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Physics Letters A 329 (2004) 130–135
PHYSICS LETTERS A
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Detrended fluctuation analysis of human brain electroencephalogram

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Received 19 January 2004; accepted 21 June 2004

Available online 6 July 2004

Communicated by J. Flouquet

Abstract

With the detrended fluctuation analysis, we investigate dynamics of human brain electroencephalogram. Long-range temporal correlation and scaling behavior are observed, and certain characteristic of the Alzheimer's disease is revealed.

PACS: 05.20.Dd; 87.10.+e

Keywords: Nonequilibrium kinetics; Electroencephalogram

In the past years, much attention of physicists has been drawn to application of physical concepts and methods to complex biological, meteorological and economic systems [1–8]. Due to strong interactions among elements in the complex systems, *long-range* spatial and/or temporal correlations are often generated. These systems are then referred to be scale-free. A feature of such systems is the power-law scaling behavior. The scaling behavior usually is rather *robust* or *universal*.

The scaling behavior in complex systems is interesting not only in physical sense. It provides an intrinsic description of the systems, and may find application or potential application [2,3,9,10]. An example of complex biological systems is the time series of heartbeat. The *detrended fluctuation analysis* (DFA) is proved to be rather powerful in tackling this kind of fluctuating dynamics [2,9]. Careful analysis with the DFA method reveals different scaling behavior of the heartbeat dynamics for the heart failure patients and healthy individuals.

Recently, dynamics of human brain electroencephalogram (EEG) has been concerned [1,11]. The authors of Ref. [1] concentrate the attention on the α

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0375-9601/$ – see front matter
doi:10.1016/j.physleta.2004.06.070

and β peaks in the o and p channels, and discover that the *amplitude* of the oscillations is long-range correlated. The DFA analysis is used for demonstrating the long-range temporal correlation and dynamic scaling behavior. However, dynamic behavior beyond the α and β peaks remains untouched.

On the other hand, the Alzheimer's disease (AD) is a popular disease for aged people, human being severely suffers from it. Diagnosis of the AD disease is rather difficult. Traditional methods for analyzing EEG, such as mean values, standard deviations, power spectrum, etc., can hardly distinguish the healthy individuals and the AD patients. Many recent activities focus on non-linear dynamics of EEG, especially with the coherence analysis [12–16].

From the view of many-body systems, long-range temporal correlations of EEG should originate from the strong interactions of the neural cells. The AD disease would weaken or even block the interactions. Therefore, one may expect that the temporal scaling behavior of EEG would be sensitive to the individuals with or without the AD disease.

In this Letter, we will generally analyze the scaling behavior of EEG dynamics with *closed* eyes, considering the α peak as a '*perturbation*'. The DFA method will be applied to EEG of both the healthy individuals and AD patients, and certain characteristic of the AD disease will be revealed.

Our experimental data are of 20 healthy individuals and 14 AD patients with closed eyes. As shown in Fig. 1, o1 and o2 channels are measured on the occipital region, p3 and p4 channels on the parietal region, t3 and t4 channels on the temporal region, c3 and c4 channels on the central region, and f3 and f4 channels on the frontal region. Measurements are performed every $1/250$ second, and last for a total time $T = 130$ seconds. If not specified, the time unit is taken to be $1/250$ second in this Letter.

In the rigorous sense, EEG dynamics is not perfectly scale-free. The α and β peaks are some kinds of time scales of the system. Therefore, Ref. [1] restricts the DFA analysis and the calculation of the auto-correlations to the magnitude of the α and β oscillations in the o and p channels.

However, the α and β peaks are not very prominent beyond the o and p channels. In addition, the central frequency and the width of the peaks are channel-

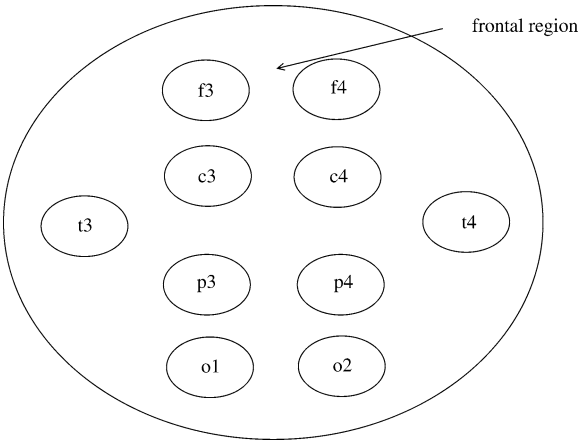

Fig. 1. Location of the 10 channels we investigate.

and individual-dependent. On the other hand, some characteristic of EEG dynamics, e.g., the effect of the AD disease, may be not centralized at the α and β peaks. Therefore, the purpose of this Letter is to go beyond the DFA analysis in Ref. [1], and to perform a relatively systematic DFA analysis of EEG dynamics.

