Application of a neural complexity measure to multichannel EEG

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Abstract

We apply a recently suggested measure for neural complexity (G. Tononi, O. Sporns, G.M. Edelman, Proc. Natl. Acad. Sci. 91 (1994) 5033), that is hypothesised to capture the interplay between two fundamental aspects of brain organisation, functional segregation and integration, to human EEG recordings. This measure is based on a weighted sum of entropy differences evaluated at different length scales of the system. A strong prediction is that this measure correlates with the conscious state of the subject, having lower values if consciousness is reduced (G. Tononi, G.M. Edelman, Science 282 (1998) 1846). It is found, however, that this neural complexity measure increases in neurological disorders where consciousness is severely reduced or absent. We discuss several possible explanations for this observation and suggest directions for future work. © 2001 Elsevier Science B.V. All rights reserved.

1. Introduction

Since the exchange of information between neurons, and therefore information processing in the brain, is (partially) realized by electronic and ionic currents along the neuronal axons and dendrites [1], the brain generates local (time-varying) electromagnetic fields. By the summed action of the dendrites connected to the neuronal cell-body membranes, excitatory and inhibitory post-synaptic potentials (EPSPs and IPSPs) are created, where the synchronous behaviour of large groups of neurons creates a net electromagnetic field, that can be measured through the intact scalp [2]. The presence of these electrical signals resulting from the neuronal activities has strongly motivated researchers to gain insight into brain function by studying this electro-magnetic brain activity with the electroencephalogram (EEG) or magnetoencephalogram.

Until about 1970, EEG interpretation was mainly heuristic and of a descriptive nature. Although since that time several papers discussed quantitative techniques to assist in EEG interpretation (for instance, [3,4]), in clinical terms the situation remained essentially unchanged. In 1985, however, Babloyantz, using techniques derived from “chaos theory”, showed that certain nonlinear measures changed during slow wave sleep, opening new directions for nonlinear quantitative EEG (Q-EEG) research [5] (for a review of several (nonlinear) Q-EEG techniques see, e.g., [6,7]). Since that time, applications of Q-EEG to several research areas have significantly increased and (potential) clinical applications have been reported, as well, such as

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the prediction of epileptic seizures [8–10], characteriza-

Recently, efforts were made to characterise the com-
plexity of neuronal interactions to increase our under-
standing of the behaviour of “complex systems”, such as the brain. Complexity is a feature of a system that may relate to different attributes, involving aspects as regularity, information processing capacity or a problem solving capability. Complexity may relate, for instance, to the spectral entropy of a time-series, where the spectral entropy $H = \sum f p_f \log(1/p_f)$, with $p_f$ the probability density function value at frequency $f$, may heuristically be interpreted as a measure of the system complexity. Note, that this complexity measure reaches high values for randomly distributed noise, and low values if there is a dominating process in the generation of the time-series. A different measure of complexity is, e.g., the number of parameters needed to fully describe a systems behaviour or characteristics. In the context of information theory, this could be the length of the shortest computer program that generates a particular bit string. Rezek and Roberts [14] presented several complexity measures for physiological signals, such as an approximate entropy and the embedding space eigenspectrum (ESES). An acceleration spectrum entropy measure was applied to human EEG data, studying the alpha rhythm desynchronisation in [16]. Measures based on (linear) dependence of the electrical field variations across the scalp were introduced by Wackerman et al. [11], who propose a spatial principal component analysis (PCA) as a global measure for the complexity of multichannel EEG recordings.

Tononi and Edelman introduce a neural complexity, $C_N$, as a measure that should reflect the interplay between functional segregation and integration within a neural system, such as the human brain [17–20]. This complexity measure satisfies the requirement that it attains a small value for both a completely random and completely regular system, contrary to complexity measures as spectral entropy, ESES or spatial PCA. It is further hypothesised that this measure obtains high values during (optimal) information processing and small values during states were this processing cannot be realized, for instance during generalised epileptic seizures, where consciousness is absent [2]. Thus far, this complexity measure has not been applied to human EEG data, however. In this study, we apply this complexity measure to EEG signals recorded from healthy subjects and neurological patients with two brain disorders, viz. epileptic seizures and severe postanoxic encephalopathy. In these disease states consciousness is severely reduced or even absent, hereby providing important real-world data to study additional characteristics of this complexity measure. The motivation for this work is two-fold. Firstly, to increase our understanding of (global) brain functioning and, secondly, to test the interesting hypothesis proposed by Tononi and Edelman that the neural complexity, $C_N$, decreases in conditions where consciousness is reduced, since information processing is lowered in these states.

