

BCI Signal Decoding

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1 Introduction

Welcome to our project, BCI Signal Decoding.

We're thrilled to have you on board as we work together on the BCI Signal Decoding project. Our aim is to decode neural activity from the brain's motor areas and to translate it into muscle control commands using Spiking Neural Networks (SNNs). This project involves working with real-world data from macaques; this data was gathered via implanted electrodes in the primary motor cortex (M1) and the dorsal region of the premotor cortex (PMd) during sequential reaching tasks.

Our goal is to use this data to build models that can contribute to advancements in brain-computer interfaces (BCIs) and prosthetic control systems. By analyzing both the neural and behavioral data, we can create systems that predict and simulate movement, providing valuable insights into motor control.

In the long term, our target is to prepare and submit a research paper to the Canadian Undergraduate Conference on AI (CUCAI) 2025, which will take place in March. This will be a fantastic opportunity to showcase our findings and share the work we've done with the broader research community.

This is a bit of a living document – we hope to keep updating it throughout the project so that all relevant information is present for anyone who would be interested in both understanding and replicating our work. The following information is correct according to our current understanding; there may be some mistakes or oversights, but we will do our best to keep this document as correct as possible.

This is largely intended to be an internal document. If you would like to distribute or share this document with anyone outside of the BCI Signal Decoding project team and/or WAT.ai Admin, please ask the TPMs for approval first.

2 Data Overview

2.1 What is our Data

The BCI Signal Decoding project utilizes the pmd-1 dataset, which contains extracellular neural recordings and kinematic data from macaques during a sequential reaching task.

Electrode data was collected from two monkeys (MM and MT) using 100-channel Utah arrays implanted in the primary motor cortex (M1) and the dorsal region of the premotor cortex (PMd) [1] [2].

2.2 Neuroanatomical Basics

As we all know, the brain is the control centre of the body. Most bodily functions are controlled by the nervous system as it sends commands throughout the body. The brain, however, is not uniform. While it is nearly impossible to attribute any one specific area to any one particular function, certain regions are dominantly involved with certain processes. In our case, M1 and PM cortices are *almost* exclusively involved in muscle control and movement planning. We owe this understanding largely to Cognitive Neuroscience — a field dedicated to the study of brain function through behavioural analysis.

For the sake of this project, we will be concentrating on Section 3 — The Motor Function Area — as depicted in Figure 1 on page 4. The motor regions of the brain are responsible for planning, controlling, and executing voluntary movements. This includes the primary motor cortex (M1), the premotor cortex (PM), and the supplemental motor area (SMA). These areas interact with the somatosensory cortex and other neighboring regions to convert environment and thought into action [4].

The M1 is located in the frontal lobe; it is the main contributor to generating neural impulses to muscles that result in movement. This is done by sending spike signals down the spinal cord to control muscle contractions and movement execution [4].

On the other hand, the PM and the SMA, located anterior to the M1, are responsible for preparing and planning movements before they are executed. They help guide movements, respond to sensory information, and coordinate the positioning of the body, especially for movements like reaching. The positioning and labels can be seen in Figure 2 on page 4 [4].

You may be wondering: how can we be sure that the region on which the electrodes were implanted correlate to arm movements? Good question. Figure 3 shows the Motor Homunculus – an approximate mapping between motor cortical regions and their corresponding muscle groups. We can intuit that the researchers collecting the original reaching task data knew where to place the electrodes by a similar principle, so the data collected should be directly correlational to the reaching task results [5]

Let's go a bit deeper into neurobiology now. As you know, the brain is made of (mostly)

