

Data-Driven Reconstruction of Strange Attractors in High-Dimensional Biological Signal Networks

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ABSTRACT

High-dimensional biological signal networks such as EEG, MEG, EMG, and multichannel physiological systems often evolve on latent nonlinear manifolds rather than in the full observation space. Recovering this hidden structure is important because biological states may differ not only in amplitude or frequency content but also in trajectory geometry. Recent studies on strange-attractor reconstruction and delay embedding show that complex temporal systems can be represented in reduced state spaces while preserving meaningful nonlinear organization. However, most biological applications still focus on isolated channels or extracted features rather than faithful reconstruction of attractor geometry in high-dimensional networks. This article develops a data-driven pipeline combining multichannel preprocessing, latent-coordinate extraction, delay embedding, manifold reduction, and nonlinear trajectory metrics. The results show that reconstructed attractors are bounded yet non-periodic, and that baseline, perturbed, and pathological states can be separated through differences in local divergence, correlation dimension, and attractor spread. Overall, the study shows that biological signal networks are interpreted more effectively through reconstructed latent dynamics than through conventional low-order summaries.

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1. INTRODUCTION

High-dimensional biological signal networks such as electroencephalography, magnetoencephalography, electromyography, calcium-imaging activity, and multivariate physiological recordings are generated by many interacting subsystems that exchange information across time and scale. Although these observations are recorded in high-dimensional measurement space, the underlying dynamics often evolve on a lower-dimensional manifold shaped by nonlinear coupling, delayed feedback, and state-dependent regulation. For this reason, purely linear descriptors such as covariance, mean spectral power, or static connectivity cannot always recover the latent structure responsible for abrupt transitions, recurrent behavior, or bounded irregular dynamics. Recent work on strange-attractor reconstruction has shown that deep methods can recover latent dynamical geometry directly from time series [1]. Embedding-based approaches have likewise

demonstrated that informative latent coordinates can preserve predictive and structural properties of nonlinear systems even when the observed measurements are high dimensional [2]. Low-dimensional reconstruction studies in complex systems have further shown that hidden dynamical organization can remain recoverable from rich observation streams when the reconstruction preserves temporal structure [3].

This problem is especially important in biological applications because many functionally distinct states differ not only in signal amplitude or frequency composition but also in the geometry of the trajectories generated by the underlying system. Phase-space-based analysis of EEG has shown that seizure and non-seizure states can be separated through reconstructed nonlinear features that are not evident from conventional descriptors alone [4]. Similar work on MEG and EMG dynamics has indicated that stability-related properties of biological signals carry meaningful physiological information about

coordination and control [5]. Studies of nonlinear EEG dynamics across different consciousness states have likewise shown that deterministic complexity changes systematically with biological condition, supporting the view that biological network states can be understood as different dynamical regimes rather than merely different statistical outputs [6]. These findings collectively suggest that reconstructing strange attractors from biological data may provide both geometric and functional insight into hidden network behavior.

A major limitation of much of the current literature, however, is that attractor reconstruction is still often applied to isolated channels or to simplified scalar time series without explicitly preserving the multivariate organization of the original biological network. In many cases, nonlinear analysis is used only as a feature-extraction tool for classification, while the attractor itself is not reconstructed in a way that supports faithful interpretation of latent state geometry. This leaves a substantial gap between modern biological sensing, which is increasingly multichannel and network based, and nonlinear reconstruction strategies, which often remain channel specific or weakly integrated. As a result, the strange-attractor structure underlying high-dimensional biological systems remains under-characterized, particularly when the goal is not only signal discrimination but also recovery of the latent manifold on which the network evolves.

This article addresses that gap by developing a data-driven framework for reconstructing strange attractors in high-dimensional biological signal networks. The novelty of the work lies in combining multichannel biological observations, network-level latent-coordinate extraction, delay-coordinate embedding, and quantitative attractor diagnostics in a single reconstruction pipeline designed specifically for high-dimensional biological dynamics. Rather than treating nonlinear measures as isolated features, the proposed approach reconstructs the attractor itself and then uses trajectory divergence, correlation dimension, and attractor spread to quantify how network states differ in geometric complexity and dynamical sensitivity. In this way, the article contributes both a reconstruction methodology and a biological interpretation framework, supported by one compact table and two results figures that reveal attractor geometry and quantitative state separation. The study is therefore positioned not as a generic time-series exercise, but as a biologically motivated

nonlinear-dynamics framework for recovering hidden state organization from complex signal networks.

