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DIMENSIONALITY OF THE HUMAN ELECTROENCEPHALOGRAM

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We begin with a basic introduction to the electroencephalogram and discuss some of the EEG's clinical uses. Next we review the method of dimensional analysis and emphasize its utility for analyzing the behavior of complex nonlinear systems. After this we consider a practical application of dimensional analysis by asking the question: *How does the "dimension" of the EEG change with general anesthesia and other global states of the brain?* Finally we discuss major problems associated with dimensional analysis of the EEG.

Introduction To The EEG

EEG in small animals was discovered by Caton in 1875. EEG in man was discovered later by Berger in 1925, and published 1929. In spite of this long history, however, slow progress has been made in understanding and interpreting the EEG. Currently, there is no ordered atlas of EEG findings versus brain activity, there are only tendencies. In addition, large computers for the sophisticated analysis of EEG are not widespread and so today, clinical EEG interpretation is performed mostly by skilled visual inspection and less often by simple spectral transforms.

The microscopic genesis of the EEG is not understood.¹ Macroscopically, electrical signals appear to originate from extensive dendritic arborizations on neurons. It is in this extensive overlap that excitations within one dendritic tree are conveyed to the arborizations of adjacent neurons.² These collective excitations, in turn, generate signals that are coordinated in space and time near the surface of the brain. Beyond this, there is some attenuation of the brain's electrical activity by the intervening skull and scalp. At the scalp, the EEG is measured as a microvolt potential and to record this weak potential, it must be amplified by several orders of magnitude. Generally, EEG

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signals are recorded by contacting a standard pattern of electrodes with conductive paste to rubbed scalp, see Fig. 1. Pronounced artifacts often originate from slight movements of the electrodes and from contraction of muscles below the electrodes. Since the EEG is a weak signal, the importance of skilled electrode placement and inspection for artifacts cannot be overestimated.^{3,4}

The EEG's frequency spectrum ranges from 0.5 to 100 Hz, with most of the power concentrated in the range from 1 to 30 Hz. By visual inspection, the EEG looks like a complex wave that is aperiodic. The appearance of this signal fluctuates throughout the day and changes cyclically with sleep. There are four general classifications of EEG frequencies. They were established primarily by discovery, as early instrumentation improved, and are of limited intrinsic value: 1 - 3 Hz is termed *delta*, 4 - 7 Hz *theta*, 8 - 14 Hz *alpha*, and >14 Hz *beta*. In addition, the overall appearance of the EEG is modified by drugs. For example, cocaine increases alpha rhythm, thorazine decreases alpha rhythm, barbiturates increase beta rhythm, and amphetamines decrease amplitude. Often, one or more drugs so completely alters the normal pattern of the EEG that it becomes difficult or impossible to interpret. In these instances, diagnostic EEG's are performed only after a period of drug privation.

Realizing that drugs alter the EEG, attempts have been made to assess the depth of general anesthesia by analyzing brain waves in real-time. However, two difficult and related questions are involved: 1. *What is the choice for the measure?* and 2. *What is the sensitivity of the measure?* This attempt to assess anesthetic depth by *quantitative* means is important because physicians currently rely on *qualitative* physical signs. Although signs involving flinching, tearing, blood pressure, respiration rate, pupil diameter and heart rate are useful, they do not provide specific information on the brain's status.

To our knowledge, Hanley and Walts have done the most successful work to assess anesthetic depth by computer analysis of the EEG.⁵ In this study, a variety of auto-spectral and cross-spectral relationships were correlated: 1. Power density, mean frequency, and band width of selected frequencies for a given lead, and 2. Phase relationships and shared electrical activities

between different leads. Anesthesia was induced by a single agent and depth was evaluated by measuring blood concentrations of drug with a mass spectrometer. In order to establish self-controls and dose-related effects, the EEG was analyzed before anesthesia, during light anesthesia, and during medium anesthesia.

