

Multivariate stochastic coupling in unknown complex systems: Informative randomness in deterministic brain-body communication

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Physiological systems exhibit deterministic behavior influenced by dynamic stochasticity, where informative random components play a crucial role in complex system interactions. Fully characterizing multivariate random components is challenging when the underlying deterministic dynamics are unknown. We propose a framework for estimating stochastic coupling in unknown deterministic systems influenced by dynamic noise. Building on a multivariate extension of approximate entropy (MAPEn), the proposed framework exploits the divergence of this metric in the presence of dynamical noise. After establishing the theoretical basis and validity of the method, which can accommodate arbitrary multivariate noise probability distributions, we show that, in the case of bivariate white Gaussian noise, the framework enables closed-form estimation of the noise covariance matrix without requiring prior knowledge of the underlying deterministic functions. To this end, we introduce an iterative algorithm to estimate the covariance matrix coefficients. The algorithm identifies the parameter values that rescale the differential behavior of multivariate MAPEn along a target curve. We validate the framework using both synthetic datasets and real physiological recordings. The analysis reveals important insights into brain-heart interactions, particularly the modulation of stochastic coupling across sleep-stage transitions through low-frequency oscillatory activity. Our results indicate that conscious wakefulness is characterized by stronger stochastic coupling, which progressively decreases during the transition to sleep. This framework opens new avenues for investigating stochastic physiological dynamics and their associated networks, with potential applications across a wide range of scientific disciplines.

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I. INTRODUCTION

Coupling and coupling strength provide quantitative information for characterizing the collective behavior of multiorgan and multisystem interactions [1–3]. Estimation of coupling functions from real experimental data typically requires prior knowledge or hypotheses about the dynamical systems under study, which are generally assumed to be either fully deterministic or stochastic [3].

Considering physiological systems as fully deterministic, i.e., systems governed by a specific set of analytical equations, coupling functions may provide detailed insights into the functional and anatomical rules that govern the systems’ interactions. In the literature, several methods have been proposed for reconstructing deterministic coupling functions from experimental data based on a variety of inference techniques, such as least-squares and kernel smoothing fits [4], dynamical Bayesian inference [5], maximum likelihood

(multiple-shooting) methods [6], and phase equations [7], among others. Although these methods often assume periodicity in physiological signals, which can be approximated through Fourier analysis by considering a limited number of oscillations, many methods may lose crucial information related to discarded frequencies, particularly in cases of nonlinearity, chaos, and dynamic stochastic perturbations.

Considering physiological systems as fully random—represented by realizations of interacting random variables—a simple but effective measure of functional coupling is the Pearson’s correlation coefficient, which quantifies statistics up to the second order. In an information-theoretic framework, statistical coupling is defined as the information transfer between variables within a random process [4,8]. Coupling functions between such systems may be provided by the joint entropy and related mutual information. While the former provides a quantification on the predictability of the joint processes, the latter indicates the amount of information shared among the network’s components or, equivalently, how much knowledge of one variable reduces the uncertainty of the others [9]. Note that the mutual information between Gaussian random variables is directly related to Pearson’s correlation coefficient in a closed-form expression. Despite their broad applicability, information-theoretic quantifiers face challenges in estimating physiological joint probability density functions and marginals, particularly when data are limited, heavily

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corrupted by noninformative noise, or when the systems are also governed by an underlying deterministic function.

Scientific literature lacks a methodological framework tailored for assessing interactions actual physiological systems, which embed both deterministic and random components. Consequently, it is unknown whether such stochastic components may be an integral part of the interacting multiorgan and multisystem dynamics. This issue is particularly relevant for brain-body communication, which comprises many recursive physiological pathways whose dynamics may not be described by explicit analytical equations.

In this study, we propose a methodological framework allowing to estimate stochastic coupling in multivariate physiological systems driven by dynamical noise, when the systems' equations are unknown. Next, without loss of generality, we formalize the problem in hybrid bivariate systems. Let us consider two dynamical systems contaminated by dynamical noise, assuming that the latter takes the form $(x_{n+1}, y_{n+1}) = T(x_n, y_n) + (\varepsilon_{n,x}, \varepsilon_{n,y})$, where the map $T : \mathbb{R}^2 \rightarrow \mathbb{R}^2$ defines a dynamical system and the pair $(\varepsilon_{n,x}, \varepsilon_{n,y})$ is a realization of a bivariate random Gaussian variable with mean $(0,0)$ and a covariance matrix Σ . Starting from the observed bivariate data, the series $\{(x_n, y_n)\}_{n=1}^N$, no methods are currently available in the literature for detecting the covariance matrix Σ , particularly the antidiagonal of Σ , which represents the correlation between the random variables ε_x and ε_y . From now on, this correlation will be called *stochastic correlation*, as it involves only the nondeterministic part of the systems.

In a previous study, we proposed a framework to estimate dynamical noise in univariate systems [10]. To this end, any physiological signal $\{z_n\}$ is regarded as the output of a hidden dynamical system described by the unknown linear or nonlinear map T , while the noise is assumed to be a realization of independent and identically distributed (IID) Gaussian random variables η_n with mean 0 and variance σ^2 , which acts on the dynamics generating series of the form $z_n = T(z_{n-1}, \dots, z_0) + \eta_n$. The nontrivial estimation of the physiological noise power σ^2 has been achieved in a closed form [10] by exploiting the theoretical divergence of the approximate entropy [11] quantifier in noisy series. Approximate entropy was originally introduced as a practical approximation of Kolmogorov-Sinai entropy for short time series [12]. While both measures aim to quantify the complexity of a system's behavior, Kolmogorov-Sinai entropy is formally defined through a limiting process over infinite trajectories, making it theoretically rigorous but impractical for real-world data. Approximate entropy, by contrast, provides a family of statistics [11] that maintain interpretability while allowing for tractable estimation from finite time series.

Here the stochastic coupling among the random components of bivariate dynamical systems—namely all the entries of the matrix Σ —are estimated independently of the map T , i.e., without making assumptions about the form of T . Leveraging previous endeavors [13], we propose a multivariate extension of the approximate entropy quantifier that considers the system's evolution along all dimensions simultaneously. Then we state a theoretical result linking the shape of the multivariate approximate entropy quantifier as a function of the radius, one of its parameters, with an expression that

contains all the terms in the matrix Σ . From this formal result, we derive a simple algorithm that accomplishes the task of retrieving the stochastic coupling among the components of bivariate systems. The method is then tested on synthetic data gathered from bivariate noisy dynamical systems and is applied to characterize bivariate physiological systems.

While our approach estimates noise as an intrinsic element of the phenomenon—one that is inherently embedded in its dynamics—related concepts have also been explored in the literature. Specifically, several studies have focused on estimating noise correlations in the brain, which refer to statistical dependencies between the seemingly random fluctuations (i.e., noise) in the activity of different neurons or neural populations. These correlations typically emerge during the encoding of neural information, as activity patterns vary across repeated exposures to the same input and become correlated across neurons [14]. Noise correlations have been employed to explain various cognitive and behavioral phenomena, including performance in discrimination tasks, the precision of spatial encoding in the hippocampus [15], their effect on classification accuracy [16], and their role in accelerating and enhancing learning processes [17].

In information theory, the concept of predictability is closely linked to conditional entropy (CE), a general statistical measure that quantifies the uncertainty of the present state given the past [18,19]. Although CE and standard differential entropy focus primarily on probabilistic dispersion, nonlinear quantifiers of dynamical complexity, such as approximate entropy (ApEn), are designed to characterize the topological properties of the underlying deterministic flow, yielding values near zero for regular dynamics and positive values for chaotic regimes. The primary goal of this research is to investigate how these specific nonlinear complexity quantifiers are structurally perturbed by intrinsic dynamical noise and to demonstrate how the continuous profile of these metrics can be exploited to extract the underlying stochastic coupling. We focus on investigating the role of stochastic coupling between neural and cardiovascular systems during sleep stage transitions, aiming to characterize the so-called brain-heart axis. Noninvasive electroencephalographic (EEG) signals are then considered as outputs of the cerebral cortex, while heartbeat dynamics derived from electrocardiographic (ECG) signals represent the output of the neuroautonomic control on the cardiovascular system. Data are gathered from a publicly available dataset comprising data from 53 healthy subjects in free-sleeping. Sleep is a fundamental condition in human physiology. According to physiological reasoning and the associated electrophysiological features, some major sleep phases might be identified, mainly the awakenings, the rapid eye movement (REM) phases, and the non-REM (NoREM) phases, which could be further subclassified. While the behavior of a single signal (e.g., EEG and ECG) features during the sleep evolution is well known, and the existence of a stochastic component in such physiological signals dynamics has been ascertained, it has never been investigated if, and eventually how, such stochastic components interact. Several studies in the literature demonstrate that sleep depth significantly impacts heart-brain functional coupling, as evidenced by directional measures such as transfer entropy, Granger causality, and related techniques. Specifically, these

interactions generally weaken progressively with increasing sleep depth, while central brain regions (C3 and C4) play a crucial role in reflecting these modulations [20]. Furthermore, heart-brain coupling appears more pronounced in some cortical areas than in others, with its strength varying across different sleep stages [21]. Bidirectional brain-heart interactions are stronger during wakefulness, which is characterized by more dominant brain-to-heart regulation [22]. Additional findings indicate that activation of the brain-heart axis during REM sleep causally contributes to conscious dreaming experiences [23]. Moreover, information transfer from heartbeat and respiration dynamics to the brain increases progressively across REM, light, and deep sleep stages, with the occipital lobe notably receiving the greatest input from the cardiorespiratory system during light sleep [24]. On another note, investigations using the maximal information coefficient revealed that brain-heart coupling exhibits patterns specific to sleep stages: The EEG- β band shows distinct coupling with heart rate variability (HRV) frequency bands depending on the sleep stage, whereas the EEG- δ band demonstrates stronger coupling with HRV during non-REM sleep [25]. Similarly, synchronization likelihood analyses have uncovered a nonlinear dynamic interdependence between HRV and sleep EEG power bands [26]. It is also well established that brain-heart interactions represent a dynamic bidirectional coupling, where neural activity modulates cardiac function and cardiac signals, in turn, influence brain dynamics [27]; cardiovascular changes observed across sleep stages—wakefulness, NoREMS, and REMS—reflect dynamic coupling between sleep-related central commands and autonomic control, although the exact neural pathways remain largely unknown and hold clinical significance [28]. Sleep is characterized by dynamic coupling between the central nervous system and the autonomic nervous system, with coordinated cortical and cardiac oscillations fluctuating across sleep stages and events, yet the causal mechanisms underlying this interaction remain unclear [29]. Finally, the coupling between heart period and blood pressure varies by sleep stage, with deep NoREMS exhibiting the strongest baroreflex-driven coupling alongside diminished influence from central autonomic commands [30]. To conclude, multidisciplinary studies reveal that bidirectional interactions between the arterial baroreflex and arousal—mediated by brainstem nuclei—form a positive feedback loop coordinating autonomic and brain state transitions, whose dysfunction may underlie cardiovascular and sleep-wake disorders such as obstructive sleep apnea and insomnia [31]. Coupling mechanisms are also critical in the brain-respiration system, influencing its interaction with the external environment across various timescales, particularly in response to dynamic external stimuli [32]. This system plays a key role in regulating mental activity and its underlying dynamics [33]. Evidence from fMRI analyses indicates that coupling between external stimuli and intrinsic neuronal timescales (INT) is preserved during sleep, with stimulus-induced modulation of INT suggesting that brain responsiveness and dynamic state transitions remain active even in unconscious states [34]. Scale-free dynamics and changes in stochasticity have also been observed in brain activity under external stimulation and anesthesia [35,36]. Moreover, neuronal entrainment has been identified as a key mechanism in

coupling brain rhythms with both internal bodily and external environmental rhythms, enabling precise coordination and modulation of information flow across multiple spatial and temporal scales in sensory and cognitive processes [37]. Next, we show that significant modulation of stochastic coupling is observed throughout healthy sleep transitions, especially sustaining functional brain-heart interactions through low-frequency oscillations.