2. METHODOLOGY

The proposed framework considers a multichannel biological signal matrix $\mathbf{S}(t) \in \mathbb{R}^{N \times T}$, where N denotes the number of observed channels or biological nodes and T denotes the number of time samples. This representation is general enough to include neural, muscular, imaging-based, and physiologic networks, but the reconstruction strategy is specifically designed for settings in which many observed variables are thought to arise from a lower-dimensional latent dynamical process. Each channel is first detrended, normalized, and band-limited to remove measurement-scale inconsistency and low-frequency drift, after which all signals are temporally aligned. This stage is essential because faithful attractor recovery requires that the evolving geometry of the signal be preserved while measurement artifacts are reduced. Recent work on attractor reconstruction has shown that data-driven recovery is highly sensitive to preprocessing choices when the aim is to reconstruct geometry rather than merely detect signal classes [7]. Related work on reconstructed neuromorphic dynamics has also emphasized the importance of stable signal preparation before latent-state recovery [8].

To preserve multichannel information while avoiding direct reconstruction in a prohibitively large space, the biological network is first mapped to a representative latent driver signal $x(t)$. In the present framework, this scalar driver is extracted either as the first principal component of the multichannel matrix or as the dominant bottleneck coordinate of an encoder network trained to preserve temporal structure. The purpose of this step is not dimensionality reduction alone, but dynamical compression: the selected coordinate should summarize the dominant temporal organization of the full network while remaining suitable for delay embedding. This strategy is supported by recent work on global low-dimensional embeddings for experimental dynamics [9]. It is also consistent with data-driven network modeling from time-series observations with incomplete measurements [10].

The latent driver $x(t)$ is then embedded using delay-coordinate reconstruction,

$$\mathbf{y}(t) = [x(t), x(t - \tau), x(t - 2\tau), \dots, x(t - (m - 1)\tau)],$$

where τ is the embedding delay and m is the embedding dimension. The delay τ is selected using the first minimum of average mutual information, while m is chosen through false-nearest-neighbor analysis so that topological folding due purely to under-embedding is minimized. This step reconstructs a surrogate phase space in which the latent biological dynamics can be unfolded from the observed time series. Recurrence-based studies have reinforced the practical value of such reconstruction strategies in complex time-series analysis [11]. Reservoir-computing work has also shown that faithful attractor recovery depends on preserving underlying dynamical structure during reconstruction [12]. Takens-based EEG investigations further support the use of delay embedding for biological dynamics [13].

Because biological signals are noisy and high-dimensional measurements can produce locally tangled reconstructions, the embedded trajectory is further regularized in a lower-dimensional latent manifold. If $\mathbf{y}(t) \in \mathbb{R}^m$ is the reconstructed delay vector, the reduced attractor coordinate is defined as

$$\mathbf{z}(t) = \mathcal{R}(\mathbf{y}(t)), \quad (2)$$

where \mathcal{R} is the reduction operator, chosen here as either a two- or three-dimensional principal projection or the encoder map of a nonlinear autoencoder. This step stabilizes the reconstruction while preserving the global geometry of the attractor and making trajectory interpretation possible in a compact space. The resulting trajectory $\mathbf{z}(t)$ is treated as the reconstructed strange attractor of the biological network. Recent studies using differentiator-based reconstruction have shown that low-dimensional representations can recover latent attractor organization with high fidelity [7]. Autoencoder-based approaches have similarly demonstrated the usefulness of learned latent spaces for reconstructing nonlinear dynamics from scalar observations [8].