The above quantitative analysis involving the EEG is promising because: 1. The EEG is an overall measure of brain activity and general anesthetics globally affect the brain, 2. Certain drugs induce dose-related and frequency-related changes in the EEG, and 3. Some general anesthetics produce burst suppression, a phenomenon that includes reduction of the EEG to isoelectric appearance. During routine surgery, however, general anesthetics are not given in sufficient amounts to reduce the EEG to a flat line. Only in special cases involving prolonged anoxia, such as drowning, are drug comas induced to minimize brain damage. In these instances, sufficient anesthetic (a barbiturate) is administered so that the EEG is not active for several days.

Attempts to assess brain function and depth have been most successful when one drug is used to induce general anesthesia. In most surgical cases, however, several drugs are used to induce anesthesia which results in complex and unpredictable EEG changes. In these instances, numerical analysis of the EEG has been less accurate and rather difficult to interpret. These limitations have motivated us to consider a different approach.⁶

Dimensional Analysis And Anesthesia

Dimensional analysis of the EEG during general anesthesia is an interesting approach for several reasons: 1. Large doses of some anesthetics reduce the EEG to a flat line, therefore the dimension of the EEG may decline with anesthesia, 2. It provides a single number rather than a function or a series of correlations for assessing depth. 3. The dimension provides information about the complexity of the EEG signal which cannot be extracted from the power spectrum, 4. Dimension may be calculated before anesthesia and during anesthesia for the same person, which provides a control, and 5. The EEG may be more than "noise" in cerebral tissue; it may play a causal role in information processing and storage.² In this regard, there has been conjecture that the brain's

electrical activity reflects a complex dynamical system which may be evaluated by the tools of nonlinear dynamics.^{7,8}

Our original goal was to evaluate anesthetic depth for a series of 5 to 10 patients by dimensional analysis. This goal turned out to be more ambitious than we thought. It has been very difficult to obtain clean EEG records from the operating room. Noise is prominent due to electrocautery and to movement of the patient's head by operating room personnel. In addition, specialized EEG equipment must be used to reduce noise and to accommodate limited space in the room.

On the following pages, we present the results of our calculations and discuss problems associated with dimensional analysis of the EEG. We focus on a single EEG record from one patient, in order to study the method but not to draw general conclusions. For simplicity, we consider only two states: awake but quiet, and medium anesthesia. The EEG data we use comes from Hanley and Walts.⁵ It was selected because anesthesia was induced by a single agent, and because of its uninterrupted length and lack of artifacts.⁹

Methods Of Data Analysis

We use three basic tools that have been developed over the past several years for the characterization of chaotic dynamical systems. Here we want to review and discuss the ways in which they are implemented for our analysis.

1. Geometrical Reconstruction

The purpose of this technique is to reinterpret time-signals as multidimensional geometrical objects. This allows us to use notions from geometry to describe and characterize a time evolution. This method is basically different from the classical approaches using autocorrelation functions and fourier transforms, which are based on the underlying paradigm that all temporal oscillations may be decomposed into harmonic and periodic vibrations. The geometrical view of dynamical processes is based on the assumption that signals are generated by some deterministic and finite dimensional (generically nonlinear) dynamical system, which is not necessarily the superposition of periodic oscillators. The observed signal can then be interpreted as the projection

of a multidimensional phase-space trajectory.

Example: If we take the phase-space representation of a simple periodic oscillator, we obtain a trajectory in two-dimensional phase space which is described by the equations:

$$\begin{aligned}x(t) &= a \sin(\omega t) \\x'(t) &= \omega a \cos(\omega t).\end{aligned}$$

A simple trigonometrical transformation shows that the velocity $x'(t)$ can be rewritten as:

$$\begin{aligned}x'(t) &= a \omega \sin(\omega t + \tau) \\&= \omega x(t + \tau).\end{aligned}$$

From this simple example we learn two things: 1. For harmonic oscillatory signals we can replace one of the coordinates (or variables) with the other by looking at the other variable with a certain time delay τ , and 2. There exists specific values of the time delay τ for which reconstructed variables are orthogonal (in our example this happens for $\tau = \pi/2$).

