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An estimation of the first positive Lyapunov exponent of the EEG in patients with schizophrenia

Dai-Jin Kim[a], Jaeseung Jeong[b], Jeong-Ho Chae[a], Seongchong Park[b], Soo Yong Kim[b], Hyo Jin Go[a,*], In-Ho Paik[a], Kwang-Soo Kim[a], Bomoon Choi[a]

[a] *Department of Psychiatry, College of Medicine, The Catholic University of Korea, Seoul, South Korea*
[b] *Department of Physics, Korea Advanced Institute of Science and Technology, Taejon 305-701, South Korea*

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Abstract

We studied the complexity of the electroencephalogram (EEG) in schizophrenic patients by estimating the first Lyapunov exponent (L1), which might serve as an indicator of the specific brain function in schizophrenia. We recorded the EEG from 25 schizophrenic patients (12 male, 13 female; age = 25.1 ± 7.0 years) fulfilling DSM-IV criteria and 15 healthy controls (9 male, 6 female; age = 27.8 ± 4.2 years) at 16 electrodes, different from previous studies which recorded the EEGs at limited electrodes. We employed a method with an optimal embedding dimension to calculate the L1s. For limited noisy data, this algorithm was strikingly faster and more accurate than previous ones. Our results showed that the schizophrenic patients had lower values of the L1 at the left inferior frontal and anterior temporal regions compared with normal controls. These results for L1 in non-linear analysis have some differences from those for power ratios in linear analysis. These suggest that the non-linear analysis of the EEGs such as the estimation of the L1 might be a useful tool in analyzing EEG data to explore the neurodynamics of the brains of schizophrenic patients.

Keywords: Schizophrenia; EEG; Non-linear analysis; Lyapunov exponent; Complexity

* Corresponding author. Tel.: +82-32-340-2142; fax: +82-32-340-2670.

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1. Introduction

Although various EEG abnormalities in schizophrenia have been reported, it is generally agreed that no pathognomonic or characteristic EEG pattern is apparent on visual inspection (Itil, 1977). A number of studies of schizophrenia with power spectral analysis over the last 20 years revealed that there are no definite specific findings for EEG in the patients (Hughes, 1996). This may reflect the forcing of such a heterogeneous disorder as schizophrenia into group statistics, neglecting different subtypes, and activities of a schizophrenic process (Hughes, 1996). One relatively stable finding is the increased beta power of the EEG in schizophrenic patients (Itil et al., 1972; Itil, 1977). The increased beta activity may appear significantly on the left side, especially in the frontal area (Hughes, 1996). Gattaz et al. (1992) showed increased activities for fast alpha and beta bands in first-onset, neuroleptic-naive schizophrenics. The other consistent finding is increased slow-wave abnormalities on the left side, especially in the left anterior temporal area and occasionally in the left frontal and parietal areas (Karson et al., 1988; Gattaz et al., 1992). However, there are many limitations in using such a linear method because of the absence of an identified metric that quantifies complex behavior of the brain.

Recent progress in the theory of non-linear dynamics has provided new methods for the study of time-series data from human brain activities (Pradhan and Dutt, 1993). Babloyantz et al. (1985) first reported that the EEG data from the human brain had chaotic attractors for sleep stages II and IV. Much research with non-linear methods revealed that the EEG is generated by a deterministic neural process (Rapp et al., 1985; Babloyantz, 1988; Röschke and Basar, 1988; Soong and Stuart, 1989). According to these reports, the EEG has a finite non-integer correlation dimension and a positive Lyapunov exponent. Furthermore, the distinct states of brain activity had different chaotic dynamics quantified by non-linear invariant measures such as correlation dimensions and Lyapunov exponents (Babloyantz and Destexhe, 1987; Babloyantz, 1988; Pijn et al., 1991; Röschke and Aldenhoff, 1991; Fell et al., 1993; Wackermann et al., 1993).