II. MULTIVARIATE STOCHASTIC COUPLING IN UNKNOWN COMPLEX SYSTEM

We introduce a theoretical framework, leveraging a formal mathematical proof, that yields closed-form estimates of second-order stochastic components in any multidimensional deterministic system. Although the underlying deterministic dynamics may be linear or nonlinear and entirely unknown, the associated time-varying stochastic components can follow arbitrary distributions and exhibit any correlation structure. The proposed methodology uses multivariate approximate entropy to estimate the covariance of bivariate stochastic components for any assumed probability density function, given a bivariate experimental time series from an unknown multidimensional dynamical system.

We exploit the multivariate approximate entropy (MApEn) profile detailed below to uncover properties of the underlying multivariate system dynamics and dynamical noise. Specifically, our goal is to detect stochastic coupling in a multidimensional time series without prior knowledge of the underlying (deterministic) dynamics. For simplicity, we illustrate the framework using a two-dimensional series $\{(x_n, y_n)\}_n$. Consider (X, T, μ) as a metric dynamical system defined on a compact subset $X \subset \mathbb{R}^2$, described by a continuous and differentiable C^1 map $T \rightarrow X$ and a T -invariant measure μ . Assume that the system is dynamically perturbed in both dimensions by correlated stochastic disturbances, represented by two sequences of IID random variables $\varepsilon_{x,n}$ and $\varepsilon_{y,n}$, identified by a covariance matrix Σ , which influence the system according to the following equation:

$$\begin{aligned} x(n+1) &= T_1(x(n), y(n)) + \varepsilon_{x,n} \\ y(n+1) &= T_2(x(n), y(n)) + \varepsilon_{y,n} \end{aligned} \quad (1)$$

where $(x(n), y(n)) \in \mathbb{R}^2$ and T_1 and T_2 are the two coordinates specified by the map T . According to the proposed MApEn extension, we state the following theorem.

Theorem 1. Let (X, T, μ) be a discrete dynamical system defined by a C^1 map T , i.e., differentiable, on a compact set $X \subset \mathbb{R}^2$ and preserving the probability measure μ , whose density h is also C^1 . Define the noise as the realization of a bivariate stochastic process $\{(\varepsilon_{x,n}, \varepsilon_{y,n})\}_n$ of independent and identically distributed random variables following a Gaussian distribution of mean $(0,0)$ and defined by the covariance matrix

$$\Sigma = \begin{bmatrix} \sigma_x^2 & \rho^\varepsilon \sigma_x \sigma_y \\ \rho^\varepsilon \sigma_x \sigma_y & \sigma_y^2 \end{bmatrix},$$

where σ_x^2 , σ_y^2 , and ρ^ε denote, respectively, the variances and the Pearson correlation of the random variables ε_x and ε_y . σ_x and σ_y denote the standard deviations of the previous random

variables. For any perturbed orbit of the system

$$\begin{pmatrix} x(\sigma) \\ y(\sigma) \end{pmatrix} = \begin{pmatrix} x_0 \\ y_0 \end{pmatrix}, T \begin{pmatrix} x_0 \\ y_0 \end{pmatrix} + \begin{pmatrix} \varepsilon_{x,1} \\ \varepsilon_{y,1} \end{pmatrix}, \\ T \left(T \begin{pmatrix} x_0 \\ y_0 \end{pmatrix} + \begin{pmatrix} \varepsilon_{x,1} \\ \varepsilon_{y,1} \end{pmatrix} \right) + \begin{pmatrix} \varepsilon_{x,2} \\ \varepsilon_{y,2} \end{pmatrix}, \dots \end{pmatrix}$$

for the MApEn it holds

$$\begin{aligned} \text{MApEn}(\{x(\sigma), y(\sigma)\}_{n=1}^{\infty}, D, r) \\ \approx -\ln[r^2/(\pi\sigma_x\sigma_y\sqrt{1-(\rho^\varepsilon)^2})] \end{aligned} \quad (2)$$

as $r \rightarrow 0^+$, for any dimensional embedding vector $D = (m_1, m_2)$ containing the embedding dimensions of the univariate time series.

Proof. Case $D = [1, 1]$: Let $X = (x_1, \dots, x_i, \dots)$ and $Y = (y_1, \dots, y_i, \dots)$ be the components of a bivariate independent and identically distributed white Gaussian process with mean $(0,0)$ and a covariance matrix described by Σ . The $\text{MApEn}([X, Y], [1, 1], r)$ is the $-\ln$ conditional probability $\mathcal{P}$ that any 2 four-dimensional vectors $z_i := (x_i, x_{i+1}, y_i, y_{i+1})$ and $z_j := (x_j, x_{j+1}, y_j, y_{j+1})$ remain r -close, given the fact that $w_i := (x_i, y_i)$ and $w_j := (x_j, y_j)$ are r -close. In symbols

$$\begin{aligned} \text{MApEn}(Y, [1, 1], r) = - \int \ln \mathcal{P}(|z_i - z_j| \leq r, \\ \text{given } |w_i - w_j| \leq r) d\mu(z_i), \end{aligned}$$

where μ is the invariant probability measure. Since the samples are independent and identically distributed, the inner term of the right hand-side of the previous term becomes

$$-\ln \mathcal{P}(|(x_{i+1}, y_{i+1}) - (x_{j+1}, y_{j+1})| \leq r), \quad (3)$$

which corresponds to

$$-\ln \mathcal{P}_\psi(B_r(0, 0)),$$

i.e., minus the logarithm of the measure of the bidimensional square of length $2r$ according to the probability density function ψ given by the difference of any two Gaussian bivariate random variables with mean 0. If r is small enough, then we obtain

$$\begin{aligned} -\ln \mathcal{P}_\psi(B_r(0, 0)) &\approx -\ln[4r^2\psi(0, 0)] \\ &= -\ln[r^2/(\pi\sigma_x\sigma_y\sqrt{1-\rho^2})] \end{aligned}$$

since

$$\psi(x, y) = (2\pi)^{-1} \det(2\Sigma)^{-1/2} \exp\left(-\frac{1}{2} \begin{pmatrix} x \\ y \end{pmatrix} (2\Sigma)^{-1} \begin{pmatrix} x \\ y \end{pmatrix}\right)$$

follows by hypothesis. By integrating with respect to the invariant measure μ , the thesis follows.

Case $D \neq [1, 1]$: The proof directly retraces the case $D = [1, 1]$. Despite the invariant measure μ is defined on a larger space, the stochastic independence of the time series samples reduces the MApEn into a simpler formula as expressed in Eq. (3). ■

The demonstration for a generic map T is achieved by following the same steps outlined in Ref. [10], which involve

considering Taylor expansions up to the fourth order of conditional probabilities, where the measures given by the noisy bivariate random variables account for the drifts of the map T .

Remark 1. The theorem holds for any multivariate system with dimension $m > 2$. Indeed, if one considers m -dimensional systems perturbed by m -dimensional noise vectors ε with zero mean $\mathbf{0}$ and covariance matrix Σ , and if ψ defines the random variable representing the difference between two realizations of the multivariate noise ε and ε' , expressed as

$$\begin{aligned} \psi(x_1, x_2, \dots, x_m) \\ = (2\pi)^{-m/2} \det(2\Sigma)^{-1/2} \\ \times \exp\left(-\frac{1}{2} \begin{pmatrix} x_1 \\ \vdots \\ x_m \end{pmatrix}^T (2\Sigma)^{-1} \begin{pmatrix} x_1 \\ \vdots \\ x_m \end{pmatrix}\right), \end{aligned}$$

then by integrating over m -dimensional balls B_r and repeating the steps of the proof, it is possible to have

$$\begin{aligned} \text{MApEn}(Y, [1, 1, \dots, 1], r) \\ = -\ln \mathcal{P}_\psi(B_r^m(\mathbf{0})) \approx -\ln[2^m r^m \psi(\mathbf{0})] \\ = -\ln[r^m (\pi)^{-m/2} \det(2\Sigma)^{-1/2}], \end{aligned}$$

thereby generalizing the formulation to an m -dimensional system.

Remark 2. In the presence of dynamical noise, the asymptotic behavior of the MApEn quantifier for small r is independent of the Gaussian noise assumption. Let ψ represent the difference between two independent noise realizations in the system, with an unspecified probability density function (p.d.f.). Assuming only that the noise variables share the same unknown p.d.f., and, applying the analytical steps from our derivation, we yield:

$$\begin{aligned} \text{MApEn}(Y, [1, 1, \dots, 1], r) = -\ln \mathcal{P}_\psi(B_r^m(\mathbf{0})) \\ \approx -\ln[2^m r^m \psi(\mathbf{0})]. \end{aligned}$$

It follows from this expression that for small-enough r , the presence of noise forces the MApEn to diverge, irrespective of the underlying probability distribution. Moreover, an increase in the system's dimensionality m leads to a linearly accelerating divergence rate, given that the evaluation of the p.d.f. at the origin, $\psi(\mathbf{0})$, remains a constant scaling factor.

A. Multivariate approximate entropy

We provide a multivariate extension of the approximate entropy quantifier and use this characterization to develop a tool for assessing the relationships between the dynamical noisy components of a multidimensional system. This approach builds on the ideas presented in Refs. [13,38–40], where a multivariate, multiscale sample entropy extension has been proposed. Starting from the q -variate time series $\{y_{h,i}\}_{i=1}^N$, $h = 1, \dots, q$:

(i) Embed the time series $\{y_{h,i}\}_{i=1}^N$ into the m -dimensional vector:

$$Y_m(i) = [y_{1,i}, y_{1,i+\tau_1}, \dots, y_{1,i+(m_1-1)\tau_1}, \\ y_{2,i}, y_{2,i+\tau_2}, \dots, y_{2,i+(m_2-1)\tau_2}, \dots, \\ y_{q,i}, y_{q,i+\tau_q}, \dots, y_{q,i+(m_q-1)\tau_q}], \quad (4)$$

forming $(N - n)$ composite delay vectors, where $i = 1, 2, \dots, N - n$ and $n = \max\{m_k\} \times \max\{\tau_i\}$. Here all the m_k embedding dimensions form the $\mathbb{R}^q$ embedding vector $D = [m_1, m_2, \dots, m_q]$; all the time delays τ_i provide the time lag vector $\tau = [\tau_1, \tau_2, \dots, \tau_q]$. The quantity $m = \sum_k m_k$ represents the dimension of the embedded vectors $Y_m(i) \in \mathbb{R}^m$.

