Modelling Absence Epilepsy Seizure Data in the NeuCube Evolving Spiking Neural Network Architecture

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***Abstract*—Epilepsy is the most diffuse brain disorder that can affect people's lives even on its early stage. In this paper, we used for the first time the spiking neural networks (SNN) framework called NeuCube for the analysis of electroencephalography (EEG) data recorded from a person affected by Absence Epileptic (AE), using permutation entropy (PE) features. Our results demonstrated that the methodology constitutes a valuable tool for the analysis and understanding of functional changes in the brain in term of its spiking activity and connectivity. Future applications of the model aim at personalised modelling of epileptic data for the analysis and the event prediction.**

***Keywords*—Spiking Neural Networks, EEG, NeuCube, Epilepsy, Childhood Absence Seizures.**

I. Introduction

Epileptogenic processes are hyper-syncronization of the electrical neural activity. It is still unknown the cause that produces them, however, there is a hypothesis that they may occur in different areas of the brain indiscriminately. The critical area associated with the epileptogenetic event is called epileptogenetic zone. This is responsible of recruiting other areas until the brain triggers the seizure. This process can be seen as a network-event rather than a local event. Thus, the problem to localise and study the epileptogenetic zone is of great interest to neuroscientists.

Spatio-temporal brain data (STBD) and especially EEG data is widely used to record changes of brain activity during seizures at a millisecond time scale. It allows us to obtain knowledge about frequency, time and space of the epileptogenetic events. When a subject experiences a seizure, he/she needs to undergo recording of several EEGs. This implies inpatient or day-hospital care and, if epilepsy is diagnosed, the patient must undergo further several EEG recordings in order to assess the evolution of the disease and to monitor the effects of the treatment. Once the EEG has been recorded (which can last for minutes to days) a careful review of the entire recording is needed, in order to detect the presence of critical events and to come up with a diagnosis. In clinical practice, while reviewing the EEG, the neurologist manually scrolls through the EEG and visually detects seizures (ictal states) and seizure-free intervals (inter-ictal states), so that he/she can evaluate the events locally in space, electrode per electrode. The neurologist usually visualizes only 20 seconds at a glance, scrolling ahead till the end of the recording. Thus working with EEG data means handling "big data" and all the technologies that can support the neurologist in dealing with this huge amount of data, will certainly improve epileptic people's quality of life as well as facilitate the neurologist's work.

Furthermore, many results reported in the literature suggest that seizures are not completely random and unpredictable events, which means that they are part of a more complex network phenomenon, a sort of "epileptogenic process" that, for unknown reasons, arises, evolves, and finally results in a seizure. Based on such hypothesis, one should wonder *when*, *where* and *why* abnormalities in the electrical activity arise in the brain and *how* they evolve, recruiting other areas. So far, no tool has been developed in order to monitor such mechanisms which, if discovered and explored, would allow for a much deeper understanding of the pathology that could lead to novel therapeutic perspectives. The core of such a tool would be an algorithm, meant for the extraction of mathematical

descriptors from EEG signals, which would be able to follow the evolution of the dynamics of cerebral electrical activity and to identify the transition from the inter-ictal stage to the pre-critical stage, thus detecting possible impending seizures (early seizure detection).

Aiming at this purpose, in this study we proposed a spatial-temporal model to study the development of seizures, in order to: automatically mark the critical events on the recording; provide deep and novel diagnostic information about brain networks (off-line utility); and to provide a warning tool in case of high probability of an impending seizure (on-line utility).

In already published studies [1], [2], we analysed spatial-temporal EEG signals based on ordinal measures of patterns through PE. There, long EEG recordings (minutes to hours before seizure onset) have been analysed in order to check whether the epileptogenic process in absence seizures actually corresponds to the model of a "jump" transition of the underlying dynamical system or a gradual transformation is detectable in advance. PE was used to convert real EEG signals into motifs neglecting amplitude of signals and excluding any dependence on the effect of the reference electrode.

