Innov Biosyst Bioeng, 2026, vol. 10, no. 2,
doi: 10.20535/ibb.2026.10.2.349634

Review

PHYSICAL PROCESSES IN BIOLOGICAL NEURONAL NETWORKS DURING EPILEPTIC SEIZURES: AN OVERVIEW OF MODELS

I. Pidvalnyi[1*], O. Sudakov[2]

[1] Taras Shevchenko National University of Kyiv, Kyiv, Ukraine
[2] Technical Center, National Academy of Sciences of Ukraine, Kyiv, Ukraine

*Corresponding author: podvalniy111@gmail.com

Received 12 January 2026; Accepted 13 May 2026

Abstract: This review summarizes approaches to epileptic seizure detection and forecasting from electroencephalographic and intracranial electroencephalographic signals, with emphasis on physically grounded modelling of neurons and neuronal networks. The topic is relevant because modern machine-learning and deep-learning methods often achieve high classification performance, but their practical use is limited by cross-patient variability, scarcity of labelled data, dependence on preprocessing, false-alarm control and insufficient interpretability of informative features. The paper systematizes the main model classes used in computational epileptology: low-dimensional phenomenological models, biophysical models of single neurons and membrane processes, network and multiscale frameworks, machine-learning approaches, and high-order numerical schemes for large-scale simulations. It is shown that biophysical and multiscale models provide deeper insight into ionic, synaptic and network mechanisms of epileptiform activity, but require many parameters and substantial computational resources. In contrast, phenomenological models, especially Epileptor and Epileptor-2, offer a rational compromise between simulation speed, parameter controllability and the ability to reproduce transitions between normal, ictal and post-ictal regimes. We argue that such models should not be considered a replacement for real EEG/iEEG recordings, but rather a controlled source of synthetic signals for feature calibration, classifier robustness testing and analysis of the physical origin of informative signal characteristics. Model-generated signals are therefore positioned as an intermediate layer between experimental recordings and machine-learning algorithms. This approach may improve the reproducibility of studies, increase the interpretability of diagnostic features and provide a basis for further development of compact systems for epileptic seizure detection and forecasting.

Keywords: electroencephalography; ictal transition; synthetic calibration data; phenomenological dynamics; Epileptor-2; bifurcation mechanisms; multiscale connectome; feature extraction; classifier calibration; transfer validation.

Introduction

Epilepsy is a widespread chronic disorder that affects more than 50 million people worldwide; in roughly one-third of patients the disease remains pharmacoresistant, leading to substantial cognitive, traumatic, and socio-economic consequences [1–5]. This heterogeneity motivates multi-scale computational modelling as a route from mechanisms to observable EEG/iEEG signatures [6]. Reliable identification of EEG precursors minutes before onset is therefore a key objective of seizure monitoring and can enable preventive intervention [7, 8]. Fig. 1 summarises a typical edge-to-cloud workflow for ambulatory intracranial EEG (iEEG) monitoring.

Today, the most common seizure detection and prediction approaches are data-driven, based on machine learning and artificial intelligence using datasets annotated by experts. Despite the impressive performance of the mentioned approaches, such as deep Convolutional Neural Network/Long Short-Term Memory (CNN/LSTM) models providing an Area Under the Receiver Operating Characteristic Curve (AUROC) of approximately 0.90 [9], their large-scale deployment is hindered by three systemic barriers. First, high false-positive rates threaten the safety of implanted warning devices; strict statistical calibration can reduce the False Positive Rate (FPR) but only at the cost of sensitivity [10, 11]. Second, models trained on limited cohorts lose accuracy sharply in cross-patient settings, motivating biologically inspired architectures that are more robust to inter-individual variability [12, 13]. Recent EEG foundation and self-supervised representation-learning approaches, including transformer-based pretraining and transfer learning, can only partly mitigate label scarcity and cross-subject shift by leveraging large-scale unlabeled recordings and fine-tuning to target cohorts [14–17]. Third, the lack of unified segmentation and annotation protocols for public EEG data sets produces methodological heterogeneity and inflated performance estimates [18, 19].

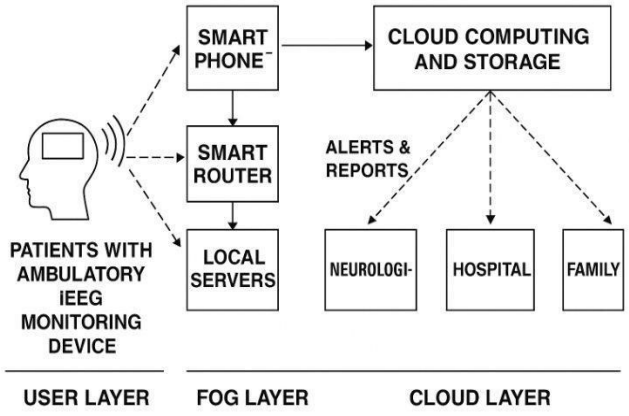

Figure 1: Conceptual three-layer architecture for ambulatory iEEG monitoring

A promising way to overcome these limitations is the controlled generation of synthetic data sets using models of neuronal networks and individual neurons. More generally, mechanistic neural modelling has been advocated as a translational bridge between multiscale observations and clinically actionable variables in neurological disorders [20]. Since the pioneering biophysical hippocampal model of Traub et al. [21], attention has shifted to low-dimensional phenomenological systems; among them, the Epileptor model realistically reproduces the "normal–seizure" transition via bifurcation mechanisms [22]. Foundational compendia similarly highlight how computer modelling can link cellular and network mechanisms to macroscopic seizure phenomenology and EEG signatures [23]. Its integration into multiscale frameworks such as the Virtual Epileptic Patient has shown the effectiveness of *in silico* simulations for modelling discharge propagation and evaluating surgical intervention [24]. Recent neurosurgical syntheses consolidate this line of work, summarising how patient-specific whole-brain dynamical models support intervention planning and virtual resection analysis in drug-resistant epilepsy [25, 26]. Nevertheless, most studies focus on qualitative waveform reproduction; only a few compare statistical descriptors quantitatively or test classifiers trained on synthetic data against *in vivo* recordings. Thus, the unresolved problem is to enhance EEG informativeness through a systematic combination of modelling and machine learning followed by validation on real signals. Unlike surveys that primarily catalogue data-driven architectures, we assess mechanistic models as controllable generators of calibration datasets and discuss how their utility should be quantified via standardized detection/forecasting metrics under transfer and leave-one-subject-out validation.

The classical reviews [27–29] describe the model's taxonomy tracing progress from early phenomenological schemes to multiscale platforms, documenting the ability of different models to reproduce critical bifurcations of the brain network. More recent synthesis papers extend this taxonomy toward clinically constrained multiscale interpretation and digital-twin workflows, including neuro-inspired EEG interpretation models [30], the phenomenological-to-biophysical continuum [31], and patient-specific seizure-spread models on cortical surfaces [32]. Yet, despite their detailed taxonomies, the authors do not actually compare the predictive capabilities of these models. Metrics such as AUROC or Area Under the Precision-Recall Curve (AUPRC) are missing. Instead of a single comparison score, it would be beneficial to present a standardized panel of clinical metrics. This panel could include AUROC for balanced data, AUPRC for imbalanced data, sensitivity/specificity, false alarms per hour for safety, detection latency for timeliness, and calibration score (Brier/ECE). These metrics should be evaluated under leave-one-subject-out validation with bootstrap confidence intervals (CI) to ensure reproducibility. Similar calibration and threshold-selection issues arise in diary-based seizure-risk forecasting, underscoring the practical importance of probability calibration beyond EEG-only pipelines [33].

Clinically oriented reviews [34–36] indicate that model-guided strategies can optimize the placement of implanted deep-brain neurostimulators and test antiseizure drugs *in silico*. Beyond stimulation and surgery, computational epilepsy models have also been proposed as *in silico* testbeds for therapy development and drug discovery pipelines [37]. However, they provide limited information on which temporal or spectral markers, such as band-limited power (δ-γ), spectral entropy, line length, autocorrelation time, rates of high-frequency oscillations, and phase-amplitude coupling, are associated with successful forecasting. Wendling explicitly calls for a multi-level "animal–*in vitro*–clinic" validation to guarantee model reproducibility [34–36]. Recent Bayesian frameworks, such as the "Virtual Epileptic Patient" and "Virtual Brain Twin," operationalize this multi-level paradigm by inferring patient-specific propagation patterns and calibrating mechanistic models using multimodal data. A representative probabilistic formulation is the Bayesian Virtual Epileptic Patient, designed to infer hidden disease variables and propagation parameters from patient data under uncertainty [38, 39]. This approach establishes a direct connection between seizure-spread modeling and forecasting-oriented evaluation.

A significant body of literature focuses on the processing of EEG and Magnetoencephalography (MEG) signals. Classical surveys [40, 41] have highlighted the considerable variability in parameters introduced during filtering and artifact removal stages. The attenuation of ocular artifacts like blinks and saccades, myogenic artifacts, cardiac/cardioballistic contamination, power-line interference (50/60 Hz and

harmonics), electrode pops/impedance shifts, motion artifacts, and slow sweat-related drifts can have a significant impact on the signal spectrum. For intracranial EEG (iEEG), stimulation artifacts are also taken into account. These artifacts often overlap with the spectral characteristics of target biomarkers: δ-θ bands for ocular/motion artifacts, β-γ bands for EMG, narrow 50/60 Hz lines for mains noise, and sub-hertz components for slow drifts. Inadequate correction of these artifacts can lead to systematic biases in power spectral density (PSD), spectral entropy, high-frequency oscillation (HFO) estimates, and other informative features, ultimately compromising downstream classification and accuracy metrics. Wearable EEG hardware and edge device pipelines may introduce additional constraints, such as motion-sensitive electrodes, limited dynamic range, and on-device preprocessing. These constraints should be taken into consideration when establishing "real-world" benchmarks for forecasting and detection [42–44].