2. Theory

The motivation for the introduction of the neural complexity measure, $C_N$, is the observation that the brain contains specialised areas, or subsystems, $x_i$, that have unique characteristics, e.g., language generation or visual perception. At the same time, this specialisation occurs in the context of functional integration, so that information of the several subsystems is available to other brain areas. Stated differently, the activity of a subsystem should be “detected” elsewhere in the brain. Clearly, these two requirements may conflict: maximal specialisation reduces integration and maximal integration interferes with specialisation.

The complexity measure introduced captures this interplay between functional segregation and global integration of the different elementary subsystems, $x_i$, with $i = 1, \ldots, N$, of the whole brain system, $X$, consisting of the collection of $N$ elementary subsystems. If we now consider the system at different length scales, or subset sizes $k$, $X^k$, that is composed of $k$-out-of-$N$ components, we can also define characteristics of these several subsets, $X^k$. Note, that different subsets with size $k$ can be realized; characteristics of the several subsets, therefore, relate to average values

1 The adjective elementary is arbitrary; we could define it as a small measurable set of neurons, for instance from which electrical signals can be obtained in a particular experiment.
over the \(N!/k!(N-k)!\) combinations of the \(k\) components.

In particular, we can define the neural complexity, \(C_N\), as [17]
\[
C_N = \sum_{k=1}^{N} \left[ \frac{k-1}{N-1} I(X) - I(X^k) \right],
\]
where the integration, \(I(X^k)\), is given by
\[
I(X^k) = \sum_{i=1}^{k} H(x_i) - H(X^k).
\]

In this expression, the collection of elementary subsystems \(x_i\) forms the subset \(X^k\); \(H(\cdot)\) is the entropy. Since \(H(X^k) \leq \sum_{i=1}^{k} H(x_i)\), where the equality holds for completely independent systems, it follows that \(I(X^{k+1}) \geq I(X^k) \geq 0\) (see Appendix A for further details).

The integration, therefore, measures the difference between the sum of the entropies of the several elementary subsystems, \(x_i\), considered independently and the entropy of the collection of these subsystems, \(X^k\). We can interpret \(I(X^k)\) a measure for deviation from statistical independency of the individual components, where \(I(X) = 0\) if the components are statistically independent; \(I(X)\) becomes maximal for complete dependency. The neural complexity measure, \(C_N\), as defined in Eq. (1), is high when, on the average, the integration of the several subsets as a function of the subset size \(k\), is smaller than the value that would be expected from a linear increase over increasing subset size. A different interpretation of a high complexity is that the mutual information between any subset of the components is high. A more elaborate exposure is presented in Appendix A and in [17,18].

3. Methods

3.1. Computer implementations

Various strategies can be used to calculate \(I(X^k)\) and \(C_N(X)\) from a set of data. Assuming that the multidimensional stationary stochastic process describing the activity of the \(N\) subsystems, \(x\), is Gaussian, all deviations from independence among the components are expressed by their cross-covariances and the integration, \(I(X^k)\), can be obtained from the cross-covariance or cross-correlation matrix according to standard formulae [21]. In particular, \(I(X^k)\) was calculated using
\[
I(X^k) = -0.5 \ln(|\text{Corr}(X^k)|),
\]
where \(|\cdot|\) denotes the determinant and Corr\((X^k)\) cross-correlation matrix of the subset \(X^k\). We denote the \(m \times k\) data matrix with similar symbolics as the subset \(X^k\), by
\[
X^k = \begin{bmatrix}
    x_1(t_1) & x_2(t_1) & \ldots & x_k(t_1) \\
    \vdots & \vdots & \ddots & \vdots \\
    x_1(t_m) & x_2(t_m) & \ldots & x_k(t_m)
\end{bmatrix},
\]
where the signals measured from the elementary subsystems \(x_i\) are denoted by \(x_i(t_n), n = 1, 2, \ldots, m\), with \(m\) the number of samples. To obtain reliable estimates of the integration for the several subset sizes \(k\), the average value for typically 30–100 random samples was calculated. Numerical analysis showed that this approximation yielded consistent values for \(C_N(X)\). We remark, that this implementation is identical to the approach presented in [17].