In this paper, we analysed the dynamics of the epileptic events through the PE topographies by means of an evolving spatio-temporal data machine (eSTDM) based on neuromorphic, brain-like information processing principles. In particular, the methodology is based on the NeuCube framework [3]. This model utilises SNN as major processing modules. Data can be classified and the SNN cube (SNNc) analysed in term of connectivity and spiking activity generated during the learning process. This helps us to study and reveal spatio-temporal patterns retained in the data and to localise them in specific areas of the brain.

The paper is constructed in the following way: section II presents the problem and the data available; section III describes the NeuCube-based methodology used for the study; section IV reports the results and the conclusions; and section V proposes future work that we are currently undertaking.

II. Epileptic EEG Analysis with SNN

A. Absence Seizures

We based our study on Childhood Absence Epilepsy (CAE) data, a common idiopathic generalized epilepsy syndrome [4], [5]. CAE is usually present in children between age of 4 and 10 years, peaking at age 6-7 years. A strong genetic predisposition is evident, with occurrence more often in girls than in boys. The very frequent absences (several to hundreds a day) exert a negative impact on an otherwise normal child. Untreated children often exhibit learning and attention difficulties because of their alterations of consciousness. The pediatric neurologists main objective is to neutralize all absences as long as the side effects.

B. EEG data Description

Standard EEG recording from a patient, diagnosed with CAE, was used for training and testing. The patient was a young girl and her age was 9. The EEG was acquired using a 16-channel device (Fp1, Fp2, F3, F4, F7, F8, T3, T4, T5, T6, C3, C4, P3, P4, O1, O2) according to the international 10/20 system with Cadwell Easy II (Cadwell Laboratories, Inc., Kennewick, WA). All channels were filtered with a pass band of 0.53-25 Hz, and digitised at a rate of 200 Hz. In total, 8 paroxysms longer than 2 seconds were identified by a board-certified clinical neurophysiologist. In total, 15.8 minutes of electroencephalogram data were recorded. All data analysis was performed using MATLAB (The MathWorks, Inc., Natick, MA).

C. Permutation Entropy

The *n*-channels EEG recording was processed by means of sliding temporal windows. The EEG samples were buffered in 5 seconds non-overlapping windows (since the sampling rate is *200Hz*, a window includes *N=1000* EEG samples), then PE was estimated, channel by channel and window by window. Each EEG time window includes *n* time series, where *n* is the number of EEG channels. This window is stored in the computer as a *nxN* matrix. Within each window, a sample of PE per channel is computed and the *n* values are arranged in a *nx1* PE vector, therefore, a *nxN* EEG matrix corresponds to a *nx1* PE vector thus a compressed temporal representation of the original time series is produced. In order to calculate PE, each time series *x* was mapped into a *m*-dimensional space, with *m* being the embedding dimension and *L* being the time lag. Vectors X_t were constructed selecting *m* equally spaced samples from *x*, starting from time point *t*:

$$X_t = [x(t), x(t+L), ..., x(t+(m-1)L)]^T \quad (1)$$

The values of X_t are reshaped in an increasing order, the time points are renamed yielding Xr_t, a reshaped version of X_t: $Xr_t = [x(t+(t_1-1)L), x(t+(t_2-1)L), ..., x(t+(t_m-1)L)]^T$ Therefore, each vector X_t can be considered uniquely mapped onto a symbol vector $\pi = [t_1, t_2, ..., t_m]$. The vector π is a sequence of time points, hence a symbol. The frequency of occurrence of each possible π is indicated as $p(\pi)$, which represents the frequency of occurrence of the specific vector π in the time series under analysis, normalized by $N-(m-1)L$ where *N* is the number of samples of the time series *x*. PE is finally computed as:

$$H(m) = -\sum_{i=1}^{m!} p(\pi_i) ln(p(\pi_i)) \quad (2)$$

Where *log* is the natural logarithm and *m!* is the number of the possible permutations. Since $H(m)$ can maximally reach $ln(m!)$, PE is generally normalized as:

$$H_n(m) = -\frac{\sum_{i=1}^{m!} p(\pi_i) ln(p(\pi_i))}{ln(m!)} \quad (3)$$

D. The NeuCube SNN Architecture

The eSTDM NeuCube was first introduced in [3], [6] and then developed in [7], [8]. It is a multi-modular computer systems designed to deal with large and fast spatio-/spectro-temporal data. It is inspired by the main biological principles that regulate learning and memory dynamics of spiking neurons that make up its network. A block digram of the eSTDM NeuCube is depicted in Fig. 1. It consists of the following modules:

- Input information encoding module.
- Input mapping module.

- 3D SNN module (the SNNc).
- Output classification/regression module.
- Gene regulatory network (GRN) module (Optional).
- Parameter optimisation module.
- Visualisation and knowledge extraction module.

The input module transforms the raw data vectors into trains of spikes. The encoded STBD is then presented to the main module, the 3D SNNc, into certain areas of the cube that have the same 3D coordinates as the data source locations during collection.

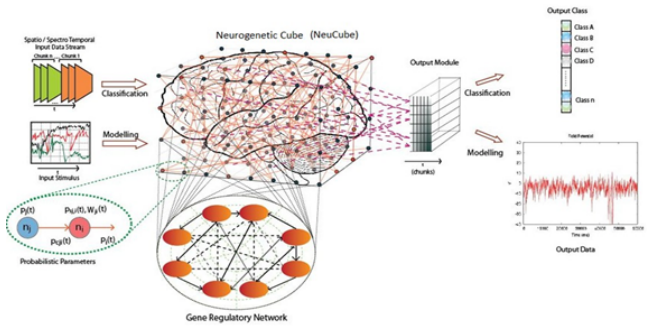

Fig. 1. A general NeuCube architecture of eSTDM for STBD modelling with its three main modules: input data encoding module; a 3D SNNc module; an output evolving classification module [3]. An optional Gene Regulatory Network (GRN) module can be incorporated if gene information is available. The spiking neurons can be implemented as the simple leaky integrate and fire model or probabilistic models (shown in the lower left section).

NeuCube has already demonstrated its ability to classify and extract new knowledge from both EEG [9]–[11] and functional magnetic resonance imaging (fMRI) [12] cognitive data and to other type of spatio-temporal data for personalised disease prognosis [13]. This paper contributes to the set of methods related to the NeuCube with the introduction of a new methodology for the analysis of spiking activity and connectivity of EEG data recorded from a CAE patient, using the information available from the PE features.

III. The Proposed NeuCube Methodology for the Study of CAE PE Data

The NeuCube-based methodology schematized in Fig. 2 consists of following procedures and parameters:

1) Every data vector was transformed into a spike train using the address event representation (AER) algorithm [14]–[16]. This is calculated as a bi-directional threshold (AER_{thr}), which is applied to the signal $x(t)$ as following:

$$AER_{thr} = \mu + s\ \sigma \tag{4}$$

where μ is the mean of the differential signal with respect to time, $x(t) = \{x_{t_2} - x_{t_1},\ x_{t_3} - x_{t_2}, \ldots,\ x_{t_n} - x_{t_{n-1}}\}$, calculated by using all samples; σ is its standard deviation; s is a scale parameter of σ. The AER_{thr} is used to generate two types of spike sequences, a positive spike train corresponding to the signal increment, which is mapped to a specific input neuron in the SNNc; a negative spike train, corresponding to the signal decline, which is mapped into another input neuron of the SNNc that is placed in the same position as the positive one.

2) Then, the input spike sequences are presented to an evolving brain-mapped network of spiking neurons - the SNNc. Each of these neurons represented the centre coordinates of a 1 cm^3 area of a human brain template known as the Talairach Atlas [17]–[19]. Thus, the SNNc reflects the number of input variables, the Brodmann area associated with them and the size of the data available.