(ii) Following Ref. [11], choose the maximum or infinity norm d as the distance between any two composite vectors $Y_m(i)$ and $Y_m(j)$, i.e., $d(Y_m(i), Y_m(j)) = \max_{l=1, \dots, m} \{|y(i+l-1) - y(j+l-1)|\}$. Two points or vectors that satisfy the previous inequality are said to be r -close.

(iii) Choose a threshold r and, for a fixed embedded vector $Y_m(i)$, define P_i as the number of the $(N - n)$ embedded vectors $Y_m(j)$ that satisfy $d(Y_m(i), Y_m(j)) \leq r$.

(iv) Calculate the frequency of occurrence $C_i^m(r) = \frac{1}{N-n} P_i$ and define

$$\phi^m(r) = \frac{1}{N-n} \sum_{i=1}^{N-n} \ln C_i^m(r)$$

as the average log-probability of having two embedded vectors sufficiently close.

(v) Extend the dimensionality of the multivariate delay vector in (4) from m to $m+q$, i.e., embed the original series into the $\mathbb{R}^{m+q}$ dimensional space where the embedding vector D' is of the form $D' = [m_1 + 1, m_2 + 1, \dots, m_q + 1]$.

(vi) Following steps (ii)–(iv) for the $m+q$ dimensional vectors, obtain $\phi^{m+q}(r)$.

(vii) Define the multivariate approximate entropy, MAPEn, as the difference:

$$\text{MAPEn} = \phi^{m+q}(r) - \phi^m(r). \quad (5)$$

MAPEn then measures the average of the log-probability that along all the q directions, *simultaneously*, any two points $y_{j,l}$ and $y_{i,l}$ remain r -close given that the previous m_l points are sufficiently r -close, $\forall l = 1, \dots, q$. Therefore, the MAPEn reflects the average divergence of the orbit along all dimensions.

B. Numerical estimation of ρ^ε

Here we present a computational method to estimate the matrix

$$\Sigma = \begin{bmatrix} \sigma_x^2 & \rho^\varepsilon \sigma_x \sigma_y \\ \rho^\varepsilon \sigma_x \sigma_y & \sigma_y^2 \end{bmatrix},$$

which defines the stochastic coupling in a bivariate noisy series (x_n, y_n) of N samples, using an embedding dimension D and a step Δr .

We make the following assumptions:

(i) The dynamics are deterministic (which may include chaotic behavior) and governed by a differentiable, bidimensional map T ;

(ii) The noise in both dimensions is dynamical and modeled as a sequence of IID Gaussian random variables with mean $(0,0)$ and covariance matrix $\Sigma = [\sigma_x^2, \rho^\varepsilon \sigma_x \sigma_y; \rho^\varepsilon \sigma_x \sigma_y, \sigma_y^2]$, where ρ^ε is defined as above;

(iii) The system dynamics are perturbed by a bidimensional dynamical noise process $\{(\varepsilon_{x,n}, \varepsilon_{y,n})\}$ with the properties described above. Any orbit of the dynamical systems takes the form:

$$\begin{pmatrix} x_n \\ y_n \end{pmatrix} = T \left(\begin{pmatrix} x_{n-1} \\ y_{n-1} \end{pmatrix}, \begin{pmatrix} x_{n-2} \\ y_{n-2} \end{pmatrix}, \dots, \begin{pmatrix} x_0 \\ y_0 \end{pmatrix} \right) + \begin{pmatrix} \varepsilon_{x,n} \\ \varepsilon_{y,n} \end{pmatrix}.$$

In the context of time-series analysis, the properties of the map T are both general and well suited to describe and model a broad class of observable phenomena. The procedure can be outlined as follows: First, we observe that σ_x^2 and σ_y^2 can be obtained by applying the univariate method described in Ref. [10] to each individual time series x_n and y_n .

Next, for each component, we perform a modified z -score standardization by subtracting the mean and dividing by the intrinsic noise level. In symbols, this yields the new series $\bar{X}$ and $\bar{Y}$, where $\bar{X} = \frac{X - \text{mean}(X)}{\sigma_x}$ and $\bar{Y} = \frac{Y - \text{mean}(Y)}{\sigma_y}$. We note that the intrinsic noise levels in $\bar{X}$ and $\bar{Y}$ are both $\bar{\sigma}_x = \bar{\sigma}_y = 1$. For clarity, we redefine $X := \bar{X}$ and $Y := \bar{Y}$.

Following the methodology outlined in Ref. [10], we then plot the multivariate approximate entropy (MAPEn) profile of the series $[X, Y]$ as a function of the tolerance r . According to the above theorem, we obtain an initial rough estimate of the quantity $\sigma_x \sigma_y \sqrt{1 - (\rho^\varepsilon)^2}$ by identifying the value $\bar{r}$ where $\text{MAPEn}(X, m, r)$ and $-\ln r^2$ exhibit the most similar slopes. Since $\sigma_x = \sigma_y = 1$, the raw quantity simplifies to $\sqrt{1 - (\rho^\varepsilon)^2}$. Finally, this estimate is refined by fitting the function $s \rightarrow \text{MAPEn}(Y, D, r) + \ln(\frac{r^2}{\pi s})$ within the interval $[0, 1]$, leading to a more accurate estimation of $\sqrt{1 - (\rho^\varepsilon)^2}$, from which the stochastic coupling ρ^ε can be derived: the stochastic coupling or correlation estimated directly from the data according to the above procedure is denoted as $\rho_{x,y}^\varepsilon$. The algorithm implementation in MATLAB is available through the link provided in the Data Availability statement.

III. EXPERIMENTAL METHODS AND DATA

This study was approved by local ethics committee of the University of Pisa.

A. Numerical analysis

Synthetic series are gathered as follows.

Given the noise realizations $\{(\varepsilon_{x,n}, \varepsilon_{y,n})\}_n$ from bivariate independent and identically distributed random variables following a Gaussian distribution of mean $(0,0)$ with covariance matrix Σ , we form the bivariate time series according to

$$\begin{aligned} x(n+1) &= T_1(x(n), y(n)) + \varepsilon_{x,n} \\ y(n+1) &= T_2(x(n), y(n)) + \varepsilon_{y,n} \end{aligned} \quad (6)$$

for the dynamical noise case.

When dynamical noise effects result in orbit divergence in bivariate maps (e.g., Hénon maps), we apply bivariate output noise to introduce stochastic correlation within the deterministic maps. This procedure first requires generating noise-free

orbits, p_n and q_n , according to the following:

$$\begin{aligned} p(n+1) &= T_1(p(n), q(n)) \\ q(n+1) &= T_2(p(n), q(n)) \end{aligned}$$

A realization of the bivariate Gaussian noise $\varepsilon_{x,n}$ and $\varepsilon_{y,n}$ is then generated, with properties defined by the matrix in Ref. [6]. Last, the correlated bivariate noise realizations are added to the clean series $p(n)$ and $q(n)$, forming the perturbed orbits $x(n) = p(n) + \varepsilon_{x,n}$ and $y(n) = q(n) + \varepsilon_{y,n}$.

In order to evaluate the performance of the method, we define five possible scenarios, including Gaussian stochastic processes, logistic maps, Hénon map, and autoregressive models of order 3 [AR(3)]. Details of the map are reported in the appendix section.

For comparison, we also include the linear Pearson's correlation coefficient $\rho_{x,y}$ from the time-series data.

We notice that the results coming from the Pearson's correlation are similar to the nonlinear mutual information quantifier I , since the latter is estimated according to the closed formula

$$I(x; y) = -\frac{1}{2} \ln \det (1 - \rho_{x,y}^2), \quad (7)$$

where $\rho_{x,y}$ is the Pearson's correlation, under the hypothesis of considering the time series jointly Gaussian—this procedure is standard practice when using mutual information and avoids the probability density functions estimations or the usage of data binning techniques.

The aim of the numerical simulation is to assess the method's reliability in discerning the different values of stochastic correlation $\rho_{x,y}^\varepsilon$ for different stochastic coupling strengths. To do so, we set the time-series length equal to 10 000 points, with $\rho_{x,y}^\varepsilon$ varying in $[0,1]$ with a step of 0.1 while we have considered four noise standard variations combinations σ_x and σ_y , simulating the interactions of different magnitude order stochastic components as follows:

(i) For the bivariate white Gaussian noise process, the logistic maps, and the system composed of the logistic map and the AR(3) model, the noise levels are defined as $\sigma_x = 0.1$ and $\sigma_y \in 0.05, 0.1, 0.15, 0.2$;

(ii) For the AR(3) models: $\sigma_x = 1$ and $\sigma_y \in 0.5, 1, 1.5, 2$;

(iii) For the Hénon map with output noise: $\sigma_x = 0.4$ and $\sigma_y \in 0.2, 0.4, 0.6, 0.8$.

The four levels of the second noisy component are denoted as $\{\sigma_{y,1}, \dots, \sigma_{y,4}\}$, independent of the chosen map. For each map, we collected 200 realizations of dynamical system orbits with stochastic coupling, divided into four groups based on the magnitudes of the individual noise components in each series of the system. For all simulations, we fixed the embedding dimension to $D = [1, 1]$ and set $\Delta r = 10^{-3}$. To assess the performance of our method, we compared it with the mean phase coherence (MPC), a phase-based synchronization index commonly used in dynamical systems analysis. Unlike directional causality measures, MPC evaluates the degree of phase synchronization between two signals, computed via the Hilbert transform. It is defined as the square root of the sum of the squared mean cosine and sine of the phase difference and takes values in $[0,1]$, where 1 indicates perfect synchronization and 0 denotes no phase locking. Under phase

independence, MPC follows a chi-squared distribution scaled by the inverse of twice the signal length [41].

We applied the MPC analysis to bivariate series generated as described above.

B. Real physiological data

The experimental dataset used in this study is part of the publicly available Montreal Archive of Sleep Studies (MASS) Dataset [42]. To ensure consistency in research protocols and devices used, we analyzed the first cohort available. This cohort consists of 53 polysomnographic (PSG) whole-night recordings, collected at the Center for Advanced Research in Sleep Medicine in Montreal, Canada.

The dataset includes 17-channel EEG recordings, with electrodes placed according to the 10-20 standard scheme and signals sampled at 256 Hz. The recorded EEG channels are C3, C4, Cz, F3, F4, Fz, F7, F8, O1, O2, P3, P4, Pz, T3, T4, T5, and T6. ECG signals were recorded using a thoracic one-lead system with a sampling frequency of 512 Hz. The subjects' ages ranged from 55 to 76 years (mean 63.5, SD 5.3) with 34 males and 19 females.