Actually, from the power spectrum $P(f)$ of EEG based on the standard Fourier transformation, one cannot conclude that the dynamics is dominated by the α or β peak, even for the o and p channels. In Fig. 2, the power spectrum is displayed for the o1 and f3 channels. Taking into account that the highest frequency in the measurements is 250 Hz, one may believe the power spectrum is power-law-like. The α peak looks like a 'perturbation' around $f \sim 10$ Hz, which is somewhat stronger in the o1 channel. Therefore, we may generally expect scaling behavior in EEG dynamics, but with possible perturbative effects of the α peak.

We first introduce the DFA method [2,9]. For a fluctuating dynamic series $dB(t')$, we construct

$$C(t') = \sum_{t''=1}^{t'} \bigl[dB(t'') - dB_{\text{ave}}\bigr]. \tag{1}$$

Here dB_{ave} is the average of $dB(t')$ in the total time interval $[1, T]$. Then we uniformly divide the interval $[1, T]$ into windows with a size of t, and linearly fit $C(t')$ to a linear function $C_t(t')$ in each window. Fi-

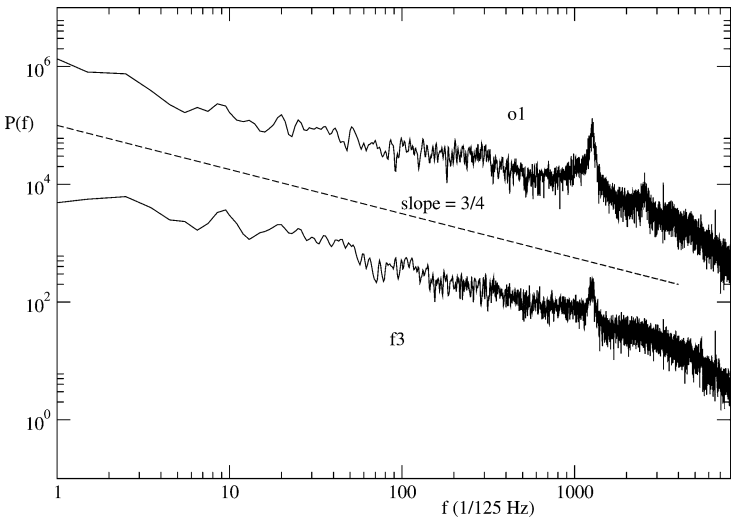

Fig. 2. Power spectra of EEG obtained with the standard Fourier transformation for the o1 and f3 channels of a single healthy individual. The slope of the dashed line is 3/4. For comparison, curves have been shifted suitably along y axis.

nally, we calculate the DFA function

$$F(t) = \sqrt{\frac{1}{T}\sum_{t'=1}^{T}\bigl[C(t') - C_t(t')\bigr]^2}. \tag{2}$$

In general, $F(t)$ obeys a power-law behavior

$$F(t) \sim t^{\theta}. \tag{3}$$

If $0 < \theta < 0.5$, $dB(t')$ is long-range anti-correlated; if $0.5 < \theta < 1.0$, $dB(t')$ is long-range correlated. $\theta = 0.5$ corresponds to the Gaussian white noise, while $\theta = 1.0$ indicates the $1/f$ noise. In the case that $dB(t')$ is long-range anti-correlated, measurements of $F(t)$ may suffer from fluctuations. To improve the measurements, we may perform the integration procedure in Eq. (1) one more time, and the DFA function $F(t) \sim t^{\theta+1}$ [2].

Denoting the EEG time series as $Y(t')$, we define the variation $dY(t') \equiv Y(t' + \Delta t') - Y(t')$. For our analysis, we take $\Delta t' = 1/250$ Hz. The DFA method can be applied to both $dY(t')$ and $Y(t')$, and the DFA function behaves like $F(t) \sim t^{\theta}$ and $t^{\theta+1}$, respectively.

In Fig. 3, the DFA function calculated with $Y(t')$ is displayed on a log–log scale. In order to achieve more accurate results, an average over all individuals has been performed. Such a 'grand-ensemble' average is also carried out in Refs. [1,11]. What we believe, however, is that this is not necessary in case the experimental data of a single individual is sufficiently long or repeated many times.

