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Quantitative measure of complexity of EEG signal dynamics

Włodzimierz Klonowski[1], **Wojciech Jernajczyk**[2], **Krystyna Niedzielska**[2], **Andrzej Rydz**[1] and **Robert Stępień**[1]

[1]Institute of Biocybernetics and Biomedical Engineering, Polish Academy of Sciences, 4 Trojden St., 02-109 Warsaw, Email: wklon@hrabia.ibib.waw.pl; [2]Laboratory of Clinical EEG, Institute of Psychiatry and Neurology, 1/9 Sobieski St., 02-957 Warsaw, Poland

Abstract. Since electroencephalographic (EEG) signal may be considered chaotic, Nonlinear Dynamics and Deterministic Chaos Theory may supply effective quantitative descriptors of EEG dynamics and of underlying chaos in the brain. We have used Karhunen-Loeve decomposition of the covariance matrix of the EEG signal to analyse EEG signals of 4 healthy subjects, under drug-free condition and under the influence of Diazepam. We found that what we call KL-complexity of the signal differs profoundly for the signals registered in different EEG channels, from about 5-8 for signals in frontal channels up to 40 and more in occipital ones. But no consistency in the influence of Diazepam administration on KL-complexity is observed. We also estimated the embedding dimension of the EEG signals of the same subjects, which turned to be between 7 and 11, so endorsing the presumption about existence of low-dimensional chaotic attractor. We are sure that nonlinear time series analysis can be used to investigate the dynamics underlying the generation of EEG signal. This approach does not seem practical yet, but deserves further study.

Key words: EEG, chaos, deterministic, nonlinear dynamics, principal components analysis, Diazepam

INTRODUCTION

Nonlinear Dynamics and Chaos Theory in EEG-signal analysis

"What is Chaos? It is this order which was destroyed during Creation of the Universe" said Polish poet and philosopher S.J. Lec. However, chaos is still present in our Internal Universe - our brain. Activity of the brain may be monitored by registration of electroencephalographic signals (EEG). Many studies have demonstrated systematic relations between EEG signal dynamics and different brain conditions, including those induced by drugs and alcohol. It has been suggested that controlling chaos in the brain may have important applications in medicine - for example, it may offer new opportunities to desynchronize the periodic behavior typical of epileptic seizures or Creutzfeld-Jacob disease. It seems that one statement remains true - it is healthy to be chaotic.

Since EEG signal may be considered chaotic, deterministic chaos theory seems to be a promissing method for supplying effective quantitative descriptors of EEG dynamics and of underlying chaos in the brain. We hope that efficacy of different drugs, and of other forms of therapy, e.g. phototherapy (in patients suffering of a very common form of depression, so called Seasonal Affective Disease, SAD), used in neuro-psychiatry, might be compared quantitatively using chaos theory, because the therapy does control chaos in the brain.

For example, the correlation dimension, D_2, estimates the number of degrees of freedom of the EEG signal, and further, it determines the number of independent variables which are necessary to describe the dynamics of the central nervous system (Roeschke and Aldenhoff 1991). Its numerical value describes the coherence of the underlying dynamics - the more coherent the system, the smaller the value of D_2. It was shown that D_2 decreases as the brain switches from α-waves activity ($D_2 \approx 6.1$, measured on the channel C4-P4) to deep sleep ($D_2 \approx 4.4$); in the pathological states the synchrony is still stronger ($D_2 \approx 3.8$ for Creutzfeld-Jacob coma and $D_2 \approx 2.05$ for "petit mal" epilepsy, on the channel C3-P3) (Gallez and Babloyantz 1991).

Computer-assisted EEG signal analysis increased the desire for effective quantitative interpretation of EEG data and of describing properties of the EEG which often cannot be perceived by human eye. We all would like to have much better understanding how our brain really works. But the "pedestrian" goal of our work is much simpler - using methods of Nonlinear Dynamics and Chaos Theory we search for as simple as possible descriptors of EEG signals, descriptors which may be relatively easily calculated and interpreted so to help doctors in appraising patient state (e.g. in studying drug abuse), in estimating therapy influence on the patient, and eventually in diagnostics. Traditional electroencephalography produces a large volume display of brain electrical activity, which creates problems particularly in assessment of long periods recording. Question arises how dynamical descriptors can be applied for the detection of the changes of the chaoticity of the brain processes measured in EEG. The number and variety of methods used in dynamical analysis has increased dramatically during the last fifteen years, and the limitations of these methods, especially when applied to noisy biological data, are now becoming apparent; their misapplication can easily produce fallacious results (Rapp 1994).