3) The neurons of the cube are initialised as a "small-world" (SW) connected networks and their initial connections weights are calculated as the product of a random number $[-0.1, +0.1]$ and the multiplicative inverse of the Euclidean distance $d(i,j)$ between a pre-synaptic i and a post-synaptic neuron j (calculated according to their (x, y, z) coordinates). 20% of these weights are randomly selected to be negative (inhibitory connections weights), while 80% are positive (excitatory connections weights). The $d(i,j)$ also depends on a distance threshold D_{thr} calculated as:

$$D_{thr} = max\ (d(i,j))\ p \tag{5}$$

where p is the SW connectivity parameter.

4) The neurons of the cube are modelled as leaky integrate and fire (LIF) neurons [20]. If an initial connection $c_{i,j}$ between two neurons is established, then, the action potential v_j of a neuron j increases according to the time and the order of the incoming spike S_i from neuron i. The v_j increases until a firing threshold Θ, then, it resets and an output spike S_j is emitted.

$$S_i = \begin{cases} 1 & \mathrm{v_j} \geq \Theta \\ 0 & \text{otherwise} \end{cases} \tag{6}$$

The membrane potential will keep to zero for the length of its refractory time (r). Between spikes, the membrane potential leaks according to the potential leak rate (l).

$$v_j(t) = \begin{cases} v_j(t-1) + w_j & \mathrm{r} = 0 \\ v_j(t-1) - l & \text{otherwise} \end{cases} \tag{7}$$

5) The SNNc is trained in an unsupervised mode using the spike timing dependant plasticity (STDP) [21] learning rule. This Hebbian rule describes the connection between two neurons as stronger as their activation persists and repeats. This is implemented as following:

$$w_j(t) = \begin{cases} w_j(t-1) \pm \alpha/\Delta t & \mathrm{t_j} \neq \mathrm{t_i} \\ w_j(t-1) & \mathrm{t_j} = \mathrm{t_i} \end{cases} \tag{8}$$

where α is the STDP learning rate, Δ_t is the time elapsed since the last spike was emitted by neuron j. If a neuron i fires before a neuron j then, its weight

$w_{j,i}$ increases, otherwise, it decreases.

6) The output classifier is trained in a supervised mode using dynamic evolving SNN (deSNN) [22] algorithm, which combines the rank-order (RO) learning rule [23] and the STDP [21] rules. According to this algorithm, every trained sample is associated to an output neuron that is connected to every other neuron of the cube. In case of the NeuCube model, the inputs to the deSNN classifier are all neurons of the SNNc. Initially, the connection weights $w_{i,j}$ between the input neuron i and the output neuron j are all set to zero. Then, according to the rank-order (RO) learning rule, they are computed as following:

$$w_{i,j} = mod^{order(i,j)} \tag{9}$$

where mod is a modulation factor and $order(i,j)$ is the order of the first incoming spike.
The new connection weights will be modified according to the spike driven synaptic plasticity (SDSP) learning rule. Implemented as:

$$w_{i,j}(t) = \begin{cases} w_{i,j}(t-1) + drift & \mathrm{S_j(t) = 1} \\ w_{i,j}(t-1) - drift & \mathrm{S_j(t) = 0} \end{cases} \tag{10}$$

where $drift$ is a parameter used to modify the connection weights and $S_i(t)$ represents the occurrence of the spikes arriving from neuron i at a time t after the first one was emitted.

7) The classification results are evaluated using repeated random sub-sampling validation (RRSV) or leave one out cross validation (LOOCV).

8) Steps (3) to (7) are repeated using different parameter values in order to optimize the classification output. The best performing model can then be recorded for further uses.

9) The trained SNNc is visualised, its connectivity and the dynamic spiking activity are analysed for a better understanding of the data and the brain processes that generated it including changes of brain functionality across conditions and subject groups.