On the contrary, there is some evidence that the EEG is not a chaotic signal of low dimension (Osborne and Provenzale, 1989; Theiler et al., 1992; Pritchard et al., 1995; Palus, 1996; Theiler and Rapp, 1996). The above studies showed that the normal resting human EEG was non-linear but did not represent low-dimensional chaos, and it may be generated from 1/f-like linear stochastic systems.

Regardless of what the true dynamics of the EEG are, non-linear analysis of the EEG to make the correlation dimension and/or the first positive Lyapunov exponent estimates have proved to be very useful in making relative comparisons of different physiological states (Rapp, 1993). Many investigations with these methods have revealed possible medical applications for non-linear analysis and have given rise to the possibility that non-linear analysis of the EEG might be a useful tool in differentiating physiological and or pathological brain states (Babloyantz and Destexhe, 1986, 1987; Frank et al., 1990; Pritchard et al., 1991, 1993, 1994; Stam et al., 1994, 1996; Besthorn et al., 1995; Fell et al., 1995; Jeong et al., 1998a; Lehnertz and Elger, 1998). In non-linear analysis, some pathological conditions such as epileptic seizures, coma, and dementia showed a decreased complexity in EEGs, whereas normal attentional states tended to show an increased complexity by the estimation of the correlation dimension (Rapp et al., 1989; Frank et al., 1990; Pijn et al., 1991; Lutzenberger et al., 1992; Pritchard et al., 1994; Stam et al., 1995; Jeong et al., 1998a; Lehnertz and Elger, 1998). In our analysis we regarded the first Lyapunov exponent as an operational definition and a measure of complexity instead of using it as an absolute measure to differentiate between periodic, chaotic or stochastic dynamics.

Classical algorithms for calculating non-linear invariant measures from the EEG data require a very large number of computations in the embedding process (Grassberger and Procaccia, 1983). Because the amount of data required for meaningful results was beyond the experimental possibilities for physiological data, there were many practical problems (Smith, 1988; Eckmann and

Ruelle, 1992). We used an algorithm of optimal embedding dimension, which was proposed by Kennel et al. (1992), to estimate the L1s fast and efficiently for finite noisy data. This algorithm is strikingly faster and more accurate than other algorithms (Jeong et al., 1998b).

A few reported studies for the non-linear analysis of the EEG of patients with schizophrenia have mainly estimated the non-linear invariant measures of the EEG in schizophrenia at small numbers of electrodes (Röschke and Aldenhoff, 1993; Koukkou et al., 1993). We tried to record the EEG from 16 channels in patients with schizophrenia in order to investigate the whole pattern of chaotic dynamics in the brain by the L1.

In Section 2, we explain the procedure for reconstructing brain dynamics from an EEG and for analyzing the EEG by linear and non-linear methods, as well as our algorithm for determining the optimal embedding dimension and for compensating for both noise contamination and edge effects. The L1 is also defined and discussed. The procedure for recording data is briefly presented. Section 6 shows the differences in the values of the L1 between the schizophrenic patients and the normal controls. In Section 7, we discuss our results with respect to both complexity in the EEG and the possibility of modeling the brain by non-linear dynamics. Our conclusions are also given in Section 7.

2. Subjects and methods

2.1. Algorithm

In non-linear analysis, first we reconstruct brain dynamics from a one-dimensional EEG by using delay coordinates and Takens' embedding theorem. Takens (1981) showed that an attractor, which is topologically equivalent to the original data set, can be reconstructed from a dynamical system of n variables x_1, x_2, x_3,..., x_n by using the

