Unfolding dimension and the search for functional markers in the human electroencephalogram

Rudolf M. Dünki
Computer Assisted Physics Group, University of Zürich, Winterthurerstrasse 190, CH-8057 Zürich, Switzerland

Gary Bruno Schmid
General Psychiatry, Cantonal Psychiatric Clinic Rheinau, CH-8462 Rheinau ZH, Switzerland

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A biparametric approach to dimensional analysis in terms of a so-called ''unfolding dimension'' is introduced to explore the extent to which the human EEG can be described by stable features characteristic of an individual despite the well-known problems of intraindividual variability. Our analysis comprises an EEG data set recorded from healthy individuals over a time span of 5 years. The outcome is shown to be comparable to advanced linear methods of spectral analysis with regard to intraindividual specificity and stability over time. Such linear methods have not yet proven to be specific to the EEG of different brain states. Thus we have also investigated the specificity of our biparametric approach by comparing the mental states schizophrenic psychosis and remission, i.e., illness versus full recovery. A difference between EEG in psychosis and remission became apparent within recordings taken at rest with eyes closed and no stimulated or requested mental activity. Hence our approach distinguishes these functional brain states even in the absence of an active or intentional stimulus. This sheds a different light upon theories of schizophrenia as an information-processing disturbance of the brain. [S1063-651X(98)05602-5]

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I. INTRODUCTION

The human electroencephalogram (EEG) comprises electric signals reflecting the underlying neural dynamics of the brain. Accordingly, EEG attracts attention from different perspectives: on the one hand, researchers from biophysics, theoretical biology, and related disciplines are mainly interested in the mechanisms generating these signals [1–3]; on the other hand, physicians and psychologists are concerned with the identification of EEG patterns related to (mal) functional brain states [4–7].

A traditional approach linking these two perspectives is the assessment of the rhythmic activity in terms of time series analysis. The classical view of the EEG assumes the signal generator to be a linear oscillating system [1,8]. This approach has led to broad application of spectral analysis and related techniques to analyze and describe the EEG. Spectral patterns have been scrutinized for clinical relevance and it has been shown that these patterns carry individually specific information [9] that remains stable over time spans of up to several years. The diagnostic relevance of the EEG, however, presents another picture. A critical evaluation of the EEG research that has taken place over the past few decades offers a disappointing perspective on these classical approaches [10].

Until now there hardly seems to be a particular feature of the human EEG that clearly indicates specific psychopathological states or syndromes. Furthermore, it has not yet been possible to make reliable psychiatric diagnoses or prognoses with the help of these classical EEG approaches [11]. Hence there is a dire need for an alternative to these approaches to EEG analysis that places demands on the intraindividual stability and functional specificity of derived quantitative measures despite the well-known problems of intraindividual variability.

More recently, nonlinear time series analysis of EEG has become a focus of interest [12]: hints for possibly chaotic attractors underlying the EEG supply theoretically oriented scientists with information on degrees of freedom and the degree of dissipation to be explained by a model while, in practice, researchers wish to find the same measures of chaos to be of clinical relevance, e.g., for diagnostic purposes.

Regarding the most popular of these quantities, namely, the so-called D_2 or correlation dimension [13], however, things do not yet seem to be very promising: The extent to which the above-mentioned individual specificity can be reproduced (i.e., the extent to which D_2 is stable) remains unclear and the extent to which EEG is distinguishable from a linear stochastic process [14–17] is still debatable. This indicates a real need to improve correlation dimension analysis.

D_2 analysis starts with reconstructing an appropriate phase space. This is usually done by embedding a single (univariate) time series in m dimensions according to the method of time delays [18]. This makes use of the same signal m times: m successive points are regarded as independent coordinates each separated by one delay time τ from the preceding one:

$$\mathbf{X}_t = x(t), x(t-\tau), \ldots, x(t-(m-1)\tau). \tag{1.1}$$

In the case that several time series are simultaneously available, a parallel embedding scheme may be used [19] whereby the various channels included are regarded to be linearly independent and are therefore taken to comprise the m dimensions:

$$\mathbf{X}_t = x_1(t), x_2(t), \ldots, x_m(t). \tag{1.2}$$

Insofar as different EEG channels may represent different signal generators, the two approaches (delay-time coordi-

nates from a single location versus parallel embedding obtained simultaneously from the whole head surface) are not necessarily expected to lead to the same numerical results. Investigations into this question might be best done with the concept of mutual dimension [20,21]. Since we are in this work only concerned with differences between functional states and not with the number of generators responsible for each state, it suffices to select one of the above two approaches and simply stick with it. The procedure in either case is the same: Once the corresponding m-dimensional vectors are built, one calculates the so-called correlation integral [13] and evaluates therefrom the correlation dimension $D_2(m)$. Normally this procedure is repeated for a given series of measurements by successively increasing the embedding dimension m. For m sufficiently large, saturation for $D_2(m)$ is expected to occur and this saturation value is taken to be the correlation dimension.