To make the spectral context explicit, we treat EEG/iEEG as a superposition of canonical frequency bands (δ: 0.5-4 Hz, θ: 4-8 Hz, α: 8-13 Hz, β: 13-30 Hz, γ: 30-80 Hz) and high-frequency oscillations (HFOs), typically reported in the ripple (≈80-250 Hz) and fast-ripple (≈250-500 Hz) ranges. Across these bands, forecasting and detection pipelines commonly quantify shifts in band power and band ratios, spectral entropy and related complexity measures, narrow-band peaks, phase-amplitude coupling, and HFO rates, features that are directly sensitive to preprocessing choices and therefore must be defined consistently. In the present review, these spectral descriptors are carried forward as part of the standardised feature panel discussed throughout the paper and linked to operational metrics (AUROC/AUPRC, false alarms per hour, latency and calibration) so that modelling choices can be compared in a quantitatively reproducible way.

Recent studies [45–48] highlight the significance of nonlinear dynamic metrics that capture the geometry and stability of system trajectories. These metrics encompass the largest Lyapunov exponent (sensitivity to initial conditions), correlation dimension (effective "fractal" complexity of the attractor), entropy measures such as Sample/Permutation Entropy (degree of irregularity), and recurrence-quantification indices (e.g., determinism, laminarity). In practice, these descriptors are computed on sliding windows after delay-coordinate embedding of the EEG time series, with robustness evaluated through surrogate-data testing. In the field of epileptology, these metrics hold direct prognostic value: pre-ictal states typically exhibit increasing instability and shifts in effective dimensionality, while ictal activity tends to become more regular and structured, leading to distinct changes in entropies and Recurrence quantification analysis (RQA) metrics. These studies also compare network dynamics metrics with deep CNN/LSTM schemes, with the latter achieving an AUROC of approximately 0.9 but retaining a "black-box" nature. Interpretable, lightweight machine learning baselines can achieve competitive performance on preclinical EEG data, thereby promoting hybrid "features + mechanistic dynamics" pipelines [49]. Conditions for the existence and collapse of chimera and solitary states in large nonlinear oscillatory networks [50–53] offer additional insights for feature extraction and explanation. These stationary states with coherence and incoherence are deemed crucial for brain network functions.

Although there are many individual works devoted to different aspects of the application of neurons and neuronal networks modeling to detection and prediction of epileptic seizures using electroencephalography signals, we found no literature reviews with comparisons of such approaches. In this paper, we provide a review of existing approaches that combine modeling of biological neurons and neuronal networks with machine learning, followed by validation on real signals, and draw conclusions on their possible further improvements. Specifically, we treat mechanistic models as controllable generators of calibration EEG/iEEG datasets and evaluate their downstream utility through clinically meaningful metrics (AUROC/AUPRC, false alarms per hour, detection latency, and calibration) under leave-one-subject-out and transfer settings. This framing highlights how low-dimensional phenomenological generators such as Epileptor-2 can complement data-driven pipelines by enabling reproducible stress-testing and feature-level interpretability. We also suggest a new approach for extension of datasets used for machine learning with the results of mathematical modeling of neuronal networks. The analyzed sources outline five essential requirements that our working scheme adheres to: (i) quantitative comparison of phenomenological systems using AUROC/AUPRC; (ii) a standardized set of spectral, statistical, and nonlinear features; (iii) integration of modeling with machine learning to improve transferability and detectability; (iv) obligatory testing on real EEG; and (v) scalable, reproducible simulation/digital-twin pipelines that can be executed on realistic network sizes and hardware constraints [54, 55].

From a dynamical-systems perspective, seizures can be framed as bifurcation-driven regime shifts: gradual changes in an excitability-like control parameter push the system across saddle-node or Hopf thresholds, converting a stable baseline into oscillatory ictal activity and, in many models, back via slow recovery variables [22, 56, 57]. Near these critical points, canonical early-warning signatures – critical slowing down, rising variance and lag-1 autocorrelation – provide physically interpretable cues for feature

design and forecasting horizons. Importantly for algorithm evaluation, the bifurcation structure provides a controllable "knob" for synthesis: by sweeping parameters around the critical manifold, one can generate calibration traces spanning subcritical, pre-ictal, ictal and post-ictal regimes with known ground-truth timing, enabling standardised testing of AUROC/AUPRC, false-alarms-per-hour, latency and calibration under transfer and leave-one-subject-out settings.

The remainder of this article is organised as follows. In Sections 2-6 we provide details on model classes from phenomenological schemes for the "normal - seizure" switch to advanced numerical techniques that can reinforce any preceding paradigm. The concluding analytical Section 7 compares the strengths and weaknesses of each group and justifies the choice of the model that best serves the stated research goal.

Benchmarking Low-Dimensional Phenomenologcal Models for Synthetic EEG

Phenomenological models of epilepsy form a methodological bridge between the brain's biophysical complexity and the need for rapid numerical analysis of seizure transitions. By intentionally reducing neuronal micro-dynamics to low-dimensional systems of differential equations, these models focus on the bifurcation scenarios that govern abrupt changes in cortical electrical activity. By "micro-dynamics" we mean the evolution of the membrane potential and the gating variables of ion channels at the single-neuron level [54]. At the same time, instead of a full, high-dimensional cellular description (ion channels, conductances, synaptic currents, slow ionic accumulations, etc.), we introduce a few effective variables that aggregate fast and slow processes and faithfully reproduce the key "normal–seizure" transition via bifurcations. In other words, the complex membrane physics is reduced to two-three ordinary differential equations with interpretable parameters (excitability, recovery, slow adaptation), enabling analysis of thresholds, stability, and the onset of oscillations. Such a strategy enables the generation of synthetic EEG signals with controlled parameters, an essential feature when evaluating classification and forecasting algorithms under limited clinical data.

The best-known representative of this class is the Epileptor [56] – originally introduced as Epileptor-2014 in [57] and still the canonical "engine" for clinical applications – which separates the dynamics into fast and slow subsystems. The fast subsystem is described by

$$\dot{x}_1 = y_1 - f(x_1) - z_1,\ \dot{y}_1 = g(x_1) - y_1 \qquad (1)$$

Where $f(x_1) = x_1^3 - ax_1$ provides a cubic nonlinearity and $g(x_1)$ implements local feedback; the slow subsystem

$$\dot{z}_1 = \mu(4(x_1 - x_0) - z_1),\ 0 < \mu \ll 1, \qquad (2)$$

governs post-ictal recovery. Despite its three-dimensional phase space, the model reproduces saddle-node and Hopf bifurcations and runs in real time. When fitted to 42 intracranial recordings, the mean Pearson correlation between simulated spike-wave envelopes and empirical EEG reached r = 0.88; the original cohort of 20 patients in [57] yielded r = 0.85, validating the model for initial clinical use. Here r denotes the Pearson coefficient,

$$r = \frac{\sum_{t=1}^{T}(x_t - \overline{x})(y_t - \overline{y})}{\sqrt{\sum_{t=1}^{T}(x_t - \overline{x})^2}\sqrt{\sum_{t=1}^{T}(y_t - \overline{y})^2}} \qquad (3)$$

computed on time-aligned envelope pairs $\{x_t\}$ (simulated) and $\{y_t\}$ (empirical), with $\overline{x}$ and $\overline{y}$ their sample means. Values near 1 indicate strong linear concordance of waveform envelopes; thus $r \approx$ 0.85-0.88 reflects high agreement between model and data. Abstracting away from cellular mechanisms (ion currents, inhibitory modulation), however, limits accuracy in scenarios where sub-cellular processes are decisive.

To partially address this limitation, in [31] added an "ionic" variable,

$$\dot{w}_1 = \alpha(x_1 - w), \qquad (4)$$

that represents slow extracellular potassium accumulation. The root-mean-square error between model output and intracranial EEG decreased from 0.42 mV to 0.19 mV, and the mean correlation rose to $r = 0.79$. Although biologically more plausible, each additional variable increases the demand for parameter data and computational cost, an important trade-off in large-scale classifier testing.

A minimalist alternative is Montgomery's model [58], based on normal-form bifurcations, that is, on canonical low-dimensional equations obtained by reducing the full dynamics near a critical point (via center-manifold reduction and rescaling) so that only the instability-driving terms remain. For a saddle-node scenario, where a stable and an unstable equilibrium collide and annihilate as a control parameter crosses a threshold, yielding abrupt onset and critical slowing down, it reduces to

$$\dot{x} = r + x^2, \qquad (5)$$

where the control parameter r measures the distance to the bifurcation and drives the system through the critical point. Across 1 000 Monte-Carlo runs the median absolute error in predicting the bifurcation time was 0.46 s. The model's theoretical transparency and low dimensionality facilitate rapid hypothesis screening, although extra terms may be required to reproduce specific EEG morphologies.

Introduction of a phase variable $\theta(t)$ with intrinsic frequency ω, coupled to an amplitude variable $A(t)$, enables explicit capture of pre-ictal rhythms [39]. A minimal phase-amplitude oscillator can be written as

$$\dot{\theta} = \omega + F(\theta, A; \theta), \quad (6)$$

$$\dot{A} = G(\theta, A; \theta), \quad (7)$$

$$x(t) = A(t)cos\theta(t), \quad (8)$$

where F and G encode the nonlinear amplitude-phase interactions and Θ denotes the parameter set. In this model, phase-space reconstruction separated pre-ictal and inter-ictal states with an AUC of 0.91 at a warning horizon ≥30 s [59]. The main shortcoming is high sensitivity to parameter values, which complicates calibration without prior spectral analysis. A practical remedy is to align modelled and empirical phases by maximising mutual information $G\ I(\phi_{model}, \phi_{EEG})$ (or related circular concordance measures) before fitting the remaining parameters, thereby regularising phase locking and improving robustness.

A further step toward clinical practice is demonstrated by the Virtual Epileptic Patient (VEP) module [60, 61], wherein Epileptor-2014 instances are embedded at nodes of an individual structural connectome, enabling *in silico* prediction of seizure spread and evaluation of resection efficacy; in a dataset of 15 patients this approach achieved 71% spatial concordance with SEEG and corresponded to postoperative Engel I outcomes in 12/15 cases.

In summary, phenomenological models offer a spectrum of compromises among simulation speed, biophysical plausibility and the ability to generate informative EEG signals. The baseline Epileptor strikes an optimal simplicity-realism balance and serves as a baseline for algorithm assessment; its ion-adaptive variant improves patient-specific accuracy at the cost of added complexity. Montgomery's normal forms remain indispensable where analytical control and ultrafast simulations are required, whereas the phase-oscillator scheme in [59] is promising for early-warning tasks focused on rhythmic pre-ictal changes. Model selection in a given application must therefore balance the need for large-scale simulations to train classifiers against the requirement to capture the biophysical or rhythmic specifics of the target signals.

