Can We Identify the What, When and Where of Sensorimotor Activity in EEG Recordings?

Marije ter Wal (m.j.terwal@tudelft.nl)
BioMechanical Engineering, ME, TU Delft, Mekelweg 2, 2628 CD Delft, NL

Ioannis Kyriazis (I.Kyriazis@tudelft.nl)
BioMechanical Engineering, ME, TU Delft, Mekelweg 2, 2628 CD Delft, NL

Alfred Schouten (A.C.Schouten@tudelft.nl)
BioMechanical Engineering, ME, TU Delft, Mekelweg 2, 2628 CD Delft, NL

Frans van der Helm (F.C.T.vanderHelm@tudelft.nl)
BioMechanical Engineering, ME, TU Delft, Mekelweg 2, 2628 CD Delft, NL

Mark van de Ruit (M.L.vandeRuit-1@tudelft.nl)
BioMechanical Engineering, ME, TU Delft, Mekelweg 2, 2628 CD Delft, NL

Abstract

The origin of sensorimotor EEG signatures remains to be elucidated. Here, we present a model that spans the gap from neuronal spiking to the EEG and is based on known cortical anatomy and physiology. We use this model to simulate EEG signals, in order to enhance the understanding of human EEG recordings during rest and motor tasks.

Keywords: sensorimotor; S1; M1; EEG; Izhikevich model; point neuron; beta bursts

Introduction

Electroencephalography (EEG) is a non-invasive and cost-effective neuroimaging technique that is readily available in both healthy and patient populations. EEG has a high temporal resolution, but low spatial discriminability, due to low sampling (typically 32-128 electrodes) and the effects of volume conduction. Its limited spatial resolution has severely constricted the contribution of EEG to dynamic network identification. In other words, the contribution of EEG in identifying the what, when and where of cortical circuits involved in cognition and disease is still in need of improvement. To improve the interpretability of EEG data, we need a better understanding of the contribution of low-level cortical activity to the signals recorded at the scalp.

Modelling EEG signals

To this end, we have developed a model of human sensorimotor cortex that bridges the gap by simulating the activity of single cells (Figure 1A), that were connected to each other via model synapses into small circuits that represent the cortical layers, and which were in turn connected into columns that represent primary sensory cortex (S1) and sensory motor cortex (M1) (Figure 1B). We simulated the EEG based on the activity in these model circuits, allowing us to compare the activity of single cells and neural ensembles to EEG signals recorded during rest and task.

Neuron Models

Individual cells were modelled as Izhikevich point neurons (Izhikevich, 2003). We used three different cell types (Figure 1A): one excitatory type (regular spiking), and two types of inhibitory cells: fast spiking model neurons to represent Parvalbumin positive (PV+) cells, and low threshold spiking model neurons to represent somatostatin positive (SOM+) cells.

As we aimed to simulate human EEG data, the excitatory cells made up 70% of the population

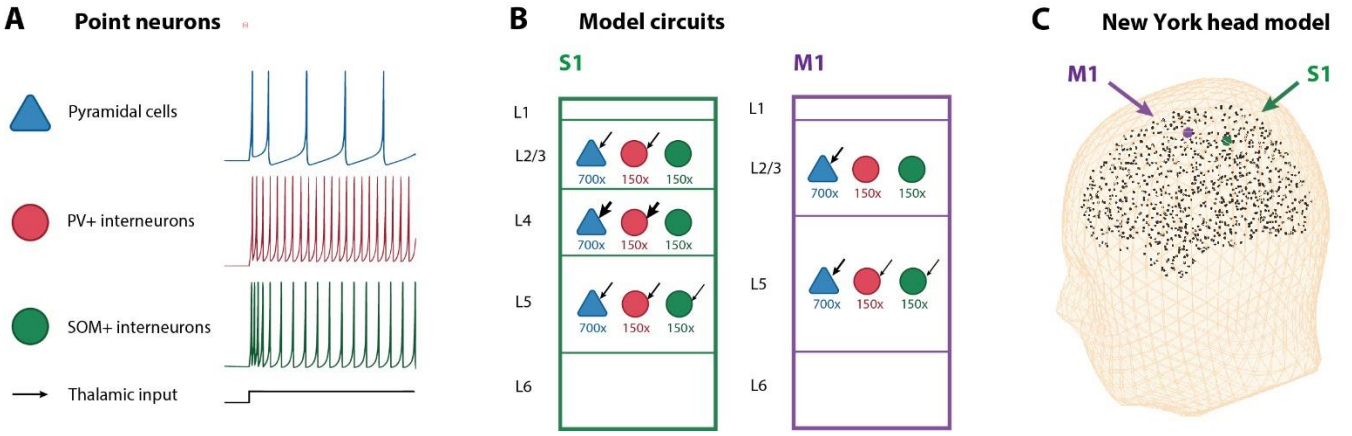

Figure 1.Model overview. A: Dynamics of the cell types in the model; B: Circuit overview of S1 and M1 models with thalamic input (black) per cell type (see A); C: Dipole locations used for computation of the EEG.

(Bakken et al., 2021; Shapson-Coe et al., 2024). Excitatory regular spiking cells represent stellate cells in L4 of S1, and pyramidal cells in all other simulated layers. The inhibitory cell types each made up half of the remaining population, mimicking their prominent contribution in cortical circuits (Bakken et al., 2021).