3.2. Simulations

To illustrate some essential properties of the integration and complexity measure, it was applied to an elementary model system, \(M\), comprised of 25 subsystems, \(x_i\), where the signals obtained from each subset are denoted by \(x_i(t_n)\) given by
\[
x_i(t_n) = a_i \sin(\omega_i t_n + \phi_i) \cos(\omega_i t_n) \cos(\omega_i^2 t_n/10) + n_i(t_n),
\]
where \(a_i\), \(\omega_i\), and \(\phi_i\) are constant for each subsystem \(x_i\), with \(i = 1, \ldots, 25\). \(n(t_n)\) is a zero mean, white noise process. This function may serve as an illustration of a system comprised of several (independent) subsystems, that may show different levels of integration by varying the values of the parameters \(a\), \(\omega\) and \(\phi\), resulting in different values of the complexity measure, \(C_N\). As discussed in the previous section, complexity should be low if the components of the system \(M\), i.e., the 25 subsystems, are either completely independent or uniformly dependent and attain larger values, otherwise. Appendix A presents more
details about this model system, including, in operational terms, the calculations that were performed.

3.3. EEG recordings

EEGs were recorded with a Brainlab digital EEG system (OSG, Belgium). Filter settings were 0.16–70 Hz, with a sampling rate of 500 Hz and 16-bit A/D precision. The EEG was recorded with Ag/AgCl electrodes placed at the Fp2, Fp1, F8, F7, F4, F3, A2, A1, T4, T3, C4, C3, T6, T5, P4, P3, O2, O1, Fz, Cz and Pz loci of the international 10–20 system and transformed to an average or source reference montage (which included all electrodes except Fp2, Fp1, A2 and A1). Impedance was kept below 5 kΩ. Signals in EEG recordings can be referenced differently. In this respect, we note that the function \( x_i(t_n) \) should primarily reflect the activity of the subsystem \( x_i \), only. In general, however, we have

\[
x_i(t_n) = f(x_i) + \sum_{j \neq i}^N g_{ij}(x_j),
\]

with \( f \) a particular function of the activity generated by the subsystem, \( x_i \), and \( g_{ij} \) a particular function that describes the additive contribution of the neuronal activities of the subsystems \( x_{i \neq j} \) on the final signal, \( x_i(t_n) \) measured. Contributions to this influence are, e.g., the reference selected and volume conduction effects.

An ideal reference montage will now significantly reduce the contribution of the neighbouring subsystems, \( \sum_{j \neq i}^N g_{ij}(x_j) \), to the signal \( x_i(t_n) \), to obtain

\[
x_i(t_n) = f(x_i) + \epsilon,
\]

with \( \epsilon \ll f(x_i) \).

We applied the commonly used average reference montage, where the voltage difference between the recording site and the average of all electrode potentials is used for the subsequent analysis and the source reference. In this latter montage, the voltage difference between the recording site and the mean voltage of the nearest neighbour recording sites (typically 3–4) is used for subsequent analysis.

Twenty-one EEGs from healthy subjects recruited from our own department were recorded. Artifact free epochs with a length of 6 s (3000 samples) were selected for further analysis, both during closed eyes (EC) and open eyes (EO) recording conditions. In addition, 3 EEGs from patients with generalized seizures and 6 EEGs from patients with a severe postanoxic encephalopathy were included in our analysis. All parameters were estimated after digital filtering of the sampled EEG in the range 0.5–30 Hz since most power (>95%) is contained in this frequency range.