A shortcoming of this approach lies in only using information for m sufficently large while discarding any information for low m. To overcome this shortcoming, the concept of ''unfolding dimension'' has been recently proposed. One hereby considers the following biparametrization [22]:

$$D_2(m) = b_0[1 - \exp(-m/m^*)]. \tag{1.3}$$

b_0 indicates the attractor dimension, while m^*, the so-called unfolding dimension, is a measure of the rate at which an attractor unfolds with increasing m. One thus takes information for low as well as for high m into account. An example is shown in Fig. 1.

This approach has already brought two interesting properties to light: first of all, applied to psychophysiological time series, it has become possible to statistically distinguish the human EEG of healthy persons from their corresponding so-called surrogate I or II data [14], i.e., data with the same Fourier spectra as the original, but without any phase correlation as one might obtain from a linear stochastic process. This was also achieved for cases in which traditional dimensional analysis failed to make this distinction (Fig. 2).

This distinction is important for a dimension algorithm because it proves the ability to retrieve information not visible within the power spectrum. On the other hand, failure to achieve this distinction would have shed doubt upon the overall relevance of dimensional analysis to the understanding of EEG signals, even though the relevance of surrogate-data testing in EEG analysis is not completely clear yet [17].

Secondly, this biparametric approach evidenced that the individual EEG of different healthy probands recorded over time did not turn out to be stochastic as monitored through the so-called confusion index [22] (see below). This nonstochasticity implies a certain statistically significant *intra*-individual stability and specificity of the human EEG in agreement with the above-mentioned findings of spectral analysis.

The distinction of the human EEG from its corresponding surrogate data as well as the nonstochasticity of the individual human EEG are prerequisites for any attempt to reach the goal of investigating functional brain states on a per individual basis by means of nonlinear dimensional analysis. Accordingly, the present work has two objectives: (i) To extend the analysis of a normative-EEG study to include up

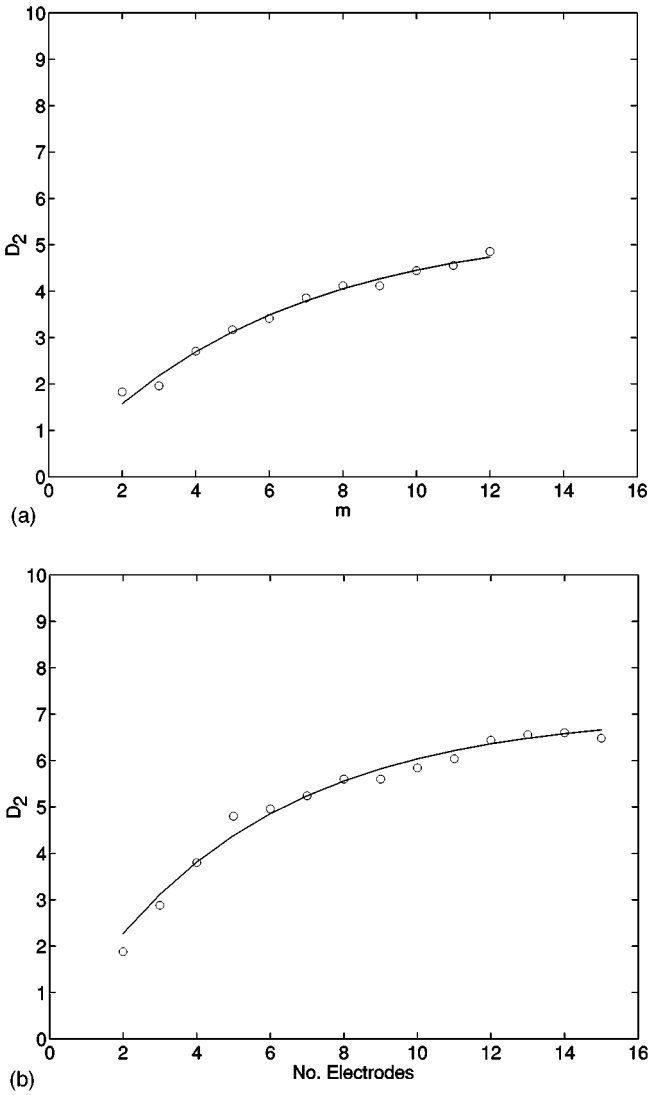

FIG. 1. Biparametric description of the curve $D_2(m)$ from two different human EEG samples. (a) The fit yields $b_0 = 5.44$, $m^* = 5.85$ (see text for explanations of symbols). The time delay embedding procedure [18] is applied. The embedding dimension m is defined as in Eq. (1.1). (b) The fit yields $b_0 = 7.04$, $m^* = 5.13$. The multichannel embedding procedure [19] is applied, i.e., each channel is assumed to be a coordinate in phase space. The embedding dimension is varied by adding the simultaneous signals from more and more electrodes.