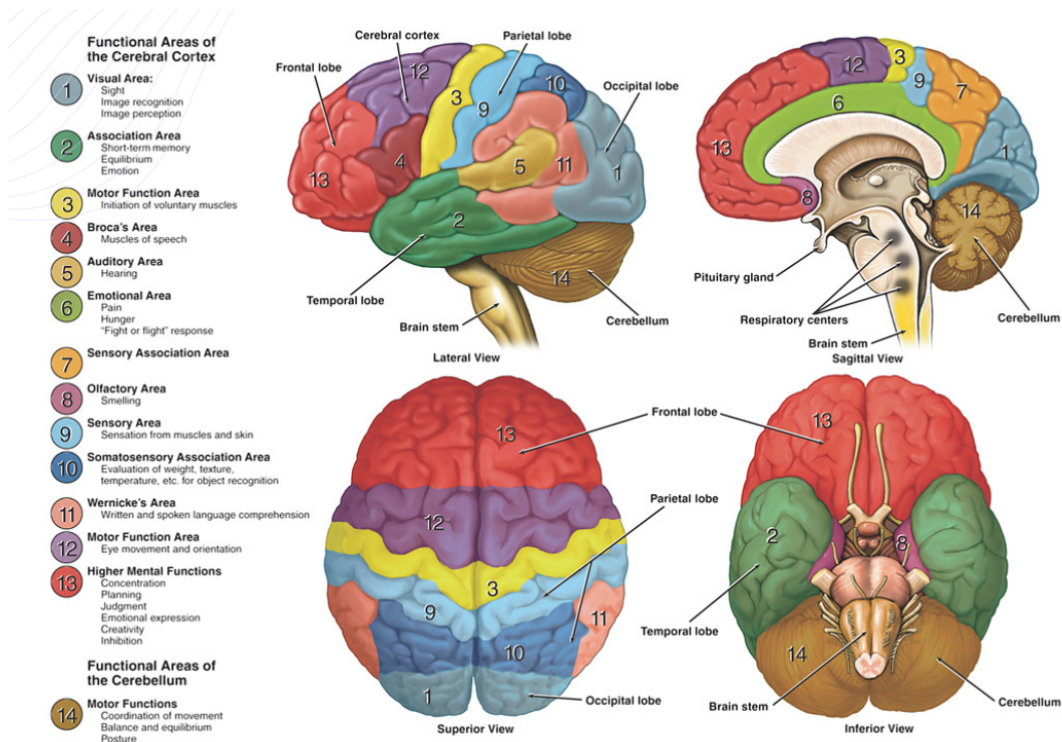


Figure 1: Diagram of Approximate Cortical Regions [3]

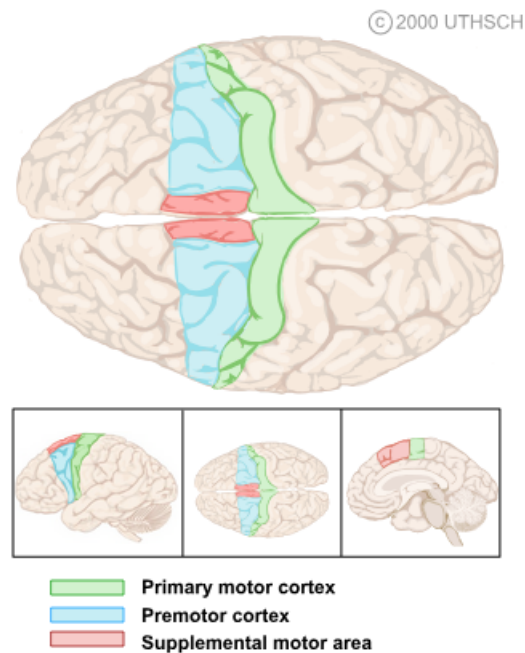


Figure 2: Motor Cortices[4]

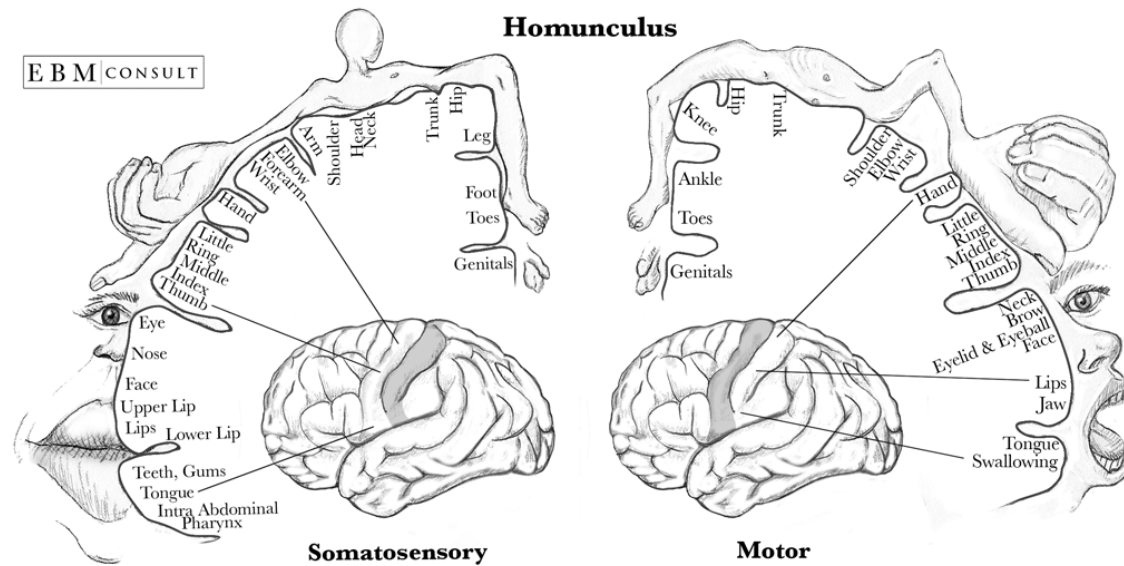


Figure 3: Motor Homunculus

nerve cells known as **neurons**. Neurons communicate by emitting electrical impulses when stimulated either by stimulus receptors or other neurons. Current is input to neurons – specifically their **soma** (nucleus) – through their **dendrites**, and spikes are emitted through their **axon**. Spikes travel through an insulating **myelin sheath** along the axon. At the axon's tips – the **telodendria** – messenger chemicals called **neurotransmitters** are released within their synaptic connections. These chemicals cause an input current to the following neurons' dendrites. Figure 4 on page 5 demonstrates a few of the typical forms neurons come in, but *many* more exist. [6]