4. Results

Results of a typical simulation, using our model system, \( M \), comprised of 25 subsystems \( x_i \), are presented in Fig. 1 (see Appendix A for more details). The complexity \( C_N \) was indeed relatively low (\( C_N < 10 \)) for a model with completely independent subsystems, each generating independent, random time-series (model A: \( a_i = 0, \) for all \( i \)). A similar low complexity was found for a nearly completely dependent system, model C, where the subsystems generated in-phase sine waves, to which a low-amplitude zero-mean noise process was added (\( a_i = 10 = \text{const}, \phi_i = 0.2; \omega_{i,1} = \omega_{i,2} = 1 = \text{const}, \) for all \( i \)). For systems comprised of subsystems with time-series where the coefficients \( a_i \) have different values for a number of subsystems, intermediate values of the integration and the highest values of \( C_N \) are found. At the same time, integration, as a measure for global coupling, was highest for system C and lowest for system A. Additional details of the model system are presented in Appendix A.

Subsequently, we applied our analysis to real-world signals recorded from healthy subjects; an example of such a recording is shown in Fig. 2. As pointed out earlier, for the final analysis, recordings from channels Fp1, Fp2, A1 and A2 were discarded since in practice, these channels often contain artifacts, for instance from eye-blinks. Therefore, 17 channels remained, from which the integration and complexity were estimated.

A typical example of a patient suffering from a severe postanoxic encephalopathy is presented in Fig. 3. In this brain disorder, severe neuronal damage has occurred due to a temporary reduction in the oxygen transfer to the brain, often by a cardiac infarction and or cardiac arrhythmia. Especially cortical neurons, that can only withstand a few minutes of oxygen depletion, are severely damaged. Typically, consciousness
Fig. 1. Average integration as a function of subset size, $k$, for three different realizations, A–C of our model system, comprised of 25 subsystems, $x_i$. The integration is smallest for realization A, that contains nearly completely independent subsystems with $a_i = 0$, for all $i$, and attains the highest value in realization C, comprised of nearly completely dependent subsystems with $a_i = 10 = \text{const}$, $\omega_{i,1} = \omega_{i,2} = 1 = \text{const}$, for all $i$. In realization B, (i) $a_i = 10$, $i = 1, \ldots, 9$; $\omega_{i,1} = \omega_{i,2} = 1$; $\phi_i = 0.2$; (ii) $a_i = 0.3$, $i = 9, \ldots, 14$; $\omega_{i,1} = 1$; $\omega_{i,2} = 0$; $\phi_i = 0$; and (iii) $a_i = 0.4$, $i = 15, \ldots, 25$; $\omega_{i,1} = \omega_{i,2} = 1$; $\phi_i = 0$. In this realization, the integration attains intermediate values and the complexity $C_N$, that equals the hatched area, attains the highest value. This system B is neither completely dependent, nor independent.

Fig. 2. EEG recording from a healthy subject, with his eyes closed. Default montage with a common reference. The several recording sites are shown at the left. The alpha rhythm recorded at O2 and O1 (the occipital regions) with a dominating frequency of approximately 10 Hz is clearly visible. Similar rhythmic activity is present at different recording sites, for instance the temporal regions (T6 and T5).

Fig. 3. EEG recording from a patient with a severe postanoxic encephalopathy. This pattern is known a “burst suppression” pattern to denote the typical variations in the presence of high voltage activity, regularly interspersed with low voltage activity.

Fig. 4. EEG recording from a patient having generalized seizures. All recording sites show periodic oscillations, with a fundamental frequency of approximately 3 Hz. is severely reduced, and usually absent, with a concurrent decrease in information processing capacity.

Finally, in Fig. 4 we present a recording of a patient suffering from a generalised seizure. At all recording sites, there are in-phase oscillations, with a fundamental frequency of approximately 3 Hz. During this recording, consciousness was not preserved, as is al-
Fig. 5. Left: Integration, $I$, as a function of subset size for the default montage (common average). A healthy control and two different patients are shown. The complexity is the area enclosed by pairs of curves, as indicated for healthy control subject, $C$. $C_{AN}$ relates to the complexity of a patient with generalized seizures and $C_{BN}$ to a patient with a severe postanoxic encephalopathy. Right: the results for a source montage.

ways the case for patients having generalised seizures, and no information processing can be realized.

