

UC Irvine

UC Irvine Previously Published Works

Title

Electroencephalographic photic driving in patients with schizophrenia and depression

Permalink

<https://escholarship.org/uc/item/74w3g0cc>

Journal

Biological Psychiatry, 41(4)

ISSN

0006-3223

Authors

Jin, Yi
Potkin, Steven G
Sandman, Curt A
[et al.](#)

Publication Date

1997-02-01

DOI

10.1016/s0006-3223(96)00473-8

Copyright Information

This work is made available under the terms of a Creative Commons Attribution License, available at <https://creativecommons.org/licenses/by/4.0/>

Peer reviewed

Electroencephalographic Photic Driving in Patients with Schizophrenia and Depression

Yi Jin, Steven G. Potkin, Curt A. Sandman, and William E. Bunney, Jr

Key Words: Electroencephalography, photic driving, harmonic, schizophrenia, depression, normal

BIOL PSYCHIATRY 1997;41:496–499

Introduction

Electroencephalographic (EEG) photic driving, a steady-state evoked potential (Regan 1989), was first reported by Andrian and Mathews (1934). It was found that repetitive photic stimulation could evoke cerebral flickering response at its fundamental frequency or the higher harmonics. Using frequency analysis, Walter and Walter (1949) demonstrated that the measured response was actually a compound of multiple harmonic components of the stimulation frequency. In many subjects, the photic responses around 10 Hz are larger in amplitude compared with those at neighboring frequencies (Jin et al 1995a; Rice et al 1989; Regan 1972; Montagu 1967).

Early studies showed that focal or lateralized cerebral diseases could cause ipsilateral attenuation of the photic driving, especially if the visual pathways are involved (Kooi et al 1957; Rodin et al 1953; Oosterhuis et al 1969). Focal lesions may also increase the amplitude of photic driving on the abnormal side (Brezny and Gaziova 1964; Cantor and Ilbag 1973). Vogel et al (1971, 1974) and Kleiber et al (1972) observed that substances known to enhance central monoamine functioning (e.g. amphetamine, monoamine oxidase inhibitors) block the EEG driving response, whereas phenothiazines that inhibit the monoamine activity increase the photic driving. Depressed patients have greater photic driving response than normal subjects (Kleiber 1972), whereas schizophrenic patients have less response than normal controls (Shagass 1972).

Using the spectrum analysis (Jin et al 1990, 1995a; Rice et al 1989), we observed that the energy of EEG photic driving in the range of alpha frequency is greatly reduced in schizophrenics. We noticed recently that the EEG changes in schizophrenics could be normalized by neuroleptic treatment, especially in the clinical responders (Jin et al 1995b). In this study we intended to replicate the previous findings and further investigate the photic driving in depressed patients to test the sensitivity and specificity of this procedure in a possible clinical use.

Methods and Materials

Subjects

Thirty-eight schizophrenic patients (M/F: 27/11, mean age: 31.4 ± 7.7 years), 19 major depressed patients (M/F: 5/14, mean age: 41.1 ± 12.2 years), and 24 normal subjects (M/F: 10/14, mean age: 30.8 ± 9.7 years) consented in the present study. There were no age difference among the three groups ($p > .05$). Contingency analysis showed that there were more female patients in the depressed group than in the normal or schizophrenic groups ($p < .01$), whereas there was no significant difference between the later two groups ($p > .05$). Patients had been drug free for 5 days or more before the EEG evaluation. Normal subjects were free of personal or family history of psychiatric illness or illicit drug use.

EEG Measurement

Subjects were tested in a standard EEG photic driving procedure (Jin et al 1990) including one resting condition and three photic stimulation conditions. The fundamental frequencies of the three pulsed stimuli were 2.4 Hz, 4.5 Hz, and 8.3 Hz, respectively.

From the Department of Psychiatry and Human Behavior, University of California, Irvine, California.

Address reprint requests to Y. Jin, MD, Department of Psychiatry and Human Behavior, UCI Medical Center, Route 88, Orange, CA 92868-3298.
Received February 7, 1996; revised September 30, 1996.

Table 1. Resting EEG of Patients with Depression ($n = 19$), Schizophrenia ($n = 38$), and Normal Controls ($n = 24$)

	Depression		Schizophrenia		Normal	
	Mean	SD	Mean	SD	Mean	SD
Fz						
δ	2049.1 ^a	1223.2	4563.5	1940.3	4018.6	1649.2
θ	1270.0	634.8	1226.3	525.4	1456.2	534.3
α	4920.2 ^a	2200.4	2209.9	1591.9	2866.5	1828.9
β	1581.0	878.0	1688.8	1170.9	1449.0	608.5
Pz						
δ	2462.5 ^a	1406.7	4004.2	1728.9	3360.4	1370.0
θ	1558.7 ^b	606.9	1200.5	485.6	1350.2	615.6
α	4122.1 ^b	1830.0	2753.1	1661.7	3414.3	1623.7
β	1642.7	1005.9	1712.1	1028.0	1646.6	777.9
Oz						
δ	1345.5 ^a	812.6	4301.8	2250.1	4907.4	3036.1
θ	975.9	404.8	851.7	515.0	937.1	592.6
α	3516.2 ^a	2104.2	2252.7	1614.7	2427.4	1884.1
β	2954.5 ^a	1106.5	2173.9 ^c	1520.6	1424.7	1109.7

ANOVA (diagnosis \times band \times locus): $F = 6.35, p < .001$.