In Fig. 3, the deviation of the curves from the power law around $t = 25$ indicates the perturbation of the α peak, which is more prominent for the o channels and becomes rather weak for the f channels. After $t \sim 100$, $F(t)$ gradually converges to the power-law behavior. From the figures, one clearly observes that the curves of the AD patients increase faster than those of the healthy individuals.

In Table 1, we have listed the values of θ measured in Fig. 3. It is somewhat subtle to estimate the errors. The errors given in Table 1 are those from the grand average. Since our data for each individual are for a total time of 130 seconds only, these errors are possibly overestimated. Both θ_n for the healthy individuals and θ_a for the AD patients take the values smaller than 0.5. Therefore, EEG dynamics is long-range anti-correlated. In addition, both θ_n and θ_a show an increasing tendency from the o channels to f channels.

From the data in Table 1, θ_n and θ_a could be distinguished just within the errors. As stated above, the errors may be somewhat overestimated. On the other hand, $\theta_a - \theta_n$ is definitely positive for every channel. Therefore, we may believe that the AD disease *does* change the interactions among the neural cells, and therefore, the dynamic scaling behavior. Essentially,

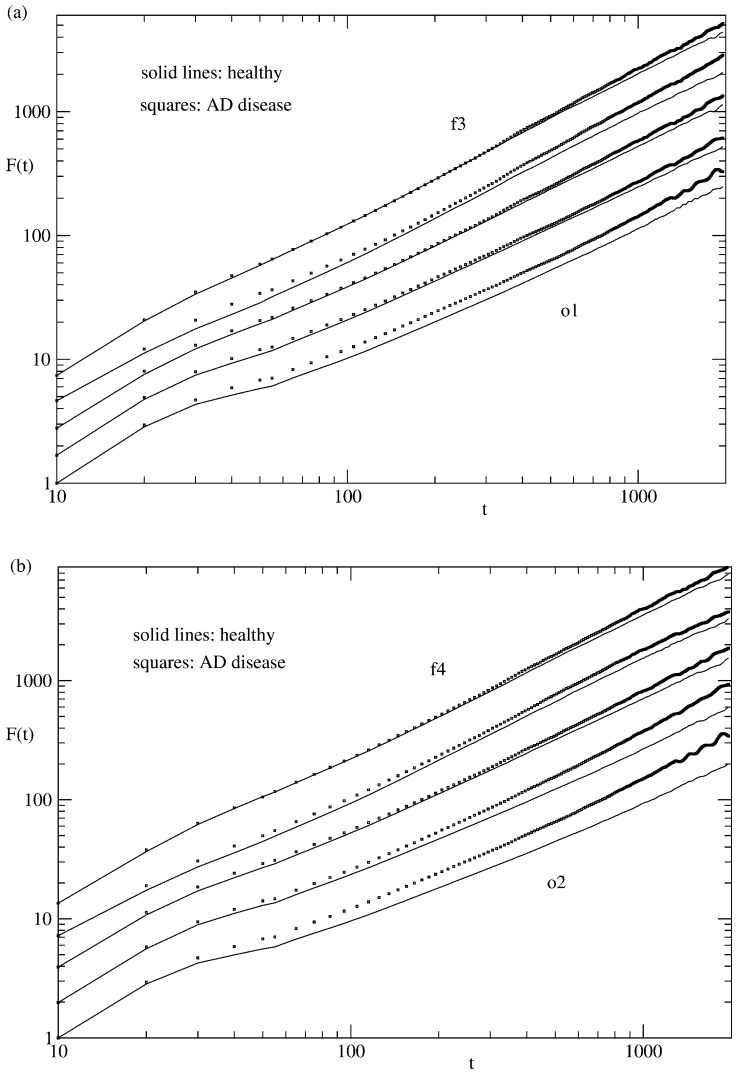

Fig. 3. DFA functions calculated with $Y(t')$. Solid lines and squares are for the healthy individuals and AD patients, respectively. From below, curves in (a) are of o1, p3, c3, t3 and f3 channels; in (b) are of o2, p4, c4, t4 and f4 channels. For comparison, curves have been shifted suitably along y axis.