The integration and complexity calculated for these three typical EEG recordings — both using the average reference and the source montage — are shown in Fig. 5. We find that the integration, $I$, is larger in the patient with a burst-suppression pattern and the patient with generalised seizures than in the healthy control. In addition, the neural complexity, $C_N$, increases in these two pathological conditions, as compared to the healthy subjects, both for the average reference and the source reference montage.

An overview of the mean values obtained for the controls ($n = 21$) and the two patient groups is presented in Table 1.

5. Discussion

In this Letter, we study the behaviour of a recently introduced measure for the complexity of the brain that may quantify the balanced integration and segregation of brain processes, as suggested by Tononi and Edelman. This neural complexity, $C_N$, attains low values for systems comprised of either completely independent or completely dependent functioning sub-systems. Such systems could be viewed as being of a low complexity, as opposed to systems where the sub-systems show both evidence of independent activity and cooperation, where $C_N$ should attain larger values. To illustrate this concept, simulations were performed using an elementary system, comprised of 25 subsystems. By varying the amount of “cooperation” between these subsystems, different values of the complexity were attained. Our simulations show that $C_N$ is indeed low both for completely independent sub-systems and nearly dependent subsystems, with values $C_N < 10$. For a more complex system, comprised of a sum of sine and cosine functions, the complexity increased, reaching a value $C_N \approx 60$. It is further illustrated that the integration $I(X)$ is low for the completely independent system and attains a high value for the nearly dependent system. These results are in agreement with the theory and the, much more extensive, simulations by Tononi and Edelman [17].

Subsequently, we calculated the integration and complexity measures for three different types of EEG signals from healthy volunteers, patients with generalised seizures and patients with a postanoxic encephalopathy, respectively. The two different pathological brain states were studied to test the hypothesis that the neural complexity decreases if information processing capacity is reduced, which holds in disorders where consciousness is severely reduced (postanoxic encephalopathy) or completely absent (generalised seizures). In patients with a postanoxic en-
cephalopathy there is severe damage to cortical neuronal groups, while during generalised seizures, there exists a pathologically increased coupling between the various brain areas.

In healthy subjects, the integration, $I(X) = 11.77 \pm 2.2$ for the default montage and $I(X)_{\text{source}} = 4.62 \pm 0.87$ for the source montage. In agreement with visual inspection and the underlying pathophysiology, we find that the integration, as a measure for dependence between the subsystems, obtains higher values in severe postanoxic encephalopathy, $I(X) = 18.3 \pm 4.7$ and $I(X)_{\text{source}} = 8.5 \pm 3.6$, and obtains a value of $I(X) = 19 \pm 4$ or $I(X)_{\text{source}} = 15 \pm 6$ during generalised seizures. A source montage will perform better than the average reference montage to reduce the contribution of subsystems $x_i \neq j$ on the signals measured from subsystems $x_j$, as follows directly from Kirchoff’s rules [22] (see also Eq. (7)). Consequently, this montage will reduce “spurious” dependency of the subsystems, and indeed all values of the integration found in the source montage are lower than those obtained using the default montage.

In healthy subjects (EO), the neural complexity, $C_n = 25.38 \pm 2.01$, and in the source montage $C_{n, \text{source}} = 12.93 \pm 1.92$. In patients with a postanoxic encephalopathy we find that $C_n = 38.2 \pm 6.3$ and $C_{n, \text{source}} = 19.7 \pm 6.4$. For patients with generalised seizures, $C_n = 39 \pm 7$ and $C_{n, \text{source}} = 32 \pm 14$.

These values of the neural complexity found are not in agreement with the recently stated predictions by Tononi and Edelman [19]. They hypothesise, that during cognitive activities involving consciousness there should be evidence for a large but distinct set of distributed neuronal groups that interact over fractions of a second much more strongly with themselves than with the rest of the brain, which should be reflected in their complexity. In addition, complexity should correlate with the conscious state of the subject, and neural complexity should be extremely low during epileptic seizures. Our current findings do not support this hypothesis and possible explanations will shortly be discussed.