to 8 artifact-free, 20-s EEG epochs and 2 channels on each of three widely separated recording days. (ii) To carry out, in the spirit of our biparametric approach, a meta-analysis of an earlier EEG-psychosis study in which—by means of conventional D_2 analysis—no statistically significant interindividual or intraindividual difference between psychosis and remission was evident [23].

The first objective is meant to substantiate the above-mentioned apparent tendency found in [22] (briefly outlined below in Sec. II) to support the idea that an individual's EEG is not stochastic over time, thus demonstrating the achievement of the above-mentioned prerequisites. We will see below that the indicated extension confirms the intraindividual stability over a period of at least 5 years whereby no pro-

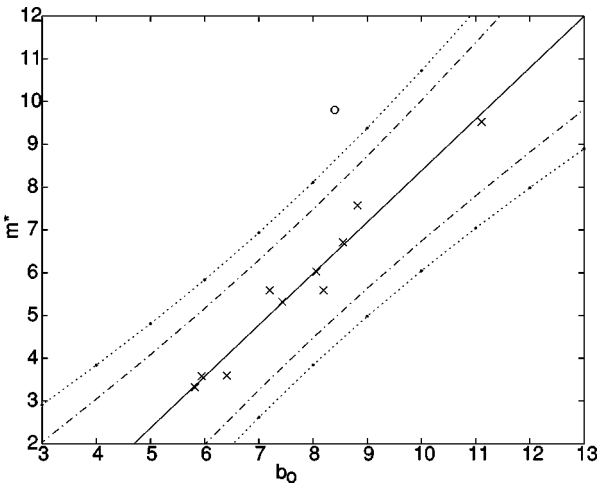

FIG. 2. Unfolding dimension m^* vs asymptotic correlation dimension b_0 [see Eq. (1.3)] for an EEG segment ($\bigcirc$) and 10 surrogates ($\times$) [delay-time embedding protocol (1.1)]. A linear regression for the latter yields the straight line (2.1) and the dashed-dotted (dotted) lines represent the 95% (99%) confidence level for the surrogates. If only the correlation dimension is considered (projection onto the abscissa), the correlation dimension of the EEG is indistinguishable from those of its surrogates. The biparametric analysis, however, reveals the nonlinear structure.

nounced channel dependence seems to be manifest. This is shown in Sec. III.

The second objective is meant to explore the idea that nonlinear analysis in terms of the biparametric approach to EEG may be useful to investigate different brain states. This is demonstrated on the particular problem of ''diagnosing'' the functional brain state of psychosis in comparison to the state of remission (full recovery) in the same person. We show in Sec. IV that the mentioned meta-analysis indicates a difference between the resting functional brain states, psychosis versus remission, even in the absence of external stimuli. This difference is mainly explained through differences in the respective unfolding dimensions.

In Sec. V, the outcome will be measured with the success of conventional dimensional analysis and advanced linear methods already used to test similar hypotheses [9,24]. By comparison, we regard the major impact of the present work to lie in its offer of a route for answering questions on the existence of stable and specific nonlinear EEG markers of nonreactive, functional brain states.

II. DATA AND METHODS

A. EEG data

Two different independent populations and analyses are involved in this investigation:

(a) An EEG normative group encompassing 23 healthy individuals (average age 28, minimum age 22, maximum age 33) recorded at rest with eyes closed at 3 different times: $t1$, $t2=t1+14$ days, and $t3=t2+5$ years as described in [22]. In the present work, we extend our previous analysis involving only one EEG channel, namely, $P3$-$O1$, to now include the EEG channels $T3$-$T5$ and $T5$-$O1$ according to the International 10-20 scheme [25] (Fig. 3) sampled at 256 Hz and low-pass filtered with 32 Hz. In addition, we now also take