Exclusion criteria included the use of antidepressant drugs, a diagnosis of depression or other major mental disorders within the last 10 years (Beck Depression Inventory score <22 and Beck Anxiety Inventory score <30), or the presence of a personality disorder. Subjects were also excluded if they had experienced concussion, traumatic brain injury, brain stroke, pulmonary disease, neurological disorder, or sleep problems (except for mild obstructive sleep apnea) in the past.

The experimental protocol required subjects to lie down freely on a bed and sleep without any other experimental manipulations during the night. The experimental procedures were approved by the local ethics committee and were conducted in accordance with the Declaration of Helsinki.

1. Sleep staging

The MASS datasets includes raw time series and the associated sleep stages. Sleep staging was performed by experienced PSG technicians according to the American Academy of Sleep Medicine (AASM) guidelines [43]. The AASM defines four main stages: awake phase, REM, and three non-REM phases, namely NoREM1, NoREM2, and NoREM3. Each stage is characterized by distinct criteria and specific physiological patterns.

For example, NoREM1 is the initial stage of sleep, characterized by a slowing of EEG power, a decrease in occipital alpha waves, and reduced muscle and eye movements, whereas NoREM2 is associated with sleep spindles and K-complexes, which are specific EEG patterns that occur only during sleep. The NoREM3 phase is marked by the dominance of low-frequency, high-amplitude EEG waves. REM sleep is associated with low-amplitude, desynchronized mixed-frequency EEG waves with occasional REMs and is typically considered to resemble wakefulness more than the NoREM phases.

2. EEG processing

EEG signal preprocessing was conducted using MATLAB R2023B software (The MathWorks Inc., 2023) and the EEGLab toolbox [44] through a semiautomated pipeline. This pipeline included band-pass filtering, bad channel rejection, independent component analysis-based artifact rejection, and average rereferencing, as recommended for BHI applications [45]. More specifically, we applied a zero-phase FIR filter (0.5–50 Hz) using a 1690-order Hamming taper, with delay automatically corrected. EEG recordings were visually inspected to identify and reject bad channels—fewer than one channel per subject on average—and to mark time windows and concurrent ECG segments corrupted by movement or other major artifacts (e.g., movement). This procedure ensured that no significant electrical or motion artifacts remained for analysis. We then performed independent component analysis (fastICA) and manually removed components corresponding to physiological artifacts (ocular, muscle, cardiac, and respiratory). Finally, data were average referenced according to established BHI guidelines [45]. Due to major artifacts, three full-night EEG recordings were discarded, yielding data from 50 individuals for further analysis. After artifact rejection, we estimated the EEG power spectral density via short-time Fourier transform with a 2-s Hamming window and 0.25-s step, producing spectrograms sampled at 4 Hz in time and 0.5 Hz in frequency. The PSD was subsequently integrated over the five frequency bands of interest, namely delta ($\delta \in [0.5\text{--}4]$ Hz), theta ($\theta \in [4\text{--}8]$ Hz), and alpha ($\alpha \in [8\text{--}12]$ Hz).

3. Heartbeat dynamics signal processing

HRV series were derived from ECG signals as the time intervals between consecutive heartbeats. Heartbeats were detected using the well-known Pan-Tompkins algorithm [46] and preprocessed to remove physiological and algorithmic artifacts using KUBIOS software [47]. The resulting series, along with the interbeat interval histograms, were visually inspected by experts. Segments containing artifacts were marked and subsequently rejected, and the corresponding EEG-derived PSD series were also labeled as artifacts.

Time-resolved spectrograms of the HRV series were estimated using the smoothed pseudo-Wigner-Ville distribution method [48]. The resulting spectrograms were integrated over the canonical HRV frequency ranges and interpolated over time to generate PSD series comparable to those derived from EEG. The HRV-related frequency bands were defined as the low-frequency (LF) band in the interval $[0.04, 0.15]$ Hz and the high-frequency (HF) band in the range $[0.15, 0.4]$ Hz. Although some controversy exists [49], this spectral representation is widely used: The LF band is considered a nonspecific marker of sympathovagal activity, while the HF band is primarily associated with parasympathetic activity [50].

4. Stochastic coupling analysis

We computed the stochastic coupling of the bivariate brain-heart physiological system by considering all possible combinations of EEG and HRV frequency bands for each of the 17 scalp channels. For each subject, the time series consisted of a maximum of 5000 samples to minimize

excessive nonstationarity, with each series created by concatenating consecutive segments from the same sleep stage. If 5000 samples were not available, then the series included the maximum number of available points. The embedding dimension was set to $D = [1, 1]$, and the radius r varied from 0 to the maximum amplitude of the series, with a Δr step size equal to $1/1000$ of the maximum series amplitude.

The objective of this analysis was to detect changes in stochastic coupling as an intrinsic feature of the brain-heart system across different sleep stages for the 17 scalp channels.

We verified that concatenating multiple series with the same stochastic matrix does not affect the proposed stochastic correlation detection, as shown experimentally in the Fig. S9 of Supplemental Material (SM) [51].

The significance of channelwise statistical differences in stochastic coupling among various sleep stages was assessed using Friedman's test when comparing more than two sleep phases, under the null hypothesis of equal medians across the conditions. Bonferroni-Holm correction for multiple comparisons has been applied. The significance threshold α was set at 0.05. Specifically, for each channel, groupwise differences were tested in three cases: (1) among all phases (i.e., awake, NoREM1, NoREM2, NoREM3, and REM) with four degrees of freedom for the test; (2) among only the sleep stages (thus excluding awakenings), with three degrees of freedom of Friedman test; and (3) among the three NoREM phases, comparing light and deep sleep stages while minimizing the impact of potential eye movement artifacts (two degrees of freedom for the test).

IV. RESULTS

A. Synthetic data

The panels in Fig. 1 illustrate the method's performance in detecting stochastic coupling in noisy dynamical systems. The upper row shows the stochastic coupling $\rho_{x,y}^e$ computed using the proposed algorithm, while the bottom row represents the Pearson correlation coefficient $\rho_{x,y}$ of the two system series, plotted against different levels of imposed stochastic correlation (x axis). The columns correspond to the different maps considered. Each panel features five lines, corresponding to the four different noise magnitudes imposed on the second component—with σ_x fixed—and the actual superimposed stochastic correlation value, depicted by the dashed red lines. Here, independently of the maps, the magnitudes are represented by levels $\sigma_{y,1}, \dots, \sigma_{y,4}$, as defined under Methods.

Overall, the proposed method provides a more accurate estimation of stochastic coupling compared to the statistical Pearson correlation. The different noise levels σ_x and σ_y do not significantly or systematically affect the performance of the proposed method.

The proposed method better detects higher stochastic correlations, as the estimations are closer to the red dashed lines representing the actual imposed values. The poorest performance for small stochastic correlations is observed in the autoregressive model, while the estimated stochastic correlations generally follow the actual trend for the other maps. Except for the logistic map in the dynamical noise regime, the estimation accuracy improves as the imposed correlation

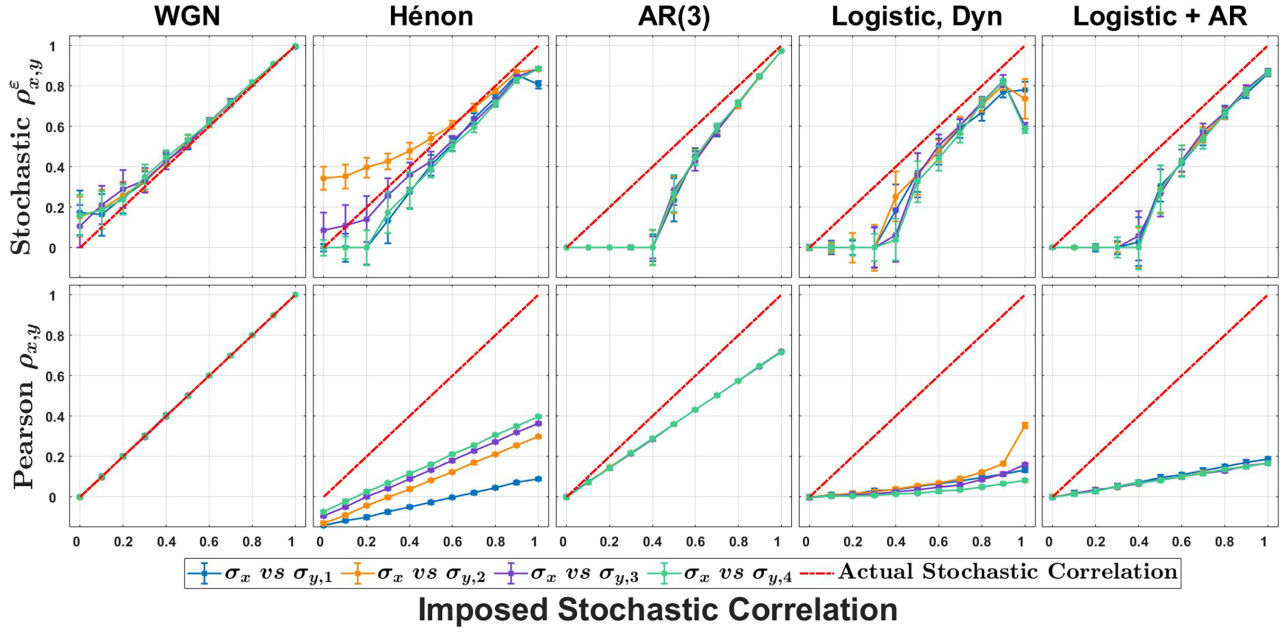


FIG. 1. Stochastic coupling estimation in synthetic data. The proposed method (top row) and the statistical Pearson coefficient (bottom row) were used to detect the imposed stochastic correlation (x axis) of bivariate noise injected into bivariate series generated by the maps specified in each column: Bivariate white Gaussian noise (WGN), Hénon map, bivariate AR(3) model, logistic maps, and a combined model of the logistic map and AR(3) model. The noise was dynamical for all maps except the Hénon map, where output noise was added. In each panel, red lines indicate the injected stochastic correlation values, while the other colored lines represent the median values $\pm$ MAD observed in 50 realizations of the maps. Each series of the bivariate recording contains $N = 10\,000$ samples. The estimations were performed using $D = [1, 1]$ and a tolerance value of $r = 0.001 \times$ largest range of the two series. Each color indicates the considered magnitudes of noise power σ_y of the second noisy bivariate component, while σ_x is kept constant. The different noise powers are detailed in the methods section and indicated here as $\sigma_{y,1}, \dots, \sigma_{y,4}$, which are defined as $[0.05, 0.1, 0.15, 0.2]$ ($\sigma_x = 0.1$) for the Gaussian processes, logistic maps, and the combined logistic-AR(3) model; $[0.5, 1, 1.5, 2]$ ($\sigma_x = 1$) for the pure AR(3) model; and $[0.2, 0.4, 0.6, 0.8]$ ($\sigma_x = 0.4$) for the Hénon map.

magnitude increases, as indicated by smaller deviations around the median values. It is noteworthy that the method also reliably estimates stochastic correlation for systems governed by two distinct dynamics, i.e., periodic (logistic map) and stochastic [AR(3)].