Table 1
θ_n and θ_a are the scaling exponents of the DFA analysis calculated with $Y(t')$ of the healthy individuals and the AD patients, respectively

	o1	o2	p3	p4	c3	c4
θ_n	0.112(23)	0.069(21)	0.102(15)	0.132(29)	0.149(16)	0.144(14)
θ_a	0.188(43)	0.200(45)	0.165(33)	0.272(59)	0.219(28)	0.224(34)
$\theta_a - \theta_n$	0.076	0.131	0.063	0.140	0.070	0.080
	t3	t4	f3	f4		
θ_n	0.181(33)	0.188(22)	0.188(16)	0.202(18)		
θ_a	0.283(45)	0.213(27)	0.261(27)	0.255(32)		
$\theta_a - \theta_n$	0.102	0.025	0.073	0.053		

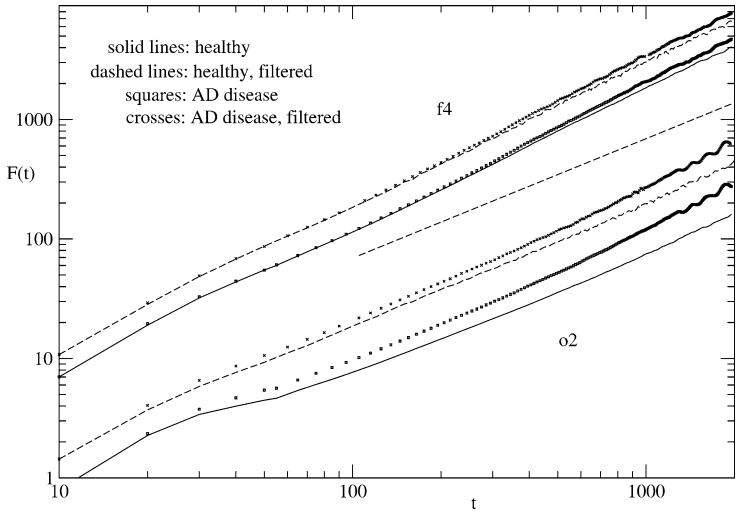

Fig. 4. DFA functions before and after filtering out the α peak. Solid lines and squares are for the healthy individuals and AD patients before filtering out the α peak, while dashed and crossed lines are after filtering out the α peak. Lower and upper groups of curves are for the o2 and f4 channels, respectively. The slope of the dashed line inbetween is 1.0.

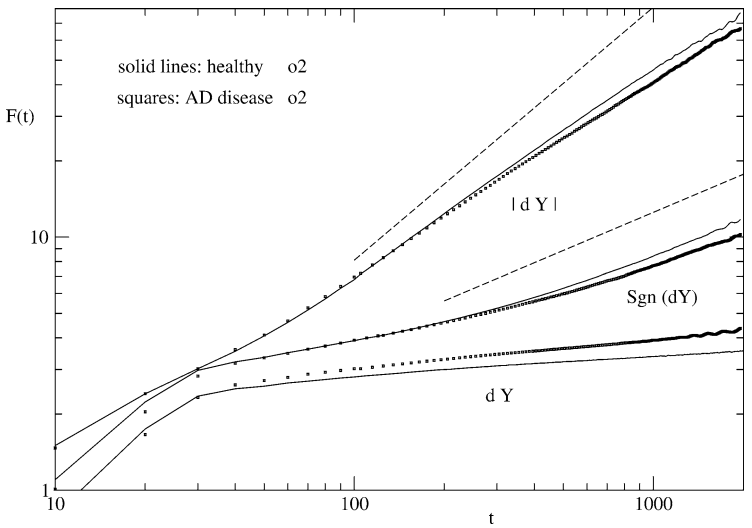

Fig. 5. DFA functions of the o2 channel calculated with $dY(t')$, $\mathrm{Sgn}(dY(t'))$ and $|dY(t')|$. Solid lines and squares are for the healthy individuals and AD patients, respectively. The slope of the lower dashed line is 0.5, while that of the upper dashed line is 1.0.

a bigger θ_a represents a slower dynamics for the AD patients. This is consistent with the previous finding that the amplitude of low frequencies in the power spectrum of EEG increases for the AD patients [12]. From the microscopic viewpoint, the slower dynamics should originate from the partial breaking down of the interactions among the neural cells. Details of this kind will be presented elsewhere.

How does the α peak affect the dynamic behavior of EEG? To answer this question, we first filter out the α peak of the power spectrum $P(f)$ in Fourier space, and then transform it back to the configuration space. We denote this refined time series as $Y_c(t')$. In Fig. 4, the DFA functions for both $Y(t')$ and $Y_c(t')$ are plotted on a log–log scale. For $Y_c(t')$, the waving around $t = 25$ is almost invisible. In later times, the curves of

$Y(t')$ and $Y_c(t')$ yield similar values of the exponent θ. Therefore, we conclude that the α peak only perturbs the dynamic behavior at early times, and does not dominate the effect of the AD disease.

