-0.030(0.284)</td>
<td>3.85(0.67)</td>
<td>3.79(0.63)</td>
<td>-0.029(0.072)^m</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>137.7(28.3)</td>
<td>32.8(20.0)</td>
<td>3.74(0.72)</td>
<td>4.09(0.40)</td>
<td>+0.153(0.294)</td>
<td>4.03(0.57)</td>
<td>4.23(0.46)</td>
<td>+0.085(0.121)</td>
</tr>
</tbody>
</table>

Schizophrenics < controls, main effect for group:
- F = 6.787, df = 1, 17, p = 0.0178
- F = 6.805, df = 1, 17, p = 0.0184
- F = 5.773, df = 1, 17, p = 0.028
- F = 8.999, df = 1, 17, p = 0.0081

Schizophrenics < controls within tasks:
- t = -3.01, df = 16.81, p = 0.008
- t = -2.46, df = 16.52, p = 0.0253

Significant differences are indicated by superscript letters.
Task Effects
In general, task main effects approached significance in all four lead-reference combinations in the expected direction, with right hemisphere desynchronization during blocks and relative left hemisphere desynchronization during reading. There were no group differences in the task effect, with a trend toward a greater task effect in schizophrenics in T-LE ($F = 3.134$, df = 3,51, $p = 0.095$). Eliminating the three subjects who had performance difficulties on BRH or BLH did not change the overall picture.

Posttreatment Asymmetry
Despite the small sample size ($n = 6$), significant effects on alpha and theta asymmetry were observed when schizophrenics were compared before and after neuroleptic treatment. Both areas of change were in P-LE and moved the patients in the direction of the control sample values. Table 2 presents the data and the results of the statistical analyses. Log alpha power ratios in treated schizophrenics were not different from those of controls and were increased across tasks, with no significant task-by-treatment interaction. Log theta power ratios showed an equivalent pattern. Although there were no significant changes in log power in either band, ratio changes seemed to reflect relatively greater right-sided power increases. Two of the patients who had received prior medication were included in this sample, both demonstrating these changes. Figure 1 displays parietal log alpha and theta power ratios across tasks for normals, the total schizophrenic group pretreatment, and the subgroup of schizophrenics studied pre- and posttreatment.

Discussion
We have demonstrated group differences in alpha (and to a lesser extent, theta) band lateralization between acutely ill, untreated schizophrenics and normal controls. These effects were diminished weeks later, when symptoms had subsided under neuroleptic treatment. In parietal alpha ratio.

Table 2. Mean Log Power (SD) and Log Power Ratios (R/L)(SD) in Alpha and Theta Bands for Six Schizophrenics Pre- and Posttreatment, Using Parietal Leads and Linked Ears Reference. Comparisons by ANOVA and Paired Differences t-test

<table>
<thead>
<tr>
<th>Condition</th>
<th>Group</th>
<th>n</th>
<th>Left</th>
<th>Right</th>
<th>Ratio*</th>
<th>Left</th>
<th>Right</th>
<th>Ratio*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blocks</td>
<td>Pretreat.</td>
<td>6</td>
<td>3.64(0.66)</td>
<td>3.47(0.61)</td>
<td>-0.070(0.057)*</td>
<td>4.30(0.48)</td>
<td>4.17(0.42)</td>
<td>-0.057(0.051)</td>
</tr>
<tr>
<td>(right hand)</td>
<td>Posttreat.</td>
<td>6</td>
<td>3.83(0.54)</td>
<td>3.86(0.45)</td>
<td>+0.013(0.075)</td>
<td>4.47(0.38)</td>
<td>4.48(0.28)</td>
<td>+0.007(0.085)</td>
</tr>
<tr>
<td>Blocks</td>
<td>Pretreat.</td>
<td>6</td>
<td>3.70(0.77)</td>
<td>3.60(0.69)</td>
<td>-0.045(0.085)</td>
<td>4.43(0.45)</td>
<td>4.24(0.39)</td>
<td>-0.079(0.060)*</td>
</tr>
<tr>
<td>(left hand)</td>
<td>Posttreat.</td>
<td>6</td>
<td>3.93(0.63)</td>
<td>3.93(0.57)</td>
<td>+0.002(0.066)</td>
<td>4.40(0.51)</td>
<td>4.44(0.41)</td>
<td>+0.018(0.064)</td>
</tr>
<tr>
<td>Eyes open</td>
<td>Pretreat.</td>
<td>6</td>
<td>3.99(0.87)</td>
<td>3.90(0.74)</td>
<td>-0.042(0.076)</td>
<td>4.14(0.48)</td>
<td>3.96(0.44)</td>
<td>-0.080(0.052)*</td>
</tr>
<tr>
<td>Read aloud</td>
<td>Pretreat.</td>
<td>6</td>
<td>4.04(0.73)</td>
<td>3.94(0.61)</td>
<td>-0.041(0.079)</td>
<td>4.53(0.54)</td>
<td>4.42(0.47)</td>
<td>-0.051(0.051)*</td>
</tr>
<tr>
<td></td>
<td>Posttreat.</td>
<td>6</td>
<td>4.10(0.47)</td>
<td>4.08(0.36)</td>
<td>-0.000(0.071)</td>
<td>4.41(0.34)</td>
<td>4.42(0.23)</td>
<td>-0.003(0.070)</td>
</tr>
</tbody>
</table>