For characterisation of EEG time series we try to adapt Karhunen-Loève transform (KL-decomposition). Previous attempts to apply signal's orthogonal expansion by KL-decomposition (cp. Fuchs et al. 1992, Jirsa et al . 1995) and related methods of principal-component analysis or singular-value decomposition (SVD) (Broomhead and King 1986, Lutzenberger et al. 1995) seemed to be promissing. But much more sceptical results have also been reported (Lamothe and Stroink 1991). We introduce a simple quantitative descriptor, so called KL-complexity, hoping that it would be a useful measure which could be used to investigate the dynamics underlying the generation of EEG signal e.g. for the detection of the changes of the chaoticity of the brain processes caused by different drugs.

Embedology

To apply chaos theory in EEG analysis it is necessary to reconstruct attractors from EEG signals. The first step is to embed data in a multidimensional phase space. Embedding of data may be considered a science or an art by itself - embedology (Sauer et al. 1991). One can embed EEG signals using simultaneous coordinates (cf. Klonowski et.al. 1997). We agree with Ott et al. (1994) that "simultaneous measurements will often give superior results, and should be used, if available". In EEG data one has records from several channels, and the signals in different channels measured at any given moment of time may serve as generalized simultaneous coordinates. Only few EEG studies used the simultaneous coordi-

nates approach, using the number of channels as the embedding dimension (Dvorak 1990, Jirsa et al. 1995). It is also possible to reconstruct attractor from a one channel record, $x[n]$ $(n=1,...,N)$ using time-delay method (derivative coordinates) (Takens 1981). Unfortunately, Takens' theorem assumes the availability of an infinite amount of noise-free data (Casdagli et al. 1991, Rosenstein et al. 1994), while in EEG analysis we have noisy, finite data sets and only short stationary epochs.

An attractor may be reconstructed using the method of principal components analysis or singular value decomposition (SVD) (Broomhead and King 1986). It is known that the highest principal component exhausts maximum of the total variance, the second exhaust maximum of residual variance, etc. So any subspace spanned on M components ($M<K$, where K is the number of EEG channels considered) with the highest eigenvalues represents maximum of the total variance of data, and therefore provides an optimal projection of the original data into a space of lower dimension (Wackerman 1996). The original time series is projected into this new coordinate system. The extension of the generated attractor into the various directions of phase space is quantified by the associated singular values. Singular values close to zero mostly correspond to instrumental noise so that the associated space dimensions can be discarded. Since the singular values are ordered in size, the minimal embedding is obtained by ignoring all dimensions with singular values close or equal to zero, i.e. below a certain threshold, often expressed as a certain percentage (noise limit) of the maximum singular value. Lutzenberger et al. (1995) used a SVD decomposition of the EEG signal to determine the number of independent dimension for the reconstruction of the attractor.

While SVD is applied to so called trajectory matrix of the signal (cf. Methods below), we prefer to use Karhunen-Loève decomposition of the covariance matrix of the EEG signal. Results of both types of signal transformations are comparable. From our point of view the covariance matrix is a better starting point, as much of modern signal processing theory is concerned with the relationship between the eigenvectors of the covariance matrix and the frequency components of the signal.

METHODS

Recording of EEG data

In our investigation we analyzed EEG signals of four male probands, normal young adult subjects, under drug-free condition and under the influence of Diazepam (an anxiolytic, sedative drug, a derivative of benzodiazepine, known also as Valium or Relanium). Subject were volunteers. Routine rest waking EEG recording were performed on each subject.