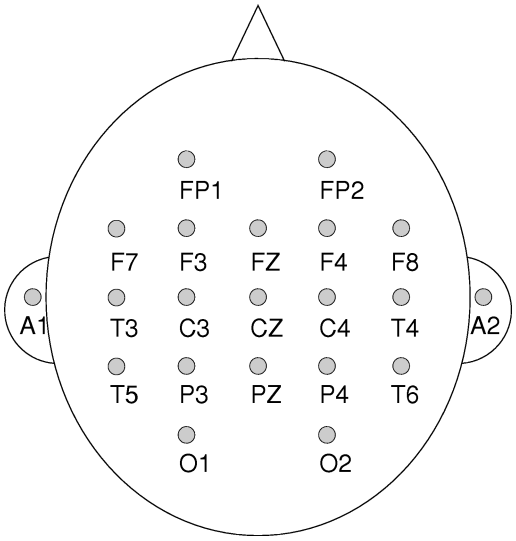

FIG. 3. Electrode positions according to the International 10-20 scheme.

several more artifact-free EEG epochs into account, namely, up to eight 20-s epochs per recording in comparison to only one epoch per day in the previous normative study.

(b) An EEG psychosis group encompassing the EEGs of 9 persons (4 males, 5 females) measured during each of two different functional brain states without medication: psychosis (average age 25.3, standard deviation 6.4) and remission (average age 26.0, standard deviation 5.1). Details as to the selection of probands, definitions of schizophrenic psychosis, and remission (i.e., full recovery from illness) are given in [24].

These data underlie Schmid and Koukkou's [23] Table 2, which has been here reinvestigated to yield the parameters b_0 and m^* for each individual and epoch in both psychosis and remission (whereby at most four 20-s artifact-free epochs per individual and state were available). Figure 4 offers an example of an EEG segment from the same person in a

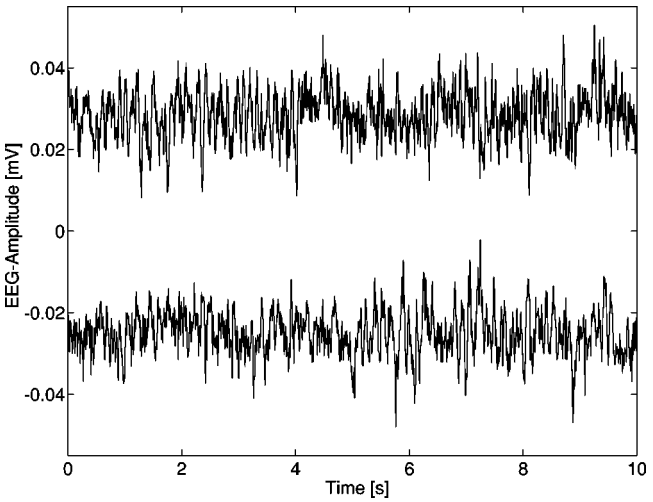

FIG. 4. EEG segments of the same proband in psychosis (upper curve) shortly after admission and in remission (lower curve) before leaving the hospital. Bipolar recording ($O1$-$T5$) measured at rest with eyes closed. For better visibility, the curves have an offset of plus and minus 25 μV, respectively.