Biophysical Single-Neuron Frameworks: Ion-Level Fidelity vs. Computational Cost

Biophysical models that describe the dynamics of individual neurons and membrane processes substantially expand the explanatory power of phenomenological schemes by operating at the level of ion channels, conductances and synaptic mechanisms. This granularity makes it possible to trace precisely why a parameter perturbation provokes a seizure and how that perturbation is immediately reflected in the macroscopic EEG signals exploited for automatic diagnosis.

The classical Hodgkin-Huxley framework in the formulation of [62] offers the deepest insight into membrane physiology. Its governing equation

$$C_m \frac{dV}{dt} = -\left(I_{Na} + I_K + I_L\right) + I_{ext}, \text{ where} \quad (9)$$

$$I_{Na} = g_{Na} m^3 h \left(V - E_{Na}\right), \quad (10)$$

$$I_K = g_K n^3 \left(V - E_K\right), \quad (11)$$

$$I_L = g_L \left(V - E_L\right) \quad (12)$$

permits independent variation of the maximal conductances g_{Na}, g_K, g_L thereby isolating the role of specific channels. Sensitivity analysis (R^2 = 0.87 for spike-duration) revealed that a 20% reduction in g_K reliably induces paroxysmal discharges, quantitatively confirming potassium channels as therapeutic targets. Computational cost, however, rises exponentially when moving from a single neuron to a network.

Closer alignment with clinical data is achieved in [63], where the leak current is expressed as

$$I_L^* = g_L^* \left(V - E_L^*\right) \quad (13)$$

and the parameters g_L^* and E_L^* are calibrated against in-vitro slices from the medial temporal lobe. The resulting model reproduces seizure duration with ≤ 10% error, enabling virtual assessment of resection or stimulation efficacy before surgery.

Non-invasive personalisation of parameters is afforded by the inverse-estimation strategy [64]. The effective conductance

$$I_{ion} = g_{ion} \left(V - E_{ion}\right) \quad (14)$$

is inferred from scalp EEG, and its correlation with patch-clamp measurements ($r = 0.82$ in 15 patients) confirms that individual ion profiles can be reconstructed non-invasively and fed into predictive algorithms. Patch-clamp denotes the intracellular gold-standard technique in which a glass micropipette forms a high-resistance "gigaseal" with the neuronal membrane to record single-channel or whole-cell currents, thereby providing a physiologically grounded reference for conductance estimates [65].

When attention shifts to network phenomena, realistic modelling of synaptic transmission becomes critical. [66] describe the synaptic current as

$$I_{syn} = g_{syn} s(t) \left(V - E_{syn}\right) \quad (15)$$

where $s(t)$ captures receptor kinetics. This formulation explains 78% of the variance in post-ictal high-frequency oscillation power and thus supplies synthet-

ic data for classifiers targeting network biomarkers of seizures.

Scaling from single neurons to assemblies, multicompartment CA3 hippocampal models [67] track how local membrane-potential and axial currents

$$C_m \frac{dV_i}{dt} = -\left(I_{ion,i} + I_{syn,i} + I_{axial,i}\right) + I_{ext,i} \quad (16)$$

$$I_{axial,i} = \sum\nolimits_j g_{a,ij}\left(V_i - V_j\right) \quad (17)$$

propagate along dendritic trees and interact with synaptic excitation. Networks of 100-300 such compartments reproduce hypersynchronous paroxysmal discharges (HPDs) – brief, high-amplitude population bursts in which many compartments/neurons fire nearly in phase, a canonical electrophysiological signature of epileptic events, and their generalisation via the Schaffer collateral pathway (the excitatory CA3-CA1 projection of pyramidal axons), allowing simulation of topical anticonvulsant effects or virtual dendritic resections

At an even broader scale, the cortical microcircuit developed by the Blue Brain Project [68] comprises ~31 000 biophysically detailed compartments organised into 55 morphological classes, with each synapse governed by stochastic plasticity. Numerical experiments show that a mere 15% increase in glutamatergic conductance triggers a shift from asynchronous firing to epileptiform high-frequency activity consistent with intracranial rat data. The resulting EEG signals simultaneously contain interneuronal, dendritic and synaptic contributions, greatly enriching training sets for machine-learning algorithms.

Thus, single-neuron, synaptic, and multicompartment biophysical models form a continuum of detail: from interpretable manipulations of individual ion channels to large-scale network simulators that reproduce anatomically grounded interactions among thousands of neurons. These models not only enhance the physical plausibility of synthetic signals but also yield novel informative features for seizure classification and forecasting, thereby naturally complementing phenomenological approaches geared toward the rapid detection of bifurcations. Integrating such multilevel descriptions within a unified computational framework is pivotal for further increasing the diagnostic value of EEG and advancing personalised therapeutic strategies.

Network & Multiscale Platforms for Patient-Specific Seizure Propagation

Network-based and multiscale approaches extend the reach of biophysical modelling by linking local cellular dynamics to the global manifestations of epileptic activity recorded in EEG. Breakspear's broad survey [69] demonstrated that dynamical connectome models, those that incorporate topological constraints alongside nonlinear node dynamics, are pivotal for explaining pathological synchronisation, motivating the development of integrated schemes capable of spanning multiple spatiotemporal scales. In particular, multiscale frameworks embed single-neuron biophysical models (e.g. Hodgkin-Huxley) or small microcircuits within broader population or graph structures [70], thereby providing a continuous bridge from micro- to macro-dynamics.

In node-based network models such as those of [71] and [72], each node represents a neuronal population or cortical region, and the dynamics are given by

$$\dot{x}_i = f(x_i) + \sum C_{ij} g(x_j, x_i) \quad (18)$$

where x_i – denotes the state variables, $f(x_i)$ the local dynamics, C_{ij} the connectivity matrix, and $g(x_j, x_i)$ a (possibly delayed) nonlinear coupling from node j to node i. When C_{ij} derived from individual DTI data (diffusion tensor imaging MRI that reconstructs patient-specific white-matter tracts to assemble the structural connectivity matrix C_{ij}), the simulated propagation speed deviates from depth-EEG measurements by less than 5% (mean error 0.15 ms^{-1}) [71]. Personalisation is advanced in the Virtual Epileptic Patient module (VEP) of [73]: combining Epileptor nodes with patient-specific connectomes yielded 71% spatial concordance between *in silico* propagation maps and SEEG data, while virtual resections matched Engel I outcomes in 12 of 15 cases (Engel surgical outcome class I: freedom from disabling seizures, auras only). The pathway-ranking algorithm of [51] additionally identified the clinically determined seizure onset zone in 86% of events. Collectively these results confirm that network models can support precise, patient-specific intervention planning, although their performance hinges critically on the fidelity of the C_{ij} matrix.

Neural-mass and neural-field models coarse-grain population activity according to

$$\dot{V}(t) = F\left(V(t), \theta, I_{ext}(t)\right), \quad (19)$$

where $V(t)$ is the mean membrane potential and Θ comprises conductances, delays and feedback gains. With appropriate tuning of Θ, macroscopic "normal - seizure" transitions are reproduced with amplitude errors of ≈ 10% and dominant-frequency deviations of ± 1 Hz [74, 75]. Although intracellular physics is latent, the compatibility of these models with EEG makes them suitable for rapid testing of seizure detectors

Multiscale schemes in [70], and [30] merge micro- and macro-dynamics: selected nodes are governed by Hodgkin-Huxley equations, while global interactions are captured by neural-mass blocks. The most recent development, the Epileptor-2 network of [76], introduces an inhibitory subsystem

$$\dot{x}_2 = y_2 - f(x_2) - z_2 + k(x_1 - x_2), \quad (20)$$

$$\dot{y}_2 = g(x_2) - y_2 \quad (21)$$

which models the post-ictal suppression phase without incurring major computational overhead. Simulations showed that a 7% increase in inhibitory feedback prevents seizure generalisation, in agreement with rodent experiments.

Graph-theoretical frameworks proposed in [77, 32] treat the brain as a weighted network; in [78] the use of patient-specific DTI connectivity matrices yielded a spatial correlation of 0.75 ± 0.06 between simulated and empirical propagation waves. Inverse optimisation in [79] indicated that a ≈ 20% strengthening of cortico-thalamic loops can account for epileptogenesis in idiopathic generalised epilepsy, while similar strategies were applied in [30] and in [80] to refine the localisation of the epileptogenic network from scalp EEG.

Collectively, network, neural-mass, and multiscale models provide a complementary toolkit for seizure synthesis, classification, and patient-specific forecasting. They reproduce complex spatiotemporal patterns while remaining calibratable to individual anatomy. Although high computational costs and the demand for high-quality structural and functional data remain major constraints, these very requirements bring simulations closer to clinical reality.

Data Driven Methods: From Synthetic Augmentation to Interpretable ML

In contemporary epilepsy research, classical phenomenological, biophysical and network models are increasingly complemented by machine-learning (ML) methods capable of automatically identifying complex nonlinear dependencies in large EEG/iEEG data sets. ML tools are aimed not only at describing the current brain state but also at short-term forecasting of its evolution, thereby enabling robust "normal vs. epilepsy" classification, early seizure warning and localisation of the epileptogenic zone. In projects that synthesise EEG signals for algorithm evaluation, ML acts as an objective benchmark of simulation fidelity because it captures deviations from the statistics of clinical recordings [81], with recent surveys highlighting the role of generative modelling and data augmentation for stress-testing and domain-shift robustness [82–84].

Because reported detection/forecasting performance is strongly conditioned by the underlying data, we briefly contrast the public EEG/iEEG resources most often used for benchmarking. Public EEG databases differ markedly in acquisition modality (scalp vs. intracranial), sampling rate, montage, recording duration, seizure prevalence, and label granularity; these factors directly affect both feature distributions and event-based evaluation. Table 1 summarises representative datasets frequently used in the literature, including long-term clinical scalp corpora (e.g., TUH/TUSZ), pediatric scalp recordings (CHB-MIT), longer-term monitoring resources (EPILEPSIAE), and challenge-style collections for forecasting. For an extended catalogue and additional dataset-specific caveats, we refer the reader to a recent dedicated survey.