Inputs and Synaptic Connections

Model neurons were connected to each other via model AMPA and GABA synapses. Synaptic connections were made randomly, following connection probabilities reported in the literature (amongst others: Beierlein et al., 2003; Hooks et al., 2013; Kätzel et al., 2011; Lefort et al., 2009; Markram et al., 2015). Connections between S1 and M1 were excitatory only. Neurons received excitatory thalamic inputs modelled as random or modulated spike trains (i.e. pulsed, sinusoidal).

Simulating the EEG

To simulate EEG signals we calculated the amplitudes of two dipoles: one for M1 and one for S1 (Figure 1C), while assuming correlated noise for the rest of the brain. S1 and M1 dipole moments were based on a scaled and time-shifted combination of the AMPA and GABA currents into the pyramidal cells in L2/3 and L5, following (Martínez-Cañada et al., 2021). Using the New York head model (Huang et al., 2016), we then computed the signals at 231 EEG electrodes.

Model Validation and Predictions

By comparing the model EEG signals to EEG signals from healthy volunteers recorded during rest, active and passive movement, we aim to: 1) provide pointers to improve the processing and network analysis of EEG recordings and 2) aid the interpretation of EEG signals originating from sensorimotor cortex. This will allow us to gain more insight into the linear and non-linear interaction that give rise to recorded EEG activity and thus improve our understanding of (changes in) network dynamics in healthy and diseased brains.

References

Bakken, T. E., Jorstad, N. L., Hu, Q., Lake, B. B., Tian, W., Kalmbach, B. E., Crow, M., Hodge, R. D., Krienen, F. M., Sorensen, S. A., Eggermont, J., Yao, Z., Aevermann, B. D., Aldridge, A. I., Bartlett, A., Bertagnolli, D., Casper, T., Castanon, R. G., Crichton, K., … Lein, E. S. (2021). Comparative cellular analysis of motor cortex in human, marmoset and mouse. *Nature*, *598*(7879), 111–119. https://doi.org/10.1038/s41586-021-03465-8

Beierlein, M., Gibson, J. R., & Connors, B. W. (2003). Two Dynamically Distinct Inhibitory Networks in Layer 4 of the Neocortex.

Journal of Neurophysiology, *90*(5), 2987–3000. https://doi.org/10.1152/jn.00283.2003

Hooks, B. M., Mao, T., Gutnisky, D. A., Yamawaki, N., Svoboda, K., & Shepherd, G. M. G. (2013). Organization of Cortical and Thalamic Input to Pyramidal Neurons in Mouse Motor Cortex. *The Journal of Neuroscience*, *33*(2), 748–760. https://doi.org/10.1523/JNEUROSCI.4338-12.2013

Huang, Y., Parra, L. C., & Haufe, S. (2016). The New York Head—A precise standardized volume conductor model for EEG source localization and tES targeting. *NeuroImage*, *140*, 150–162. https://doi.org/10.1016/j.neuroimage.2015.12.019

Izhikevich, E. M. (2003). Simple model of spiking neurons. *IEEE Transactions on Neural Networks / a Publication of the IEEE Neural Networks Council*, *14*(6), 1569–1572. https://doi.org/10.1109/TNN.2003.820440

Kätzel, D., Zemelman, B. V., Buetfering, C., Wölfel, M., & Miesenböck, G. (2011). The columnar and laminar organization of inhibitory connections to neocortical excitatory cells. *Nature Neuroscience*, *14*(1), 100–107. https://doi.org/10.1038/nn.2687

Lefort, S., Tomm, C., Sarria, J.-C. F., & Petersen, C. C. H. (2009). The Excitatory Neuronal Network of the C2 Barrel Column in Mouse Primary Somatosensory Cortex. *Neuron*, *61*(2), 301–316. https://doi.org/10.1016/j.neuron.2008.12.020

Markram, H., Muller, E., Ramaswamy, S., Reimann, M. W., Abdellah, M., Sanchez, C. A., Ailamaki, A., Alonso-Nanclares, L., Antille, N., Arsever, S., Kahou, G. A. A., Berger, T. K., Bilgili, A., Buncic, N., Chalimourda, A., Chindemi, G., Courcol, J.-D., Delalondre, F., Delattre, V., … Schürmann, F. (2015). Reconstruction and Simulation of Neocortical Microcircuitry. *Cell*, *163*(2), 456–492. https://doi.org/10.1016/j.cell.2015.09.029

Martínez-Cañada, P., Ness, T. V., Einevoll, G. T., Fellin, T., & Panzeri, S. (2021). Computation of the electroencephalogram (EEG) from network models of point neurons. *PLOS Computational Biology*, *17*(4), e1008893. https://doi.org/10.1371/journal.pcbi.1008893

Shapson-Coe, A., Januszewski, M., Berger, D. R., Pope, A., Wu, Y., Blakely, T., Schalek, R. L., Li, P. H., Wang, S., Maitin-Shepard, J., Karlupia, N., Dorkenwald, S., Sjostedt, E., Leavitt, L., Lee, D., Troidl, J., Collman, F., Bailey, L., Fitzmaurice, A., … Lichtman, J. W. (2024). A petavoxel fragment of human cerebral cortex reconstructed at nanoscale resolution. *Science*, *384*(6696), eadk4858. https://doi.org/10.1126/science.adk4858