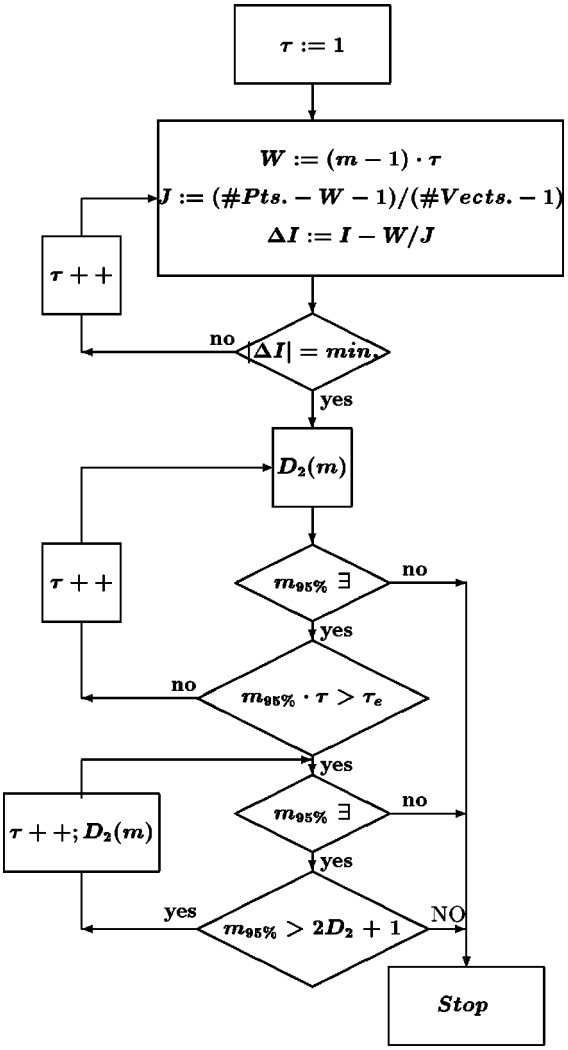

FIG. 5. Delay-time protocol: *#Pts*, number of points within the time series; *#Vects*, number of vectors to be included in the analysis; m, embedding dimension; τ, delay time; I, length of Theiler's [26] window as in [22]; W, time window spanned by one embedding vector; J, time window between two successive embedding vectors; τ_e, time at which the autocorrelation function drops below $1/e$ of its initial value; $m_{95\%}$, minimal embedding dimension; $m_{95\%}$ $\exists$; $D_2(m_{95\%})$ observable; NO, normal exit for chaotic systems; no, disturbance of the signal (e.g., due to noise) may force these exits.

psychotic state and then later in remission.

B. Computational methods

The algorithm used for both studies mentioned above has already been described in great detail [22]. Because of its importance to the present work, we briefly mention here the essential properties and primary results of this algorithm.

Special features of our algorithmic method encompass an operator-user–independent, automatic, and reproducible specification of both an ''optimal'' time delay τ for calculating embedding vectors as well as an ''optimal'' (scalar invariant) plateau region for the extraction of the correlation dimension D_2. The former is based on simultaneous considerations of Theiler's window parameter [26], the signal's autocorrelation time τ_e, and certain requirements concerning minimal embedding dimension (Fig. 5). In the case of our EEG data, τ was suggested to be 1. The set of D_2 values for a sequence of embedding dimensions m is then parametrized according to Eq. (1.3). The outcome for test systems assessed this way is shown in Table I. The detailed description, the many tests for compatibility with other recent embedding criteria [27–29] and techniques estimating delay times [30–32], the assessment of error bars, and outcomes are presented in [22].

TABLE I. Dimension estimates for several test systems.

System	b_0 [:= $D_2(m=\infty)$]	m^*
Henon [a]	1.23	0.58
Rössler [a]	1.89	0.53
Lorentz [a]	2.08	1.07
NMR signal [b]	3.2	2.4 [c]
Mackey-Glass [d]	7.0	4.3

[a]See Table 1 in [22].
[b]Hyperchaotic region [J. Simonet (private communication)]; see [40] for details.
[c]From assessments $D_2 = b_*\{1-\exp[(-m/m^*)^\gamma]\}$. To estimate m^*, we made a m^* vs γ plot for various $\gamma>1$ and extrapolated it to $\gamma=1$.
[d]$\tau=100$; $T=0.1$ [41] employing the scheme of Ding *et al.* [27]. The result is the average from several runs with 7000 vectors equidistantly sampled from (a) 140 000 and (b) 210 000 iterations.

In this representation, the parameter m^* is referred to as the ''unfolding dimension'' insofar as it indicates the rate at which an attractor unfolds as the embedding dimension increases (i.e., the initial rate in Fig. 1). For the sake of completeness, we note that the exponential form of the unfolding described in Eq. (1.3) was found heuristically. We therefore do not exclude the possibility that similar parametrizations might also work (see, e.g., Table I, footnote c). Furthermore as is the case with D_2, m^* is not immune to false specifications of the delay time τ.

From a theoretical point of view, the relation between b_0 and m^* allows certain inferences about minimal embedding criteria [22]. Special emphasis has been given to the b_0-m^* relation via two different approaches. The principle underlying these approaches becomes obvious from Fig. 2. We have found that the relation between b_0 and m^* can be expressed in terms of linear regression:

$$m^* = s b_0 + i, \tag{2.1}$$

using values of b_0 and m^* calculated either from different segments of (i) surrogate data belonging to one particular EEG segment [14] and (ii) different segments of real data from a particular proband. The parameters s and i are then determined from such a sequence of pairwise measurements (b_0, m^*). In the first, rather formal approach (i), the latter are assumed to be outcomes of different realizations of the same process, describable as a biparametric population through Eq. (2.1). Standard statistical techniques are then used to test whether the outcome of the original EEG segment might also belong to this population or if it should rather be regarded as distinct. An example illustrating this approach is shown in Fig. 2. Our biparametric description thus allows for a successful distinction of raw EEG from its surrogates even in

cases when traditional dimensional analysis does not enable such discrimination. Hence our treatment provides information deducible neither from power spectra nor from standard (uniparametric) dimensional analysis.