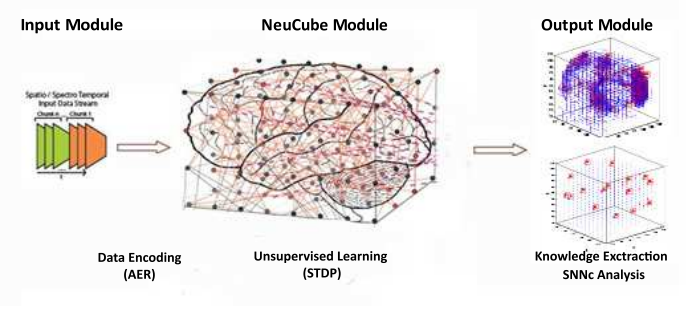

Fig. 2. A graphical representation of the NeuCube-based methodology used for PE data modelling and brain connectivity analysis.

IV. Results and Discussion

For our experimental case study, data was first classified, to establish the effectiveness of the PEs as classification features. Then, the SNNc was visualised and analysed in a step-wise mode to study the activity and connectivity generated during learning.
As shown in Fig. 3, the 187 PE vectors calculated from the 16-channel EEG data corresponded to 7 ictal (PE 2-5, 25, 34-41, 63-67, 95-99, 129-132 and 179-183) and 8 equivalent interictal states. This data was propagated through the cube generating a spiking activity according to the time and frequency of the encoded spike trains. Thus, a trained NeuCube represented a persons dynamic model of epileptic event progression over a short period of time.

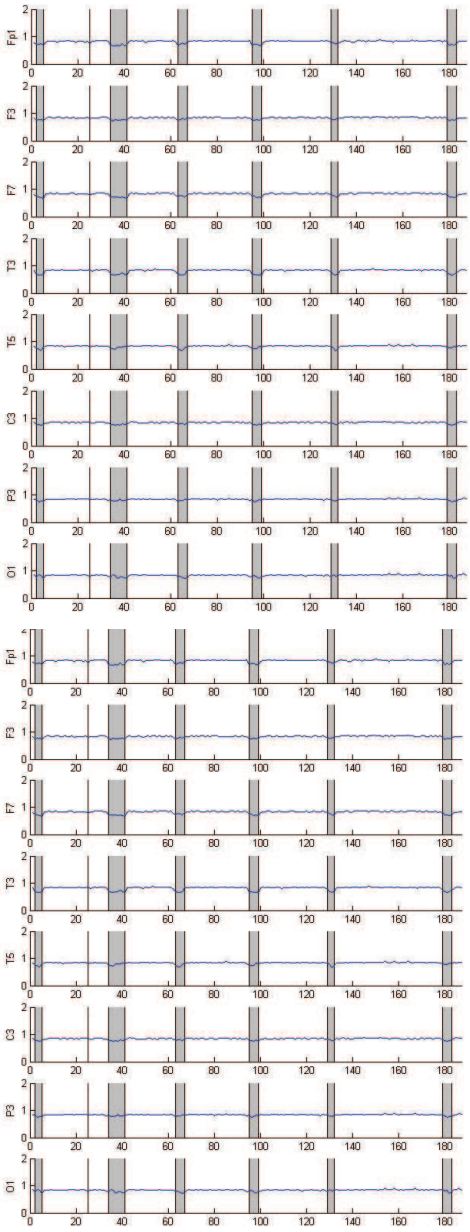

Fig. 3. Sequence of PE obtained from the 16-channel EEG data. In grey colour are the PE corresponding to the ictal state, while the other PE are associated to the interictal states.

A. Classification

For the classification experiments, the 187 PE levels were divided into the two respective classes. Excluding the samples containing just one of the PE topography, we obtained 6 samples for class 1 (ictal state) and 7 samples for class 2 (interictal state).
The encoded spike trains, obtained from the 187 PE vectors, were entered into a 3D grid of 1471 LIF neurons by entering them to the 16 corresponding brain-mapped input neurons. Data was first learnt in the SNNc in an unsupervised way and then classified *via* supervised learning method in a trained deSNN classifier. The classification results were evaluated using LOOCV and they are reported in table I.

TABLE I. NEUCUBE CLASSIFICATION RESULTS EXPRESSED AS ACCURACY PERCENT.