^a Indicates the means that are significantly different from both groups ($p < .05$) by t test.

^b Indicates the means that are significantly different from one of the two groups ($p < .05$) by t test.

^c Indicates the mean that is significantly different from normal controls ($p < .05$) by t test.

EEG electrodes were placed at Fz, Pz, and Oz and referenced to linked mastoids. Electro-oculograms (EOGs) from outer canthi were recorded to monitor the eye movements. Eight artifact-free EEG epochs for each condition were converted by a fast Fourier transform (FFT) routine. Four consecutive resting frequency bands named δ (0.6–4.0 Hz), θ (4.1–7.0 Hz), α (7.1–12.0 Hz), and β (12.1–35.0 Hz), and four harmonics of the EEG photic driving in the α frequency band (7.2 Hz, 8.3 Hz, 9.0 Hz, and 9.6 Hz) were calculated.

Results

Resting Condition

Three-way analysis of variance (ANOVA) (diagnosis \times band \times placement) with repeated measures of four frequency bands and three electrode locations of the resting EEG power revealed a significant interaction among these main effects ($F_{12,468} = 6.35, p < .001$). The post hoc t test for each individual band is summarized in Table 1. Depressed patients exhibited significantly higher energy in α and lower energy in δ activities than schizophrenic patients and normal controls, whereas there were no differences of the four bands between schizophrenic and normal subjects except for the β band in the occipital lead.

Photic Stimulus Condition

Main effect of diagnosis, electrode placement, and EEG harmonic at each stimulus condition was tested by repeated-measures ANOVA. Results showed an overall difference among the groups ($F_{2,78} = 7.36, p < .001$) and a significant interaction among the main effects ($F_{12,468} = 2.43, p < .01$). Table 2 displays means and post hoc t test for individual harmonic variables at each electrode. Depressed patients had greater power

density of photic driving than normal subjects and schizophrenic patients, whereas schizophrenic patients had even lower energy than normal controls.

Separate analyses of depression data were conducted to clarify the possible gender influences on the EEG activities, since there were more female patients in the depressed group than the others. Although there were no significant differences in total power of resting EEG ($F_{1,17} = 1.76, p > .05$), alpha activity ($F_{1,17} = 3.57, p > .05$), or EEG photic driving ($F_{1,17} = 3.67, p > .05$) between the two gender groups, female patients showed slightly lower energy in the resting α (mean: 3744.3) and photic driving (mean: 631.3) than male patients (mean resting α : 2450.1; mean photic driving: 996.5). Therefore, the differences between the diagnostic groups with higher resting α activity and EEG photic driving in depression cannot be attributed to the gender effect.

A discriminant function was also calculated to define a weighted EEG component that best predicts the diagnosis to which a case belongs. In contrast to the prior probability 33.3%, the overall percentage of correct classification using these functions was 71.6%. The specificity/sensitivity ratios for depression and schizophrenia were 1.3 (93.9%/74.0%) and 0.8 (74.4%/92%), respectively.

Discussion

There is evidence that synchronous discharges of cortical cell assemblies are driven by afferent thalamic inputs, which in turn are controlled by the inhibitory inputs from the substantia reticularis in the midbrain (Andersen and Andersson 1968; Speckmann and Caspers 1979). Incorporating the present findings with our previous hypothesis that lower EEG alpha activity and photic driving in schizophrenics indicate a deficit of thalamic function (Rice et al 1989; Jin et al 1990, 1995a, 1995b), we

Table 2. EEG Photic Driving in Patients with Depression ($n = 19$), Schizophrenia ($n = 38$), and Normal Subjects ($n = 24$)

	Depression		Schizophrenia		Normal	
	Mean	SD	Mean	SD	Mean	SD
Fz						
7.2 Hz	283.8	191.7	165.5 ^a	113.3	250.9	133.2
8.3 Hz	1119.5 ^a	990.4	490.2	345.3	631.8	569.0
9.0 Hz	1437.8 ^a	1401.4	386.3 ^a	450.5	494.9	407.8
9.6 Hz	905.6 ^a	845.6	242.9 ^a	338.8	317.5	255.0
Pz						
7.2 Hz	315.2	155.3	193.5 ^a	137.7	250.4	117.0
8.3 Hz	865.4	736.7	613.0	424.1	794.0	509.4
9.0 Hz	681.4 ^b	538.1	464.5	448.8	494.1	323.7
9.6 Hz	557.1 ^a	415.8	322.9	372.9	300.0	225.1
Oz						
7.2 Hz	238.7	148.3	221.3	232.7	288.3	332.4
8.3 Hz	911.4	739.3	734.3	696.9	844.6	749.1
9.0 Hz	832.0 ^a	577.9	606.4	642.8	575.8	573.9
9.6 Hz	580.7 ^a	575.5	349.7	340.0	368.0	347.6

ANOVA (diagnosis \times frequency \times locus): $F = 2.43$, $p < .01$.