Complexity is affected by the nature of the measurements used, and will be influenced by noise in the data. The presence of uncorrelated noisy data will emulate a system which is more independent, in the limit creating a cross-correlation matrix diag(1, . . . , 1), and will therefore reduce the value of the complexity. If the assumption that the functions measured are primarily generated by the local subsystem $x_i$ only, as stated in Eq. (7), is not satisfied, the cross-correlation matrix obtained will contain values skewed to those that would be obtained from a system with relatively more dependent subsystems. This may reduce the values of the complexity found, as well.

To correct for the contributions of the functions $g$ (see Eq. (7)), a source reference was applied. This did not change the observation, however, in the sense that $C_N$ remained higher in the pathological brain states where information processing is severely reduced or

Table 1
Integration and complexity for the different groups studied

<table>
<thead>
<tr>
<th></th>
<th>Control, EC</th>
<th>Control, EO</th>
<th>BS</th>
<th>SZ</th>
</tr>
</thead>
<tbody>
<tr>
<td>$(n = 21)$</td>
<td>$(n = 21)$</td>
<td>$(n = 6)$</td>
<td>$(n = 3)$</td>
<td></td>
</tr>
<tr>
<td>Average I(X)</td>
<td>12.35 ± 1.67</td>
<td>11.77 ± 2.20</td>
<td>18.3 ± 4.7</td>
<td>19 ± 4</td>
</tr>
<tr>
<td>$C_n$</td>
<td>26.86 ± 2.49</td>
<td>25.38 ± 2.01</td>
<td>38.2 ± 6.3</td>
<td>39 ± 7</td>
</tr>
<tr>
<td>Source I(X)$_{\text{source}}$</td>
<td>4.69 ± 0.76</td>
<td>4.62 ± 0.87</td>
<td>8.5 ± 3.6</td>
<td>15 ± 6</td>
</tr>
<tr>
<td>$C_{n, \text{source}}$</td>
<td>13.74 ± 2.35</td>
<td>12.93 ± 1.92</td>
<td>19.7 ± 6.4</td>
<td>32 ± 14</td>
</tr>
</tbody>
</table>

The errors relate to a single standard deviation. EO = eyes open, EC = eyes closed, BS = burst suppression and SZ = seizures. Average relates to the average reference montage; source to the source reference montage.
The major factor that may be responsible for our findings, however, is probably related to the characteristics the entropy measure used, as will now be discussed with regard to the analysis and interpretation of the epilepsy data. Although it is generally believed that during generalized seizures all brain areas are pathologically coupled, $C_N$ is higher than in the control subjects. A detailed inspection of the EEG recording obtained during an epileptic seizure shows in particular a strong coupling at a single frequency (here, approximately 3 Hz), while at the same time the several signals clearly contain different harmonics. This latter is caused by a variety of factors, such as the different orientations of the cortical neurons, that yield different projections of the brain’s electro-magnetic field at the scalp [2]. In addition, the passing of the cortical EEG signal through the cerebrospinal fluid, dura mater, bone, galea and scalp has a strong attenuating effect on the amplitude and phase of the original signal. All these factors will contribute to the value of the neural complexity, $C_N$ since the integration, as defined by Eq. (3), is based on the calculation of the cross-correlation function of the measured time series. If the recorded amplitudes at all different recording sites were strictly linearly related, i.e., $A_i = c_{ij} A_j$, the complexity would indeed attain a low value. Stated differently, although the signals show a coherence $\approx 1$ at the fundamental frequency $f \approx 3$ Hz, the correlation is not maximal, being a function of all frequencies and their phases present in the signals. Therefore, the current (local) measure used, i.e., the raw time-series, may not be the parameter that represents the best a particular characteristic, being the local information processing activity of the elementary subsystem $x_i$. Stated more precisely: the probability density function of the “raw signals”, used in the calculation of the entropy (by the cross-correlation matrix), relates to the amplitude distribution of the signals, and may not represent the “correct” measurement function.