Conversely, the linear Pearson coefficient (and consequently the mutual information) fails to properly capture the injected level of correlation in dynamical systems. It is highly underestimated for the logistic map with dynamical noise, the Hénon map with output noise, and the mixed model of the logistic map and autoregressive model. However, as expected, the linear coefficient is precise and accurate for the bivariate white Gaussian process and the autoregressive models. Further results of the stochastic coupling estimation in case of bivariate uniform noise and of bivariate correlated Gaussian noise can be found in Secs. 1 and 2 of the Supplemental Material [51]. Details of the error propagation of $\rho_{x,y}^\varepsilon$ as function of the noise standard deviations σ_x and σ_y are reported in Sec. 3 of SM [51].

For the method comparison with the MPC, Fig. 2 depicts the median MPC computed across 50 trials for each fixed value of Pearson's coupling $\rho \in [0, 1]$, including confidence intervals.

Results indicate that MPC effectively tracks ρ in purely stochastic systems (e.g., Gaussian noise or AR models), showing similar trends to Spearman correlation (see Fig. 1). However, it fails to capture coupling changes in systems with chaotic or heterogeneous dynamics (e.g., Hénon or

logistic-AR pairs), where it remains near zero. In the case of regular dynamics (e.g., logistic-logistic), MPC slightly increases with stronger coupling, especially under low noise, reflecting underlying periodic structure.

These findings suggest that, while MPC may detect phase locking in simple stochastic systems, it does not reliably quantify noise-driven coupling in more complex or mixed-dynamics scenarios. Our method, instead, demonstrates greater sensitivity to variations in stochastic correlation, as highlighted in Fig. 1.

B. Stochastic brain-heart communication

Experimental results on stochastic coupling were obtained using real data from brain and heartbeat dynamics in healthy individuals recorded across various sleep stages. For brevity, this section presents results for brain-heart oscillations in the EEG- δ band and in the sympathovagal (HRV-LF band) and vagal (HRV-HF band) activity, as shown in Figs. 3 and 4. Results for other frequency bands are provided here in Sec. 5 of the SM [51].

Panels (a) of Figs. 3 and 4 illustrates the variations in stochastic coupling (top row) and Pearson's linear correlation coefficient (bottom row) in terms of the channelwise p values derived from the nonparametric Friedman test for paired samples. Comparisons were made across all five phases (i.e., awake, NoREM1, NoREM2, NoREM3, and REM), only sleep stages (excluding the awake phase), and only

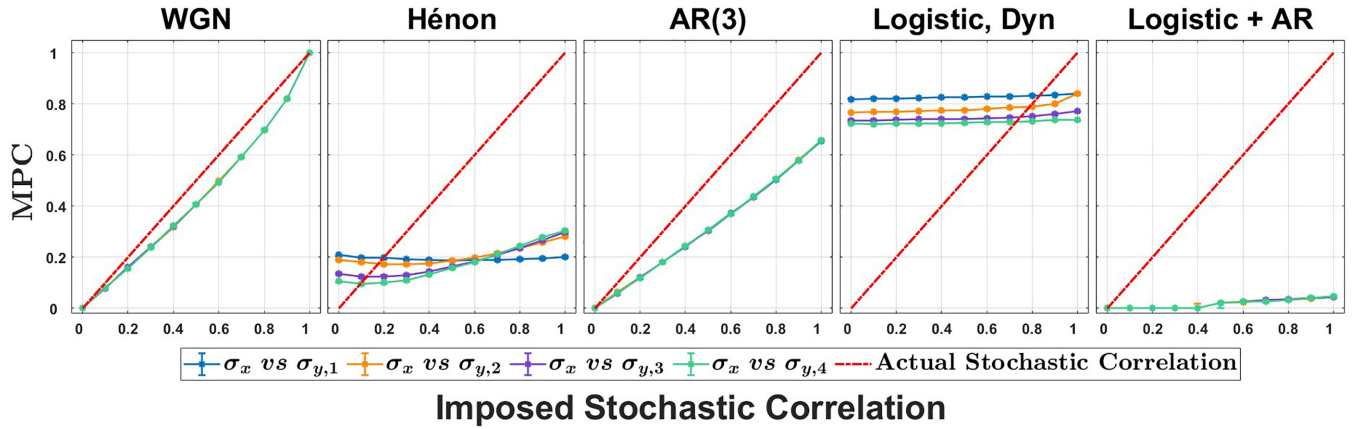


FIG. 2. Median MPC across varying stochastic coupling strengths ρ . Confidence intervals computed across 50 realizations of 10 000 samples. Maps considered are pure Gaussian noise, Hénon, logistic and AR(3) models.

NoREM stages (excluding REM). Degrees of freedom are 4, 5 and 2, respectively. White areas indicate a lack of significance, while colored areas denote statistically significant groupwise differences, with p values < 0.05 . The more intense the color, the lower the associated p values. Panels (b) shows the median distributions among subjects for stochastic cou-

pling (first row) and Pearson's linear correlation coefficient (second row) across the five sleep stages. For each column, subjectwise distributions are shown using boxplots, where each point represents the channelwise median for a specific subject, and topoplots, which display a topographical distribution of the median value across subjects. Channelwise details

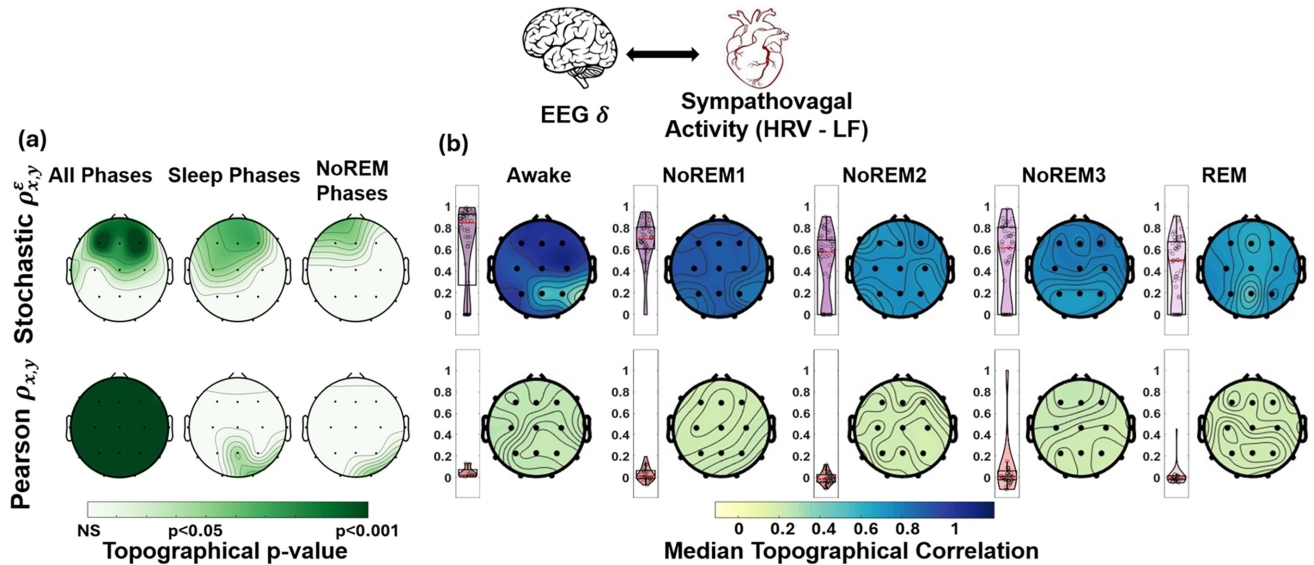


FIG. 3. Stochastic coupling in brain-heart-interplay data. Panel (a) presents a topographical representation of the groupwise statistical analysis results in terms of p values for the variations in stochastic coupling, $\rho_{x,y}^{\epsilon}$ (top row), and Pearson's linear correlation coefficient, $\rho_{x,y}$ (bottom row), across different sleep stages. Bonferroni-Holm correction has been applied with a threshold $\alpha = 0.05$. The analysis considers the spectral power signals of the heart-brain system in the LF and δ bands for HRV and EEG signals, respectively. The first column shows the results of the groupwise nonparametric Friedman test for paired samples (four degrees of freedom) across all experimental phases (i.e., awake, NoREM1, NoREM2, NoREM3, and REM). Similarly, the second column displays results for sleep phases only (excluding the awake phase), while the third column reports results considering only NoREM phases, respectively, with three and two degrees of freedom. White areas indicate no significant differences among sleep stages ($p > 0.05$), while green areas indicate statistically significant differences, with darker shades representing lower p values. Panel (b) illustrates the median distributions among subjects for stochastic coupling (top row) and linear Pearson's correlation coefficient (bottom row) across the five sleep stages. For each column, subjectwise distributions are depicted using boxplots, where each point represents the channelwise median for a specific subject. Additionally, topoplots display the topographical distribution across the scalp of the median values among subjects. As shown in the color bar, blue areas on the topoplots denote higher median correlations, while yellow and lighter areas correspond to lower median coupling values. The series of the 50 subjects here considered have at most $N = 5000$ points. Each measurement of the stochastic coupling and of the Pearson's correlation coefficient from data were taken from distinct samples.