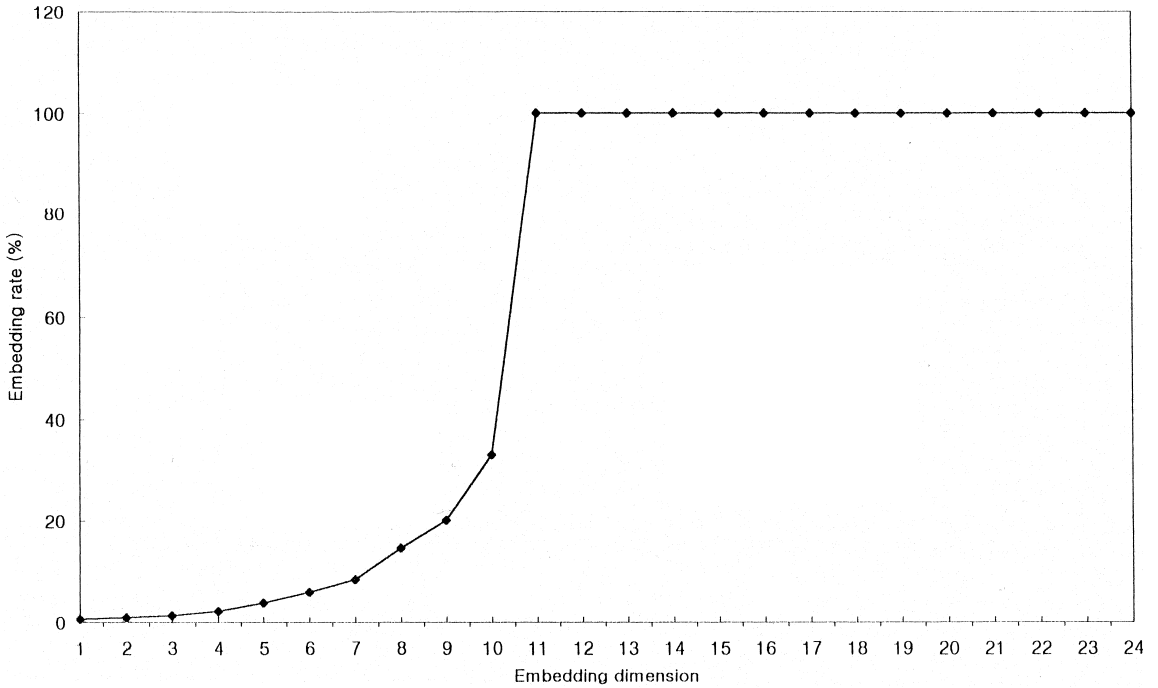

Fig. 1. The embedding rate as a function of embedding dimension for 16384 EEG data points at T4 in a normal control. The optimal minimum embedding dimension for calculating the L1 was selected as 11 in this case.

so-called delay coordinates $y(t) = [x_j(t), x_j(t + T),...,x_j(t + (d - 1)T)]$ from a single time series x_j and by performing an embedding procedure, where d is the embedding dimension. The purpose of time-delay embedding is to unfold the projection back to a multivariate state space that is a representation of the original system (Takens, 1981; Eckmann and Ruelle, 1985). For the time delay T, we used the first local minimum of the average mutual information between the set of measurement $v(t)$ and $v(t + T)$. Mutual information measures the general dependence of two variables (Fraser and Swinney, 1986).

We used the minimum (optimal) embedding dimension in the reconstruction procedure by the algorithm based on the idea that in the passage from dimension d to dimension $d + 1$, one can differentiate between points on the orbit that are 'true' neighbors and those on the orbit which are 'false' neighbors (Kennel et al., 1992). A false neighbor is a point in the data set that is a neighbor solely because we are viewing the orbit (the attractor) in too small an embedding space ($d < d_{\min}$). When we have achieved a large enough embedding space ($d \geq d_{\min}$), all neighbors of every orbit that points in the multi-variate phase space will be true neighbors. We define the embedding rate as the ratio of the true neighbors to the neighbors in the embedding dimension. Fig. 1 shows a typical example of the embedding rate as a function of the embedding dimension for 16 384 EEG data points at $T4$ in a normal control. The proper minimum embedding dimension was selected as 11 in this case. Next, we can estimate the L1 by calculating them only in the minimum embedding dimension, which is different from the conventional method (Wolf et al., 1985).

Lyapunov exponents estimate the mean exponential divergence or convergence of nearby trajectories of the attractor in the phase space. The first Lyapunov exponent, the highest value of the Lyapunov exponents of the attractor, reflects the sensitive dependence on the initial conditions, which is then a measure of dynamical complexity.