First we recorded the EEG under drug-free conditions; then we applied an oral dose of 5.0 mg Valium and 1 h. later we recorded EEG again. The signals were re-

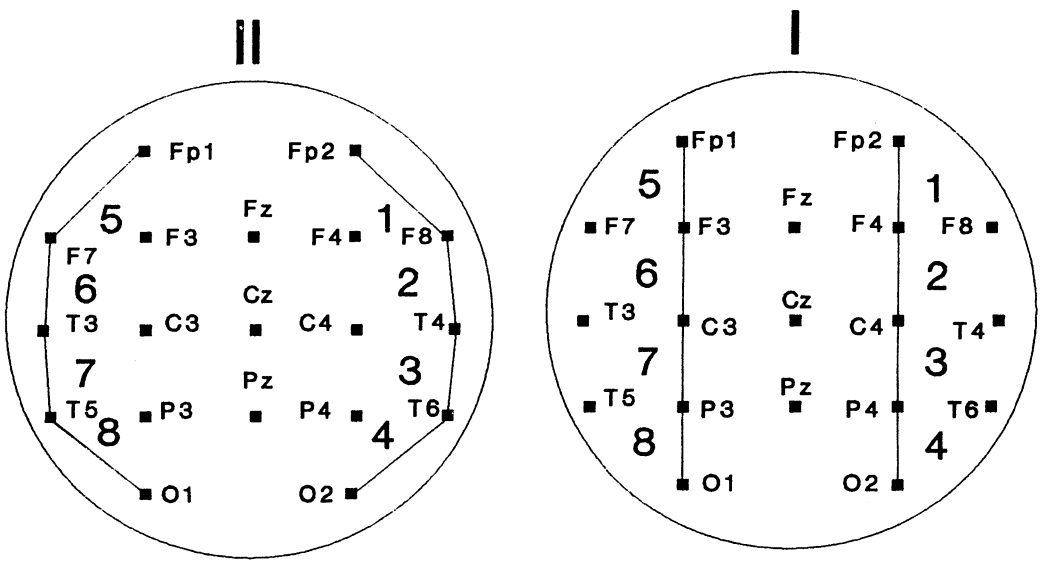

Fig. 1. The montage of 16 bi-polar connections (schemes I and II together) of the 10/20 placing system used in the present study. Head seen from above, nose up.

corded in the Laboratories of Clinical EEG, Institute of Psychiatry and Neurology, Warsaw. The EEG was acquired into a microcomputer system made by P.I.M. ELMIKO (Warsaw), based on PC Pentium. Signals were recorded on 16 channels, with standard 10/20 electrode placing (montage shown on Fig. 1). Digital EEG data were acquired at a sampling rate of 128 Hz per channel. No special selection of artifact-free time epochs was performed on the records prior to the computer analysis.

Signal analysis based on KL-decomposition

Our method is similar to that proposed by Ould Henoune and Cerf (1995). The digitalized EEG signal recorded on a certain channel is represented by the time series, $x(n)$. We choose certain number I ; we take first I values in the original series and we form a column vector $v_1=[x_1,...,x_I]$; then we take next I values and we form a column vector $v_2=[x_{I+1},...,x_{2I}]$ etc., each time moving I values to the right in the original series. This way Q vectors v_q are formed, where Q is the biggest integer smaller than (N/I). Trajectory matrix of the signal $A(t) = Q^{-1/2}[v_1, v_2 ,..., v_Q]^T$, and the IxI covariance matrix $A^T \cdot A$ are then formed. Then the KL-transform of the covariance matrix is performed.

The program named IntegralOperator which performs tasks described above were written by A. Rydz in structured 'C'. The numerical code is based on the book "Numerical Recipes in 'C'". The win version uses only the first 3,000 samples of data in the input file, whereas unwin version uses the whole available record, in the analyzed cases about 12,000 points. The program was implemented under UNIX and run on Silicon Graphics.

For each of the 16 signals, recorded from each subject before and after drug administration we calculated covariance matrices ranging from 10 by 10 to 150 by 150. The maximum number of terms in KL-expansion is equal to the covariance matrix dimension I , and is defined by the programmer for each run. The eigenvalues of the covariance matrix are then ordered and only

TABLE I

KL-complexity of multi-channel EEG (I = 60)