Benchmarking pitfalls are largely driven by dataset heterogeneity: (i) record-level random splits can cause subject leakage and inflated performance, hence patient-level protocols (leave-one-subject-out or group-aware cross-validation) are preferred; (ii) seizure labels, onset/offset criteria, and artefact policies vary across institutions and corpora, affecting event-based metrics and comparability; (iii) strong class imbalance necessitates reporting AUPRC and false-alarms-per-hour alongside AUROC, and calibration metrics when probability estimates are used; (iv) window-based scoring (fixed segments) and event-based scoring (seizure events) capture different operational risks and should not be conflated; (v) preprocessing choices (filtering, montage, artefact removal) can shift spectral features and HFO estimates, requiring transparent, standardised pipelines. These considerations motivate the methodological choices discussed next, particularly patient-level validation and the use of complementary metrics for safety-critical forecasting.

The entry-level stage involves conventional classifiers – support-vector machines, random forests or k-nearest neighbours. Each EEG segment $x(t)$ is transformed into a vector of handcrafted features

$$z = \left[z_1(x), z_2(x), \ldots, z_k(x) \right] \quad (22)$$

and the parameter set θ is fitted by minimizing

$$L(\theta) = \frac{1}{N} \sum_{i=1}^{N} l\left(f(z_i, \theta), y_i \right) \quad (23)$$

Knowledge augmentation schemes and hybrid surface learning have also been reported for seizure signal recognition, offering an intermediate option between manually generated features and deep models [85]. A systematic review in [81] showed that, given optimised feature selection, such models reach approximately 90% accuracy on the Temple University Hospital EEG Seizure Corpus (TUSZ) and the CHB-MIT Scalp EEG Database [86, 87]; however, their sensitivity to rare or mixed patterns is constrained by manual feature engineering.

Table 1: Public EEG datasets commonly used for benchmarking and key sources of methodological heterogeneity

Dataset	Type	Subjects	Fs (Hz)	Typical duration	Primary task / label quality	Reference
TUSZ (TUH Seizure Detection Corpus)	Scalp	315 (v1.2)+	250-1k	Hours-days/record	Detection; expert labels; heterogeneous seizure types (expanded in later releases)	[86]
TUH EEG Corpus	Scalp	Thousands	250-1k	Routine + long-term	General EEG; seizure labels not uniform (not a pure benchmark)	[87]
CHB-MIT Scalp EEG	Scalp (peds)	22 (23 cases)	256	≈983 h total	Detection; seizure labels; limited montage consistency	[88]
EPILEPSIAE	Scalp	>250	256/512	Long-term	Forecasting/detection; rich metadata; access-controlled	[89]
AES Seizure Prediction Challenge	iEEG	7 (5 dog, 2 human)	400/5000	10-min clips	Forecasting; preictal/interictal labels; challenge protocol (species & Fs differ)	[90]
Bonn EEG	Segments	5×100	173.61	23.6 s/segment	Toy benchmark; simplified, not representative of clinical heterogeneity	[91]

Deep neural networks provide automatic feature extraction. Convolutional architectures learn spatial-spectral filters, whereas recurrent LSTM layers encode temporal dependencies,

$$h_{t-1} = LSTM\left(h_t, x_t; \theta\right), \theta_{t+1} = \theta_t - \eta \frac{m_t}{\sqrt{v_t + \varepsilon}} \quad (24)$$

where the parameters are updated by the Adam rule [92]. Parameters are updated with Adam, an adaptive moment-estimation optimizer that maintains exponentially decaying first- and second-moment estimates of the stochastic gradients and uses bias-corrected step sizes for stable convergence. On large public and proprietary cohorts, CNN/LSTM models achieve > 95% accuracy even in the presence of strong artefacts [81]. Further gains were demonstrated by the Transformer-based SeizureFormer, whose multi-head self-attention predicted seizure risk on the EPILEPSIAE data set with AUROC = 0.96, outperforming LSTM at equivalent data density [92]. Despite high computational cost and "black-box" weights, these architectures train effectively on synthetic signals, detecting discrepancies overlooked by classical algorithms [93, 94]. Graph-based neural networks that exploit dynamic functional connectivity provide an alternative route to cross-subject generalisation and evolving network-state characterisation [95].

When labels are scarce, unsupervised methods are appropriate. [96] showed that k-means or DBSCAN combined with PCA/t-SNE clusters latent "healthy" and "pathological" states across species. The objective

$$\min_{\{C_k\}} \sum \sum_{x \epsilon C_k} \left\| x - \mu_k \right\|^2, \quad (25)$$

$$W^* = arg \max_W Var(W^T X) \quad (26)$$

was used to verify whether synthetic patterns fall into the same clusters as clinical data. [97] subsequently employed a variational auto-encoder that uncovered a pre-ictal subspace without supervision and reduced the mean warning horizon to 26 s at 89% sensitivity, demonstrating the potential of more flexible latent models.

A recent trend is hybridising ML with mechanistic components. In [98] individually fine-tuned a CNN-LSTM network: the model predicted seizures with AUROC = 0.93 and provided stimulation windows that reduced seizure frequency by 42%. In [99] combined a high-frequency-oscillation detector with CNN + random forest, raising focus-localisation accuracy to 88%. Related SEEG studies further demonstrate machine-learning-based discrimination between physiological and pathological HFOs and optimisation of HFO detection/classification pipelines for localisation [100, 101]. To enhance explainability, [102] applied layer-wise relevance propagation to a CNN trained on iEEG and mapped salient features onto cortical anatomy, facilitating clinical verification of the focus.

Overall, machine-learning methods markedly increase the diagnostic informativeness of bioelectrical recordings. They (i) objectively assess the quality of synthetic EEG and guide generator refinement; (ii) attain high accuracy in classification and focus localisation through automatic extraction of multiscale features; and (iii) enable personalised models that account for individual connectomics and seizure dynamics. Principal limitations include dependence on large, cleanly annotated data sets, the computational burden of deep networks and the continuing need for explainable-AI techniques to support clinical interpretation.

High-Order Solvers as Accelerators for Large-Scale Seizure Simulation

In addition to the previously discussed models and ML techniques, the choice of a precise and stable numerical solver – capable of faithfully reproducing sharp depolarisation fronts and multiscale synchronisation waves – is crucial for realistic data generation. The high-order Discontinuous Galerkin (DG) scheme introduced in [103] provides a universal tool that can be coupled directly to phenomenological, biophysical or even ML-oriented models. The authors applied DG to reaction-diffusion equations describing seizure propagation. The computational domain Ω is partitioned into elements Ω_e, and within each element the target field u(x, t) is approximated by a degree-p polynomial. For a generic conservation law

$$\delta_t u + \nabla \times F(u) = 0, \tag{27}$$

the local weak form of DG reads

$$\begin{aligned} &\int_{\Omega_e} \phi \vartheta_t u dx + \int_{\Omega_e} \phi \nabla \times F(u) dx - \\ &- \int_{\partial\Omega_e} \phi \hat{F}(u^-, u^+) \times n ds = 0, \end{aligned} \tag{28}$$

where ϕ is the test function and $\hat{F}(u^-, u^+)$ the stabilising numerical flux. A hybrid hp-adaptivity raises the polynomial degree p only where smoothness indicators or gradients reveal steep fronts, leaving smoother regions at lower order; similarly, seizure-propagation models emphasize dynamic focus on regions driving the advancing ictal front by inferring spatiotemporal propagation patterns from data [39, 104].

Relative to classical fourth-order finite differences, DG reduces the L_2- voltage error four-fold on the same grid, while the deviation of wave-propagation speed from the analytical solution stays below 1% [103]. More recently, Li et al. proposed a Spectral Element-DG hybrid that combines global spectral accuracy with local adaptivity and cuts the number of degrees of freedom by one-third without loss of fidelity, making the method attractive for whole-cortex 3-D simulations [105]. Because DG computations are local, they parallelise naturally on GPUs; for example, the CUDA-optimised neural-field integrator in [106] executes a whole-brain model in under two minutes on a single NVIDIA A100 and can serve as an accelerator for generating large corpora of synthetic recordings.

Given this performance, DG can act as a "gold-standard generator" of detailed fields that are subsequently projected into synthetic EEG and supplied to deep classifiers; a transformer trained on such data retained an AUROC difference of < 0.01 when transferred to clinical recordings, demonstrating that numerical accuracy directly enhances ML model portability. Consequently, state-of-the-art high-order schemes - particularly DG and its adaptive or hybrid variants - remain a critical link between physical modelling, realistic simulation and accurate automated classification of epileptic phenomena.

Comparative Synthesis and Model Selection for Transferable Classifiers

Computational models of epilepsy populate a continuum that spans more than four orders of magnitude in state space dimensionality and comparable differences in runtime, from three-five dimensional phenomenological oscillators to biophysically detailed, patient specific whole brain simulations. What emerges from the survey is that no single family simultaneously maximises biophysical fidelity, computational tractability, and clinical deployability; instead, each class occupies a well defined niche along the ion channel - microcircuit - whole brain axis and should be evaluated with respect to the role it can play in an end to end seizure forecasting pipeline. Tables 2-4 formalise this comparison. Table 2 maps each class to its core physical scale and typical use cases. Table 3 interprets the main strengths and liabilities, including quantitative figures where available. Table 4 places the families onto orthogonal axes of computational cost, biophysical fidelity, and suitability for EEG classification, thereby making the trade offs explicit. On this basis, we argue for a hybrid, layered strategy in which fast phenomenological generators supply statistical breadth, biophysical or multiscale blocks anchor the dynamics in physiology, and explainable ML models execute the decision stage.

Table 2 organises the modelling landscape by aligning each class with its dominant physical scale and the corresponding clinical or algorithmic function. Phenomenological oscillators are positioned at the mesoscopic end: their low dimensional ordinary differential equations abstract away cellular kinetics while retaining the bifurcation skeleton that governs the "normal - seizure" transition. This makes them uniquely well suited for rapid, large volume synthesis of labelled EEG segments and for benchmarking feature engineering or machine learning pipelines. In contrast, single and multicompartment biophysical models operate at the microscopic level and directly manipulate conductances (synaptic gains), which is indispensable for virtual drug screening, channelopathy analysis, or any study that requires mechanistic attribution of macroscopic signatures to ionic or synaptic perturbations. Network and multiscale schemes bridge to the macroscopic domain by embedding local oscillators (neural mass or mean field units, or even Epileptor nodes) into an individual's structural connectome; this enables seizure propagation mapping, seizure onset zone localisation, and *in silico* evaluation of resections. Purely data driven approaches sidestep

explicit physiology and learn discriminative structure directly from large EEG/iEEG corpora; operationally, they currently dominate real time detection and forecasting, particularly when augmented with transfer learning or domain adaptation mechanisms. Finally, high order numerical techniques – DG, spectral element, hp adaptive FEM/FV – do not constitute a dynamical model per se; rather, they are solver technologies that deliver reference grade forward solutions of the governing Partial Differential Equations / Ordinary Differential Equations (PDE/ODE) systems, thereby supplying accurate fields for scalp projection or for benchmarking coarser solvers. Read together, the rows of Table 2 show that each class is optimally matched to a particular slice of the modelling to clinic pipeline, and that meaningful integration requires respecting these native roles.