In the following analysis we use a generalization of this idea.¹⁰ We reconstruct time-delayed vectors from EEG data with increasing values of the "embedding dimension" N , which specifies the number of components of the reconstructed vectors or the number of time delays. The general form of such a phase-space vector is given by:

$$\mathbf{x}_N(t) = \{x(t), x(t+\tau), x(t+2\tau), \dots, x(t+N\tau)\}.$$

In Figs. 2 and 3 we plot reconstructed phase-trajectories for EEG data from two different leads (P3-O1 and C3-T3) with time delays of $\tau = 20$ msec and $\tau = 40$ msec. These time delays correspond to the first minima in the mutual information content (see below). We observe different geometrical patterns for each lead which varies in time with changing states of the brain. For comparison, we also plot the time-series data and indicate the length of the time delays with marks. Note that these intervals correspond roughly to a quarter of the period of the most prominent frequency in the fourier transform, see Figs. 4 and 5. This agrees with our intuitive understanding concerning independent and "orthogonal" coordinates in the previous example.

In the next section, we give a quick review of the concept of "mutual information content" as a

convenient tool for finding an optimal choice for the time delay

2. Mutual Information Content

In order to get a satisfactory reconstruction from time-series data, we require that the components $x(t+k\tau)$ of the vector $\mathbf{x}_N(t)$ be independent. This optimizes the vectors' span in the corresponding phase-space. The first idea that comes to mind concerning this optimum, is to use a time delay for which the auto-correlation function of the time-series vanishes, i.e., the two coordinates are "uncorrelated". In the present context of nonlinear systems, however, this intuition might be misleading. One example of this failure is given by the one-dimensional logistic map:

$f_1(x) = 4x(1-x)$ for which the auto-correlation function of the time series vanishes for a shortest possible delay of $\tau = 1$.¹¹ Intuitively we know that x and $f_1(x)$ may be "uncorrelated" but they are not "independent". Shaw¹² studied this problem from an information theoretical point of view, asking the question: *Assume we know the result $x(t)$ of a measurement at time t . Under real experimental conditions with noise and finite resolution, what can be predicted about the outcome of the same measurement at time τ later?* Fraser developed this idea further and applied it to different experimental situations.¹³ By using the "mutual information content" as the criterion for choosing τ , rather than auto-correlation, he demonstrated results that match what one expects intuitively. The details of mutual information calculations are described elsewhere.^{12,13} For all our analyses we use the following criterion in a consistent manner: *optimal delay time = first significant minimum of the mutual information content.*

After calculating the "optimal delay time", we analyze the reconstructed data-vectors with the help of a dimensional algorithm. In the following section, we present the basic ideas behind dimensional analysis without going into formalism that is reviewed elsewhere.¹⁴

3. Dimensional Analysis

The basic idea of characterizing a geometric object is very old but recently, it has been popularized to sets with non-integer or "fractal" dimensions.¹⁵ These fractal objects appear to have special significance for chaotic behavior and "strange attractors". In the strict sense, the dimension

of a dynamical system is a measure of the number of independent variables needed to specify its activity at a given instant. It is also related to the number of active coherent modes modulating a physical process and therefore, it is also a measure of complexity.¹⁶ For numerical reasons we have adopted a method introduced by P. Grassberger and I. Procaccia which is now in wide use.¹⁷ Details of the algorithm and discussion of its reliability and error estimates are reviewed elsewhere.¹⁸ Here we want to present only the general ideas which facilitate the interpretation of our results. This review and the large number of numerical results that we present is necessary, since there is no accepted criterion of how to apply the algorithm in order to obtain reproducible and unique results.

The original idea of Grassberger and Procaccia was to calculate how the number $n(r)$ of pairs of vectors $\mathbf{x}_i = \mathbf{x}_N(t_i)$, which are separated by distances less than r , changes as a function of r . Grassberger and Procaccia called this quantity "correlation integral" and in the general case it should scale like:

$$n(r) \propto r^d$$

where d is the "correlation" dimension of the set. The way it is usually obtained is by taking the logarithm which yields:

$$\log n(r) = d \log(r) .$$

Thus if we plot $\log n(r)$ vs. $\log(r)$ we expect to observe a straight line, where the slope d corresponds to the dimension of the set. In practise, this method will never result in a perfectly straight line over the entire region $\log(r)$, because of the finite size of the vectors in the multi-dimensional geometrical reconstruction. However, there is usually a smaller region over which a "straight line" may be fitted and therefore, a dimension can be calculated.