NeuCube Classification Results	
Ictal State	100
Interictal State	71
Overal Accuracy	**86**

The high classification accuracy (100% for class 1 and 71% for class 2) obtained *via* the NeuCube methodology proved the model ability to manifest this difference and also that the two classes were in fact two distinguishable groups when using PE values as features. Table II reports the NeuCube parameters used to obtain these results.

TABLE II. PARAMETER SETTING USED TO OBTAINED THE RESULTS REPORTED AS NEUCUBE CLASSIFICATION ACCURACY.

SNNc Parameter Setting		
Threshold of Firing: 0.5	Potential Leak Rate: 0.002	STDP: 0.5
Refractory Time: 2.5	SW distance: 0.15	AER Threshold 0.003
deSNN mod: 0.4	deSNN drift: 0.25	Time to Train: 1

These parameters were found after running a grid search in which 100 SNNc network configurations were evaluated regarding their test accuracy using RRSV (50% of the data was used for training and 50% for testing).

B. SNNc Analysis

In this case, the 187 PE sequences were used to train a NeuCube model in a step-wise manner. We looked for the first time at the dynamic of both spiking activity and connectivity during learning using the information available from the PE topographies. This threw more light on the functional changes in the brain provoked by the epileptic event and more importantly it helped to locate where these changes took place.
To analyse and visualise the cube activity, we have used again a 3D SNNc of 1471 brain-mapped spiking neurons. The data was learnt in the SNNc in an unsupervised way and then the SNNc activity was analysed and interpreted for a better understanding of the data and to identify differences between brain states.
Figure 4 show the NeuCube evolution over the 187 time points obtained during STDP learning. Table III reports the parameters settings used to obtain these results.
On the entire series of PE, we are interested in the spiking activity and connectivity generated by each of the 6 ictal states and the 7 relative interictal states. The first two sequences of

TABLE III. PARAMETERS SETTINGS USED FOR THE STEP-WISE SNNC ANALYSIS.

SNNc Analysis Parameter Setting		
Threshold of Firing: 0.5	Potential Leak Rate: 0.002	STDP: 1
Refractory Time: 2	Long Dist. Possibility: 0.01	Weight Threshold 0.08

PE topographies associated with a seizure (PE 2-5 and PE 25) do not show a particular change in terms of connectivity or spiking activity. The third series of PE related to an epileptic event (PE 34-41) represents the longest sequence of PE topographies. Here, the F4 electrode appears to be the focal point in term of enhanced connectivity. Also, new connections are formed in the (right) frontal polar area, electrode Fp2. The subsequent ictal state (PE 63-67) provokes the establishment of new connections in the right hemisphere especially in the anterior region of the frontal area, but also by the left hemisphere, at F7, F3 and FP1 positions. By the next sequence of ictal PE topographies (PE 95-99), the new connections have now consolidated and also new are formed by the (right) occipito-parietal area, channels O2 and P4. By the next ictal section (PE 129-132), most of the connections generated by the (right) frontal and occipito-parietal cortex and the (left) centro-temporal area keep consolidating, while the central regions are not affected. The last series of PEs associated with a seizure (PE 179-183), clearly show that most of the connections in the SNNc model are formed around the input neurons especially in the right frontal portion of the brain, while no connections are evolved in the central line at all.
Given a certain electrode and its PE level, we did not expect high PE to be linked to high degree of connections, we only expect PE to match the complexity degree of the electrical activity of the cerebral region covered by that electrode. PE levels can give us information that are local in space, depicting the topography over the scalp. It is a way to see which electrode showed similar PE levels, but we obtained information about brain connectivity only through our proposed NeuCube model.
Neurologists address the frontal-temporal regions as “critical” in absence seizure patients and this seems to be in line with our modelling results and in particular with the ictal states associated to the PE 67, 99, 132, 183.