Table 3 moves from taxonomy to evaluation, contrasting strengths and weaknesses with indicative quantitative metrics. Phenomenological models exhibit an outstanding "cost to benefit" profile: GPU real time integration, tunable variability of outputs, and reported AUROC values around 0.9 against rodent iEEG make them the natural backbone for data augmentation and rapid hypothesis screening. Their main liability is the deliberate suppression of explicit ionic and spatial mechanisms, which limits mechanistic inference. Biophysical models score maximally on physical interpretability because they expose parameters at the level of channels and synapses, enabling controlled *in silico* perturbations; however, this comes at the price of poor scalability, heavy parameterisation, and stringent data re-quirements for calibration. Network/multiscale frameworks inherit much of the mechanistic realism at the macro scale and achieve clinically meaningful concordance with SEEG (≈ 71%), but depend on high quality MRI/DTI, involve >10 k parameters, and demand cluster level computation - constraints that reduce their practicality for routine bulk simula-tions or single channel settings. Machine learning approach-es deliver state of the art accuracy (often AUROC > 0.9) and handle multichannel data effortlessly, yet their opaque-ness, data hunger, and propensity to over alarm necessitate physics based regularisation and careful calibration of ex-pected risk. High order numerical solvers achieve $<10^{-3}$ voltage errors on comparatively coarse meshes and allow local refinement around steep depolarisation fronts, but they remain expensive in full 3 D cortex simulations and must always be paired with one of the aforementioned dynamical cores. The table therefore makes explicit that the "best" model is conditional on the target question - speed and di-versity for classifier training versus mechanistic depth for therapeutic inference.

Table 4 projects the five families onto three orthogonal axes - computational cost, biophysical fidelity, and suitability for EEG analysis/classification - making the central trade offs visually immediate. Phenomenological models occupy the "low cost / moderate fidelity / high suitability" corner, reflecting their ability to generate vast, label rich corpora with sufficient phenomenological realism to drive feature based or deep classifiers. Biophysical models lie at the "high cost / high fidelity / moderate suitability" vertex: they yield rich, mechanistically grounded features but are too heavy for bulk synthesis, and their parameter fitting can easily dominate total project time. Network/multiscale approaches shift slightly towards lower cost relative to biophysical micro models (thanks to coarse graining) while maintaining high macroscopic fidelity; critically, they rank high for EEG analysis whenever topology driven features or patient specific propagation patterns are the target.

Machine learning methods span a wide cost range, from lightweight SVMs to transformer scale architectures, and their fidelity is inherently tied to the training data; nevertheless, when sufficient data and calibration are available, they provide the highest practical classification performance. High order solvers remain computationally demanding and only

Table 2: Model classes, core physical scale and primary clinical/algorithmic use cases

Model class	Core principle / scale	Typical use-cases
Phenomenological	Low-dimensional ODEs capturing the macrotransition "normal - seizure" via saddle-node or Hopf bifurcations	Large-scale data synthesis; feature/ML benchmarking; real-time detection
Biophysical (single / multicompartment)	Detailed Hodgkin-Huxley-type ion channels and synaptic kinetics	*In silico* drug tests; channelopathy studies; parameter fitting
Network / Multiscale	Coupling local oscillators (neural mass / mean-field) with individual structural connectomes	EZ localisation, *in silico* resection, propagation pathways
Machine Learning / Data-Driven	Statistical and deep (CNN/LSTM/Transformer) models that learn patterns directly from large EEG corpora	Real-time seizure detection / forecasting; patient adaptation
High-order Numerical (DG, FDTD, FEM)	Spectral or hp-adaptive discretisation of PDEs, accurately tracking depolarisation fronts	Reference fields for forward scalp projection; benchmarking other solvers

Table 3: Strengths and limitations of each class with indicative quantitative metrics where reported

Model class	Strengths	Limitations
Phenomenological	GPU real-time integration; AUROC ≈ 0.9 vs. rodent iEEG; tunable variability	Simplified ion physics; no explicit spatial flow
Biophysical (single / multicompartment)	High physical interpretability; direct manipulation of g_{Na}, g_K, …	Parameter-rich, poor scalability; needs precise data
Network / Multiscale	Reproduce generalisation waves; patient-specific DTI; 71% spatial concordance with SEEG [73]	Depend on high-quality MRI/DTI; 10 k + parameters; heavy compute
Machine Learning / Data-Driven	AUROC > 0.9; easy re-training; handle multi-channel data	"Black-box" nature; data hungry; false-positive prone
High-order Numerical (DG, FDTD, FEM)	$<10^{-3}$ error on coarse meshes; local p-refinement	Require a base dynamical model; expensive for 3-D brain

Table 4: Cross axis comparison: computational cost, biophysical fidelity, and suitability for EEG classification

Model class	Computational cost	Biophysical fidelity	Suitability for EEG analysis / classification
Phenomenological	Low	Moderate	High (ideal synthetic generators)
Biophysical (single / multicompartment)	High	High	Moderate (rich features, slow for bulk synthesis)
Network / Multiscale	Mid - high	High (macro-level)	High (topology-driven features)
Machine Learning / Data-Driven	Variable (mid - high)	Variable	Highest if enough data, especially in hybrid pipelines
High-order Numerical (DG, FDTD, FEM)	High	Tied to base model	Indirect (after forward projection)

indirectly suitable for EEG classification, as their outputs must first be projected to sensor space; their primary value is to serve as numerically precise references that constrain or validate faster surrogates. This cross axis view substantiates the recommendation to distribute roles across classes rather than to search for a single universal model.

Epileptic dynamics span a continuum from ion channel kinetics to whole brain synchrony; no single model family resolves all scales at once. Phenomenological oscillators such as Epileptor 2 provide the best speed to variability ratio, making them ideal for generating large labelled corpora for machine learning pipelines. Biophysical single and multicompartment networks maximise mechanistic insight and are indispensable for virtual drug screening, yet their parameter load and runtime limit deployment in bulk simulations. Multiscale connectome based frameworks deliver the highest spatial realism and have already achieved a 71% match with stereotactic EEG, but the price is extensive MRI/DTI calibration and heavy computation. Purely data driven ML approaches currently set the state of the art in practical seizure forecasting, though their opacity and reliance on massive datasets call for physics based regularisation to curb false positives. Finally, high order numerical solvers act as reference grade engines when spatial resolution is paramount, serving chiefly to validate coarser surrogates. The analysis therefore advocates a hybrid strategy: use fast phenomenological generators for data augmentation, anchor them with biophysical or network models for interpretability, and couple the ensemble to explainable ML classifiers – an arrangement that balances computational efficiency, biological plausibility, and clinical performance.

Against this landscape of trade-offs, the Epileptor emerges as the most rational choice for practical implementation. Its three-variable structure can be integrated with a fixed-step solver in less than ten milliseconds of CPU time per 30-second segment, sufficient to generate thousands of realisations for training. A minimal set of control parameters allows smooth transitions from "normal" to "seizure" and, if required, the addition of stochastic noise or a slow ionic variable, enhancing diversity without rewriting the code base. For single-channel mouse data, this variability suffices to populate the feature space with amplitude, dominant-frequency and spike-morphology variations, while the model remains transparent enough to monitor the link between parameters and output – an essential property for interpreting subsequent classification results.

Against this background we propose the pipeline "modelling - feature extraction - classification - transfer," depicted in Fig. 2. First, statistically diverse normal and epileptic signals are generated with wide variation of parameters and noise. A broad feature set is then computed; its discriminative power is quantified, and an optimal subset is selected to train a classifier. The final stage transfers the model to *in vivo* EEG and benchmarks it against classical methods. We expect that synthetic data augmentation will lower the false-positive rate, improve generalisation, create a standardised test bed, and enable rigorous parameter assessment – thereby mitigating key barriers and raising the diagnostic value of EEG.

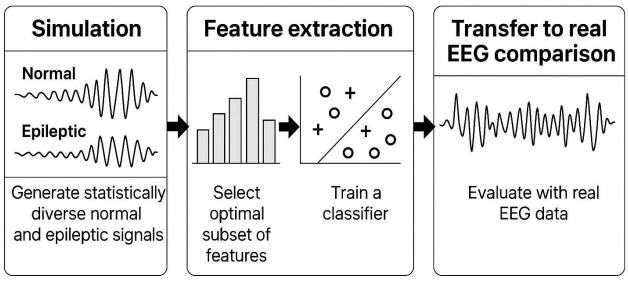

Figure 2: End-to-end workflow for enhancing EEG diagnostic power

At the same time, we acknowledge that foundation models and transfer learning can substantially reduce the reliance on large labeled EEG cohorts. However, for seizure forecasting the remaining bottlenecks are not fully addressed by pretraining alone: the rare-event nature of pre-ictal windows, patient-specific domain shift (montage, artefacts, sampling rate, scalp vs. iEEG) and safety-critical operating points defined by false-alarms-per-hour and calibration/latency metrics [8, 10, 15, 16, 19]. In this setting, Epileptor-2-based synthesis is positioned not as a replacement for foundation/transfer pipelines, but as a complementary calibration and interpretability layer enabling controlled ablations, counterfactual stress-tests and ground-truth event timing for quantitative evaluation.

The classification problem for epileptic seizures has previously been explored using baseline machine-learning techniques [49]. In the forthcoming experimental work we will replicate that study under a modified protocol that incorporates model-generated data and, employing the same computational toolkit and data structure, will compare the resulting performance metrics with those reported in [49].