We conclude that a straightforward implementation of this neural complexity measure $C_N$, using standard EEG recording techniques and the cross-correlation of the time-series as a measure for their dependency, does not show decreased values in pathological conditions with lowered consciousness levels where information processing capacity is reduced. However, application of this interesting concept to high-spatial-resolution recordings and/or using different measurement functions, such as zero-crossing intervals or phase information, may allow a definition of neural complexity that does strongly correlate with the information processing capacity of the brain and the clinical condition of (neurological) patients. Currently, these alternatives are being studied in their application to a similar neural complexity measure.

Appendix A

A.1. Entropy definition, $H$

The intuitive notion of entropy is that it is the logarithm of the number of possible states that the system can occupy. Thus, if $X$ is a discrete variable, so that it can only have values $x_1, x_2, \ldots, x_K$, and each of the $K$ values occurs with a probability $p_k$, then the entropy is

$$H = -\sum_{m=1}^{K} p_k \log_2 p_k. \quad (8)$$

This expression has a natural generalization to the case where $x$ has a continuous range of values, so we have a probability distribution function $P(x)$ rather than a discrete set of probabilities. In this continuous case,

$$H = -\int P(x) \log_2 P(x) \, dx. \quad (9)$$

The problem with measuring the entropy of continuous variables, however, is that the “number of possible states” is infinite and Eq. (9) will not converge to a finite value. In practice, however, we are primarily interested in entropy differences. Considering two continuous systems, $A$ and $B$, we find for their entropy
difference, using Eq. (9) that
\[ H_A - H_B = - \int \frac{\log_2[P_A(x)]}{\log_2[P_B(x)]} dx, \] (10)
which is a well defined quantity. In our application, the entropy differences are being calculated using Eqs. (2) and (3).

We remark that in the current interpretation, the several states of the system are the values of the amplitudes of the signals measured at the scalp, i.e., the probability density function relates to the amplitude distribution of the recorded signals. In this appendix, we will illustrate in more detail the behaviour of \( C_N \) for systems that are comprised of subsystems with varying levels of dependency. In addition, we present the operational procedure applied in our calculations.

A.2. Application of the neural complexity measure to a model system

Consider an elementary model system, \( \mathcal{M} \), comprised of 25 elementary subsystems, \( x_i \), where the signals measured from each subsystem \( x_i \) are denoted by \( x_i(t_n) \) as
\[ x_i(t_n) = a_i \sin(\omega_{i,1} t_n + \phi_i) \cos(\omega_{i,2} t_n) \cos(\omega_{i,2} t_n/10) + n_i(t_n), \] (11)
where \( a_i \), \( \omega_{i,1} \), \( \omega_{i,2} \) and \( \phi_i \) are constant for each subsystem \( x_i \), with \( i = 1, \ldots, 25 \), and \( n_i(t_n) \) a zero mean white noise process.

We will now construct three different model systems \( \mathcal{M} \) to represent three different levels of dependence between the several elementary subsystems, and discuss the calculation of the integration, \( I(X) \) and neural complexity, \( C_N \) for these three systems: these three model systems are illustrated in Fig. 6, where we indicated a random selection of a subset with size \( k = 3 \), as well.

A.2.1. Model A: complete independence

In this realization, all subsystems are independent, each modeled as a zero mean, white noise process:
\[ x_i(t_n) = n_i(t_n). \] (12)

The data matrix \(^2 X^k \), being a collection of \( k \) of the elementary subsystems, is given by
\[ X^k = \begin{bmatrix} x_1(t_1) & x_2(t_1) & \ldots & x_k(t_1) \\ \vdots & \vdots & \ddots & \vdots \\ x_1(t_m) & x_2(t_m) & \ldots & x_k(t_m) \end{bmatrix}, \] (13)
where \( m \) is the amount of samples. Note, that the indices of the subsystems for values of \( k < N \), with \( N \) the size of the model system \( \mathcal{M} \), are randomly assigned to each of the original subsystems, as was shown in the illustration Fig. 6.