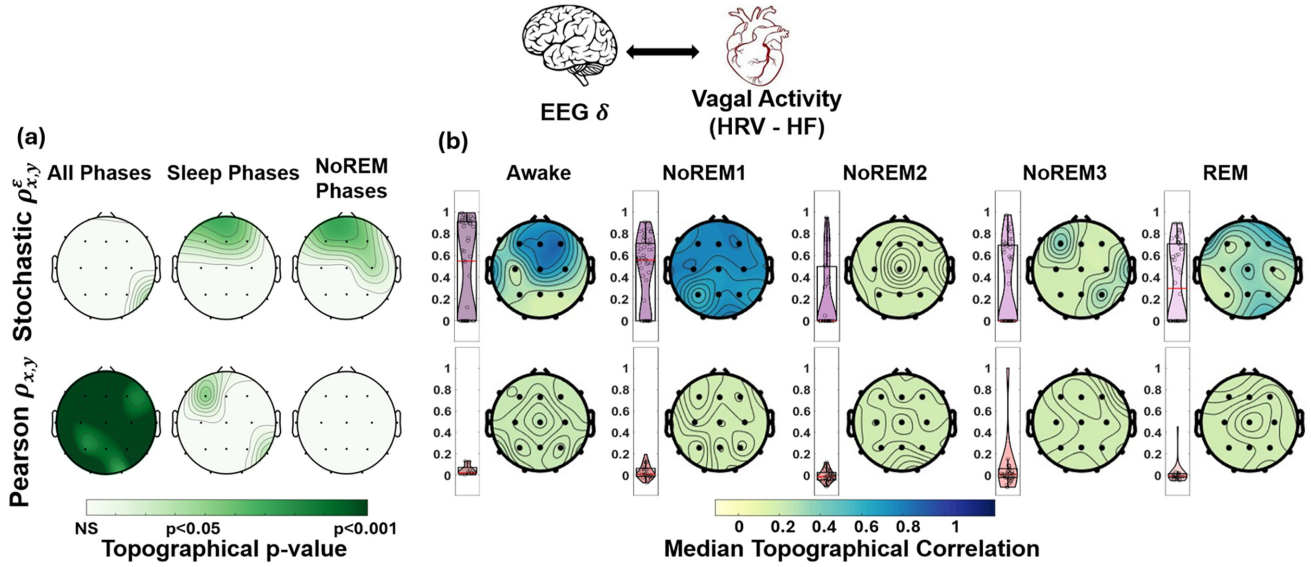


FIG. 4. Stochastic coupling in the brain-heart axis: HRV-HF and EEG- δ oscillations. Panel (a) presents the statistical significance analysis using p values for the variation of stochastic coupling $\rho_{x,y}^{\epsilon}$ (top row) and Pearson's correlation $\rho_{x,y}$ (bottom row) across different sleep stages. The analysis is based on the spectral power signals of the heart-brain system in the HRV-HF and EEG- δ bands. Each EEG signal consists of 17 channels. Bonferroni-Holm correction has been applied with a threshold $\alpha = 0.05$. The first column shows the results of the groupwise nonparametric Friedman test for paired samples (four degrees of freedom) across all experimental phases (i.e., awake, NoREM1, NoREM2, NoREM3, and REM). Similarly, the second column displays results for sleep phases only (excluding the awake phase), while the third column reports results considering only NoREM phases, respectively, with three and two degrees of freedom. White areas indicate no significant differences among the sleep stages, while greener areas indicate higher significance ($p < 0.05$). Panel (b) illustrates the median distributions of stochastic coupling (top row) and Pearson's correlation (bottom row) among subjects across the five sleep stages (AWAKE, NoREM1, NoREM2, NoREM3, and REM) using boxplots and topoplots. In the boxplots, each point represents the channelwise median for each subject, while the topoplots depict the median distribution across various scalp regions. As shown in the color bar, blue areas in the topoplots represent higher median correlation values, while yellow and lighter regions correspond to lower median coupling values. The series gathered from the 50 subjects have at most $N = 5000$ points. Each measurement of the stochastic coupling and of the Pearson's correlation coefficient from data were taken from distinct samples.

of the stochastic coupling estimation can be found in Tables S1–S6 of Sec. 5 of SM [51].

For the coupling between cardiac sympathovagal (HRV-LF) and EEG- δ oscillations, stochastic coupling variations seem predominantly in frontal and central areas and are consistent across all comparisons, being most pronounced when all phases are considered. This behavior is further confirmed by the decreasing trends in the boxplots and topographical representations in the first row of Fig. 3(b). The awake and REM phases show, on average, the highest and lowest median values, respectively. Between these two extremes, stochastic coupling gradually decreases from higher values in the light sleep NoREM1 phase to lower values in the deeper NoREM3 stage. Conversely, the Pearson correlation coefficients [bottom row of panel Fig. 3(a)] show significant scalp variations among stages only when the awake phase is considered. No significant variations are observed among sleep stages. Unlike stochastic coupling, Pearson's linear correlation coefficient remains consistently close to 0, as shown in Fig. 3(b).

For the coupling between cardiac vagal (HRV-HF) and EEG- δ oscillations, stochastic coupling fluctuations are predominantly localized over frontal and central electrode sites and remain significant across all comparisons, with the most extensive coupling detected when all sleep-wake phases are considered. Median topographical trends are highest during

wakefulness, decrease through NoREM1 and NoREM2, reach their minimum in NoREM3, and partially recovers in REM. In contrast, Pearson correlation coefficients exhibit no systematic stage-dependent variations, remaining near zero at all scalp locations and across all sleep stages. This result confirms that only the stochastic coupling metric captures the random dynamic brain-heart interactions across sleep stages.

The results for the stochastic coupling between heartbeat and EEG- θ and EEG- α oscillations are extensively reported in Figs. S10–S13 of the SM [51], which follow the same structure as Figs. 3 and 4. Specifically, Fig. S10 of SM [51] presents the estimation of stochastic coupling for oscillations in the θ band of the EEG and the LF band of cardiovascular dynamics, showing a consistent decreasing trend in stochastic coupling, primarily concentrated in the frontal scalp region. Pearson's correlation, in contrast, remains close to 0, with some variability in the occipital areas across different sleep phases. A similar trend is observed in the EEG- α band during sympathovagal activity in Fig. S11 of SM [51], although greater variability in stochastic coupling is noted in the occipital region, while the Spearman's correlation coefficient shows little variation.

For vagal activity (HRV HF band), stochastic coupling is mostly variable in the frontal region for the EEG- θ in Fig. S12 of SM [51] and EEG- α bands in Fig. S13 of SM [51],

with higher values observed during wakefulness, NoREM1, and REM. Pearson's correlation, while still close to 0, shows higher variability during wakefulness, especially in the slower EEG- θ band (Fig. S12 of SM [51]). In other cases, no significant variations were detected.

V. DISCUSSION

We proposed a methodology for characterizing stochastic interactions within multivariate physiological systems. Specifically, we focused on the brain and heartbeat dynamics, where the systems can be modeled as the sequence $\{x_n, y_n\}$ with $(x_{n+1}, y_{n+1}) = T(x_n, y_n) + (\varepsilon_{n,x}, \varepsilon_{n,y})$. Here the map $T : \mathbb{R}^2 \rightarrow \mathbb{R}^2$ defines a dynamical system, and the pair $(\varepsilon_{n,x}, \varepsilon_{n,y})$ represents a realization of a bivariate Gaussian random variable with mean $(0, 0)$ and a covariance matrix Σ . By considering noise variation as an intrinsic feature of the system, we established the stochastic correlation, focusing specifically on the coupling terms in the matrix Σ that define the stochastic components. The proposed framework assumes a hybrid structure in which the bidimensional physiological system evolves under deterministic dynamics perturbed by dynamical stochastic components. Importantly, the noise considered here is not additive or external to the system but rather *dynamical* in nature, i.e., integrated within the system's evolution and modulating its state at each iteration. The case of additive noise, which could bias the proposed methodology, is not explicitly modeled here, as such noise components can generally be mitigated through proper initial preprocessing and standard artifact removal steps prior to applying the proposed framework. Specifically, rather than being simply superimposed onto an unperturbed signal, the noise is embedded within the system such that the dynamics follow the model $x_{n+1} = T(x_n) + \varepsilon_n$, where T is a deterministic map and $\{\varepsilon_n\}_n$ represents correlated stochastic perturbations. This contrasts with the additive noise case, typically modeled as $y_n = x_n + \varepsilon_n$, where noise is introduced after the deterministic evolution. In physiological systems, such dynamical noise is increasingly recognized as an intrinsic feature rather than an external disturbance [52–56]. This perspective motivates the investigation of noise-driven coupling as a meaningful marker of physiological interdependence, potentially shaped by shared endogenous or exogenous influences. Within this context, we introduce a methodological framework to estimate the covariance of multivariate dynamical noise in a bivariate systems, without requiring prior knowledge of the underlying deterministic dynamics. By focusing on dynamical noise, our method captures a complementary dimension of interdependence—orthogonal to that revealed by traditional metrics such as Pearson correlation, mutual information, or mean phase coherence, which generally assume either purely stochastic or purely deterministic sources. If instead the system were dominated by additive or multiplicative noise, then the methodological assumptions would not hold: additive noise lies outside the embedded dynamics model, while multiplicative noise alters the deterministic structure, thereby violating the stationarity assumption underlying our framework.

To achieve this estimation, we introduce a multivariate extension of the well-known approximate entropy quantifier (MApEn) and demonstrated a result that links this quantifier to

the entries of the covariance matrix Σ . We then implemented an algorithm to estimate the stochastic coupling present in Σ and validated it on noisy dynamical systems with various levels of imposed stochastic correlation. To efficiently evaluate the MApEn profile over a range of tolerance values, the algorithm implements the cumulative histogram method [57]. This allows for the simultaneous computation of MApEn across multiple tolerances, maintaining a time complexity of $O(n^2)$ at the expense of higher memory usage, thus avoiding the computational bottleneck of sequential evaluations. To fully appreciate the theoretical foundations of the proposed methodology, it is useful to contextualize it against standard information-theoretic approaches. The ApEn is conceptually related to the CE of the present given the past. In the specific setting of a linear bivariate Gaussian AR system, this entropy can be analytically expressed as $H = \frac{1}{2} \ln((2\pi e)^2 \det(\Sigma_\varepsilon))$, explicitly linking predictability to the covariance matrix of the innovations Σ_ε [18,19]. While this represents a well-established standard result connecting CE to noise covariance in linear systems, our framework operates under a fundamentally different paradigm. Rather than estimating a single CE value, our approach exploits the asymptotic divergence profile of the MApEn across varying spatial tolerances r . Because ApEn is fundamentally a nonlinear quantifier—capable of distinguishing between regular and chaotic deterministic flows—analyzing its perturbation profile allows us to quantify the stochastic coupling even in strongly nonlinear, unknown dynamical systems where standard linear AR analytical expressions do not directly apply. Finally, we applied the method to real data representing the interplay between cardiovascular and cortical dynamics, mimicking a bivariate physiological system, across different EEG and HRV frequency bands.

Intrinsic physiological noise can disrupt underlying dynamics and introduce bias when applying complexity measures to distinguish different pathophysiological conditions. Given the complex nature of physiological systems, it is challenging to construct a model that encompasses all possible variables, making it difficult to isolate or remove the intrinsic stochastic component. For these reasons, estimating the amount of noise is essential when analyzing physiological data.

From the synthetic data, it emerged that the lower the stochasticity, the better the proposed method performs: It outperforms both linear Pearson's correlation and mutual information in detecting stochastic coupling when the time series are not purely stochastic but are governed by nontrivial dynamics. While the computationally simple statistical correlation is effective for bivariate white Gaussian processes and autoregressive models, it proves entirely inadequate for noisy time series generated by dynamical systems. Conversely, the proposed method is highly effective in the presence of deterministic dynamics, where linear correlation fails but also performs well in cases of pure stochasticity, with estimated values closely matching the actual imposed values. Notably, the method still works properly when two distinct dynamics are considered, i.e., regular and stochastic series generated by the logistic map and the AR(3) model, respectively. The only drawback is the potential difficulty in recognizing small stochastic correlations, particularly in AR(3) models. This issue may stem from the relatively small number of

samples, which may not yield a more accurate MAPEn quantifier, thereby leading to small interactions between noisy components being perceived as independent in a reduced sample size—the theorem holds for processes of infinite length.

In this context, deterministic dynamics play a fundamental role, as their presence invalidates statistical and stochastic methods for detecting correlations by altering the statistical distribution of the time-series samples when they are considered purely stochastic. Our method accounts for both the presence of dynamics and its perturbation due to noise, which could be either output noise or dynamical noise (the latter irreversibly destroys the underlying dynamics), thereby identifying stochastic correlation where classical statistical methods fail.