Having adressed the distinction between real data and its corresponding surrogates for a given EEG segment, we now turn to (ii). This second, clinically related approach to the b_0 versus m^* relation was found from analysis of EEG segments of the same person obtained over five years. Here, the concept of the so-called ''confusion index'' has been applied [22]. This quantity can be thought of as an ordinal distance measuring how near the outcomes of one and the same person are situated to each other. The smaller this distance is, the more related are the outcomes. It has been shown that this concept applied to a threefold estimation of the correlation dimension of several persons indicated stochasticity, i.e., no hints for a person's EEG to be longitudinally stable. However, when applying the same approach to the regression lines (2.1) defined through the three b_0-m^* outcomes, such longitudinal stability could indeed be retrieved (see especially Fig. 4 from [22]). Based on these tests, we decided to choose this algorithm to assess the aforementioned stability and functionality of the human EEG. Note that we do not intend to analyze here the surrogate data problem for data collected over an extended time period. This would require us to explore the extent to which any longitudinal information displayed by a nonlinear measure could be available from a linear description of the underlying EEG. We concentrate instead on retrieving this longitudinal information by means of our nonlinear approach. To know that longitudinal information exists is the prerequisite for studying linear and/or nonlinear performance behavior.

III. ASSESSMENT I: LONGITUDINAL STABILITY AND INTRAINDIVIDUAL SPECIFICITY

The results for the extended EEG normative study are summarized in terms of mean and standard deviation per day and channel [Table II(a), Fig. 6] as well as correlations made intraindividually between days 1 and 2, 1 and 3, and 2 and 3 [Table II(b)].

To ensure that only reliable correlations enter into the statistics, certain precautions had to be taken: correlation coefficients may be considerably biased in the presence of unreliable (b_0, m^*) pairs. Hence the assessment of slope s and intercept i from the (b_0,m^*) pairs is crucial for our analysis. To assure reliability, each slope s from the set of values $\{s\}$ had to satisfy certain quality selection criteria:

(1) Fit reliability: only properly resolved pairs (b_0, m^*) were considered (i.e., b_0 smaller than the cutoff value indicating nonconvergence, no two successive unresolved scalar invariant plateaus for $m \geqslant 9$, total squared difference between fit and experiment < 2.5).

(2) Statistical reliability: (a) From the EEG epochs of a given day, channel and person (maximum: 8), at least 5 such (b_0, m^*) pairs must be available and $\max(b_0) - \min(b_0) > 1$ fulfilled. (b) The correlation coefficient $\rho(b_0, m^*)$ for the regression curve fitted to these pairs must be greater than 0.85.

Failure of one of these criteria led to rejection of the slope estimate for correlation analysis in roughly 40 % of the cases. Since standard statistical tests rely upon a normal distribution, the apparently skewed distribution of the slopes s (according to the Bowmann-Shenton test [33]) has been corrected with an empirically found variance stabilizing transformation: $s\_ := s/[1+(s/c)^a]^{1/a}$. The constants a and c are chosen such that the distribution of the tranformed slopes $s\_$ is not significantly different from a normal distribution. The transformed slopes thus fulfill the requirements for standard statistical tests. Accordingly, this approach has advantages over the alternative use of the confusion index mentioned above, where ad hoc assumptions on the distribution entering into the analysis had to be made.

TABLE II. (a) Mean (μ) and standard deviation (σ) of the transformed ($a = 10$, $c = 2.5$, see text) slopes $s\_$, for channel 1 ($T3$-$T5$) and channel 2 ($T5$-$O1$) (No. $s\_$ is the number of transformed slopes available for analysis). (b) Intraindividual correlations ρ of $s\_$ between different days (ΔTime is the time span between day 1 and day 2; Level is the significance level).

(a)

Day	Channel	$\mu(s\_)$	$\sigma(s\_)$	No. $s\_$
$t1$	1	1.75	0.35	5
$t1$	2	1.72	0.41	10
$t2$	1	1.55	0.23	9
$t2$	2	1.59	0.26	13
$t3$	1	1.70	0.28	10
$t3$	2	1.68	0.19	9

(b)

Day 1	Day 2	ρ	Level	No. $s\_$	ΔTime
$t1$	$t2$	0.69	$>95\%$	10	$14d$
$t1$	$t3$	0.76	$\geqslant 95\%$	7	$5y+14d$
$t2$	$t3$	0.46	n.s.	11	$5y$
$\langle t1+t2\rangle$	$t3$	0.51	$>95\%$	16	$5y/5y+14d$