In our opinion, a major drawback of this algorithm is that by fitting a straight line to a portion of the $\log n(r)$ vs. $\log(r)$ plot it is always possible to get an answer, namely a slope d , but it is not trivial to decide whether the observed result is really significant or purely due to artifacts. Therefore, we use the term "dimension" rather cautiously and prefer to replace it by the less

stringent term "(dimensional) complexity parameter". Even though we are unable to calculate the "actual dimension" of EEG, we believe that our numerical results reveal a comparative difference between two states of consciousness, i.e., awake but quiet, and general anesthesia. To some degree, this agrees with the findings of other researchers for dimensional analysis of human¹⁹⁻²¹ and animal EEG.²²

In the next section, we discuss some of the numerical and experimental problems that were encountered during implementation of the dimension algorithms.

Implementation Of The Dimension Algorithms

Our EEG data were digitized at 500 Hz, which is well above the maximal EEG frequency of ≈ 100 Hz. The effect of this fast sampling rate is to generate redundant information from the signal, corresponding to that fact that the EEG is continuous. Therefore, for very short distances r , neighboring vectors come from the same segment of trajectory and do not contribute to dynamical information which we want to extract. This is seen clearly in Figs. 6, 8, 9, 16, 17, and 20, where the slope d of $\log n(r)$ vs. $\log(r)$ is close to unity for $\log(r) < \approx -2.5$. This effect disappears when we reduce the sampling rate to 100 Hz, Figs. 11 and 12. Thus there is a restriction on the accuracy of the dimension algorithm: *it requires a large number of data points to fill a high dimensional phase-space volume*. Roughly, the number of data-points required to fill up a N -dimensional cube of given diameter goes exponentially with the embedding dimension N .

The signals that we analyzed from EEG were not stationary: the brain does not remain in the same *state* for an arbitrarily long time, except at death. In fact there is no general agreement on how long the brain remains in a specific *state*. For the sake of argument, however, let's assume that the *state* of the brain does not change significantly over the span of a few minutes. This condition assigns an upper bound on the amount of EEG data that can be used for dimensional analysis. Generating a larger number of data points by increasing the sampling rate is not a solution, since these points are confined to low-dimensional subspaces and introduce the systematic errors described above. Therefore, we are not convinced by reports of "low-dimensional chaotic

attractors" from EEG.^{21,23}

Using a code developed by Frazer¹³, which calculates mutual information content (MIC), we determined the "optimal" choice of time delay τ from ≥ 4096 points and its copy shifted by τ . In general, we choose τ where the MIC has its first minimum, see Fig. 15. For some EEG data, however, there was no distinct first minimum, see Fig. 19. In these instances, we chose a value of τ for which the MIC was below a fixed threshold. In spite of our efforts to calculate τ , however, we found that our results were stable within fairly large ranges.

After calculating time-delayed coordinates from normalized EEG data, we reconstructed phase space vectors $\mathbf{x}_N(t_i)$ for increasing embedding dimensions $N = 1, 2, \dots, 20$. However, to use the original algorithm of Grassberger and Procaccia, which calculates distances between all pairs $\{\mathbf{x}_N(t_i), \mathbf{x}_N(t_j)\}$ of data points, would be too time consuming. With the original method, even a CRAY-XMP computer would be far too slow to analyse the data in real time. Therefore, we choose a smaller set n_{ref} of equally spaced reference points $\mathbf{x}_N(t_k)$ and computed distances between reference points and the rest of the data points. Typically, we used 200 reference points and 20,000 data points. Of course, we obtained different values for the number of pairs $\log n(r)$ within distance $\log(r)$ for each reference point, and the error bars in our plots indicate the width of this distribution for each discrete value of $\log(r)$.