Neurons maintain a resting membrane reference potential of around -70 millivolts (mV), where the inside of the neuron is negatively charged compared to the outside. When a

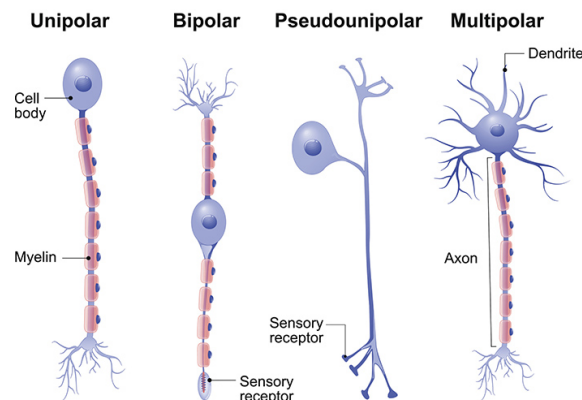


Figure 4: A Few Typical Neurons[6]

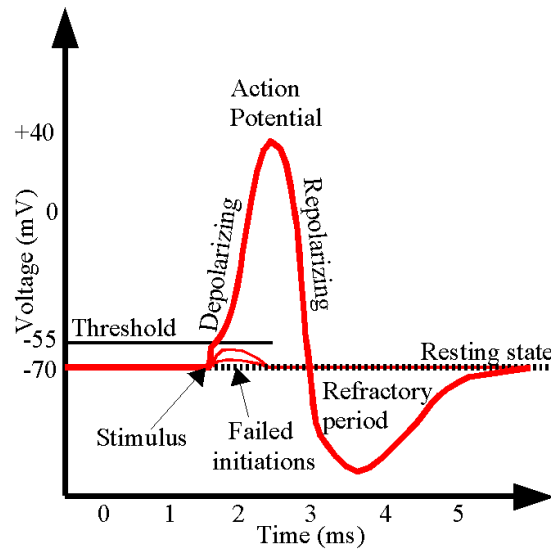


Figure 5: An Example Action Potential

neuron receives sufficient stimuli (like input from other neurons), it depolarizes—meaning its voltage becomes less negative—by allowing positively charged ions, such as sodium (Na^+), to flow into the cell. If this depolarization reaches a threshold (usually around -55 mV), it triggers an action potential – otherwise known as a spike.

During an action potential, the neuron rapidly changes its voltage:

1. **Depolarization:** The neuron's voltage spikes to about $+30$ to $+40$ mV as Na^+ ions rush in.
2. **Repolarization:** Shortly after, potassium (K^+) channels open, allowing K^+ ions to flow out of the neuron, bringing the voltage back down to a negative state.
3. **Hyperpolarization:** The voltage briefly overshoots the resting potential, making the inside of the neuron more negative than usual before returning to around -70 mV.

The pmd-1 dataset is comprised of spikes recorded from the neurons in the primary motor cortex (M1) and the dorsal region of the premotor cortex (PMd) of macaques. Each spike in the data represents a single action potential fired by a neuron, reflecting its activity over time. By analyzing these spikes alongside the kinematic data (such as movement and reach targets), we can (hopefully) decode how neural signals correlate with motor behavior, which is essential for understanding how the brain controls movement and for developing brain-computer interface (BCI) systems.