Posttreatment > pretreatment, main effect:

* = 3.876, df = 5.5, $p = 0.0312$

Posttreatment < pretreatment, within task effect:

* = 3.611, df = 5.5, $p = 0.0157$

Posttreatment < pretreatment, within task effect:

* = 3.325, df = 5.5, $p = 0.0237$

Posttreatment < pretreatment, within task effect:

* = 3.508, df = 5.5, $p = 0.0175$

Posttreatment < pretreatment, within task effect:

* = 3.715, df = 5.5, $p = 0.0157$
four lead-reference combination during blocks. There were no group task effect in schizophrenics and the three subjects who had the overall picture.

On alpha and theta asymmetry and after neuroleptic treatment, in the direction of the control of the statistical analyses. Log values from those of controls by-treatment interaction. Log values there were no significant differences to reflect relatively greater received prior medication were.

Figure 1 displays parietal log of the total schizophrenic group pre- and posttreatment.

Figure 1. Parietal log alpha and theta power ratios across tasks for normals, the total schizophrenic group pretreatment, and the subgroup of schizophrenics studied before and after treatment.

treatment. In parietal and temporal EEGs recorded with a linked ears reference, schizophrenic alpha ratios reflected proportionally more left-sided than right-sided alpha power than normals in both resting and active task conditions. All significant differences in alpha power between normals and untreated schizophrenics were over the right side. This effect was more pronounced in parietal than temporal leads. With clinical improvement under neuroleptics, we found normalization of EEG asymmetry in schizophrenic patients. This finding is consistent with ratio reversals after neuroleptic administration reported by d’Elia et al. (1977) and Etevenon et al. (1979).

In contrast, Giannitrapani and Kayton (1974) found more right-sided alpha (9 cycle/sec activity, to be more specific) in temporal leads of schizophrenics. Iacono (1982) found no difference in alpha asymmetry between schizophrenics and controls. Geunther and Breitling (1985) and Morstyn et al. (1983a) reported lateralized differences in EEGs between schizophrenic patients and normals, but not in the alpha band. However, patients in these three studies were clinically stable and predominantly medicated rather than acutely ill and untreated, as ours were.

However, our findings are in conflict with those of some workers. In the Fenton et al. (1980) study, both acute schizophrenic and control groups had similar patterns of lateral alpha asymmetry, and medicated patients were not different from untreated patients or controls. Recording conditions were quite different between groups, with controls studied in a medical examining room at a retail store. Shagass et al. (1983), using predominantly unmedicated schizophrenics, reported no difference in asymmetry measures between normal and schizophrenics in parietal leads, and differences in the opposite direction from our findings in central leads. Matousek et al. (1981) also found results similar to those of Shagass et al. (1983) when examining amplitude spectra, but did not specify the medication status of their patients. Morihisa et al. (1983) also did not report differences in alpha lateralization between schizophrenics and normals.
Our data on task-related EEG asymmetries do not confirm Flor-Henry’s (1976) contention that schizophrenic patients manifest less change in alpha power ratio between verbal and spatial task conditions, nor do they seem to be consistent with Gur et al.’s (1985) blood flow findings of left hemisphere activation during spatial tasks in schizophrenia. This raises the possibility that performance, attention, and motivational factors may have been responsible for some of the previous findings. A larger sample with tighter control over noncognitive task variables would be necessary to resolve this question more convincingly. Nonetheless, our results are not supportive of the hypothesis of anomalous hemispheric activation during spatial versus verbal tasks.