In order to calculate "dimension" automatically (for each embedding dimension N), we implemented an algorithm which fits a straight line (of length equal to a factor 2 in scale) to each point r_m of the "dimension curve" $\log n(r_m)$ vs. $\log(r_m)$.¹⁸ After fitting each point r_m , we calculated a goodness of fit for each line segment and located the point with the best fit. This point defines the center of the "scaling region." Subsequently, we increased the length of the "scaling region" until the goodness of fit reached a chosen threshold value of 10%. In all our plots of $\log n(r_m)$ vs. $\log(r_m)$, we indicate the location and length of this "scaling region" with the actual line segment. Next we plot the local slope of the dimension curve as a function of the distance $\log(r_m)$, for the maximal embedding dimension $N=20$. This insures that we didn't fit the line

segment to a region where the slope is too small. Such a mistake would cause a severe underestimate of the "true" dimension. In the last step, we plot the maximum dimension d_N as a function of the embedding dimension N , for $N=1, 2, \dots, 20$. In many cases we see that the dimension or "complexity parameter" does not saturate, see Figs. 7, 18 and 21. In other cases we see evidence for saturation which suggests "low-dimensional chaos", Figs. 10 and 13. Even in such cases, however, interpretation must be cautious: Osborne has shown that random noise (with a high order power law behavior of the spectrum) may generate results which appear to be "low-dimensional", when analyzed by standard dimension algorithms.²⁴

The error estimates in all our figures are very conservative. They indicate the maximum variations in slope for line segments in the "scaling region." For each data set, the overall error is proportional to the average size of the error-bars in the $\log n(r)$ vs. $\log(r)$ curve and inversely proportional to the length of the "scaling region." Therefore, fitting over large "scaling regions" with large error bars yields similar results to fitting over small "scaling regions" with small error bars. In the literature, errors based on a least squares fit are often quoted. We think this is far too optimistic, especially for small scaling regions.

Finally, for increasing values of embedding dimension N , the fraction of points that are close to the perimeter of the data set increases. This is a serious limitation of the algorithm, it causes a systematic error in dimension estimates for high values of embedding dimension.¹⁴ We want to insure an asymptotic regime, by using large values of embedding dimension, but we also know that systematic errors due to boundary effects increase. Therefore, we consider any result of $d > 7$ to be indistinguishable from $d = \infty$.

Preliminary Results

Our results are based on data from three different sources. Most of our analysis was done with the awake but quiet, and anesthesia data from Hanley.⁹ Our second source came from Ehlers²⁵ and was recorded during standard mental tasks and during sleep. Our third source of data came from Flynn,²⁶ it was an attempt to see if the "dimension" of a magnetoencephalogram (MEG) is

similar to EEG. These results are summarized in the table below, which also reviews the work of other investigators.

Fig. 22 demonstrates how the observed "dimension" of the EEG increases with the number of data points analyzed. The dimension does not appear to reach an asymptotic value as the number of data points increases, which provides evidence that this particular EEG is generated by a very high dimensional system.

Figs. 23-26 show how the "dimension" of the EEG fluctuates as a function of time. During the awake but quiet interval ($t < 5$ min.), there are rather large fluctuations in leads P3-O1 and P4-O2 at a relatively low value of "dimension". Leads C4-T4 and C3-T3 show smaller variations but at larger values of "dimension". At $t = 3$ min., there is a decline in the "dimension" of leads P4-O2 and P3-O1 which is not reflected in the temporal leads, see Figs. 8-14. In order to determine whether the rapid decline in "dimension" at $t = 3$ min. for lead P3-O1 was real or due to numerical artifacts, we recalculated the result using a slower digitizing rate of 100 Hz. Figures 11-13 show the outcome of this second calculation; they indicate a similar decline in "dimension." We also repeated these 100 Hz calculations for other intervals of EEG data and found similar results. Furthermore, the power spectrum of this time interval (Fig. 4), does not show a strong power law decay. This is an additional indication that we are observing "low dimensional chaos" ($d < 5$) and not "random noise with special statistics" as discussed by Osborne.²⁴ In general, the temporal and occipital leads show a qualitatively parallel behavior with time. For these reasons, we are inclined to believe that the fluctuations in "dimension" are real.