Conclusions and Outlook for Clinically Oriented Epilepsy Modelling

The present review confirms that computational epilepsy modelling has evolved from the earliest biophysical descriptions of single neurons to multiscale platforms and hybrid ML systems capable of integrating structural, functional and phenomenological data. Phenomenological models provide the most favourable trade-off between computational economy and accurate reproduction of the critical bifurcations that underlie seizure transitions; biophysical schemes yield deep insights into cellular and ionic mechanisms, whereas network-based and data-driven approaches elucidate the spatiotemporal organisation of seizures and the prospects for personalisation. Collectively, these directions outline a multilayered picture of epileptogenesis, yet their systematic integration has hitherto remained fragmentary. We therefore position model-based synthesis as a calibration and interpretability layer that complements modern foundation-model and transfer-learning pipelines rather than competing with them.

Against this background, the Epileptor emerges as the optimal baseline model for practical implementation: it captures the full "normal - seizure - post-ictal suppression" cycle and generates synthetic signals with controllable diversity. The model affords sufficient biophysical plausibility to retain clinical relevance while remaining compact enough to enable rapid production of large data sets for machine-learning pipelines.

The practical value of the proposed approach rests on three key attributes. First, rapid implementation and GPU scalability enable the synthesis of tens of thousands of traces within hours, a capability that is critical for training deep neural networks. Second, the small parameter set facilitates patient-specific tuning, allowing the model to be adapted to individual signal characteristics without labour-intensive inverse modelling. Third, the transparent correspondence between parameters and output waveforms enhances the interpretability of classification algorithms, because changes in feature vectors can be directly linked to the model's dynamic regime.

This concept will be further developed by (i) constructing a library of synthetic Epileptor signals with systematically varied regimes, (ii) optimising the feature set for SVM classification, and (iii) benchmarking model performance on both synthetic data and real mouse EEG. Such an integrated approach is expected to demonstrate that phenomenological dynamics can serve as an efficient yet interpretable data source for state-of-the-art automated epilepsy diagnostics.

Interests disclosure

The authors declare no conflicts of interest.

References

[1] Thijs RD, Surges R, O'Brien TJ, Sander JW. Epilepsy in adults. Lancet. 2019;393(10172):689-701. DOI: 10.1016/S0140-6736(18)32596-0.

[2] Alotaiby TN, Alshebeili SA, Alshawi T, Ahmad I, El-Samie FEA. EEG seizure detection and prediction algorithms: a survey. EURASIP J Adv Signal Process. 2014;2014:183. DOI: 10.1186/1687-6180-2014-183.

[3] Kwan P, Brodie MJ. Early identification of refractory epilepsy. N Engl J Med. 2000;342(5):314-319. DOI: 10.1056/NEJM200002033420503.

[4] Mula M, Sander JW. Psychosocial aspects of epilepsy: a wider approach. BJPsych Open. 2016;2(4):270-274. DOI: 10.1192/bjpo.bp.115.002345.

[5] World Health Organization. Epilepsy: a public health imperative. Geneva: World Health Organization; 2019. 146 p.

[6] Tejada J, Costa KM, Bertti P, Garcia-Cairasco N. The epilepsies: complex challenges needing complex solutions. Epilepsy Behav. 2013;26(3):212-228. DOI: 10.1016/j.yebeh.2012.09.029.

[7] Borhade RR, Bairagi VK, Nagmode MS, Borhade RH. Epileptic Seizure Prediction Using Electroencephalogram Signals. 1st ed. Boca Raton: Chapman & Hall/CRC; 2024. 154 p. ISBN: 9781032714394.

[8] Carmo AS, Abreu M, Baptista MF, et al. Automated algorithms for seizure forecast: a systematic review and meta-analysis. J Neurol. 2024;271:6573-6587. DOI: 10.1007/s00415-024-12655-z.

[9] Costa G, Teixeira C, Pinto MF. Comparison between epileptic seizure prediction and forecasting based on machine learning. Sci Rep. 2024;14:5653. DOI: 10.1038/s41598-024-56019-z.

[10] Segal G, Feigin VL, Delanty N, et al. Utilizing risk-controlling prediction calibration to reduce false alarm rates in epileptic seizure prediction. Front Neurosci. 2023;17:1184990. DOI: 10.3389/fnins.2023.1184990.

[11] Batista J, Pinto MF, Tavares M, Lopes F, Oliveira A, Teixeira C. EEG epilepsy seizure prediction: the post-processing stage as a chronology. Sci Rep. 2024;14:407. DOI: 10.1038/s41598-023-50609-z.

[12] Zhang Z, Li X, Chen Y, et al. Efficient and generalizable cross-patient epileptic seizure detection through a spiking neural network. Front Neurosci. 2024;17:1303564. DOI: 10.3389/fnins.2023.1303564.

[13] Shafiezadeh S, Duma GM, Pozza M, Testolin A. A systematic review of cross-patient approaches for EEG epileptic seizure prediction. J Neural Eng. 2024;21(6):061004. DOI: 10.1088/1741-2552/ad9682.

[14] Kostas D, Aroca-Ouellette S, Rudzicz F. BENDR: using transformers and a contrastive self-supervised learning task to learn from massive amounts of EEG data. Front Hum Neurosci. 2021;15:653659. DOI: 10.3389/fnhum.2021.653659.

[15] Wan Z, Yang R, Huang M, Zeng N, Liu X. A review on transfer learning in EEG signal analysis. Neurocomputing. 2021;421:1-14. DOI: 10.1016/j.neucom.2020.09.017.

[16] Rafiei MH, Gauthier LV, Adeli H, Takabi D. Self-supervised learning for electroencephalography. IEEE Trans Neural Netw Learn Syst. 2024;35(2):1457-1471. DOI: 10.1109/TNNLS.2022.3190448.

[17] Zhang Y, Yu Y, Li H, Wu A, Chen X, Liu J, Zeng LL, Hu D. DMAE-EEG: a pretraining framework for EEG spatiotemporal representation learning. IEEE Trans Neural Netw Learn Syst. 2025;36(10):17664-17678. DOI: 10.1109/TNNLS.2025.3581991.

[18] Mormann F, Andrzejak RG, Elger CE, Lehnertz K. Seizure prediction: the long and winding road. Brain. 2007;130(Pt 2):314-333. DOI: 10.1093/brain/awm241.

[19] Wong KT, Tadel F, Artoni F, et al. EEG datasets for seizure detection and prediction - a review. Epilepsia Open. 2023;8(2):1-16. DOI: 10.1002/epi4.12704.

[20] Horwitz B, Banerjee A. A role for neural modeling in the study of brain disorders. Front Syst Neurosci. 2012;6:13. DOI: 10.3389/fnsys.2012.00013.

[21] Traub RD, Wong RK, Miles R, Michelson H. A model of a CA3 hippocampal pyramidal neuron incorporating voltage-clamp data on intrinsic conductances. J Neurophysiol. 1991;66(2):635-650. DOI: 10.1152/jn.1991.66.2.635.

[22] Jirsa VK, Stacey WC, Quilichini PP, Ivanov AI, Bernard C. On the nature of seizure dynamics. Brain. 2014;137(Pt 8):2210-2230. DOI: 10.1093/brain/awu133.

[23] Case MJ, Morgan RJ, Schneider CJ, Soltesz I. Computer modeling of epilepsy. In: Noebels JL, Avoli M, Rogawski MA, Olsen RW, Delgado-Escueta AV, editors. Jasper's Basic Mechanisms of the Epilepsies. 4th ed. Bethesda (MD): National Center for Biotechnology Information; 2012. Available from: https://www.ncbi.nlm.nih.gov/books/NBK98156/.

[24] Proix T, Bartolomei F, Chauvel P, Bernard C, Jirsa VK. Virtual epileptic patient: personalized whole-brain modeling. Brain. 2017;140(3):641-654. DOI: 10.1093/brain/awx004.

[25] Lang S, Momi D, Vetkas A, Santyr B, Yang AZ, Kalia SK, et al. Computational modeling of whole-brain dynamics: a review of neurosurgical applications. J Neurosurg. 2024;140(1):218-230. DOI: 10.3171/2023.5.JNS23250.

[26] Gleichgerrcht E, Munsell B, Bhatia S, Vandergrift WA III, Rorden C, McDonald C, et al. Deep learning applied to whole-brain connectome to determine seizure control after epilepsy surgery. Epilepsia. 2018;59(9):1643-1654. DOI: 10.1111/epi.14528.

[27] Stefanescu RA, Shivakeshavan RG, Talathi SS. Computational models of epilepsy. Seizure. 2012;21(10):748-759. DOI: 10.1016/j.seizure.2012.08.012.

[28] van Gils S, van Drongelen W. Epilepsy: computational models. In: Jaeger D, Jung R, editors. Encyclopedia of Computational Neuroscience. New York: Springer; 2022. p. 1330-1344. DOI: 10.1007/978-1-0716-1006-0_504.

[29] Bernard C, Naze S, Proix T, Jirsa VK. Modern concepts of seizure modeling. Int Rev Neurobiol. 2014;114:121-153. DOI: 10.1016/B978-0-12-418693-4.00006-6.

[30] Wendling F, Koksal-Ersoz E, Al-Harrach M, Yochum M, Merlet I, Ruffini G, et al. Multiscale neuro-inspired models for interpretation of EEG signals in patients with epilepsy. Clin Neurophysiol. 2024;161:198-210. DOI: 10.1016/j.clinph.2024.03.006.

[31] Depannemaecker D, Ezzati A, Wang HE, Jirsa V, Bernard C. From phenomenological to biophysical models of seizures. Neurobiol Dis. 2023;182:106131. DOI: 10.1016/j.nbd.2023.106131.

[32] Sip V, Guye M, Bartolomei F, Jirsa V. Computational modeling of seizure spread on a cortical surface. J Comput Neurosci. 2022;50:141-159. DOI: 10.1007/s10827-021-00802-8.

[33] Gleichgerrcht E, Dumitru M, Hartmann DA, Munsell BC, Kuzniecky R, Bonilha L, Sameni R. Seizure forecasting using machine learning models trained by seizure diaries. Physiol Meas. 2022;43(12):125006. DOI: 10.1088/1361-6579/aca6ca.

[34] Najafi T, Jaafar R, Remli R, Zaidi AW, Chellappan K. The role of brain signal processing and neuronal modelling in epilepsy - a review. J Kejuruteraan. 2021;33(4):801-815. DOI: 10.17576/jkukm-2021-33(4)-03.