We calculate the first positive Lyapunov exponent L1 by applying a modified version of the Wolf algorithm (Wolf et al., 1985) and by following a proposal by Frank et al. (1990). Essentially, the Wolf algorithm computes the initial vector distance di of two nearby points and evolves its length at a certain propagation time. If the vector length df between the two points becomes too large, a new reference point is chosen with properties minimizing the replacement length and the orientation change. Now, the two points are evolved again and so on. After m propagation steps, the first positive Lyapunov exponent results from the sum over the logarithm of the ratios of the vector distances divided by the total evolving time:

$$L_1 = \frac{1}{m} \sum_{i=1}^{m} \frac{\ln \frac{df_i}{di_i}}{\mathrm{EVOLV} \cdot dt \cdot \ln 2} (\mathrm{bits}/\mathrm{s}) \qquad (1)$$

where dt, di, and df are the sampling interval, and the initial and the final separations between the points in the fiducial trajectory and in the nearest-neighbor trajectory separated in time by ith EVOLV step, respectively (Wolf et al., 1985). A more detailed description of the procedure is presented in our previous articles (Jeong et al., 1998a,b).

3. Subjects

Subjects were 25 schizophrenic patients (12 men, 13 women; age = 25.1 ± 6.96 years, mean ± S.D.) fulfilling DSM-IV (American Psychiatric Association, 1994) criteria and 15 healthy controls (9 men, 6 women; age = 27.8 ± 4.24 years, mean ± S.D.). None of the patients had a history of neurological disorder or drug or alcohol abuse. The effects of neuroleptic drugs expressed as chlorpromazine (CPZ)-equivalent doses (American Psychiatric Association, 1997) were analyzed with demographic data. The patients were taking neuroleptic medication with a mean dosage of 353.97 ± 319.36 mg (CPZ-equivalent). The 15 control subjects were healthy individuals with no history of psychiatric or neurological disease who were selected from 30 volunteers. All subjects

were right-handed and showed moderate amplitude (30–100 μV) on EEG background activities.

4. Data acquisition and analysis

The EEGs were recorded from the 16 scalp loci of the international 10–20 system. With the subjects in a relaxed state with closed eyes for 32.768 s (16 384 data points), data were recorded for the analysis and digitized by a 12-bit analog-digital converter in an IBM PC. Recordings were made under the eyes-closed condition in order to obtain as much stationary EEG data as possible. The sampling frequency was 500 Hz. Potentials from 16 channels (F_7, T_3, T_5, F_{p1}, F_3, C_3, P_3, O_1, F_8, T_4, T_6, F_{p2}, F_4, C_4, P_4, and O_2) against 'linked earlobes' were amplified on a Nihon Kohden EEG-4421K using a time constant of 0.1 s. All data were digitally filtered at 1–35 Hz in order to remove the residual EMG activity. Each EEG record was checked by inspection to be free from EOG and movement artifacts and to contain minimal EOG activity. Whenever a decrease in vigilance was detected on the ongoing EEG, the technician instructed the subject to open his or her eyes, and a short pause was allowed, if needed, to minimize drowsiness. The recordings were obtained at approximately the same time of day (usually in the afternoon).

The EEG was digitized and fast-Fourier transformation was performed. To calculate EEG power, the frequency spectrum was divided into 0.2-Hz bands and collapsed into EEG frequency bands of delta (1.0–3.9 Hz), theta (4.0–7.9 Hz), alpha (8.0–12.9 Hz), and beta (13.0–35.0 Hz). Each power value represented 5 s, and we analyzed 30 s of recording per case. Moreover, we designated these power values as average percentages of total power (Coben et al., 1983). These were normally called delta, theta, alpha, and beta power ratios.

The first step in non-linear analysis was to construct phase space using the delay coordinates proposed by Takens (1981). We used the time delays calculated by the method of mutual information to reconstruct the attractor. Time delays of 46–58 ms and embedding dimensions of 13–19 were used for the schizophrenic patients, and time delays of 28–32 ms and embedding dimensions of 11–19 were used for the control subjects.