Analysis of real data indicates that when noise is considered an intrinsic feature of the functional brain-heart axis, stochastic coupling reveals new interactions that are not captured by classical statistical methods. Sleep is a nonstationary physiological process. To preserve local stationarity, we segmented the recordings into 30-s epochs and classified them according to standard sleep stages (Wake, NREM1, NREM2, NREM3, and REM). Given that the proposed method requires a large number of samples to detect stochastic coupling, segments belonging to the same sleep stage were concatenated to ensure sufficient statistical power.

This approach assumes that signals within each stage are governed by a common underlying process, modulated by sleep-dependent dynamical noise. Therefore, concatenating segments from the same stage does not distort the coupling estimation, as each fragment is treated as a distinct realization of the same stage-specific dynamics. As illustrated in Fig. S1 of SM [51], this strategy preserves coupling information: the estimate obtained from the concatenated sequence closely matches the coupling level embedded in the original individual subseries. This confirms that stage-specific modulation of dynamical noise can be reliably inferred from pooled data. While a time-varying implementation using a sliding window could have been adopted—such as in our previous work on univariate noise [58]—our chosen strategy ensured robust estimation by maximizing the available data within each sleep stage. This demonstrates that the brain-heart axis dynamics is governed by deterministic dynamics, driven by underlying physiological noise. In particular, such noise may result from a stochastic coupling between neurocortical and neurocardiac activities and significantly vary across sleep stages. Multivariate physiological noise in the brain-heart axis seems, in fact, more prominent in frontal and central areas when functionally linking slow δ EEG frequency and sympathovagal cardiac activity (i.e., the HRV-LF band). These variations are more evident during the wakefulness phase, where the stochastic coupling strength appears to be the highest. This coupling decreases throughout the NREM sleep stages, reaching its minimum during the REM phase. On the other hand, the Pearson's correlation coefficient detects low values only. To enhance the interpretability of our findings, we relate our empirical results to the existing literature on brain-heart coupling. Consistent with prior studies, our data confirm that brain-heart interactions progressively diminish with increasing sleep depth, particularly in central cortical regions (e.g., C3 and C4) [20]. This observation aligns with previous re-

ports of sleep-stage-specific modulation of cortical-autonomic dynamics [21], wherein wakefulness tends to exhibit stronger top-down regulation from the brain to the heart [22,59]. Stage-dependent characteristics are also evident in the associations between EEG frequency bands and HRV components. For instance, beta-band activity displays distinct coupling profiles across sleep stages, whereas delta-band activity shows stronger coupling with HRV during non-REM sleep [25]. Nonlinear analyses further highlight the complexity of these interactions, revealing dynamic dependencies between cardiac function and EEG power [26]. Across sleep stages—including wakefulness, NoREMS, and REMS—cardiovascular fluctuations seem to track transitions in central neural activity, underscoring the bidirectional nature of brain-heart communication [27,59]. Additionally, the coupling between heart period and blood pressure varies with sleep depth, with deeper NoREMS showing stronger baroreflex-driven control and reduced cortical autonomic modulation [30]. These patterns support the view that sleep stages govern coordinated oscillations between the central and autonomic nervous systems, although the underlying causal mechanisms remain incompletely understood [29].

We emphasize that while our MAPEn formulation serves as a robust tool for estimating stochastic structure in multidimensional time series, it is not the primary methodological innovation. Rather, the core theoretical contribution lies in the closed-form estimation of the covariance matrix of bivariate stochastic components for any assumed PDF, regardless of the underlying (unknown) dynamical system. Nonetheless, we underscore that the multivariate extension of entropy estimation is far from trivial, since various multidimensional entropy definitions capture distinct aspects of system complexity and synchronization. As highlighted in the proper framework setting in SM [51], our formulation explicitly incorporates the structure of the noise—treated as intrinsic to the signal—by linking the slope of the ApEn curve to the underlying covariance of the stochastic components. This allows for robust noise quantification and coupling estimation across a wide range of conditions, including non-Gaussian (e.g., uniform) and temporally correlated noise with $1/f$ spectral scaling. These findings demonstrate the flexibility and generalizability of our framework, while also supporting its application in physiological domains such as neurocardiac dynamics, where the noise structure is often complex and not analytically tractable.

Slow drifts in the deterministic component—such as circadian rhythms or other environmental influences—can generally be treated as additive noise and are typically identifiable and removable using established techniques. However, validating the assumptions underlying our method—particularly the Gaussianity and correlation structure of bivariate dynamical noise—is challenging when working with real physiological data. Testing for Gaussianity of dynamical noise is inherently difficult, especially in the absence of an analytical model describing the underlying process. This limitation is common in physiological recordings. The lack of a well-defined generative model, combined with the system's dynamic nature, can bias the quantification of residuals between unperturbed and noise-perturbed trajectories. This challenge is further exacerbated by the intrinsic nature of

dynamical noise, which actively influences the system's evolution at each time step. When the governing dynamics are unknown, the stochastic components may exhibit time-varying distributions shaped by the system itself. For instance, nonlinear systems may yield drastically different trajectories in response to small stochastic perturbations at each iteration. Given that not all noise sources—or their statistical properties—are fully known, we adopt a general and agnostic approach, assuming that perturbations may span the full frequency spectrum, from high to low frequencies. The Gaussianity assumption is widely employed in physiological studies due to the central limit theorem: The cumulative effect of multiple independent noise sources tends to result in Gaussian-distributed perturbations. This assumption is consistent with empirical physiological observations and is supported by the literature [56], which explores the complex biological origins of physiological noise and its inherent uncertainty. Demonstrating that the noise follows a $1/f$ spectrum is similarly nontrivial, and the characterization of $1/f$ noise in neurocardiovascular systems remains open to interpretation. While serial correlation is frequently associated with a $1/f$ spectral profile in these signals, it is not always clear whether this behavior arises from the noise component alone or from the full signal, that is, the combination of deterministic dynamics and stochastic fluctuations. This distinction is crucial: If the entire signal exhibits long-range dependency, then it may result from the system's inherent dynamics rather than from its stochastic input. For example, studies such as Ivanov *et al.* [60] and Grigolini *et al.* [61] link the observed $1/f$ or $1/f^\alpha$ spectral structure in physiological signals to complex deterministic dynamics. Additionally, $1/f$ spectra have been associated with healthy physiological states and shown to degrade in pathological conditions [62]. More recently, $1/f$ behavior has been attributed to nonlinear interactions between the parasympathetic and sympathetic branches of the autonomic nervous system [63].

In this context, the assumption of IID noise is not restrictive, as the observed $1/f$ scaling may emerge from the system's deterministic dynamics rather than from the statistical properties of the noise itself.

While classical results in dynamical systems suggest that approximately 10^d to 30^d samples are sufficient to characterize a d -dimensional deterministic attractor [64], these assumptions do not hold in our context. The neurophysiological signals under analysis are deterministic but perturbed by stochastic components, which fundamentally alter the structure of the attractor. Since noise can be modeled as an infinite-dimensional process, no finite theoretical embedding dimension can fully capture its influence, and thus a substantially larger number of samples is required. Moreover, the theoretical bounds mentioned above apply to monovariate systems and are typically used to characterize qualitative behavior rather than to quantify noise-driven effects. In our case, the objective is to trace how stochastic components influence the dynamics—both in terms of magnitude and correlation—by analyzing the shape of the MApEn profile. This requires estimating the conditional probabilities that embedded vectors remain close under the joint influence of the system's evolution and the noise perturbation. As shown in Fig. 5, increasing the number of samples leads to a more detailed

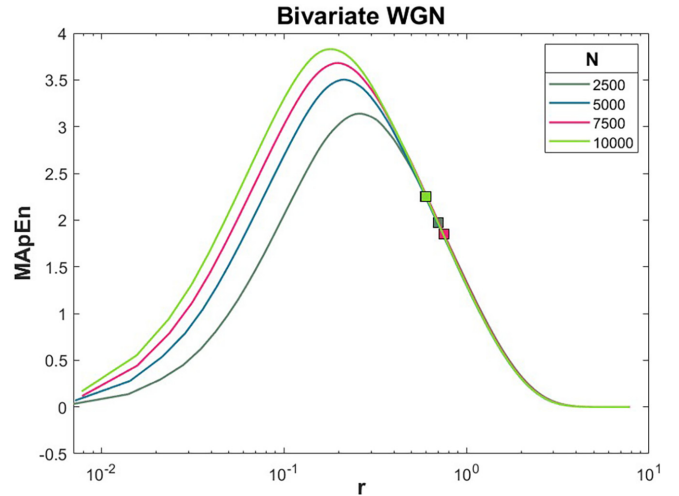


FIG. 5. MApEn as a function of the number of samples. Increasing the sample size improves the resolution of the entropy profile, especially under low embedding dimensions. The squares denote the points founded by the algorithm for stochastic coupling estimation.

and refined MApEn profile, especially when low embedding dimensions are used. Larger datasets help resolve the fine structure induced by stochastic fluctuations, whereas smaller datasets tend to overestimate coupling effects—reflected by a leftward shift of the profile markers.

Finally, higher embedding dimensions require an even greater number of samples to accurately capture noise-induced drifts in the dynamics. Based on extensive testing, we found that using 10 000 samples in combination with small embedding dimensions provides a good trade-off, yielding stable and reliable estimates of noise influence.

The embedding dimension used in our analysis was fixed at $D = [1, 1]$, selected to ensure both computational tractability and reliable estimation of the conditional probabilities required for constructing the MApEn profile. Larger embedding dimensions are desirable for capturing all stochastic correlations. While the proposed theorem applies to any embedding dimension, the numerical analysis was conducted using $D = [1, 1]$.