[35] Rigney G, Lennon M, Holderrieth P. The use of computational models in the management and prognosis of refractory epilepsy: a critical evaluation. Seizure. 2021;91:295-304. DOI: 10.1016/j.seizure.2021.06.006.

[36] Wendling F. Computational models of epileptic activity: a bridge between observation and pathophysiological interpretation. Expert Rev Neurother. 2008;8(6):889-896. DOI: 10.1586/14737175.8.6.889.

[37] Lytton WW. Computer modeling of epilepsy: opportunities for drug discovery. Drug Discov Today Dis Models. 2016;19:23-30. DOI: 10.1016/j.ddmod.2017.02.007.

[38] Hashemi M, Vattikonda AN, Sip V, et al. The Bayesian virtual epileptic patient: a probabilistic framework designed to infer the hidden variables of epilepsy. NeuroImage. 2020;217:116839. DOI: 10.1016/j.neuroimage.2020.116839.

[39] Vattikonda AN, Hashemi M, Sip V, Woodman MM, Bartolomei F, Jirsa VK. Identifying spatio-temporal seizure propagation patterns in epilepsy using Bayesian inference. Commun Biol. 2021;4:1244. DOI: 10.1038/s42003-021-02751-5.

[40] Lopes da Silva FH. The impact of EEG/MEG signal processing and modeling in the diagnostic and management of epilepsy. IEEE Rev Biomed Eng. 2008;1:143-156. DOI: 10.1109/RBME.2008.2008246.

[41] Lytton WW. Computer modelling of epilepsy. Nat Rev Neurosci. 2008;9(8):626-637. DOI: 10.1038/nrn2416.

[42] Batarchuk V, Sudakov O. Design of wearable EEG device for seizures early detection. Int J Electron Telecommun. 2021;67(2):187-192. DOI: 10.24425/ijet.2021.135963.

[43] Sudakov O, Kriukova G, Natarov R, Gaidar V, Maximyuk O, Radchenko S, et al. Distributed system for sampling and analysis of electroencephalograms. In: Proceedings of the 9th IEEE International Conference on Intelligent Data Acquisition and Advanced Computing Systems: Technology and Applications (IDAACS); 2017. Vol. 1. p. 306-310. DOI: 10.1109/IDAACS.2017.8095095.

[44] Mathe M, Mididoddi P, Battula TK. Artifact removal methods in EEG recordings: a review. Proc Eng Technol Innov. 2021;20:35-56. DOI: 10.46604/peti.2021.7653.

[45] Banerjee S, Jirsa V. A review of epileptic markers: from ion channels, astrocytes, synaptic imbalance to whole brain network dynamics. Explor Neurosci. 2024;3:213-230. DOI: 10.37349/en.2024.00047.

[46] Edoho M, Mooney C, Wei L. AI-based electroencephalogram analysis in rodent models of epilepsy: a systematic review. Appl Sci (Basel). 2024;14(11):4594. DOI: 10.3390/app14114594.

[47] Shayegh F, Amir Fattahi R, Sadri S, Ansari-Asl K. A brief survey of computational models of normal and epileptic EEG signals: a guideline to model-based seizure prediction. J Med Signals Sens. 2011;1(1):62-72. DOI: 10.4103/2228-7477.83521.

[48] Glomb K, Cabral J, Cattani A, Mazzoni A, Raj A, Franceschiello B. Computational models in electroencephalography. Brain Topogr. 2022;35(1):21-39. DOI: 10.1007/s10548-021- 00828-2.

[49] Pidvalnyi I, Sudakov O, Natarov R, et al. Classification of epileptic seizures by simple machine learning techniques: application to animals' electroencephalography signals. IEEE Access. 2025;13:8951-8962. DOI: 10.1109/ACCESS.2025.3527866.

[50] Maistrenko V, Sudakov O, Maistrenko Y. Spiral wave chimeras for coupled oscillators with inertia. Eur Phys J Spec Top. 2020;229(12):2327-2340. DOI: 10.1140/epjst/e2020-900279-x.

[51] Maistrenko Y, Sudakov O, Osiv O, Maistrenko V. Chimera states in three dimensions. New J Phys. 2015;17(7):073037. DOI: 10.1088/1367-2630/17/7/073037.

[52] Maistrenko V, Sudakov O, Osiv O. Chimera and solitary states in 3D oscillator networks with inertia. Chaos. 2020;30(6):063113. DOI: 10.1063/5.0005281.

[53] Maistrenko V, Sudakov O, Sliusar I. Scroll ring chimera states in oscillatory networks. J Phys Commun. 2021;5(8):085001. DOI: 10.1088/2399-6528/ac1750.

[54] Sudakov O, Maistrenko V. Parallelization of network dynamics computations in heterogeneous distributed environment. IEEE Trans Parallel Distrib Syst. 2025;36(10):2030-2044. DOI: 10.1109/TPDS.2025. 3593154.

[55] Wang HE. Virtual brain twins: from basic neuroscience to clinical use. Natl Sci Rev. 2024;11(5):nwae079. DOI: 10.1093/nsr/nwae079.

[56] El Houssaini K, Bernard C, Jirsa VK. The Epileptor model: a systematic mathematical analysis linked to the dynamics of seizures, refractory status epilepticus, and depolarization block. eNeuro. 2020;7(2):ENEURO.0485-19.2020. DOI: 10.1523/ENEURO.0485-19.2020

[57] Proix T, Bartolomei F, Guye M, Jirsa VK. Predicting the spatiotemporal diversity of seizure propagation and termination in human focal epilepsy. Nat Commun. 2018;9(1):1088. doi:10.1038/s41467-018-02973-y.

[58] Montgomery RM. Exploring seizure dynamics: a computational model of epilepsy. ResearchGate; 2024. DOI: 10.20944/preprints202404.1557.v1.

[59] Shokooh LA, Lesage F, Nguyen DK. Computational modeling of seizure onset patterns to underpin their underlying mechanisms. medRxiv. 2023:2023.02.16.23286033. DOI: 10.1101/2023.02.16.23286033.

[60] Kini LG, Bernabei JM, Mikhail F, Hadar PN, Shah P, Khambhati AN, et al. Virtual resection predicts surgical outcome for drug-resistant epilepsy. Brain. 2019;142(12):3892-3905. doi:10.1093/brain/awz303.

[61] Wang HF, Scholly J, Triebkorn P, Sip V, Medina Villalon S, Woodman MM, et al. VEP atlas: an anatomic and functional human brain atlas dedicated to epilepsy patients. J Neurosci Methods. 2021;348:108983. DOI: 10.1016/j.jneumeth.2020.108983.

[62] Bressloff PC. Single neuron modeling. In: Bressloff PC, editor. Stochastic Processes in Cell Biology. Lecture Notes on Mathematical Modelling in the Life Sciences. New York: Springer; 2014. p. 3-62. DOI: 10.1007/978-1-4614-8866-8_1.

[63] Ahn S, Jun SB, Lee HW, Lee S. Computational modeling of epileptiform activities in medial temporal lobe epilepsy combined with in vitro experiments. J Comput Neurosci. 2016;40(3):295-313. DOI: 10.1007/s10827-016-0614-8.

[64] Najafi T, Jaafar R, Remli R, Zaidi WAW, Chellappan K. A computational model to determine membrane ionic conductance using electroencephalography in epilepsy. Phys Sci Forum. 2023;5(1):45. DOI: 10.3390/psf2022005045.

[65] Hamill OP, Marty A, Neher E, Sakmann B, Sigworth FJ. Improved patch-clamp techniques for high-resolution current recording from cells and cell-free membrane patches. Pflugers Arch. 1981;391(2):85-100. DOI: 10.1007/BF00656997.

[66] Gandolfi D, Boiani GM, Bigiani A, Mapelli J. Modeling neurotransmission: computational tools to investigate neurological disorders. Int J Mol Sci. 2021;22(9):4565. DOI: 10.3390/ijms22094565.

[67] Lazarewicz MT, Migliore M, Ascoli GA. A new bursting model of CA3 pyramidal cell physiology suggests multiple locations for spike initiation. Biosystems. 2003;67(1-3):129-137. DOI: 10.1016/S0303-2647(02)00071-0.

[68] Markram H, Muller E, Ramaswamy S, Reimann MW, Abdellah M, Sanchez CA, et al. Reconstruction and simulation of neocortical microcircuitry. Cell. 2015;163(2):456-492. DOI: 10.1016/j.cell.2015.09.029.

[69] Breakspear M. Dynamic models of large-scale brain activity. Nat Neurosci. 2017;20(3):340-352. DOI: 10.1038/nn.4497.

[70] Woldman W, Terry JR. Multilevel computational modelling in epilepsy: classical studies and recent advances. In: Validating Neuro-Computational Models of Neurological and Psychiatric Disorders. Cham: Springer International Publishing; 2015. p. 161-188. DOI: 10.1007/978-3-319-20037-8_7.

[71] Naze S, Bernard C, Jirsa V. Computational modeling of seizure dynamics using coupled neuronal networks: factors shaping epileptiform activity. PLoS Comput Biol. 2015;11(5):e1004209. DOI: 10.1371/journal.pcbi.1004209.

[72] Karoly PJ, Kuhlmann L, Soudry D, Grayden DB, Cook MJ, Freestone DR. Seizure pathways: a model-based investigation. PLoS Comput Biol. 2018;14(10):e1006403. DOI: 10.1371/journal.pcbi.1006403.

[73] Junges L, Lopes MA, Terry JR, Goodfellow M. The role that choice of model plays in predictions for epilepsy surgery. Sci Rep. 2019;9(1):7351. doi:10.1038/s41598-019-43871-7.

[74] Cosandier-Rimele D, Merlet I, Bartolomei F, Badier JM, Wendling F. Computational modeling of epileptic activity: from cortical sources to EEG signals. J Clin Neurophysiol. 2010;27(6):465-470. DOI: 10.1097/WNP.0b013e3182005dcd.

[75] Wendling F, Benquet P, Bartolomei F, Jirsa V. Computational models of epileptiform activity. J Neurosci Methods. 2016;260:233-251. DOI: 10.1016/j.jneumeth.2015.03.027.