		P_1				P_2				P_3				P_4			
		unwin		win		unwin		win		unwin		win		unwin		win	
		b	a	b	a	b	a	b	a	b	a	b	a	b	a	b	a
1	Fp2 -F8	7	7	8	7	9	13	14	23	7	5	5	4	7	10	6	15
2	F8 - T4	20	13	25	17	21	30	28	27	13	8	13	12	5	17	5	23
3	T4 - T6	28	35	25	29	21	25	22	22	42	38	43	41	30	20	25	20
4	T6 - O2	32	33	27	29	24	23	21	21	24	29	24	26	21	23	18	22
5	Fp1 - F7	7	7	13	7	12	16	23	25	7	7	14	5	10	11	11	34
6	F7 - T3	36	18	27	20	21	27	25	23	13	14	28	11	17	27	23	48
7	T3 - T5	37	31	28	28	22	23	20	21	26	21	26	22	42	37	45	32
8	T5 - O1	36	33	28	31	22	21	20	19	25	24	24	24	34	21	38	19
9	Fp2 - F4	7	7	7	7	8	15	15	27	8	8	8	6	8	9	12	20
10	F4 - C4	25	21	24	20	20	25	24	25	21	20	21	23	11	18	17	22
11	C4 - P4	31	31	30	30	23	24	21	23	27	27	25	25	17	23	15	23
12	P4 - O2	27	31	24	28	23	23	23	22	21	23	21	23	17	21	14	21
13	Fp1 - F3	7	7	11	7	13	18	21	24	7	7	14	9	9	11	15	22
14	F3 - C3	7	17	22	19	23	29	25	24	24	17	25	24	11	14	12	16
15	C3 - P3	13	31	29	27	18	21	21	21	28	29	28	27	26	25	27	21
16	P3 - O1	35	31	28	30	17	22	21	21	23	25	23	24	29	21	30	20

P_S (s = 1,...,4) - subject's index; unwin - calculated from the whole available record; win, calculated from the epoch of the first 3,000 points; b, recorded before administration of the drug; a, recorded 1 h after administration of a single dose of Valium.

those no smaller than 0.01 of the largest one are retained; the total number of such eigenvalues, L, is then calculated. Since L is a measure of signal complexity, we proposed to call it the KL-complexity. We repeated the calculations for several values of the covariance matrix dimension, I, from 10 in steps equal 10 up to 150 (i.e. covariance matrices ranging from 10 by 10 to 150 by 150), until the greatest value of the KL-complexity obtained is about 66% of the value of I and does not change with furter increase of I. The values of L (for $I = 60$) are given in Table I.

Calculation of embedding dimension

Question arises would KL-complexity of a signal may be used as an estimate of the embedding dimension, D_E, when the signal is used for attractor reconstruction by time-delay method. To check this, we computed embedding dimension, D_E, for several channels (Fp2-F8, Fp1-F7, C3-P3, C4-P4, T5-O1, and T6-O2) which are supposed to be the most interesting from a point of view of a neurologist.

Taking the signal on the given channel in a time series representation, $x(n)$, where x is the signal amplitude at the discrete time moment n, we first construct vectors in the phase space by time delay method:

$$y(n) = [x(n), x(n+\tau), x(n+2\tau),..., x(n+(d_E-1)\tau)] \quad (1)$$

where time delay, τ, was calculated by the first zero crossing of the autocorrelation function:

$$Corr(\tau) = \sum_i x_i x_{i+\tau} \Big/ \sum_i x_i^2 \quad (2)$$

The embedding dimension, D_E, was determined by saturation of correlation dimension, D_2:

$$D_2 = \lim_{\varepsilon \to 0} \frac{\log C(\varepsilon)}{\log(\varepsilon)} \quad (3)$$

where correlation integral $C(\varepsilon)$ is defined as

$$C(\varepsilon) = \frac{2}{N(N-1)} \sum_{m=1}^{N} \sum_{n=m+1}^{N} \Theta(\varepsilon - \|\mathbf{y}(m) - \mathbf{y}(n)\|) \quad (4)$$

and $\|...\|$ denotes Euclidean norm of the vector, and $\Theta(z)$ is the Heaviside's step function:

$$\Theta(z) = \begin{cases} 1 \text{ if } z>0 \\ 0 \text{ otherwise} \end{cases} \quad (5)$$

That is, we repeat calculations of D_2 for subsequent values of d_E in (1); the value of d_E such that D_2 does not change any more when d_E further increases is assumed to be the embedding dimension D_E. The values of D_E are given in Table II.

RESULTS

The results of our calculations are summarized in Tables I and II.

