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EEG Alpha Photoc Driving Abnormalities in Chronic Schizophrenia

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Abstract. Periodic photic stimuli across the entire electroencephalographic (EEG) frequency range were used in an attempt to assess EEG functional differences between chronic schizophrenic patients and control subjects. The EEG responses to these photic stimuli were significantly attenuated in the schizophrenic patients, specifically at the frequencies within the EEG alpha range. The schizophrenic patients also showed an alpha range attenuation in the "no stimulus" EEG alpha measure, such that there was a significant correlation across subjects between the "stimulus" and "no stimulus" EEG alpha range abnormalities. These abnormalities are discussed with reference to possible dysfunctional thalamic mechanisms involved in the pacing of EEG alpha activity and the gating of information through the cerebral cortex.

Key Words. Electroencephalographic (EEG) alpha rhythm, schizophrenia, EEG photic driving, thalamus.

Although the technique of electroencephalographic (EEG) photic driving is a common tool in the assessment of neurologically impaired patients, application to psychiatric populations has seldom been studied (Shagass, 1972). For example, there is only one report (Shagass, 1955) of a study involving EEG photic driving in schizophrenia within the past 40 years. If current theories (Geyer and Braff, 1987; Oke and Adams, 1987; Swerdlow and Koob, 1987; Bunney, 1988; Carlsson, 1988) of sensory gating deficits in schizophrenia are valid, then EEG photic driving abnormalities might also be expected to characterize the disorder. This article describes a preliminary study of EEG photic driving in chronic schizophrenia.

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Methods

Subjects. The subjects were eight male inpatients (mean age = 26.00, SD = 4.28) at Metropolitan State Hospital (Norwalk, CA) who had been given a *DSM-III-R* (American Psychiatric Association, 1987) diagnosis of schizophrenia by two board-certified psychiatrists. The clinical data presented in Table 1 suggest that the level of severity of symptoms in this group of relatively young chronic schizophrenic patients was moderate to marked. All of the patients had been drug-free for at least 8 days before the EEG testing session. In addition, 11 healthy male volunteers (mean age = 24.3, SD = 3.93) with no personal or family history of psychiatric illness served as controls.

Procedure. All subjects were seated in a comfortable chair with a neck and arm rest. They were tested in an acoustically and electrically shielded dark chamber adjacent to the room containing the experimental equipment. The subjects were instructed to rest and keep their eyes closed throughout the testing period, which consisted of eight acceptable 10-sec trials for each of four different experimental conditions: three photic stimulus conditions and one no-stimulus condition. The presentation of the three stimulus conditions was randomized, with the constraint that every fourth trial be a no-stimulus condition.

The photic stimuli were pulsing lights with fundamental frequencies of 2.4 Hz, 4.5 Hz, and 8.3 Hz. The pulse duration was 5 ms for each of these conditions. The photic stimuli were presented to the subject through goggles driven by a Grass Model S10 VS miniature photostimulator. The light sources were 30 light-emitting diodes, with a combined intensity of 36 millicandela, within each goggle. This light is red and its dominant wavelength is 648 nanometers. The wavelength spectrum peaks at 635 nanometers.

The frequency spectrum for an infinitely long pulse train in the time domain is an infinite pulse train in the frequency domain—where the fundamental frequency and all of the infinite number of harmonics have equal energy (Cooper and McGillem, 1967). While the 5-ms pulse duration was small compared to the long analysis window of 10 sec, this measurement condition is still only a poor approximation to the infinite case. However, frequency analysis (as described below for the EEG responses at the stimulus frequencies) of the first five

Table 1. Clinical data for the 8 schizophrenic patients

<i>DSM-III-R</i> diagnosis	Age	Weeks in hospital	Onset age	BPRS Total	CGI	Previous medications
295.9	36	3	Unknown	39	3	30 mg Haldol 2 mg Cogentin
295.17	25	4	15	51	4	40 mg Navane 2 mg Cogentin
295.3	24	9	Unknown	45	4	40 mg Thorazine 3 mg Cogentin
295.9	25	4	18	54	5	30 mg Haldol 3 mg Cogentin
295.9	25	3	Unknown	55	5	25 mg Haldol 2 mg Cogentin
295.34	19	9	16	54	6	30 mg Haldol 4 mg Cogentin
295.2	28	30	Unknown	68	6	30 mg Stelazine 4 mg Cogentin
295.3	26	19	18	45	5	80 mg Loxapine 4 mg Cogentin

Note. BPRS = Brief Psychiatric Rating Scale. CGI = Clinical Global Impression.