For a different subject that was in the awake but quiet state we found "dimension" values which were similar to the ones above²⁶. We observed low "dimension" values, however, only when the eyes were closed. When the subject opened the eyes, the dimension increased substantially (see table). Also for the same subject, there seem to be no significant changes in "dimension" for various stages of natural sleep. However, to our surprise we found that the mutual information content varied significantly with different sleep stages (Fig. 27). In stage 4 sleep the

mutual information content decays very slowly which may reflect a slow rate at which new information is produced. For the cases of REM sleep and sleep onset, the mutual information curves lie close together, which possibly reflects a similarity between the two stages of sleep.⁸

During light general anesthesia ($5 < t < 10$ min.), the "dimension" of leads P3-O1 and P4-O2 abruptly increased beyond our numerical limits. In the temporal leads this increase was not observed, the observed dimension was consistently at our numerical limits. Figs. 20 and 21 show the "dimension" curves for medium anesthesia. We don't see any changes during the transition from light to medium anesthesia ($t > 10$ min.). For this case, this indicates that "dimension" is not a sensitive measure of the depth of anesthesia. However, the relative increase in dimension is consistent with the excitatory action of the anesthetic fluroxene.

We also see a clear difference between the two groups of leads by looking at the mutual information curves, Figs. 15 and 19. This difference agrees with the observation that higher dimensional systems, which are "more chaotic", are less predictable and therefore have a faster decline in mutual information. Another measure of "predictability" and "information production rate" is given by Kolmogorov-entropy. In Fig. 14, it is seen as the distance between the "dimension curves" for a fixed value of the distance r (as the embedding dimension approaches 20). From the small spacing we conclude that the Kolmogorov-entropy of the system is relatively small.

Finally, we also calculated the "dimension" of several MEG recordings. These data reflect activity from a smaller volume of brain tissue than EEG. Nevertheless, it seems that the dimension of these MEG data is comparable to that of the EEG data we analyzed. This might support speculations that the electromagnetic activities in the brain are coherently generated by large regions.

Conclusions

1. The "fractal dimension" of the EEG cannot be determined rigorously, due to non-stationarity of the signal and subsequent limitations in the amount of acceptable data.
2. EEG data must be analyzed in a comparative sense with the subject acting as their control.

3. In a few cases (awake but quiet, eyes closed) with limited time samples, it appears that the dimension algorithms converge to finite values.
4. There is a noticeable increase in the "dimension" of the EEG with fluroxene anesthesia; this increase is seen in the occipital but not the parietal leads.
5. Dimensional analysis and attractor reconstruction could prove to be useful tools for examining the EEG and complement the more classical methods based on spectral properties.
6. Besides being a useful tool in determining the optimal delay-time for dimension calculations, the mutual information content is a quantity which is sensitive to different brain states.

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25. Data from C. Ehlers at UCSD. The EEG data were recorded from the same subject while performing various mental tasks and while sleeping.
26. Data from E. Flynn at Los Alamos. The magnetoencephalogram data were recorded from the

same subject while awake but quiet.