[76] Jirsa VK, Depannemaecker D, Proix T, et al. Epileptor-2: dynamical inhibition during seizure progression. Proc Natl Acad Sci U S A. 2023;120(37):e2301241120. DOI: 10.1073/pnas.2301241120.

[77] Richardson MP. Large scale brain models of epilepsy: dynamics meets connectomics. J Neurol Neurosurg Psychiatry. 2012;83(12):1238-1240. DOI: 10.1136/jnnp-2011-301944.

[78] Taylor PN, Kaiser M, Dauwels J. Structural connectivity based whole brain modelling in epilepsy. J Neurosci Methods. 2014;236:51-57. DOI: 10.1016/j.jneumeth.2014.08.010.

[79] Sinha N, Dauwels J, Kaiser M, Cash SS, Westover MB, Wang Y, et al. Computer modelling of connectivity change suggests epileptogenesis mechanisms in idiopathic generalised epilepsy. Neuroimage Clin. 2019;21:101842. DOI: 10.1016/j.nicl.2019.101655.

[80] Maliia MD, Koksal-Ersoz E, Benard A, Calas T, Nica A, Denoyer Y, et al. Localization of the epileptogenic network from scalp EEG using a patient-specific whole-brain model. Netw Neurosci. 2025;9(1):18-37. DOI: 10.1162/netn_a_00418.

[81] Miltiadous A, Tzimourta KD, Tzallas AT, et al. Machine learning algorithms for epilepsy detection based on published EEG databases: a systematic review. IEEE Access. 2023;11:130112-130130. DOI: 10.1109/ACCESS.2022.3232563.

[82] Farooq MS, Zulfiqar A, Riaz S. Epileptic seizure detection using machine learning: taxonomy, opportunities, and challenges. Diagnostics (Basel). 2023;13(6):1058. DOI: 10.3390/diagnostics13061058.

[83] Habashi AG, Azab AM, Eldawlatly S, Aly GM, et al. Generative adversarial networks in EEG analysis: an overview. J Neuroeng Rehabil. 2023;20:40. DOI: 10.1186/s12984-023-01169-w.

[84] Martins FM, Gonzalez Suarez VM, Villar Flecha JR, Garcia Lopez B. Data augmentation effects on highly imbalanced EEG datasets for automatic detection of photoparoxysmal responses. Sensors (Basel). 2023;23(4):2312. DOI: 10.3390/s23042312.

[85] Zhang X, Dong S, Shen Q, Zhou J, Min J. Deep extreme learning machine with knowledge augmentation for EEG seizure signal recognition. Front Neuroinform. 2023;17:1205529. DOI: 10.3389/fninf.2023.1205529.

[86] Shah V, von Weltin E, Lopez S, McHugh JR, Veloso L, Golmohammadi M, et al. The Temple University Hospital Seizure Detection Corpus. Front Neuroinform. 2018;12:83. DOI: 10.3389/fninf.2018.00083.

[87] Obeid I, Picone J. The Temple University Hospital EEG Data Corpus. Front Neurosci. 2016;10:196. DOI: 10.3389/fnins.2016.00196.

[88] Guttag J. CHB-MIT Scalp EEG Database (version 1.0.0). PhysioNet. 2010. DOI: 10.13026/C2K01R.

[89] Klatt J, Feldwisch-Drentrup H, Ihle M, et al. The EPILEPSIAE database: an extensive electroencephalography database of epilepsy patients. Epilepsia. 2012;53(9):1669-1676. DOI: 10.1111/j.1528-1167.2012.03564.x.

[90] Brinkmann BH, Wagenaar J, Abbot D, et al. Crowdsourcing reproducible seizure forecasting in human and canine epilepsy. Brain. 2016;139(6):1713-1722. DOI: 10.1093/brain/aww045.

[91] Andrzejak RG, Lehnertz K, Mormann F, Rieke C, David P, Elger CE. Indications of nonlinear deterministic and finite-dimensional structures in time series of brain electrical activity: dependence on recording region and brain state. Phys Rev E. 2001;64(6 Pt 1):061907. DOI: 10.1103/PhysRevE.64.061907.

[92] Kingma DP, Ba J. Adam: a method for stochastic optimization. In: Proceedings of the 3rd International Conference on Learning Representations (ICLR); 2015. arXiv:1412.6980.

[93] Zhu R, Pan W, Liu J, Shang J. Epileptic seizure prediction via multidimensional transformer and recurrent neural network fusion. J Transl Med. 2024;22(1):895. DOI: 10.1186/s12967-024-05678-7.

[94] Liu X, Li C, Lou X, Kong H, Li X, Li Z, et al. Epileptic seizure prediction based on EEG using pseudo-three-dimensional CNN. Front Neuroinform. 2024;18:1354436. DOI: 10.3389/fninf.2024.1354436.

[95] Li Z, Hwang K, Li K, Wu J, Ji T. Graph-generative neural network for EEG-based epileptic seizure detection via discovery of dynamic brain functional connectivity. Sci Rep. 2022;12:18998. DOI: 10.1038/s41598-022-23656-1.

[96] Dallmer Zerbe I, Khambhati AN, Davis KA, et al. Computational modeling allows unsupervised classification of epileptic brain states across species. Sci Rep. 2023;13:14063. DOI: 10.1038/s41598-023-39867-z.

[97] Kashyap N, Tripathi M, Bhattacharya S, Pandey S. Unsupervised pre-ictal state identification in human EEG using variational autoencoders. Front Neurosci. 2022;16:1045732. DOI: 10.3389/fnins.2022.1045732.

[98] Dubcek T, Levenstein D, Buzsaki G, et al. Personalized identification, prediction, and stimulation of neural oscillations via data-driven models of epileptic network dynamics. arXiv. 2023:2310.13480. DOI: 10.48550/arXiv.2310.13480.

[99] Weiss SA, Banks GP, McKhann GM, et al. Localizing epileptogenic regions using high-frequency oscillations and machine learning. Biomark Med. 2019;13(5):409-418. DOI: 10.2217/bmm-2018-0335.

[100] Li Z, Zhao B, Hu W, et al. Machine learning-based classification of physiological and pathological high-frequency oscillations recorded by stereoelectroencephalography. Seizure. 2023;113:58-65. DOI: 10.1016/j.seizure.2023.11.005.

[101] Monsoor T, Zhang Y, Daida A, et al. Optimizing detection and deep learning-based classification of pathological high-frequency oscillations in epilepsy. Clin Neurophysiol. 2023;154:129-140. DOI: 10.1016/j.clinph.2023.07.012.

[102] Gabeff V, Teijeiro T, Zapater M, Cammoun L, Rheims S, Ryvlin P, et al. Interpreting deep learning models for epileptic seizure detection on EEG signals. Artif Intell Med. 2021;117:102084. doi:10.1016/j.artmed.2021.102084.

[103] Saglio CBL, Pagani S, Corti M, Antonietti PF. A high-order discontinuous Galerkin method for the numerical modeling of epileptic seizures. arXiv. 2024:2401.14310. DOI: 10.48550/arXiv.2401.14310.

[104] Proix T, Jirsa VK, Bartolomei F, Guye M, Truccolo W. How do parcellation size and short-range connectivity affect dynamics in large-scale brain network models? Neuroimage. 2016;142:135-149. doi:10.1016/j.neuroimage.2016.06.016.

[105] Hesthaven JS, Warburton T. Nodal Discontinuous Galerkin Methods: Algorithms, Analysis, and Applications. New York (NY): Springer; 2008. DOI: 10.1007/978-0-387-72067-8.

[106] van der Vlag MA, Hernando JB, Woodman MM, et al. RateML: a code generation tool for brain network models. Front Netw Physiol. 2022;2:826345. DOI: 10.3389/fnetp.2022.826345.

І. Підвальний [1], О. Судаков[2]

[1] Київський національний університет імені Тараса Шевченка, Київ, Україна
[2] Технічний центр, Національна академія наук України, Київ, Україна

ФІЗИЧНІ ПРОЦЕСИ В БІОЛОГІЧНИХ НЕЙРОННИХ МЕРЕЖАХ ПІД ЧАС ЕПІЛЕПТИЧНИХ НАПАДІВ: ОГЛЯД МОДЕЛЕЙ

Анотація: У статті наведено огляд підходів до виявлення та прогнозування епілептичних нападів за електроенцефалографічними та інтракраніальними електроенцефалографічними сигналами з акцентом на фізично обґрунтоване моделювання нейронів і нейронних мереж. Актуальність теми зумовлена тим, що сучасні алгоритми машинного та глибокого навчання демонструють високі показники класифікації, однак їх практичне застосування обмежується міжпацієнтною варіабельністю, дефіцитом розмічених даних, залежністю від попередньої обробки, проблемою хибних тривог і недостатньою інтерпретованістю ознак. У роботі систематизовано основні класи моделей, які застосовуються в обчислювальній епілептології: низьковимірні феноменоло-

гічні моделі, біофізичні моделі окремих нейронів і мембранних процесів, мережеві та багатомасштабні підходи, методи машинного навчання, а також високоточні чисельні схеми для великомасштабного моделювання. Показано, що біофізичні та багатомасштабні моделі забезпечують глибше пояснення іонних, синаптичних і мережевих механізмів епілептиформної активності, але потребують великої кількості параметрів і значних обчислювальних ресурсів. Натомість феноменологічні моделі, зокрема Epileptor та Epileptor-2, забезпечують раціональний компроміс між швидкістю моделювання, керованістю параметрів і здатністю відтворювати перехід між нормальним, іктальним та постіктальним режимами. Обґрунтовано, що такі моделі доцільно використовувати не як заміну реальних ЕЕГ/iEEG-даних, а як джерело контрольованих синтетичних сигналів для калібрування ознак, перевірки стійкості класифікаторів і аналізу фізичного походження інформативних характеристик. Запропоновано розглядати модельно згенеровані сигнали як допоміжний рівень між експериментальними даними та алгоритмами машинного навчання. Такий підхід може підвищити відтворюваність досліджень, покращити інтерпретованість діагностичних ознак і створити основу для подальшої розробки компактних систем виявлення та прогнозування епілептичних нападів.

Ключові слова: електроенцефалографія; іктальний перехід; синтетичні калібрувальні дані; феноменологічна динаміка; Epileptor-2; біфуркаційні механізми; мультимасштабний конектом; виділення ознак; калібрування класифікатора; трансферна валідація.