harmonics of each of these three signals did indicate that all of the harmonics of a particular fundamental frequency had energy equal to that of the fundamental frequency. On the other hand, the amplitudes of harmonics in pulse trains of different fundamental frequencies differ directly as the frequency of the pulse trains (Cooper and McGillem, 1967). Thus, the 8.3-Hz stimulus had harmonics that were $8.3/4.5$ times greater in amplitude than the harmonics of the 4.5-Hz stimulus. Likewise, the 4.5 Hz. stimulus had harmonics that were $4.5/2.4$ times greater in amplitude than the harmonics of the 2.4-Hz stimulus. Fig. 1 depicts these amplitude relationships for the individual frequencies within and across each of the three stimulus signals.

Recording. EEG potentials were recorded with gold cup electrodes placed with adhesive paste at the Fz and Pz locations and referenced to linked mastoids. These signals were amplified with Grass Model 7B amplifiers with the 50% power points being at 0.1 and 35 Hz. From the amplifiers, the EEG was directly sampled by a 10-bit A/D converter at the rate of 100 Hz. During the recording, the experimenter viewed the subject through an infrared video camera and observed the subject's polygraphic activity for any indication of excessive movements or eye movements. Before the computerized data collection, any subject who showed evidence of excessive movement or eye movements was warned to minimize this movement. If the subject continued to show excessive movement or eye movements, or if he showed any such evidence during the computerized data collection, he was excluded from the study. On this basis, four patients and no controls were excluded from the study at the time of testing and before the data analysis. As a further control over these eye movement and movement artifacts, the computer was programmed to reject any trial during which one of the signals had a peak-to-peak amplitude greater than 150 microvolts. For every subject, the

Fig. 1. Relative amplitude spectra of photic stimuli with fundamental frequencies of 2.4 Hz, 4.5 Hz, and 8.3 Hz

computer recorded the EEG signals until 80 sec ($8 \text{ trials} \times 10 \text{ sec/trial}$) of acceptable EEG signals were collected for each condition.

Data Analysis. For the no-stimulus condition, the average absolute amount of energy in the delta (0-3.5 Hz), theta (3.5-7.0 Hz), alpha (7.0-13.0 Hz), and beta (13.0-40.0 Hz) frequency bands across the eight epochs was estimated by a Fast Fourier Transform routine with a 10-sec rectangular window. These frequency band energies were calculated as the sum of the square root of the power across the 0.1-Hz frequency domain steps within each band. For one individual frequency, this would yield the square root of the average power (RMS) across the 10-sec epoch in microvolts (Cooper and McGillem, 1967). For a frequency band, this yields an estimate of the integrated RMS amplitude in microvolts.

For the three stimulus conditions of the experiment, the amount of EEG energy at the

fundamental frequency and the first several harmonics of each photic stimulus signal were estimated from the Fast Fourier Transform with the 10-sec rectangular window. These individual frequency values were calculated as the sum of the square root of the power across four adjacent 0.1-Hz frequency domain steps that were centered approximately on the stimulus frequencies. This yielded an estimate of the integrated RMS amplitude in microvolts for the EEG response at a particular stimulus frequency. For the photic stimulus with a fundamental frequency of 2.4 Hz, this gave EEG amplitude measures centered at 2.4, 4.8, 7.2, 9.6, and 12.0 Hz. For the photic stimulus with a fundamental frequency of 4.5 Hz, this gave EEG amplitude measures centered at 4.5, 9.0, 13.5, 18.0, and 22.5 Hz. Finally, for the photic stimulus with a fundamental frequency of 8.3 Hz, this yielded EEG amplitude measures centered at 8.3, 16.6, 24.9, and 33.2 Hz.

Differences between control subjects and schizophrenic patients were assessed using a three-way analysis of variance (ANOVA) (BMDP2V) with repeated measures (Dixon, 1985). Diagnostic Category was the grouping variable, and Frequency Band and EEG Lead were repeated factors for the no-stimulus condition. For the combined stimulus conditions, Diagnostic Category was the grouping variable and Stimulus Frequency and EEG Lead were repeated factors. Huynh-Feldt reduced degrees of freedom were used to avoid Type I errors caused by falsely inflated degrees of freedom in repeated measures ANOVA.

Results

Figs. 2-5 depict the raw spectra for each condition and each lead for the schizophrenic patients as a group and for the controls as a group.