V. CONCLUSION AND FUTURE WORK

The goal of the proposed study has been to develop a personalised model able to properly learn over time epileptic events in terms of space and time, so that the information can be dynamically visualised and analysed and possibly the epileptogenetic event predicted.
Our results demonstrated that the methodology constitutes a valuable tool for epileptic EEG data analysis and understanding. However, more extensive evidence is needed to establish the feasibility of a purely data driven diagnosis method for CAE diagnosis. So far, our results are promising and NeuCube is planned to be used for molecular and genetic analysis of the disease and as a personalised model for the understanding of functional changes in the brain and for the prediction of epileptic events from new data.

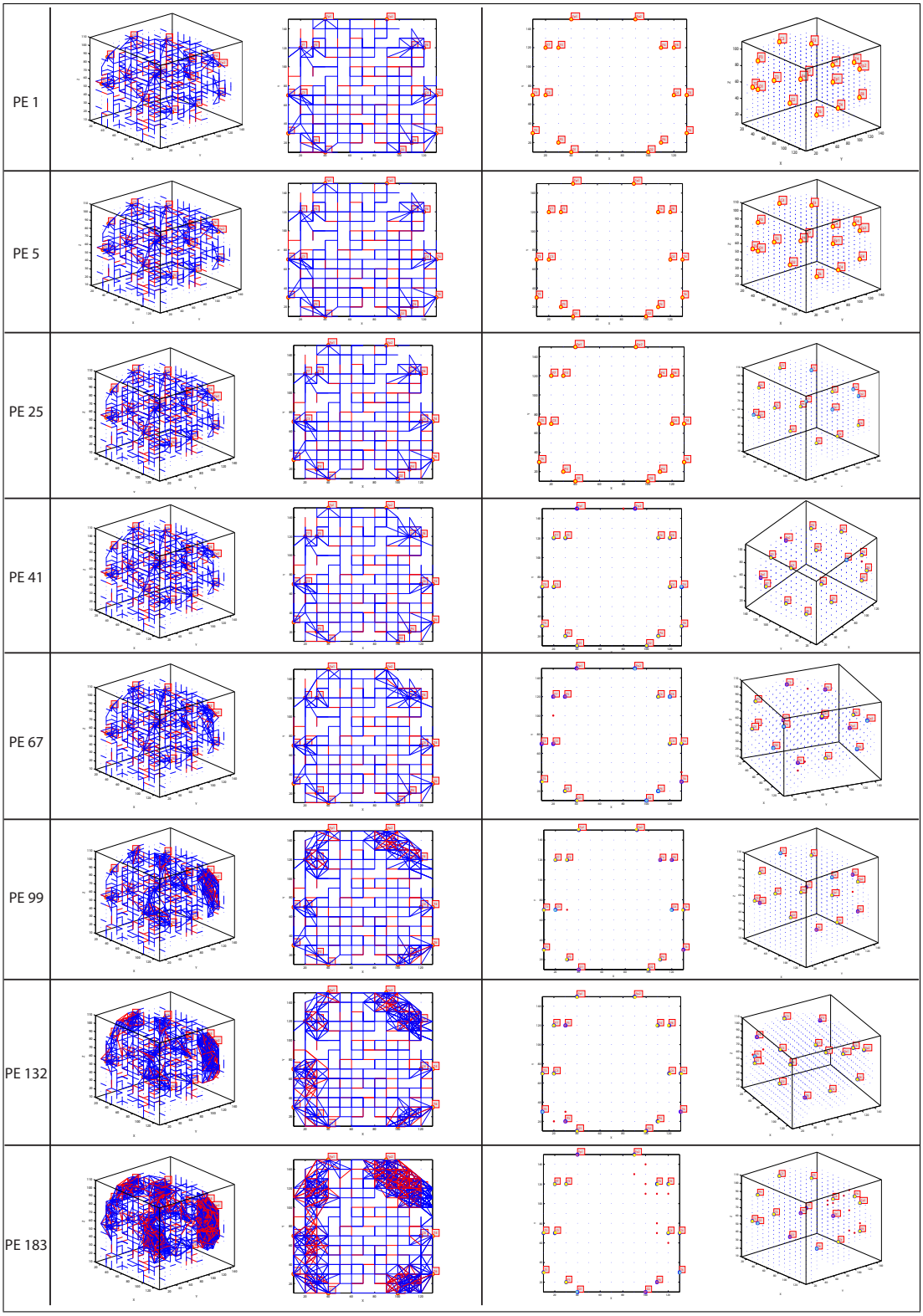

Fig. 4. Step-wise SNNc activity during STDP learning on the 187 PE topographies. The figure shows the evolution of connectivity of the 3D SNNc and its 2D plane. The top image presents the SNNc connectivity obtained from the first PE associated to the initial interictal state (PE 1). The subsequent SNNc images result from training the cube with one PE associated to the seven following ictal events (PE 5, 25, 41, 67, 99, 132 and 183). (left) Blue lines are positive connections (excitatory synapses), while red lines are negative connections (inhibitory synapses). The input neurons are identified by their labels corresponding to the 16 EEG channels. (right) The active spiking neurons are identified in red, while in blue the inactive neurons. Also, in magenta are the positive spiking input neurons and in cyan the negative spiking inputs. Yellow identifies the absence of a spike in the input neurons.

NEUROGENETIC NEUCUBE FOR THE ANALYSIS OF NEURORECEPTOR ACTIVITY RELATED TO CAE

A neurogenetic model of a neuron is proposed and studied in [24], [25]. It utilises information about how the main neuroreceptors, and the genes that expressed them, affect the spiking activities of a neuron. This model can be also optimised using a Gene/Protein Regulatory Network (GRN), controlling the dynamic interactions between genes/proteins over time, which affects the post synaptic potential of the network's neurons.