In contrast to the findings of Flor-Henry (1976), Serafetinides et al. (1981), Coger et al. (1979), and Morihisa et al. (1983), we did not find evidence of lateralized increases in high beta in either temporal or parietal regions. A sample selection factor may have been responsible for our negative findings, as Serafetinides et al. (1981) found a left-sided augmentation of high beta only in patients with predominantly “verbal” rather than “emotional” symptoms. Our current sample is too small to adequately test this possibility.

Low alpha power over the right hemisphere might reflect increased right hemisphere activation in untreated schizophrenics; a model proposed by several authors suggests that in acute schizophrenic episodes, a weakened left hemisphere surrenders perceptual and cognitive control to the right (Sugerman et al. 1973; Shimkunas 1978). Changes in EEG asymmetry associated with neuroleptic treatment may result from pharmacological effects on lateralized mesocortical dopamine systems (d’Elia et al. 1977; Schonfeld and Glick 1981; Mintz et al. 1982). However, several unresolved issues render these interpretations only preliminary. Some non-EEG studies suggest relatively increased left-sided brain activity in schizophrenics as compared to normals (Sheppard et al. 1983; Gur et al. 1985), although others are consistent with our own findings (Wolkin et al. 1985). As all of the six patients involved in the medication part of our study improved clinically, predominantly right-sided alpha attenuation may have been a nonspecific concomitant of increased anxiety, not schizophrenic per se (Goldstein 1979). However, some recent evidence suggests that anxiety is associated with increased left hemisphere activation (Tyler and Tucker 1982).

As group differences in asymmetry were only revealed with the linked ears reference, our results suggest that the effect of recording montage on spectral values, and in particular, on spectral asymmetry, has been underappreciated in clinical studies. All electrode placements we used (including the ears) were electrically active, and EEG records all measured potential differences between two active sites. The magnitude of the differential signal recorded is a function of both the degree to which the activity at the two electrodes reflects the same underlying generators and the associated phase difference between the electrode sites for each generator. In our ongoing work, we are using multiple electrode recordings together with source localization methodologies (for example, see Hjorth 1975) to help disentangle these variables as they relate to schizophrenic EEG asymmetry. In the meantime, the topographic implications of our findings and those of others remain uncertain.

Although we are encouraged by our initial results, we recognize significant methodological shortcomings, such as small sample size, lack of controls for agent or duration of treatment, and the use of only a few recording sites. Despite our care in monitoring the performance of our subjects on cognitive tasks, our conclusions must be limited by the lack of correspondence between tasks in variables other than verbal/spatial processing. These include the amount and type of motor output required, stimulus characteristics, the manner of stimuli required. Finally, paraclinical and cultural factors. We
m Flor-Henry's (1976) con-
alpha power ratio between.
consistent with Gur et al.'s
spatial tasks in schizo-
motive factors.
A larger sample with tighter
the hypothesis of anomalous
inides et al. (1981), Coger et
ence of lateralized increases
ple selection factor may have
n. et al. (1981) found a left-
ominantly "verbal" rather than
ate this possibility.
ct increased right hemisphere
ere surrenders perceptual and
ranas (1978). Changes in EEG
from pharmacological effects
. et al. (1977); Schonfeld and Glick
ses render these interpretations
ely increased left-sided brain
et al. et al. (1985),
all of the
mproved clinically, predomi-
concomitant of increased
ever, some recent evidence
emisphere activation (Tyler and
ated with the linked ears reference,
 spectral values, and in par-
rical clinical studies. All electrode
ere active, and EEG records all
the magnitude of the differential
ctivity at the two electrodes
ase difference between the
re using multiple electrode
, for example, see Hjorth 1975)
phrenic EEG asymmetry. In
ings and those of others remain

recognize significant method-
controls for agent or duration
nvoice our care in monitoring
conclusions must be limited by
than verbal/spatial processing.
ried, stimulus characteristics,
the manner of stimulus presentation, relative task difficulty, and the extent of eye scanning
required. Finally, patients and controls were not matched for education, intelligence, or
factors. We plan to address these limitations in forthcoming studies.

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Introduction

Polygraphic studies of rapid eye movement drug-free movement latency and of eye movement were embodying an aspect of the elaboration of the...

Case Selection

The group of patients years, who were diagn...

From Psychiatric Clin Pyschosomatic Psychiatry
Address reprint requests to Dr. 02-957 Warsaw, Poland.
Received March 1, 1983; revis...