Theoretically, application of the DFA analysis to $dY(t')$ should yield $F(t) \sim t^{\theta}$. In Fig. 5, $F(t)$ of the o2 channel is displayed for the healthy individuals (lower solid line) and the AD patients (lower squared line). The slops measured after $t \sim 100$ give $\theta_n = 0.071$ and $\theta_a = 0.118$, and confirm those values obtained with $Y(t')$. In general, however, application of the DFA analysis to $Y(t')$ here offers less fluctuating results.

To further understand the intrinsic behavior of EEG dynamics, we decompose $dY(t')$ into the magnitude series $|dY(t')|$ and the sign series $\mathrm{Sgn}(dY(t'))$ [2]. The DFA method is then applied to the series of $|dY(t')|$ and $\mathrm{Sgn}(dY(t'))$. In Fig. 5, $F(t)$ of the o2 channel calculated with $|dY(t')|$ is displayed for the healthy individuals (upper solid line) and the AD patients (upper squared line). Asymptotic slopes of the curves of the healthy individuals and AD patients give $\theta_n = 0.77$ and $\theta_a = 0.74$, respectively. These values indicate that the time series $|dY(t')|$ is long-range correlated. The power-law behavior of $F(t)$ calculated with $\mathrm{Sgn}(dY(t'))$ emerges only in later times. Anyway, the asymptotic slopes of the curves are around 0.4, indicating $\mathrm{Sgn}(dY(t'))$ is long-range anti-correlated. In Fig. 5, one may observe that the effect of the AD disease is more prominent in the time series $\mathrm{Sgn}(dY(t'))$ than in $|dY(t')|$.

To confirm our analysis with the DFA method, we have also calculated the auto-correlation function $A(t)$ of $|dY(t')|$. We observe a power-law decay $A(t) \sim t^{-\gamma}$, and find different slopes for the healthy individuals and the AD patients.

In conclusions, we have investigated dynamics of human brain EEG with closed eyes. Even though it is perturbed by the α peak, long-range anti-correlations (or correlations) are revealed with the detrended fluctuation analysis. The scaling exponent θ of the AD patients is different from that of the healthy individuals.

Acknowledgements

Work supported in part by DFG (Germany) under Grant No. TR 300/3-3, NNSF (China) under Grant Nos. 10325520 and 70371069 and SRFDP (China). The authors deeply thank Q.Y. Tong for the experimental data and H.J. Guo for discussions.

References

[1] K. Linkenkaer-Hansen, V.V. Nikouline, J.M. Palva, R.J. Ilmoniemi, J. Neurosci. 21 (2001) 1370.
[2] Y. Ashkenazy, P.Ch. Ivanov, S. Havlin, C.K. Peng, A.L. Goldberger, H.E. Stanley, Phys. Rev. Lett. 86 (2001) 1900.
[3] V. Schulte-Frohlinde, Y. Ashkenazy, P.Ch. Ivanov, L. Glass, A.L. Goldberger, H.E. Stanley, Phys. Rev. Lett. 87 (2001) 068104.
[4] K. Koscielny-Bunde, A. Bunde, S. Havlin, H.E. Roman, Y. Goldreich, H.J. Schellnhuber, Phys. Rev. Lett. 81 (1998) 729.
[5] R.N. Mantegna, H.E. Stanley, Nature 376 (1995) 46.
[6] V. Plerou, P. Gopikrishnan, H.E. Stanley, Nature 421 (2003) 130.
[7] F. Ren, B. Zheng, Phys. Lett. A 313 (2003) 312.
[8] B. Zheng, T. Qiu, F. Ren, Phys. Rev. E 69 (2004) 646115.
[9] C.K. Peng, S. Havlin, H.E. Stanley, A.L. Goldberger, Chaos 5 (1995) 82.
[10] N. Sapir, R. Karasik, S. Havlin, et al., Phys. Rev. E 67 (2003) 031903.
[11] P. Gong, A.R. Nikolaev, C. van Leeuwen, Neurosci. Lett. 336 (2003) 33.
[12] C. Besthorn, H. Förstl, C. Geiger-Kabisch, H. Sattel, T. Gasser, U. Schreiter-Gasser, Electroenceph. Clin. Neurophysiol. 90 (1994) 242, and references therein.
[13] C. Besthorn, H. Sattel, C. Geiger-Kabisch, R. Zerfass, H. Förstl, Electroenceph. Clin. Neurophysiol. 95 (1995) 84.
[14] L. Leocani, G. Comi, J. Clin. Neurophysiol. 16 (1999) 548.
[15] J. Jeong, J.C. Gore, B.S. Peterson, Clin. Neurophysiol. 112 (2001) 827.
[16] J. Jeong, T.H. Chae, S.Y. Kim, et al., J. Clin. Neurophysiol. 18 (2001) 58.