The L1s were calculated for all subjects in all channels. The proper evolving time (EVOLV) was selected by using $1/e$ spectral frequency and was approximately 200 ms. The calculation of the L1 naturally depends on the time over which a trajectory is evaluated. After 200 propagation steps, the values converge at an interval of $\pm 0.9\%$ around the final value of the L1.

5. Statistical analysis

The data were analyzed by using SPSS (6.0 release version). Results of group data are expressed as mean $\pm$ S.D. Group differences between the schizophrenic patients and control subjects were evaluated by using Student's t-test. A two-tailed probability of less than 0.05 was considered to be statistically significant. Pearson correlation coefficients were used to examine the relationship of CPZ-equivalent dosage and the Lyapunov exponent of the patients with schizophrenia.

6. Results

The results of linear and non-linear analyses of the EEG for the patients with schizophrenia and for the normal control subjects in all channels are summarized in Table 1 (see, also, Fig. 2). Schizophrenic patients had lower average values of the L1 at the left inferior frontal (F_7: $P = 0.012$) and anterior temporal electrodes (T_3: $P = 0.025$) compared with controls. The differences between the values of the L1 at the F_7 and T_3 channels are approximately 0.5 units. These results clearly show that for each subject, but different channels, the values of the L1 do not vary within broad ranges. To consider the antipsychotic drug effect, we estimated the relationship between CPZ-equivalent dosage and the value of L1. The L1

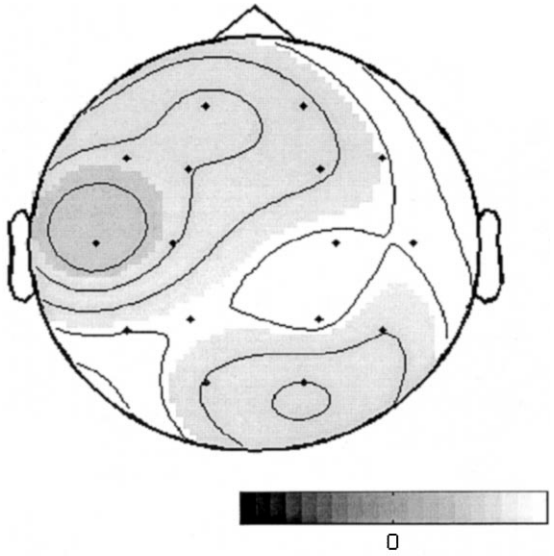

Fig. 2. Topological maps of the average values of L1 of the EEG for (a) schizophrenics and (b) normal controls.

does not correlate with the CPZ-equivalent dosage except at O_2 (Table 2). In the spectral analysis, schizophrenics had more alpha activity at F_4, F_7, F_{p1}, T_4, T_6, and O_1, less beta activity at F_7 and more theta activity at F_4, F_{p2}, T_6, C_3, C_4, and P_4 compared to controls (Table 1). However, the results of the spectral analysis are not consistent with those of the non-linear analysis.

7. Discussion

Non-linear analysis offers new tools for the investigation of information processing in the brain by analysis and classification of EEG signals. Compared with conventional spectral analysis, non-linear analysis of the EEG gives us information that reflects the dynamic properties of the whole brain.

The first positive Lyapunov exponent provides a new axis in terms of non-linear systems theory to analyze the dynamic attributes of EEG data (Fell et al., 1993). The L1 estimates the mean exponential divergence or convergence of nearby trajectories in phase space. The calculation of the first positive Lyapunov exponent expressing sensitive dependence on initial conditions provides much support for the hypothesis that the system under investigation is chaotic. There have been many publications about the evaluation of the Lyapunov exponent of EEG segments in epileptic seizures, electroconvulsive therapy, sleep, and Creutzfeld–Jakob coma (Babloyantz and Destexhe, 1987; Frank et al., 1990; Röschke and Aldenhoff, 1993).