Our results [cf. Table II(a), Fig. 6] reveal no significant

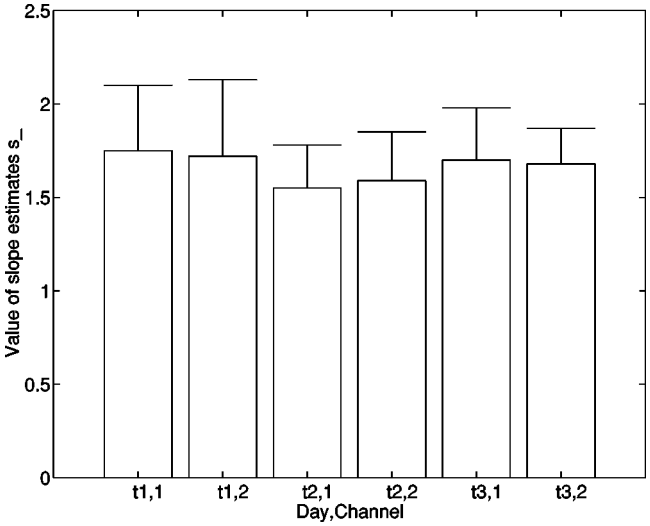

FIG. 6. Statistics of the transformed slopes $s\_$ [see Eq. (2.1) and text to Sec. III]. Mean values (bars) plus one standard deviation (T). Day, channel labelings: channel 1 = $T3$-$T5$, channel 2 = $T5$-$O1$; $t1$ = reference day, $t2 = t1+14$ days; $t3 = t2+5$ years.

differences between channels or days. However, they showed significant correlations between the outcomes of healthy probands intraindividually between recording days [cf. Table II(b)]. This is in agreement with earlier findings based on advanced linear methods [9,34]) and reveals a statistically significant relation of a person's EEG—recorded under these conditions—to itself over periods spanning up to *five* years. Hence these slopes are to some degree both intraindividually specific and longitudinally stable and may thus serve (in the statistical sense) as an individual's marker.

IV. ASSESSMENT II: SPECIFICITY OF FUNCTIONAL BRAIN STATE

Table 2 of [23] evidences a statistically significant difference in $\Delta D_2(m) := \langle D_2(m:P) - D_2(m:R) \rangle$, interindividually, between the functional states, psychosis (P) and remission (R), in terms of the permutation of all individuals for each of circa 8 to 10 embedding dimensions m per EEG channel. The fact remains, however, that for different functional states, the differences between the asymptotic values of D_2, namely, b_0 in the above exponential fit, were not found to be statistically significant. This leaves open the question as to whether or not the above-mentioned difference in $\Delta D_2(m)$ can be explained in terms of our biparametric approach.

As a first attempt toward an answer to this question, we applied our biparametric approach to the values underlying the presentation of the above-mentioned Table 2 of [23]. In particular, we employed the same selection criteria used for correlation as in the normative study of Sec. III [an exception: data set size required that we relax the number of (b_0, m^*) pairs to 4]. As a result, we have indeed found a significant difference in the mean of all $D_2(m)$ curves between psychosis and remission, thus confirming the above-mentioned evidence based upon Table 2 of [23]. Moreover, we did not find any single value of m at which the mean difference indicated an opposite sign, i.e., the difference $\delta(m:P-R) := \langle D_2(m:P) \rangle - \langle D_2(m:R) \rangle$ was always positive (cf. Table III). Under the null hypothesis of having no difference at all, we would expect this finding to occur with probability $\leqslant 0.01$. We thus accept this difference and ask about its interpretation.

The biparametric view offers three separate explanations, namely, a difference in the asymptotic correlation dimension b_0, a difference in the unfolding dimension m^*, or a difference in both. The qualitative behavior of the set of $\delta(m:P-R)$ differences strongly favors the second explanation because of the higher differences for low or intermediate m (cf. Table III). Akaikes information criterion (AIC) offers a way to decide this question quantitatively. The AIC tries to find a balance between decreasing the sum of squares (a desired property, because the difference between fit and data becomes smaller) and increasing the number of parameters needed for such a fit (an unwanted property, because it bears the risk of overfitting). The balance (best solution) is monitored through the minimum in AIC [35]. When explaining the mean $D_2(m)$ curves in terms of our model, we have found satisfactory agreement when either only m^* (AIC of -22.0, best solution) or both m^* and b_0 (AIC of -21.7) were assumed to be unequal. The worst agreement was obtained when assuming a difference in b_0 (AIC of -19.0) only. This suggests that the difference is primarily due to the unfolding properties rather than to the correlation dimension itself. The fact that b_0 enters as a simple factor and m^* as a factor in an exponential function does not affect this conclusion because one fits the models to a curve given *a priori* and the amount of the parameter difference plays no role, i.e., the potientially richer behavior due to changes of m^* does not enter.