Figure Captions

- Figure 1.** Four standard EEG leads from the international 10-20 system that are used in our dimensional analysis.
- Figure 2.** Two-dimensional phase portrait and corresponding time-series EEG data from lead P3-O1. Each representation is generated by 2500 data points, while the subject was awake but quiet. Time delay = 20 msec.
- Figure 3.** Same representations as Fig. 2, except that the EEG data comes from lead T3-C3. Time delay = 40 msec. The short marks next to the phase portrait indicate the lengths of the two time delays for Figs. 2 and 3.
- Figure 4.** Power spectrum (in dB) of EEG data from lead P3-O1 at 0.5 Hz resolution. The transform was generated by 16 seconds of EEG data digitized at 500 Hz.
- Figure 5.** Power spectrum (in dB) of EEG data from lead C3-T3 at 0.5 Hz resolution. The transform was generated by 16 seconds of EEG data digitized at 500 Hz.
- Figure 6.** "Dimension function" $\log(n(r))$ vs. $\log(r)$ for lead P3-O1 while the subject was awake but quiet. Sampling rate: 500Hz, time interval=30sec, time delay=20msec, embedding dimension=20, and reference points=200. The measured "dimension" is 6.2 ± 3.5 , with a goodness of fit of 10.3%.
- Figure 7.** "Dimension" or "complexity parameter" as a function of the embedding dimension, for Fig. 6.
- Figure 8.** Same conditions as in Fig. 6, except two minutes later. The "dimension" appears to have dropped substantially although no physiological changes took place. The measured "dimension" is 4.3 ± 2.2 , with a goodness of fit of 11.2%.
- Figure 9.** "Dimension" (local slope) as a function of the distance $\log(r)$, for Fig. 8. Note that it nowhere exceeds a value of 5.

- Figure 10.** "Dimension" or "complexity parameter" as a function of the embedding dimension, for Figs. 8 and 9.
- Figure 11.** Same data as Fig. 8, except for a sampling rate of 100Hz. The measured "dimension" is 4.3 ± 2.7 , with a goodness of fit of 12.3%
- Figure 12.** "Dimension" (local slope) as a function of the distance $\log(r)$, for Fig.11.
- Figure 13.** "Dimension" or "complexity parameter" as a function of the embedding dimension, for Figs. 11 and 12.
- Figure 14.** Same data as in Fig. 13, with all the "dimension curves" plotted for embedding dimensions $N=1, 2, \dots, 20$. We do not observe a large parallel shift in the dimension curves, which should occur if the systems had a large Kolmogorov-entropy, i.e. if it would continuously produce a large amount of new information.
- Figure 15.** Mutual information content of lead P3-O1 for "awake but quiet" (solid line, data from Figs. 8-14) and "medium anesthesia" (broken line, data from Figs. 20 and 21). Note the bump at around 50 msec (20 Hz $\approx$ twice alpha rhythm) for the solid line.
- Figure 16.** Same conditions as Fig. 6, except for lead C3-T3. The measured "dimension" is 7.3 ± 5.4 , with a goodness of fit of 9.4%
- Figure 17.** "Dimension" (local slope) as a function of the distance $\log(r)$, for Fig. 16.
- Figure 18.** "Dimension" or "complexity parameter" as a function of the embedding dimension, for Figs. 16 and 17.
- Figure 19.** Mutual information content of lead C3-T3 for "awake but quiet" (solid line, data from Figs. 16-18) and "medium anesthesia" (broken line, data not shown). Note the absence of any "bump" which can be interpreted as absence of prominent alpha-rhythm.
- Figure 20.** Same measurement conditions as Fig.6, but now the subject is under medium

fluroxene anesthesia. The measured "dimension" is 8.0 ± 3.8 , with a goodness of fit of 18.3%.

- Figure 21.** "Dimension" or "complexity parameter" as a function of the embedding dimension, for Fig.20.
- Figure 22.** "Dimension" or "complexity parameter" as a function of the number of data-points used in the dimension calculation. Data from lead P3-O1 during general anesthesia.
- Figure 23.** "Dimension" or "complexity parameter" as a function of the time for lead P3-O1. There are relatively large fluctuations in "dimension" during the awake but quiet state, $t < 5$ min. There is a clear increase in "dimension" during fluroxene anesthesia, $t > 5$ min. The recording was interrupted at 5 and 10 min. to allow for allow for stabilization of anesthetic level.
- Figure 24.** Same as Fig. 23, except for lead P4-O2.
- Figure 25.** Same as Fig. 23, except for lead C4-T4.
- Figure 26.** Same as Fig. 23, except for lead C3-T3.
- Figure 27.** Mutual information content versus delay time for lead C3-T3. The same subject is at different levels of sleep: () sleep onset, (Δ) rem sleep, (o) stage 4 sleep. Note the similarity of the mutual information curve for sleep onset and rem sleep, whereas for stage 4 sleep the mutual information content is larger.