To our knowledge, there is no distinct correlation between spectral estimates and non-linear measures such as L1 and D2 even though non-linear dynamical measures contain the information on linear properties of the signal as well as non-linear properties. Babloyantz and Destexhe (1986) reported that the bandwidth of the power spectrum of the EEG may be proportional to its correlation dimension. However, this effect is not reproduced in our study. Non-linear measures reflect the dynamical and non-linear properties of the signal, while the power spectrum depends on the statistical and linear properties of the signal.

There are some previous studies on complex behaviors of the schizophrenic brain. King et al. (1984) reported the results of a dynamic model of the central dopaminergic neuronal system and suggested that the chaotic solutions of the dynamic equations correlate with the increased vari-

Table 1
Comparisons of the first positive Lyapunov exponents, alpha activities, beta activities, theta activities, delta activities of the patients with schizophrenia and normal controls (independent samples *t*-tests)

Location	The first Lyapunov exponent					Alpha activities (%100)					Beta activities (%100)				
	Schizophrenia ($N = 25$)		Control ($N = 15$)		T	Schizophrenia ($N = 25$)		Control ($N = 15$)		T	Schizophrenia ($N = 25$)		Control ($N = 15$)		t
	Mean	S.D.	Mean	S.D.		Mean	S.D.	Mean	S.D.		Mean	S.D.	Mean	S.D.	
F_3	3.76	0.63	3.72	0.64	−0.23	0.364	0.138	0.346	0.185	2.01	0.315	0.116	0.327	0.109	0.96
F_4	3.93	0.67	3.97	0.44	0.23	0.315	0.069	0.374	0.081	2.33*	0.342	0.099	0.385	0.123	0.98
F_7	3.71	0.49	4.38	0.46	4.26**	0.316	0.097	0.393	2.54*	0.319	0.051	0.378	0.082	2.72*	0.192
F_8	4.14	0.41	4.84	0.33	1.94	0.322	0.092	0.344	0.115	0.93	0.354	0.117	0.383	0.103	0.91
F_{p1}	3.71	0.48	3.96	0.57	1.49	0.317	0.056	0.392	0.047	3.52**	0.360	0.128	0.386	0.131	0.63
F_{p2}	3.91	0.54	4.09	0.49	1.09	0.312	0.115	0.301	0.112	−0.71	0.343	0.121	0.376	0.114	0.46
T_3	3.37	0.49	3.93	0.39	3.78**	0.307	0.079	0.284	0.123	−1.19	0.312	0.112	0.339	0.117	1.51
T_4	4.19	0.55	4.14	0.59	−0.21	0.314	0.143	0.398	0.063	2.92**	0.327	0.113	0.334	0.117	0.88
T_5	4.16	0.47	4.12	0.68	−0.26	0.338	0.048	0.345	0.078	0.41	0.396	0.125	0.421	0.109	1.51
T_6	3.98	0.62	4.23	0.33	1.70	0.302	0.104	0.365	0.106	4.93**	0.370	0.089	0.395	0.098	1.90
C_3	3.75	0.54	4.03	0.51	1.65	0.370	0.087	0.387	0.115	0.56	0.373	0.146	0.399	0.121	1.01
C_4	4.22	0.38	4.18	0.37	−0.38	0.321	0.078	0.369	0.117	0.31	0.397	0.126	0.431	0.134	0.57
P_3	4.17	0.51	4.36	0.30	1.48	0.328	0.084	0.336	0.112	0.32	0.296	0.139	0.308	0.137	0.49
P_4	4.24	0.59	4.48	0.23	1.76	0.342	0.166	0.364	0.093	0.37	0.361	0.084	0.382	0.097	1.72
O_1	3.95	0.62	4.04	0.44	0.48	0.391	0.030	0.314	0.069	−2.22*	0.293	0.118	0.304	0.112	0.48
O_2	3.76	0.72	3.76	0.61	−0.02	0.367	0.108	0.347	0.087	−1.37	0.255	0.104	0.291	0.108	0.94

Table 1 (*Continued*)