Since \( X^k \) is composed of independent samples, the correlation matrix of \( X^k \), \( \text{Corr}(X^k) \), will approximate the unit matrix of order \( k \), \( \text{diag}_k(1, \ldots, 1) \). Therefore, we find for the value of the integration \( I(X^k) \) that
\[ I(X^k) \approx -0.5 \ln(\text{det}(\text{diag}_k(1, \ldots, 1))) = -0.5 \ln 1 = 0 \] (14)
for all values of \( k \). The neural complexity, therefore, will also approach zero for this system comprised of completely independent subsystems, as is directly clear from Eq. (1).

Using \( I(X^k) = \sum_{i=1}^{k} H(x_i) - H(X^k) \) we can also argue that, given the independence, the entropy of the system with subset size \( k \) is the sum of the entropies of the elementary systems \([23]\), i.e.,
\[ H(X^k) = \sum_{i=1}^{k} H(x_i), \]
and therefore \( I(X) = 0 \), in agreement with the fact that the integration is a measure for the amount of dependence present.

A.2.2. Model C: (nearly) complete dependence

Nearly complete dependence was modeled using
\[ x_i(t_n) = 10 \sin(t_n) \cos(t_n/10) + 0.1 n_i(t_n) \] (15)
for all subsystems.

Using \( I(X^k) = \sum_{i=1}^{k} H(x_i) - H(X^k) \) we argue that, if and only if there exists complete dependence, the entropy of the system with subset size \( k > 1 \) equals the entropy of any elementary system, i.e.,
\[ H(X^k) = H(x_i) = \gamma = \text{const}. \]
Therefore \( I(X^k) = \sum_{i=1}^{k} \gamma - \gamma = (k - 1) \gamma \), showing a linear dependence on the

\(^2 \) We denote the data matrix with similar symbolics as the subset with size \( k \).
subset size. Using the expression for $C_N$ in Eq. (1) we find

$$C_N = \sum_{k=1}^{N} \left[ \frac{k-1}{N-1} (N-1)^{\gamma} - (k-1)^{\gamma} \right] = 0. \tag{16}$$

Note, that the integration, calculated from $I(X^k) = -0.5 \ln |\text{Corr}(X^k)|$, approaches infinity if the subsystems become completely dependent, since the determinant of $X^k$ will approach zero. The complexity measure, however, is zero, due to Eq. (16). We recall that in the case of completely independent subsystems, the complexity was zero because the integration was zero for all subset sizes $k$.

### A.2.3. Model B: intermediate (in)dependence

In this case, the integration is finite, and will not be a linear function of the subset size, since linear
dependence is only found in completely dependent subsystems, as pointed out previously.

For complete independency we found that \( H(X_k) = \sum_{i=1}^k H(x_i) \), the latter being an upper bound for the entropy, since the entropy for intermediate dependency cannot be larger than the entropy for a complete independent system. Therefore, in the case of intermediate dependency, it holds that \( H(X_k) < \sum_{i=1}^k H(x_i) \), for all \( k \), where \( H(X_{k-1}) < H(X_k) \). If this latter inequality would not hold, the system would be completely dependent, contradicting our assumption of intermediate (in)dependency. This implies for the integration that \( I(X_k) \geq 0 \) with \( I(X_{k-1}) < I(X_k) \), i.e., the integration increases monotonically with increasing subset size \( k \). But as stated previously, the integration cannot increase linearly, because this is only found in a completely dependent subsystem. Therefore, the integration will show a parabolic behaviour as a function of the subset size, as illustrated in, e.g., Fig. 1.

### A.3. Outline of the calculations

The calculation of the integration is performed as follows:

1. Select randomly a sufficiently large collection of subsets with size \( k \) from the \( N!/k!(N-k)! \) possibilities, and create a collection of matrices \( X_k \), starting with \( k = 1 \);
2. Calculate the integration, \( I(X^k) \), for each subset in this collection of subsets with size \( k \), using Eq. (3);
3. Calculate, from the previous findings, the average value of the integration for the collection of subsets with size \( k \);
4. Repeat the procedure for \( k = k + 1 \), up till \( k = N \).

After having obtained the average values of the integration for all the collections of the subsets with size \( k \), the neural complexity, \( C_N \), is calculated using Eq. (1).

### References