TABLE III. Difference $\delta(m:P-R)$ $[=\langle D_2(m{:}\mathrm{P})\rangle - \langle \mathrm{D}_2(m{:}\mathrm{R})\rangle]$ between the mean value of psychosis $[D_2(m:P)]$ and the mean value of remission $[D_2(m:R)]$ vs embedding dimension. A peak for intermediate embedding dimensions and a decay for higher embedding dimensions are in good agreement with the outcome of the difference of two biparametric curves with different unfolding dimensions but equal correlation dimensions. The third column gives the difference obtained with the best solution (minimum AIC with $b_0=5.4$, $m^*_{\mathrm{psychosis}}=6.17$, $m^*_{\mathrm{remission}}=6.90$).

m	$\delta(m:P-R)$	Minimum AIC solution
03	0.03	0.17
04	0.40	0.20
05	0.29	0.21
06	0.24	0.22
07	0.34	0.22
08	0.36	0.22
09	0.05	0.21
10	0.25	0.20
11	0.03	0.19
12	0.25	0.18

V. DISCUSSION

The outcome of our first objective—to extend our normative EEG study—satisfactorily supports the idea mentioned at the beginning of the Introduction, namely, that it is possible to relate human EEG to the individual from whom it is obtained and that this relation remains stable over time. This means that biparametric analysis in terms of the b_0 versus m^* relationship displays evidence for the longitudinal stability and intraindividual specificity of the human EEG, at least over 5 years. This fact conforms to earlier results obtained with advanced linear approaches [34,9]: depending on the spectral parameter, correlations between 0.43 and 0.93 with a median of 0.76 were found [36]. Thus our nonlinear method can be considered to at least redisplay important findings of advanced linear methods. As outlined above, the indication of intraindividual stability is an essential prerequisite for the search for diagnostic markers on a per individual basis.

The outcome of our second objective allows us to show by means of the biparametric analysis that human EEG exhibits a difference between functional states of the brain. Accordingly, the functional states of remission and psychosis can be considered to be different even under resting conditions. This result is not trivial to achieve because one has to retrieve a relatively small effect almost blurred by a large amount of intraindividual variability [23] (due, for example, to uncontrolled cognition, etc.) We thus emphasize that the

*intra*individual distinction of the two functional states remains to be proven.

On the one hand, this is in agreement with the results of [24] according to which average values of certain EEG spectral parameters turned out to be different between groups of patients recorded during psychosis and remission under activated conditions. On the other hand, the impact of our finding goes further: our results indicate a difference between unmedicated psychosis and remission in the functional state of resting EEG with eyes closed. Without diving too deep into brain science, we may point out certain possible implications of these findings: the differences found here between the functional states of psychosis and remission in EEG recorded at rest seem to indicate a difference in the dynamics of the EEG generator independent of any particular controlled information processing. This complements other efforts, which focus primarily upon EEG recorded under activated conditions (cf. [37]). Furthermore, an indication of dynamically different resting brain states, schizophrenia versus remission, in the absence of external activation or information acquisition may point to permanent differences concerning uncontrolled cognition or may even be a hint to problems on some deeper functional level.

Whatever the exact biomedical explanation might be, such differences are not expected by theories explaining schizophrenia primarily as a disturbance of mental directive attention. These theories hypothesize that differences in EEG reactivity between functional states of schizophrenia and remission are expressions of disturbances to activated or intentional information processing of the brain and have been investigated in detail elsewhere from both a classical, spectral-analytical [24] and nonlinear, dynamical [37,38] point of view.

With regard to methodology, our biparametric b_0-m* approach introduces important new perspectives of relevance for the dimensional analysis of EEG in cases where the classical uniparametric approach seems questionable. We would like to emphasize that our results indicate the superiority of this biparametrization over classical dimensional approaches and suggests its use especially for analyses of biomedical time series.

VI. OUTLOOK

The question as to what extent the longitudinal information displayed by our nonlinear measure might already be available from a linear description of the underlying EEG is not adressed here and may be the focus of a forthcoming study. We intend furthermore to follow up the apparent dynamic intraindividual difference between functional brain states found here for the EEG recorded under the condition of quiet wakefulness with eyes closed in (unmedicated) psychotic and remitted schizophrenic patients. In addition we also want to investigate EEG epochs recorded under defined activational conditions with our approach. Hence our intention is (1) to improve discrimination intraindividually between psychosis and remission and (2) to test, in view of our results, certain hypotheses concerning schizophrenia as an expression of a disturbance to the passive, quiet wakefulness of the brain, on the one hand, and to the active or intentional information processing of the brain, on the other (cf. [39]).

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