In Table I one may observe that KL-complexity of the signal differs from about 5-8 for signals in frontal channels up to 40 and more in occipital ones. In general, the smallest KL-complexity is seen on frontal channels, where in healthy subjects there is no dominant wave frequency and frequency spectrum is relatively uniform. KL-complexity tends to increase towards the back of

TABLE II

Embedding dimension

		P_1		P_2		P_3		P_4	
		b	a	b	a	b	a	b	a
1	Fp2 - F8	8	8	9	10	9	8	7	10
4	T6 - O2	11	10	10	10	10	9	N.C.	9
5	Fp1 - F7	11	8	11	10	N.C.	8	11	11
8	T5 - O1	11	10	9	9	10	11	11	8
11	C4 - P4	11	11	10	10	10	11	N.C.	N.C.
15	C3 - P3	11	11	10	9	10	11	11	9

scalp, where the α-activity is usually much greater. Such pattern of changes is highly conserved when one compares for the given subject results obtained from shorter EEG epoch of 3,000 points (win version) with those obtained from the whole EEG record which was about 4 times longer, over 12,000 points (unwin version). It means that the applied method is not very sensitive to record's artifacts, since the artifacts has not been eliminated from the records before analysis.

Unfortunately it is seen from the Table I that no consistency in the influence of Diazepam administration on KL-complexity is observed, neither between different channels in one subject, nor between the same channels in different subjects.

In Table II one may observe that embedding dimension lies between 7 and 11, so endorsing the presumption about existence of low-dimensional chaotic attractor. This remains true also for the channels for which KL-complexity is quite high.

DISCUSSION

KL-decomposition like SVD-decomposition may be used to decompose an epoch of a multichannel EEG into multiple linearly independent (temporally and spatially noncorrelated) components, or features; the original epoch of the EEG may be reconstructed as a linear combination of the components; by omission of some component waveforms from the linear combination, a new EEG can be reconstructed, differing from the original in useful ways - for example features such as ictal or interictal discharges can be enhanced (Lagerlund et al. 1997).

The number of significant eigenvectors in KL-decomposition can be related to the number of original components forming a signal, but there is not a one-to-one correspondence between these eigenvectors and the individual components; furthermore, many, many eigenvectors may be needed to faithfully represent even a single source, if that source is nonstationary. We agree with Lamothe and Stroink (1991) that generally it would be inappropriate to ascribe any physiological significance to the data resulting from such KL-decomposition.

We think that KL-complexity, i.e. the number of terms in the KL-expansion of the covariance matrix necessary to characterize the signal with given accuracy, may be considered a simple phenomenological descriptor of the EEG signal dynamics. If so, it may be noticed that EEG signals in different channels depict properties of very different KL-complexity.

This big differences in KL-complexity between channels raise doubts about justification of chaos-theoretical approach using simultaneous coordinates for EEG signal analysis. It also raise questions in what extent is the information about EEG dynamics equivalent when extracted from different channels.

Hence, the method is not appropriarte for the purpose for which it was originally proposed, i.e. for assessing drug influence on the subject. Comparison of Tables I and II also demonstrates that KL-complexity is not a good estimate of the embedding dimension.

So, the obtained results are mainly negative. But we do think that negative results are also of importance (Klonowska and Klonowski 1988). Maybe it should exist "Journal of Negative Results", which unlike "Journal of Irreproducible Results" should be a serious scientific journal. Pushing for publication of positive *versus* negative results is an unethical or even fraudulent practice. Any prejudging what is "positive" and what is "negative", and not reporting the "negative" results, may be against scientific progress and scientific integrity.

KL-complexity and standard chaotic quantifiers we have tried (correlation dimension, Lyapunov exponents) seem to show no consistent pattern of changes when EEG-signals before and after therapy are compared (Stępień and Klonowski 1999). This is because of tremendous variability between individual subjects and non-stationarity of EEG signal. No statistical elaboration of the results will give more plausible conclusion. So these quantifiers are not suitable for therapy assessment and it is necessary to search for other EEG-signal quantifiers which could be more suitable for this purpose. We are sure that nonlinear time series analysis can be used to investigate the dynamics underlying the generation of EEG signal. This approach does not seem practical yet, but deserves further study. One of promissing nonlinear quantifiers on which we work in our Lab seems to be fractal dimension of EEG-signal itself, calculated directly from the time series representation, $x(n)$, of the signal, without necessity of constructing trajectory matrix, covariance matrix, and the system's phase space (Klonowski et al. 1999).

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