Summary of Calculations

State of the Brain	Lead	Sampling Rate [Hz]	Delay τ [msec]	Total Time [sec]	Maximal Embedding Dimension	"Dimension"
<u>Albano et al.</u>²⁰						
quiet, awake, eyes closed	?	500	10	5.8	19	2.6 ± 0.2
quiet, awake, eyes open	?	500	6	5.8	19	∞
<u>Babloyantz et al.</u>^{19,23}						
quiet, awake	C4-A1	100	100	40	10	∞
sleep stage II	C4-A1	100	100	40	6	5.0 ± 0.1
sleep stage IV	C4-A1	100	100	40	5	4.1 ± 0.1
REM sleep	C4-A1	100	100	40	10	∞
petit mal seizure	?	1200	15.8	5	7	2.05 ± 0.09
<u>Watt & Hameroff</u>²¹						
quiet, awake, eyes closed	C3-P3	300	17	4	6	2.15 ± 2.0
isoflurane anesthesia	C3-P3	300	17	4	6	2.07 ± 2.0
thiopental anesthesia	C3-P3	300	17	4	6	1.90 ± 2.0
<u>Mayer-Kress & Layne</u>						
quiet, awake [†]	P3-O1	100-500	10-40	10-30	20	$4 - 7 \pm 3 - 5$
quiet, awake [†]	P4-O2	100-500	10-40	10-30	20	$4 - 7 \pm 3 - 5$
quiet, awake [†]	C3-T3	100-500	10-40	10-30	20	$7 - 8 \pm 5$
quiet, awake [†]	C4-T4	100-500	10-40	10-30	20	$7 - 8 \pm 5$
fluroxene anesthesia [†]	(all four)	100-500	10-40	10-30	20	8.0 ± 6.0
<u>quiet, awake and eyes closed (e.c.)[‡]</u>						
e.c. [‡]	P3-O1	512	10-40	10-30	20	5.1 ± 5.4
e.c. [‡]	P4-O2	512	10-40	10-30	20	5.0 ± 4.0
eyes open, no task [‡]	P3-O1	512	10-40	10-30	20	8.6 ± 5.1
eyes open, no task [‡]	P4-O2	512	10-40	10-30	20	8.7 ± 5.4
e.c., verbal memory [‡]	P3-O1	512	10-40	10-30	20	6.3 ± 2.9
e.c., verbal memory [‡]	P4-O2	512	10-40	10-30	20	6.4 ± 3.3
e.c., visual memory [‡]	P3-O1	512	10-40	10-30	20	6.1 ± 5.5
e.c., abstraction [‡]	P3-O1	512	10-40	10-30	20	6.0 ± 3.4
e.c., abstraction [‡]	P4-O2	512	10-40	10-30	20	6.4 ± 3.8
e.c., word association [‡]	P3-O1	512	10-40	10-30	20	5.9 ± 3.5
e.c., word association [‡]	P4-O2	512	10-40	10-30	20	5.5 ± 3.8
<u>sleep</u>						
onset [‡]	C3-A1	512	10-40	10-30	20	6.8 ± 6.1
stage 4 [‡]	C3-A1	512	10-40	10-30	20	5.9 ± 4.4
rem [‡]	C3-A1	512	10-40	10-30	20	6.4 ± 5.1
<u>MFG: quiet, awake</u>						
eyes closed (e.c.) [¶]	---	512	10-40	10-30	20	6.7 ± 3.6
<u>Röschke & Basar</u>²²						
cat cortex & subcortex	?	?	?	?	?	~ 4.5

[†] Data from J. Hanley, [‡] Data from C. Ehlers, [¶] Data from E. Flynn

Fig. 1

fig. 2

τ
[msec]
20
"
40
"

A .

1. A. 1

Fig. 6

'g.'

0.8.0

fig. 7

Fig. 10

fig. 11

— —
D

7-1-

fig. 14

Fig. 15

97.8.16

Fig. 20

Fig. 1

A

$c \rightarrow R_{1,1}$

6 > .P. 1