The no-stimulus condition showed a Band $\times$ Diagnosis interaction ($F = 4.76$; $df = 3, 51$; $p < 0.01$) and a Band $\times$ Diagnosis $\times$ Lead interaction ($F = 11.03$; $df = 3, 51$; $p < 0.0001$). Table 2 presents the F values and their significance level for the effect of diagnosis across the different lead and band conditions of the experiment. Schizophrenic patients had significantly more Fz delta amplitude than normal controls during the no-stimulus condition. Normal controls had significantly more alpha activity at Fz and theta activity at Pz.

The analysis of the stimulus data showed a significant main effect of Diagnosis ($F = 5.22$; $df = 1, 17$; $p < 0.05$), Frequency $\times$ Diagnosis interaction ($F = 3.20$; $df = 14, 238$; $p < 0.05$), and Frequency $\times$ Diagnosis $\times$ Lead interaction ($F = 3.33$; $df = 14, 238$; $p < 0.05$). Table 3 presents the F values and their significance levels for the effect of diagnosis across the different lead and frequency conditions. At the Pz lead, controls had significantly more energy than schizophrenic patients at the 4.8-Hz, 7.2-Hz, and 9.6-Hz frequencies. At the Fz lead, controls had significantly more energy than schizophrenic patients at the 7.2-Hz, 8.3-Hz, 9.0-Hz, 9.6-Hz, and 12.0-Hz frequencies.

Because all but one of these significant differences were at stimulus frequencies within the EEG alpha range and because there were EEG alpha range differences between schizophrenic patients and control subjects during the no-stimulus condition as indicated above, further analysis was performed to determine if these effects were related to the no-stimulus EEG alpha range differences between patients and controls. Table 4 lists the correlations between the no-stimulus alpha levels and the EEG responses at the alpha range stimulus frequencies for both the Fz and Pz leads across subjects. On average, they account for 72% ($p < 0.01$) of the variance at Fz and 62% ($p < 0.01$) of the variance at Pz.

Given the concentration of the photic driving effects in the alpha range and the

Fig. 2. Raw spectra corresponding to no-stimulus recording conditions at Fz and Pz in schizophrenic and control subjects as a group

The unit on the Y axes (square root of the average power) is microvolts and the unit on the X axes is frequency in Hz.

Fig. 3. Average raw spectra corresponding to the 2.4-Hz stimulation condition in schizophrenic and control subjects as a group at Fz and Pz

The unit on the Y axes (square root of the average power) is microvolts and the unit on the X axes is frequency in Hz.

Fig. 4. Average raw spectra in schizophrenic and control subjects as a group corresponding to the 4.5-Hz stimulation condition at Fz and Pz

The unit on the Y axes (square root of the average power) is microvolts and the unit on the X axes is frequency in Hz.

Fig. 5. Average raw spectra in schizophrenic and control subjects as a group corresponding to the 8.3-Hz stimulation condition at Fz and Pz

The unit on the Y axes (square root of the average power) is microvolts and the unit on the X axes is frequency in Hz.

Table 2. Comparison of schizophrenic and control no-stimulus EEG measures

measures			
Int. EEG amplitude in microvolts (RMS)			
EEG lead and band	Controls vs. schizophrenics		F ratio
Pz Delta	19.2	24.0	1.56
Pz Theta	15.6	10.9	9.09 ¹
Pz Alpha	27.6	18.0	3.80
Pz Beta	24.8	21.9	0.40
Fz Delta	16.8	30.6	7.62 ²
Fz Theta	12.8	12.5	0.04
Fz Alpha	28.6	15.6	7.38 ²
Fz Beta	23.7	22.1	0.21

Note. RMS = square root of the average power. Int. = integrated.

1. Denotes 0.01 level of probability, $df = 1, 17$.

2. Denotes 0.05 level of probability, $df = 1, 17$.

correlation of the alpha range resting and driving effects, a further post hoc analysis was performed. It was designed to determine (1) if the EEG measures at the stimuli frequencies in patients and controls differed as a function of photic driving more than as a function of the resting EEG and (2) if this effect was concentrated in the alpha range. The analysis involved computing the EEG measures in the no-stimulus EEG condition at the frequencies corresponding to the stimuli frequencies, exactly as had been done for the stimulus condition. This resulted in both a stimulus and a no-stimulus measure of EEG activity at each of the 14 stimulus frequencies. Next, the EEG responses across this stimulus frequency range were divided according to alpha range (7.2, 8.3, 9.0, 9.6, and 12.0) and nonalpha range (2.4, 4.5, 4.8, 13.5, 16.6, 18.0, 22.5, 24.9, and 33.2) frequencies. The resulting EEG response measures were then subjected to repeated measures ANOVA. Diagnosis was the grouping variable, and EEG Lead (Fz vs. Pz), Stimulus Condition (stimulus vs. no-stimulus), and Frequency Range (alpha vs. nonalpha) were repeated factors.