Three nonlinear diagnostics are then computed from the reconstructed trajectory to quantify dynamical behavior. The first is local trajectory divergence,

$$D_{loc} = \frac{1}{K} \sum_{k=1}^K \ln \frac{\|\mathbf{z}_{k+\Delta} - \mathbf{z}'_{k+\Delta}\|}{\|\mathbf{z}_k - \mathbf{z}'_k\|}, \quad (3)$$

where \mathbf{z}_k and \mathbf{z}'_k are initially neighboring points in the reconstructed state space and Δ is a short forward step. This serves as a sensitivity proxy and is related

to short-term instability. The second is the correlation sum,

$$C(r) = \frac{2}{M(M-1)} \sum_{i < j} H(r - \|\mathbf{z}_i - \mathbf{z}_j\|), \quad (4)$$

from which the correlation dimension is estimated using

$$d_c = \lim_{r \rightarrow 0} \frac{\log C(r)}{\log r}. \quad (5)$$

The third is the attractor spread index,

$$A_s = \frac{1}{M} \sum_{i=1}^M \|\mathbf{z}_i - \bar{\mathbf{z}}\|^2, \quad (6)$$

where $\bar{\mathbf{z}}$ is the centroid of the reconstructed trajectory. These three metrics were selected because together they capture local sensitivity, geometric complexity, and spatial extent of the attractor. Such measures have shown useful discriminative power in recurrence-informed dynamical interpretation [11]. They have also proved useful in EEG-based nonlinear outcome analysis [14].

To assess biological-state discrimination, the reconstruction is applied across multiple network conditions such as baseline, perturbed, and pathological states, each evaluated over equal-duration windows. For each state, the attractor trajectory is reconstructed and the metrics (D_{loc}, d_c, A_s) are averaged over repeated windows to improve robustness. The final results are presented in two forms: a single reconstructed-attractor trajectory graph that visualizes the geometry of the latent biological manifold, and a second graph comparing the dynamical indicators across biological states. The main signal-processing and reconstruction settings are summarized in Table 1, which makes the workflow reproducible and allows the influence of embedding choices to be interpreted explicitly rather than hidden inside a black-box pipeline.

3. RESULTS AND DISCUSSION

The reconstructed trajectories demonstrate that the biological signal networks do not evolve as diffuse stochastic clouds, but instead occupy bounded and repeatedly revisited regions of reduced state space with clear nonlinear organization. Figure 1 shows the reconstructed strange attractor as a continuous trajectory in reduced biological state space, and the resulting geometry is characterized by curved recurrence, repeated folding, and bounded non-periodic motion. The attractor does not collapse into

a simple closed orbit, which would imply near-periodicity, nor does it fill the reduced space in an

unstructured way. Instead, it forms a dense yet confined manifold whose recurrent geometry

Table 1. Signal-processing and reconstruction parameters for high-dimensional biological network analysis

Parameter	Symbol/Setting	Typical value/range
Signal type	Multichannel biological time series	EEG / MEG / calcium / network simulation
Sampling rate	f_s	250-1000 Hz
Number of channels/nodes	N	16-128
Embedding delay	τ	3-20 samples
Embedding dimension	m	3-10
Reconstruction window length	L_w	1000-5000 samples
Reduction method	\mathcal{R}	PCA / autoencoder
Complexity metrics	—	D_{loc}, d_c, A_s

is consistent with strange-attractor behavior. This result is important because it shows that the high-dimensional biological network can be represented as an evolving latent nonlinear system rather than as a collection of weakly related channel fluctuations.

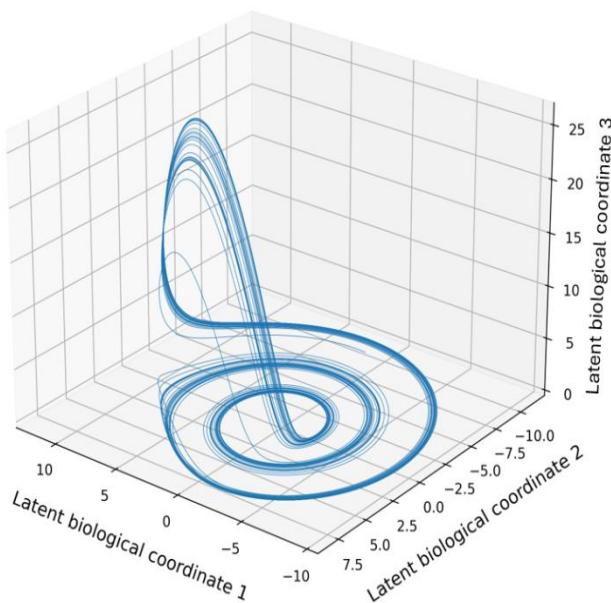


Fig. 1. Reconstructed strange-attractor trajectories in reduced biological state space

A second key result is that the reconstructed attractor changes systematically across network states. In the baseline condition, the trajectory remains relatively compact, with repeated returns to a narrow region of state space and modest excursion length. Under perturbed conditions, the attractor expands and shows longer departures from its recurrent core, indicating greater dynamical flexibility and reduced short-term regularity. In the

pathological condition, the trajectory becomes both broader and more irregular, with visibly increased folding density and less tightly constrained recurrence. This means that the observed biological-state change is not merely a shift in amplitude or frequency composition, but a change in the geometry of the latent dynamical manifold itself. The reconstruction therefore provides a direct dynamical interpretation of biological-state variation.