The purpose of the method is to depict and estimate the noise-induced drift that alters the dynamics of bivariate signals. This is achieved by analyzing the divergence of the multivariate approximate entropy as the tolerance parameter begins to reveal the influence of noise, with a particular focus on how these effects shape the MApEn profile. Adopting larger embedding dimensions is not strictly necessary, even though they can help reduce correlations. From a dynamical perspective, the key objective is to estimate how two nearby embedded vectors are separated at each integration step by the combined action of system dynamics and noise. There is currently no theoretical result that guarantees an optimal embedding dimension for noisy or noise-drifted signals, as the stochastic component resides in an infinite-dimensional space. A low embedding dimension is chosen because higher dimensions require substantially more data to accurately estimate the necessary conditional probabilities—especially under noisy conditions—something that is often infeasible

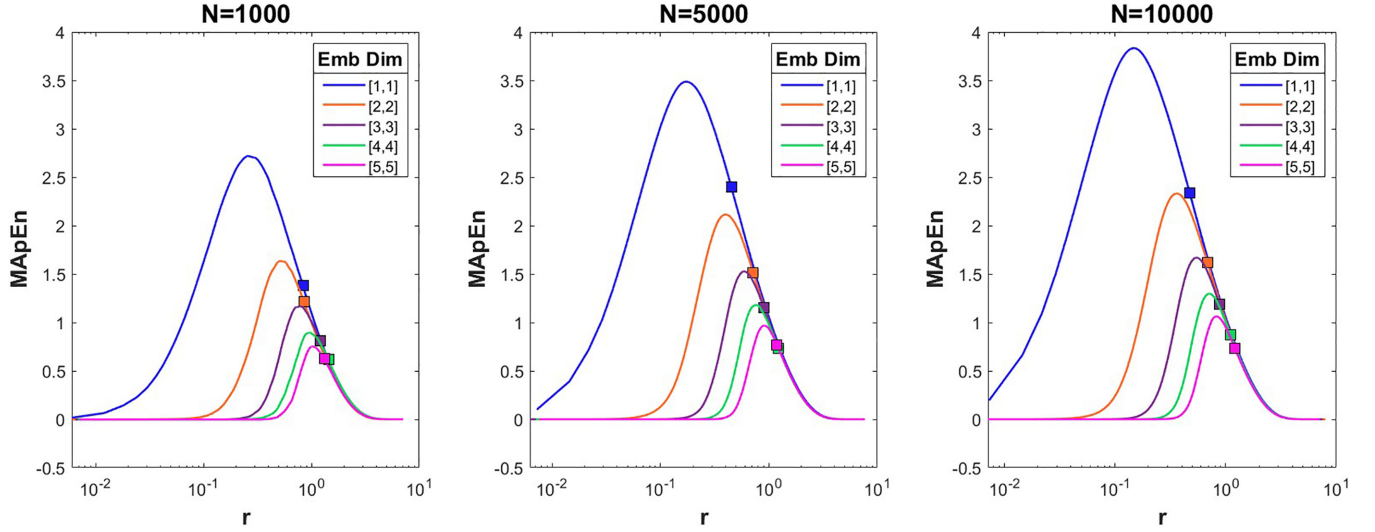


FIG. 6. MAPEn for different embedding vectors and number of samples.

in practical scenarios. Specifically, when using low tolerance values, high embedding dimensions, and limited sample sizes, the likelihood of identifying noise-drifted embedded vectors that remain close becomes very low. This issue is further compounded in multidimensional signals, where the challenge of identifying similar patterns is significantly heightened by differences in underlying dynamics and the presence of noise in each component.

Figure 6 graphically illustrates this by depicting the multivariate approximate entropy profile as a function of tolerance for a bivariate Gaussian signal, for varying sample counts ($N = 1000, 5000$, and 10000) and increasing embedding dimensions. Higher embedding dimensions flatten the MAPEn curve due to a shortage of matches in high-dimensional reconstructed spaces, caused by noise drift. Fewer samples exacerbate this phenomenon, resulting in lower MAPEn profiles.

Experimentally, even though the result holds for any embedding dimension vector and any infinite bivariate signal, increasing the embedding dimension is also associated with an overestimation of the quantity $s \rightarrow \text{MAPEn}(Y, D, r) + \ln(\frac{r^2}{\pi s})$ (squares in the plots). This overestimation, used to retrieve the injected amount of correlation, produces an underestimation of the actual coupling. Therefore, increasing the embedding dimension, while theoretically valid, leads to an underestimation of the coupling measure due to the inability to trace a sufficiently accurate approximate entropy profile.

Finally, the method does not capture any form of causality between the systems under investigation. Instead, it strictly reflects the coupling of the stochastic component. In this sense, it can be viewed as analogous to mutual information or the Pearson correlation coefficient, but applied exclusively to the stochastic part—disregarding the deterministic dynamics, for which alternative measures exist to assess coupling (e.g., mean phase coherence). Regarding the tolerance radius r , our estimation procedure does not rely on a single fixed value.

Instead, MAPEn profiles were computed across a range of tolerance values spanning the full amplitude of the time series, with resolution step Δr . The stochastic correlation ρ^e was then extracted by identifying the tolerance value that satisfies the main condition outlined in the manuscript, as detailed in the section *numerical estimation of ρ^e* . In summary, the estimated stochastic coupling is derived from the geometric properties of the MAPEn profile evaluated over a sequence of tolerance values, rather than from any fixed parameter setting. Limitations of the proposed method include its nonapplicability of the method to ultrashort time series, as the estimation is valid only for infinitely long series. Because the underlying theorem strictly holds in the asymptotic limit of infinite time series, finite sample sizes inevitably introduce an estimation bias, which can particularly affect the detection of very small stochastic correlations. While standard statistical estimators often allow for explicit finite-sample corrections, our algorithmic approach extracts the noise covariance directly from the nonlinear divergence profile of the MAPEn. Since the finite-sample bias of approximate entropy is highly nonlinear and intrinsically tied to the unknown deterministic flow of the system, a universal analytical bias adjustment cannot be easily implemented. Overcoming this limitation through algorithmic bias compensation or advanced surrogate-based corrections will be a primary focus of our future research. Additionally, it only accounts for bivariate dynamical noise—extending it to higher dimensions would entail a high computational cost and require very long recordings, assuming that the stochastic component remains stationary. Moreover, high-dimensional dynamics could propagate noise effects, which could distort the stochastic coupling, as shown in Sec. 3 of SM [51]. Although the proposed method is effective in detecting high levels of stochastic correlation, it may also overestimate coupling in systems characterized by negligible noise but complex deterministic dynamics, such as the Hénon map. This behavior can be attributed to the inherently chaotic nature of the Hénon map, which is known for its sensitivity to

initial conditions, fractal structure, and self-similarity (strange attractor) [65]. When noise levels are low, chaotic dynamics may emulate stochastic fluctuations, resulting in apparent—but structurally induced—correlations. Since each iteration of the map involves both state variables, these spurious effects can manifest as weak coupling signals. Nonetheless, the method finds a natural application in cardiovascular (HRV) recordings, which have been recently addressed as a regular system but highly contaminated by dynamical noise [66]. Although our proposed framework is strictly model-free, future research will explore quantitative comparisons with model-based techniques like parametric identification and residual covariance estimation.

Brain signals are inherently complex, and low-dimensional models are insufficient to fully capture or characterize their fluctuations. Our method does not rely on a fixed model, even when using a low embedding dimension for the MApEn profile. Rather, it is designed to characterize coupling by capturing aspects of the systems dynamics. The choice of a low embedding dimension is driven by sample constraints—not by theoretical limitations—since the underlying theorem holds universally, independent of dimensionality. Importantly, our method remains effective even for purely noisy and highly complex time series with infinite Kolmogorov-Sinai entropy. No finite embedding dimension is truly appropriate for such infinite-dimensional systems, whose behavior cannot be meaningfully represented by a simple dimension such as 2. In this context, the embedding dimension serves as a practical tool for noise detection, rather than a strict descriptor of the system's full dynamics.

While prior studies have established correlations between physical activity (e.g., HRV) and brain signal complexity or variability, our study goes further by investigating whether these correlations arise from deterministic dynamics or from stochastic components—considering noise as an intrinsic carrier of information.

Our framework is theoretically independent of any specific governing equations. It focuses on how the stochastic component influences the shape of the MApEn profile, which serves as a multivariate complexity quantifier. Therefore, the observed decrease in coupling with increasing sleep depth should be interpreted as a reflection of the phenomenon's stochastic structure—not of its deterministic dynamics—aligning with existing literature.

In the study of complex physiological systems, what is mathematically modeled as intrinsic dynamical noise does not represent measurement error. Rather, it encapsulates the cumulative effect of countless unobserved biological processes, hidden variables, and background regulatory mechanisms that continuously perturb the observable deterministic dynamics. Consequently, a high stochastic coupling serves as a macroscale indicator of background physiological integration, suggesting the presence of a shared, unmeasured systemic driver that deeply synchronizes the interacting systems. While this study demonstrated that significant modulation of stochastic coupling occurs during sleep transitions, linking cortical and cardiac oscillations, future research should focus on identifying the specific physiological components (e.g., particular biochemical, mechanical, or neural pathways) that mediate and generate such informative randomness. Thus

far, we can assert that stochastic coupling comprises all the dynamics involved in brain-heart interactions that are not directly represented by EEG and HRV series. Consequently, the observed variations in the stochastic components may be attributed to factors such as blood pressure [23] and associated mechanoreceptor [67], as well as other biochemical dynamics (e.g., serotonin and neuropeptide Y), among others.

VI. CONCLUSION

We introduced a framework for estimating stochastic coupling in hybrid multivariate systems, where unknown deterministic dynamics are driven by dynamical noise. By applying this technique to both synthetic and real physiological data, we demonstrated that stochastic coupling reveals new insights into complex physiological interactions, such as the brain-heart axis, particularly during sleep stage transitions. The results highlight the significant variations in stochastic coupling strength across different sleep phases, reflecting the evolving nature of neurocardiac interactions.

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The authors have no conflicts of interest to declare. All co-authors have seen and agree with the contents of the manuscript and there is no financial interest to report.

DATA AVAILABILITY

The experimental dataset used in this study is part of the publicly available Montreal Archive of Sleep Studies (MASS) dataset [42]. The MATLAB code for the stochastic coupling estimation is available online [68].

APPENDIX

The stochastic coupling estimator is validated on synthetic data whose underlying dynamics are known *a priori*. We designed five benchmark scenarios spanning different dynamical conditions—stochastic, regular, and chaotic and different coupling configurations, so as to verify that the method correctly discriminates interacting from non-interacting systems across regimes. The scenarios are defined as follows:

- (i) T_1 and T_2 are equal to 0, i.e., the series are realizations of pure correlated Gaussian stochastic processes;
- (ii) T_1 and T_2 are logistic, regular maps with parameter 3.5 and different initial conditions, perturbed by dynamical noise,

simulating regular but not interacting systems. The choice of the parameter 3.5 has been selected due its periodic behavior, mimicking periodicity of physiological signals, like the heart-beat time intervals;

(iii) T_1 and T_2 are the components of the chaotic Henón map with parameters $a = 1.4$ and $b = 0.3$ perturbed by output noise, representing interacting noisy systems. The choices of a and b determine the chaotic behavior;

(iv) T_1 and T_2 are the autoregressive models of order 3 [AR(3)] whose coefficients are, respectively, $a_1 = -0.4$, $a_2 = 0.2$, $a_3 = 0.1$ and $b_1 = 0.2$, $b_2 = -0.3$, $b_3 = 0.2$. These are typical systems where the innovation (or dy-

namical noise) is the main source of variability among the systems' realizations. The choice of the parameters guarantees stability of the system. There is no specific reason for the order of the AR model, since it has been proved [69] that the monovariate noise estimation needed for the stochastic coupling approach is efficient also for higher AR model order; and

(v) T_1 and T_2 correspond to the logistic map with parameter $\lambda = 3.5$ and an autoregressive model of order 3 [AR(3)] with coefficients $c_1 = 0.2$, $c_2 = -0.3$, $c_3 = 0.2$, respectively. This setup represents a scenario where the two systems exhibit distinct and noninteracting dynamics.

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