	Theta activities (%100)					Delta activities (%100)				
	Schizophrenia $N = (25)$		Control ($N = 15$)		T	Schizophrenia ($N = 25$)		Control ($N = 15$)		t
	Mean	S.D.	Mean	S.D.		Mean	S.D.	Mean	S.D.	
F_3	0.126	0.043	0.102	0.033	−1.38	0.195	0.098	0.225	0.087	0.32
F_4	0.185	0.057	0.127	0.059	−2.67*	0.158	0.073	0.114	0.103	−0.34
F_7	0.192	0.060	0.168	0.078	−0.13	0.173	0.097	0.061	0.032	−2.90*
F_8	0.121	0.043	0.091	0.049	−0.42	0.203	0.057	0.192	0.41	0.24
F_{p1}	0.144	0.067	0.122	0.059	−1.25	0.179	0.056	0.100	0.065	−0.91
F_{p2}	0.124	0.047	0.076	0.031	−3.72**	0.221	0.076	0.247	0.79	0.77
T_3	0.137	0.064	0.142	0.048	0.85	0.244	0.065	0.235	0.077	−0.63
T_4	0.122	0.065	0.114	0.041	−0.13	0.237	0.087	0.154	0.089	−0.98
T_5	0.141	0.493	0.129	0.054	−0.68	0.125	0.076	0.105	0.043	−0.37
T_6	0.115	0.051	0.072	0.033	−2.15*	0.213	0.078	0.168	0.072	−0.52
C_3	0.157	0.061	0.094	0.034	−3.15**	0.100	0.065	0.120	0.46	0.25
C_4	0.115	0.066	0.079	0.024	−2.78*	0.167	0.087	0.121	0.062	−0.78
P_3	0.118	0.058	0.132	0.062	0.21	0.258	0.043	0.224	0.72	−0.47
P_4	0.102	0.054	0.074	0.033	−2.47*	0.195	0.071	0.180	0.064	−0.35
O_1	0.116	0.043	0.097	0.063	−1.72	0.219	0.052	0.285	0.059	0.57
O_2	0.119	0.046	0.092	0.037	−1.69	0.259	0.077	0.270	0.063	0.23

*Two-tailed $P < 0.05$.
**Two-tailed $P < 0.01$.

Table 2
Correlation coefficient and *P*-value between chlorpromazine-equivalent dosage and Lyapunov exponent of patients with schizophrenia[a]

Location	Chlorpromazine-equivalent dosage	
	Pearson correlation coefficient	Significance (*P*-value)
F_3	−0.353	NS
F_4	−0.194	NS
F_7	−0.185	NS
F_8	−0.161	NS
F_{p1}	−0.330	NS
F_{p2}	−0.156	NS
T_3	−0.007	NS
T_4	0.076	NS
T_5	−0.234	NS
T_6	0.071	NS
C_3	−0.012	NS
C_4	−0.200	NS
P_3	−0.339	NS
P_4	0.013	NS
O_1	−0.267	NS
O_2	−0.550	−0.004[b]

[a] NS, not significant.
[b] $P < 0.05$.

ability of behavior in schizophrenics. Koukkou et al. (1993) reported that the correlation dimension of the temporal–parietal EEG differed between first-episode schizophrenics and normal controls. It was significantly higher in the patients than in the controls. They suggested that the higher dimensional complexity of functional brain mechanisms in schizophrenics vs. normal control subjects is reminiscent of the loosened organization of thought and suggestions of certain superior abilities in the patients. Elbert et al. (1992) calculated the two descriptive measures (complexity and mobility) proposed by Hjorth and dimensional complexity of EEG from schizophrenic and normal subjects. They reported that most schizophrenic patients exhibited higher frontal dimensional complexity values than central ones. They suggested that the simultaneous perception of the delusional and the real world by schizophrenic patients might be based on increased information-processing power expressed through a higher correlation dimension, in which the frontal and the central dynamics are dissociated even in the relaxed waking state.