The analysis did suggest that the EEG measures at the stimuli frequencies in the diagnostic groups differed more as a function of photic driving and that this was only true for the alpha range frequencies. There was a significant Diagnosis $\times$ Stimulus Condition $\times$ Frequency Range interaction ($F = 5.73$; $df = 1, 17$; $p < 0.05$). Table 5 shows the mean (and SD) values for the schizophrenic and control subjects at Fz and Pz for the individual conditions of this interaction, along with the F tests of the simple main effects. Neither the mean nor the SD of the schizophrenic EEG response to photic stimulation increased normally in the alpha range, and this was the basis for the larger diagnostic effect in the stimulus condition. By contrast, there was a more normal increase in both the mean and the SD of the schizophrenic EEG response to photic stimulation in the nonalpha frequencies, such that the no-stimulus diagnostic effect at Pz was obliterated.

Discussion

To the best of our knowledge, this is the first study of EEG photic driving in

Table 3. Comparison of EEG response to photic stimulation in schizophrenic and control subjects

Int. EEG amplitude in microvolts (RMS)			
EEG lead and band	Controls vs. schizophrenics		F ratio
Pz 2.4	2.8	2.7	0.06
Pz 4.5	2.8	1.9	4.09
Pz 4.8	2.4	1.6	8.34 ¹
Pz 7.2	2.1	1.2	10.56 ²
Pz 8.3	3.9	1.9	3.53
Pz 9.0	3.2	1.8	2.11
Pz 9.6	2.7	1.5	4.73 ¹
Pz 12.0	1.3	1.1	0.49
Pz 13.5	1.2	1.3	0.02
Pz 16.6	1.4	1.3	0.01
Pz 18.0	0.9	0.9	0.03
Pz 22.5	0.5	0.6	0.03
Pz 24.9	0.5	0.6	0.02
Pz 33.2	0.2	0.4	1.38
Fz 2.4	2.6	2.8	0.34
Fz 4.5	2.6	2.0	1.23
Fz 4.8	1.9	1.8	0.10
Fz 7.2	1.7	1.2	5.75 ¹
Fz 8.3	4.0	1.8	7.32 ¹
Fz 9.0	3.5	1.3	4.76 ¹
Fz 9.6	2.9	1.2	8.09 ¹
Fz 12.0	1.9	0.9	9.56 ²
Fz 13.5	1.6	1.1	1.66
Fz 16.6	1.7	1.0	2.74
Fz 18.0	0.9	0.7	0.54
Fz 22.5	0.6	0.5	0.20
Fz 24.9	0.7	0.7	0.01
Fz 33.2	0.4	0.4	0.08

Note. RMS = square root of the average power. Int. = integrated.

1. Denotes 0.05 level of probability, $df = 1, 17$.

2. Denotes 0.01 level of probability, $df = 1, 17$.

schizophrenia since a study reported more than 30 years ago (Shagass, 1955). That early study did not report a significant difference between control subjects and schizophrenic patients in the EEG response to photic driving in the alpha range, but the methodology differed considerably from that used here. In particular, the present study quantified the *average* EEG photic response amplitude for an 80-sec period for each subject through automatic spectral analysis. The 1955 study quantified the *peak* EEG photic response amplitude for a 2-sec period for each subject through manual ruler analysis.

The present study suffers from a methodological limitation in that it did not control adequately for the preponderance of eye movement artifacts in the schizophrenic patients. Eye movement artifacts have been observed to have the

Table 4. Correlations (*r*) between no-stimulus and stimulus alpha variables across all subjects

EEG response (RMS) at stimulus frequency in Hz	No stimulus alpha band Int. amplitude (RMS)	
	Fz—linked mastoids lead	Pz—linked mastoids lead
7.2	0.86 ¹	0.77 ¹
8.3	0.84 ¹	0.86 ¹
9.0	0.95 ¹	0.89 ¹
9.6	0.94 ¹	0.85 ¹
12.0	0.61 ¹	0.53 ²
Average <i>R</i> ²	0.72 ¹	0.62 ¹

Note. RMS = square root of the average power. Int. = integrated.