^a Indicates the means that are significantly different from both groups ($p < .05$) by t test.

^b Indicates the mean that is significantly different from one of the two groups ($p < .05$) by t test.

suggest that the increased α EEG and photic driving in depressed patients may related to the abnormal arousal mechanisms mediated by the reticular activating system (Lindsley et al 1949).

The current study replicates our previous finding of lower EEG photic driving in schizophrenics and shows a robust increase in the response in depressed patients. The high sensi-

tivity and specificity of the test in differentiating the diagnostic groups suggest a potential clinical application.

This work was supported in part by FIRST Award MH-49237 (to Dr. Jin).

References

- Adrian ED, Mathews BHC (1934): The Berger rhythm: Potential changes from the occipital lobes in man. *Brain* 57:355-385.
- Andersen P, Andersson SA (1968): *Physiological Basis of the Alpha Rhythm*. New York: Meredith Corp.
- Brezny I, Gaziova M (1964): Late high voltage EEG responses to slowly repeated flashes. *Electroencephalogr Clin Neurophysiol* 16:383-387.
- Cantor FK, Ilbag F (1973): Facilitation of photic response by focal lesions. *Electroencephalogr Clin Neurophysiol* 34:77-79.
- Jin Y, Potkin SG, Rice D, et al (1990): Abnormal EEG responses to photic stimulation in schizophrenic patients. *Schizophr Bull* 16:627-634.
- Jin Y, Sandman CA, Wu JC, Bernat J, Potkin SG (1995a): Topographic analysis of EEG photic driving in normal and schizophrenic subjects. *Clin Electroencephalogr* 26:102-107.
- Jin Y, Potkin SG, Sandman C (1995b): Clozapine increases EEG photic driving in clinical responders. *Schizophr Bull* 21:263-268.
- Kleiber E, Broverman D, Vogel W, et al (1972): Effects of estrogen therapy on plasma MAO activity and EEG driving responses of depressed women. *Am J Psychiatry* 128:1492-1498.
- Kooi KA, Eckman HG, Thomas MH (1957): Observations on the response to photic stimulation in organic cerebral dysfunction. *Electroencephalogr Clin Neurophysiol* 9:239-250.
- Lindsley DB, Bowden JW, Magoun HW (1949): Effect upon the EEG of acute injury to the brain stem activating system. *Electroencephalogr Clin Neurophysiol* 1:475-486.
- Montagu JD (1967): The relationship between the intensity of repetitive photic stimulation and the cerebral response. *Electroencephalogr Clin Neurophysiol* 23:152-161.
- Oosterhuis HJGH, Ponsen L, Jonkman EG, et al (1969): The average visual response in patients with cerebrovascular disease. *Electroencephalogr Clin Neurophysiol* 27:23-34.
- Regan D (1972): *Evoked Potentials in Psychology, Sensory Physiology and Clinical Medicine*. London: Chapman and Hall.
- Regan D (1989): *Human Brain Electrophysiology: Evoked Potentials and Evoked Magnetic Fields in Science and Medicine*. New York: Elsevier, p 7.
- Rice D, Potkin SG, Jin Y, et al (1989): EEG alpha photic driving abnormalities in chronic schizophrenia. *Psychiatry Res* 30:313-324.
- Rodin EA, Bickford RG, Svien HJ (1953): Electroencephalographic findings associated with subdural hematoma. *Arch Neurol Psychiatry* 69:743-755.
- Shagass C (1972): *Evoked Brain Potentials in Psychiatry*. New York: Plenum Press.

- Speckman E-L, Caspers H (eds) (1979): *Origin of Cerebral Field Potentials*. Stuttgart: Thieme.
- Vogel W, Broverman DM, Kleiber E (1971): EEG responses in regularly menstruating women and in amenorrheic women treated with ovarian hormones. *Science* 172:388-391.
- Vogel W, Broverman DM, Kleiber E, et al (1974): EEG driving responses as a function of monoamine oxidase. *Electroencephalogr Clin Neurophysiol* 36:205-207.
- Walter VJ, Walter WG (1949): The central effects of rhythmic sensory stimulation. *Electroencephalogr Clin Neurophysiol* 1:57-86.