Our study is very different from previous studies that performed non-linear analysis of the EEG in schizophrenic patients. First, we estimated the L1 in 16 channels, while previous studies estimated the correlation dimension in limited channels. Röschke and Aldenhoff (1993) reported a statistically significant decrease of the correlation dimension during sleep stage II and rapid eye movement (REM) sleep in schizophrenic patients. Röschke et al. (1993) also reported that the L1 in REM sleep was significantly increased in schizophrenic patients compared with control subjects. From these findings, they suggested that the correlation dimension meant complexity and the Lyapunov exponent implied flexibility; therefore, their results pointed to altered non-linear brain dynamics during REM sleep in schizophrenia. In our results, we showed that schizophrenic patients in waking states had lower values of the L1 at the left inferior frontal and anterior temporal electrodes compared with controls. Those findings suggest that the complex activities at the left fronto-temporal area in a schizophrenic brain are lower than those in normal ones. We cannot

simply say that our results are opposite to the findings of Röschke and Aldenhoff (1993), since we measured the EEG of schizophrenic patients during waking relaxed states whereas they measured it during sleep states.

However, we found that there are significantly different complex behaviors between the brains of normal and of schizophrenic patients. Spectral analysis of the EEG showed that schizophrenics had more alpha activity and less beta and theta activities compared to controls on some electrodes. These results are consistent with other studies of spectral analysis in schizophrenia except beta activity (Karson et al., 1988; Gattaz et al., 1992), but a limitation of this study was the fact that antipsychotic medication was uncontrolled and may have affected EEG in the beta frequency band. These results differed from our non-linear analysis. Our results of decreased complex activities at the left fronto-temporal area in the schizophrenic brain are consistent with the findings of hypofrontality and hypotemporality reported in EEG studies on schizophrenia (Gattaz et al., 1992; Hughes, 1996), cerebral blood flow (Ingvar and Franzén, 1974; Paulman et al., 1990; Andreasen et al., 1992; Catafau et al., 1994), magnetic resonance imaging (Barta et al., 1990), and positron emission tomography (Buchsbaum et al., 1982, 1984). Investigations using power spectral analyses also suggested slowing of brain electrical activities, especially in the frontal regions (Morihisa et al., 1983; Morstyn et al., 1983). Several lines of evidence, including our results, imply that the frontal cortical area is dysfunctional in schizophrenia. Abnormalities affecting this region seem to be linked to impaired motivation, asocialization, and complex problems. The temporal lobe is also implicated in schizophrenia (Crow, 1990). Studies applying magnetoencephalography (MEG) in schizophrenia are consistent with a left-sided disturbance originating from the superior temporal gyrus region (Reite et al., 1989).

Our study revealed the hypofrontality and hypotemporality of the complexity in the left hemisphere of schizophrenic brains. It may be that the schizophrenic brain has decreased information processing and less flexible neural networks in the left fronto-temporal area.

One limitation of our study was the fact that antipsychotic medication was uncontrolled. Patients were taking neuroleptic medication with a range of CPZ-equivalent doses, 0–1000 mg. Although there was no correlation between the L1 and neuroleptic dosages and Itil et al. (1972) also reported no effect of medication on EEG activities in patients with schizophrenia, our findings cannot be taken as proof of the absence of drug effects. We speculate that the presently observed patterns of the L1 in the patients cannot easily be attributed to the effects of medication, but rather involve the psychotic processes. This speculation, however, must be substantiated by future comparisons of medicated and unmedicated patients. Although our present study is in a preliminary stage of development, its clear results encourage further investigation of the complexity in the brain of patients with schizophrenia.

In conclusion, our results showed that schizophrenic patients in waking states had lower values of L1 at the left inferior frontal and anterior temporal electrodes compared with controls. These results suggested that the left frontal and temporal areas may be key to the etiology of schizophrenia due to lowered information processing. The implications of complexity in the brain may shed light on our understanding of the brain and its pathological condition. Especially, non-linear measures of the electrophysiological activities in the brain may offer unique and fruitful perspectives for understanding important features of patients with schizophrenia.

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