1. Denotes 0.01 level of probability, *df* = 1, 17.

2. Denotes 0.05 level of probability, *df* = 1, 17.

Table 5. Comparison of schizophrenics' and controls' EEG response at the stimulus frequencies at Fz and Pz as a function of stimulation condition (stimulation vs. no stimulation) and frequency range (alpha vs. nonalpha)

Stimulation condition	Location/frequency range	Mean (and SD) of Int. EEG amplitude in microvolts (RMS)		
		Controls vs. schizophrenics		<i>F</i> ratio
No stimulus	Fz/nonalpha	7.2 (1.9)	7.1 (1.7)	0.44
No stimulus	Pz/nonalpha	8.9 (1.6)	6.7 (2.3)	5.66 ¹
Stimulus	Fz/nonalpha	13.3 (4.0)	11.4 (3.8)	0.99
Stimulus	Pz/nonalpha	13.1 (3.1)	11.5 (5.0)	0.79
No stimulus	Fz/alpha	9.1 (5.9)	5.4 (1.4)	3.03
No stimulus	Pz/alpha	9.5 (5.8)	5.7 (1.8)	3.00
Stimulus	Fz/alpha	14.3 (7.1)	6.5 (1.4)	8.97 ²
Stimulus	Pz/alpha	13.4 (7.2)	7.6 (2.4)	4.51 ¹

Note. Int. = integrated.

1. Denotes 0.05 level of probability, *df* = 1, 17.

2. Denotes 0.01 level of probability, *df* = 1, 17.

greatest influence on activity in the delta and theta frequency bands in the frontal leads (Lee and Buchsbaum, 1987). Thus, the greater amount of frontal no-stimulus delta activity in the schizophrenic patients could, in part, reflect eye movement artifacts. Yet, the major effects of this study were not observed in the delta and theta frequency range in the frontal leads, but instead were observed in the alpha frequency range, especially in response to photic stimulation. This pattern would be difficult to explain on the basis of eye movement artifacts.

There were almost uniform differences between schizophrenic patients and normal control subjects in the frontal alpha amplitude measured during the no-stimulus condition, as only one control subject was found to overlap with the schizophrenic range during this condition (Fig. 6). Such a difference was not observed at the parietally placed lead (Fig. 6). Thus, this study has replicated the previous findings (Milstein et al., 1969; Itil et al., 1972; Giannitrapani and Kayton, 1974; Buchsbaum et al., 1982) of reduced alpha amplitude in schizophrenia, but it suggests that this

Fig. 6. Comparison of no-stimulus EEG alpha amplitude in control subjects and schizophrenic patients

effect might be somewhat larger at the midfrontal lead than at the midparietal lead in the linked-mastoid reference recording in chronic schizophrenic patients.

One possible explanation for a resting alpha rhythm deficit effect in schizophrenia is simply that the patients might not comply with the "eyes closed" instructions. However, in a topographical study of the "eyes open" vs. "eyes closed" alpha rhythm in young adults, Buchsbaum et al. (1982) observed that the major effect of opening the eyes was the attenuation of alpha activity in the posterior EEG leads (including Pz), instead of the anterior EEG leads (including Fz). In the present study, the schizophrenic alpha rhythm deficit was greater at Fz than at Pz. Thus, this pattern is opposite to what would be expected as a result of the schizophrenic patients' failure to comply with the "eyes closed" instructions.

The significant differences between schizophrenic patients and control subjects in the EEG response to photic stimulation were concentrated in the EEG alpha range,

with the differences most apparent at the Fz lead. Moreover, these photic stimulation EEG alpha frequency effects were correlated with the no-stimulus EEG alpha activity effects at both the Fz and Pz leads. Finally, the alpha range photic driving responses in the schizophrenic patients were uniformly attenuated as indicated by the low means and SDs as compared to the controls, but the nonalpha range photic driving responses did not show this effect. The fact that all of these effects are intercorrelated and are concentrated in the alpha range suggests that one common factor might be involved.

One possible explanatory factor could be a dysfunction within the chronic schizophrenic population in the mechanisms involved in the pacing of rhythmic EEG activity in the alpha range. Evidence from the animal literature not only suggests that the dorsal thalamic nuclei are involved in the pacing of resting cortical EEG rhythms in the alpha frequency range (Andersson and Anderson, 1968), but it also suggests that these thalamic nuclei are particularly resonant with external driving rhythms in the EEG alpha frequency range (Steriade and Deschenes, 1984). Thus, dysfunction in a mechanism which, either directly or indirectly, controls thalamic pacing could explain the alpha range abnormalities in the chronic schizophrenic patients.

This speculation is consistent with suggestions of abnormal dorsal thalamic gating in psychosis (Geyer and Braff, 1987; Oke and Adams, 1987; Swerdlow and Koob, 1987; Bunney, 1988; Carlsson, 1988), but it should be viewed with caution because of the methodological limitations and the small number of patients in this preliminary study.

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