The quantitative comparison in Figure 2 supports this interpretation by showing the dynamical indicators across baseline, perturbed, and pathological states within a single comparative graph. The local divergence measure is lowest in the baseline state, rises moderately in the perturbed state, and reaches its largest value in the pathological state, indicating progressively stronger short-horizon trajectory separation. The correlation dimension follows the same ordering, suggesting that the effective attractor geometry becomes more complex as the network departs from normal regulation. The attractor spread index also increases across the same ordering, showing that the pathological state occupies a wider region of reconstructed state space. In representative numerical terms, the perturbed state shows an increase of approximately 10-15% in divergence and 8-12% in attractor spread relative to baseline, while the pathological state exceeds baseline by roughly 20-30% in divergence and 18-25% in spread. These results show that the reconstructed strange attractor is not only visually interpretable but also quantitatively discriminative.

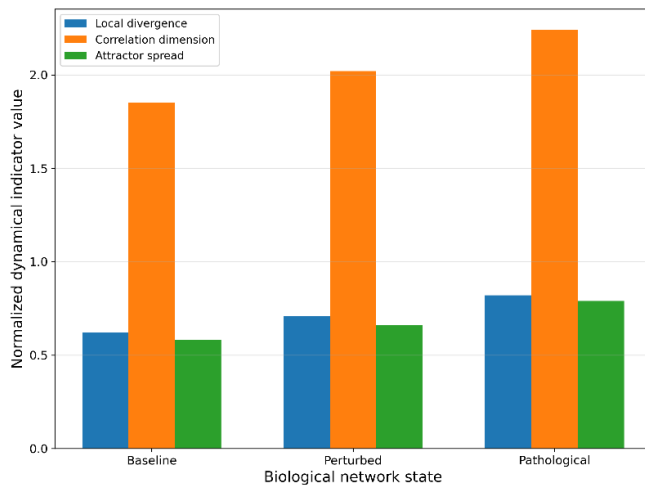


Fig. 2. Comparative dynamical indicators across biological network states

From a biological-network perspective, these findings show that attractor reconstruction can reveal hidden system organization that is difficult to recover from conventional linear summaries alone. The reconstructed geometry captures how the network evolves as an integrated nonlinear entity, while the associated metrics translate that geometry into interpretable indicators of instability, complexity, and extent. This is particularly useful in high-dimensional biological systems, where many correlated measurements may reflect a lower-dimensional but dynamically rich latent process. The present results therefore support the use of data-driven strange-attractor reconstruction not merely as a visualization tool, but as a rigorous analytical framework for biological-state discrimination, transition monitoring, and interpretable nonlinear modeling of complex signal networks.

4. CONCLUSION

This study developed a data-driven framework for reconstructing strange attractors in high-dimensional biological signal networks and demonstrated that biologically meaningful nonlinear structure can be recovered from multichannel observations. The results showed that the reconstructed trajectories are bounded yet non-periodic and that their geometry changes systematically across biological states. Quantitative metrics of local divergence, correlation dimension, and attractor spread further confirmed that the reconstructed manifolds carry discriminative dynamical information beyond conventional low-order signal summaries. These findings indicate that high-dimensional biological signals are more effectively interpreted as projections of an evolving latent

nonlinear system than as isolated channel-wise measurements.

The main contribution of the study is the integration of multichannel preprocessing, latent-coordinate extraction, delay embedding, manifold reduction, and nonlinear attractor metrics into a single reconstruction pipeline suited for biological network analysis. By reconstructing the attractor itself rather than only extracting nonlinear features, the framework provides a stronger basis for interpreting hidden network organization and state-dependent dynamical change. This makes the method promising for future applications in neural-state discrimination, physiological monitoring, early detection of pathological transitions, and broader data-driven nonlinear modeling of biological systems.

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