In this work, we used Hebbian learning rules to adapt the synapse amongst neurons, specifically the STDP as a way of competition for control of the timing of postsynaptic action potentials. However, the synapses adaptation can also be implemented through dynamic mechanisms involving the growth or decay of some neuroreceptors. They affect the spiking activity of a neuron such as *fast excitation*, *fast inhibition*, *slow excitation*, and *slow inhibition*. In turn, each neuroreceptor is affected by other neuroreceptors, therefore, the contribution of each neuroreceptor g_r to a neuron synapse in a time t might be computed as:

$$g_r(t) = g_r(t-1) + f(g_1(t-1), g_2(t-1), ..., g_n(t-1)) \quad (11)$$

where $g_i(t-1)$ is the last state of an ith receptor, and the function f is the positive/negative contribution to g_r. Such that the synaptic weight $g_{w_{ij}}$ between a neuron n_i and a neuron n_j is calculated as the difference of the sum of the excitatory receptor gains g_e and the sum of the inhibitory receptor gains g_h:

$$g_{w_{ij}} = \sum_{i=1}^{n} g_e - \sum_{i+1}^{n} g_h \quad (12)$$

This model, called Neurogenetic Neucube (NeuCube$_{NG}$), has been proposed in [26]. It can automatically balance the synaptic strengths making postsynaptic firing irregular but sensitive to presynaptic potentials such as the STDP like rules.

We plan to apply the NeuCube$_{NG}$ to CAE data to study how the regulation of GABA-mediated inhibitory mechanisms, which are believed to be involved in the neural hypersyncronization responsible of type of epilepsy, can affect the post synaptic potential of a neuron [27]. This type of mechanisms are mediated by pharmacological treatments [28] and we believe the NeuCube$_{NG}$ could be of great help for clinicians to analyse the possible changes in neural connectivity.

NEUCUBE FOR THE PREDICTION OF EPILEPTIC SEIZURES

NeuCube can be used also for dynamic predictive modelling based on neuromorphic learning. The acquisition of more CAE data and their comparison to healthy subjects can be used to create such model that learns the progression of the epileptic events of a person over time. The brain-like mapping of the cube can be used to study the specific characteristics of the epileptic events, the area of the brain where the event occurs and eventually to predict future epileptogenetic events based on the initial information.

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