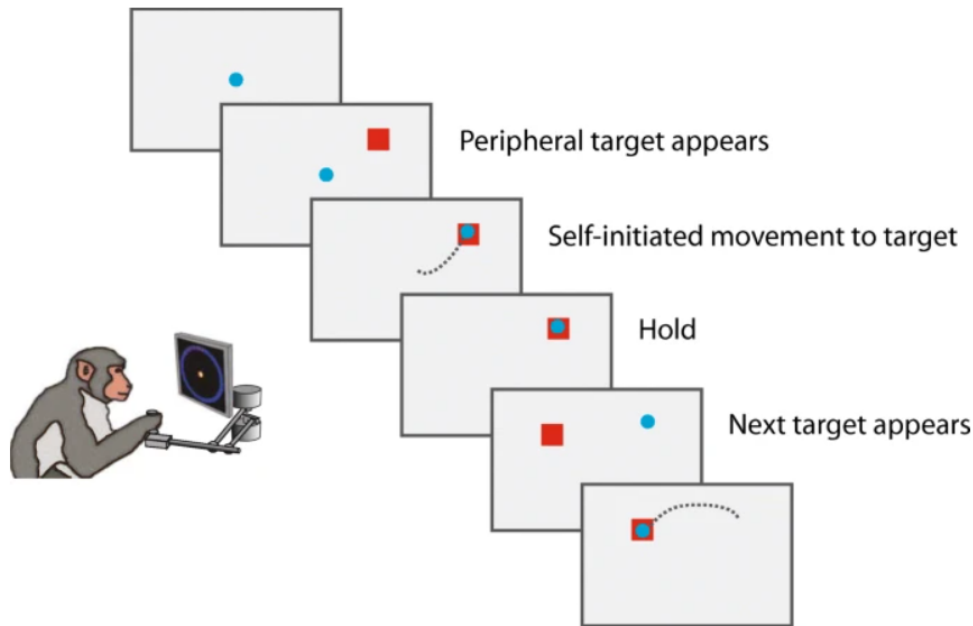


Figure 6: Experiment Schematic from Lawlor et al. [2]

2.3 Data Collection

The data collected in these experiments consists of neural recordings and movement data from macaque monkeys performing a reaching task. The goal was to understand how neural activity correlates with voluntary movements. By recording both the arm movements of the monkeys and the neural activity in specific brain regions (M1 and PMd), researchers were able to capture detailed information on how the brain generates motor commands

Neural data was collected using implanted Utah arrays, consisting of 100 microelectrodes each, surgically placed in the primary motor cortex (M1) and dorsal premotor cortex (PMd) of the monkeys. These microelectrodes penetrated the surface of the brain, allowing for the recording of extracellular electrical activity, which includes action potentials from multiple neurons. The key feature was that the high density and small size of the electrodes allowed for precise recording of neural signals.

During the experiments, the monkeys performed reaching tasks where they controlled a cursor on a screen using a planar manipulandum, which constrained their arm movements to a horizontal plane within a specified workspace of about 20 cm x 20 cm. These constraints were crucial to ensure consistent and measurable movements for accurate data collection [2]. A schematic of the experimental setup is shown in Figure 6 on page 7.

During the task, the monkeys were required to make a sequence of four reaches to targets appearing on the screen. Although there were minimal time restrictions, each successful reach was followed by a brief pause at the target before the next target appeared.

This setup helped to generate discrete movement events, which could then be linked to corresponding neural activity.

This controlled environment, where movements were somewhat restricted and consistent, allowed for better alignment between observed movements and recorded neural data, ensuring that the variability in neural activity was primarily related to the movement planning and execution rather than extraneous factors [1].

2.4 Reading the Data

The pmd-1 dataset is organized into MATLAB files containing neural and behavioral data from macaque monkeys. The data comes in two main forms: raw and processed. The following section provides an overview on what is available within the dataset. For additional reference, see the provided dataset description [1].

2.4.1 Raw Data

Each raw data file corresponds to a single experimental session and contains the complete, minimally processed data. These files include:

1. **M1 Structure:** Contains neural data from the primary motor cortex (M1), with spike information for each recorded neuron.
2. **PMd Structure:** Analogous to the M1 structure but for neurons recorded from the dorsal premotor cortex (PMd).
3. **trial_table:** A table with metadata about each trial in the session, such as start times, target appearances, movement onsets, and trial results (e.g., successful or failed).
4. **cont:** Kinematic data like position, velocity, and acceleration, are recorded over time.

You can find sub-structures within M1 and PMd:

1. **units:** Each neuron's spike times, waveforms, and metadata, such as the electrode number it was recorded from.
2. **sg:** Information to identify the unit across the session.

2.4.2 Processed Data

The processed data files are more refined and contain pre-aligned and down-sampled data focusing on each reaching movement. Each processed file has a single structure called Data, which includes:

1. kinematics: Contains the position, velocity, and acceleration of each reach.
2. neural_data_M1 and neural_data_PMd: Spiking data for each reach, represented as a count of spikes per time bin for every neuron in M1 and PMd, respectively.
3. block_info and trials: Metadata about each trial and which trials were included in the processed data.
4. Additional fields specifying timestamps, reach information (e.g., start and end times, directions), and target cue details.

2.5 Spiking Neural Networks

While not strictly necessary to know for this project, it is valuable to understand how exactly Spiking Neural Networks function and the principles behind their implementations. This section will explore a variety of relevant concepts to facilitate your understanding of the SNNs that are implemented through Nengo.

2.5.1 Artificial Neurons

You may be more familiar with neurons in the context of traditional NNs. These neurons are based loosely on biological neuron function, though they fail to accurately capture the dynamics of biological neural activity.

For example, consider a neuron with a ReLU (Rectified-Linear Unit) activation layer. Supposing it had some input, its instantaneous output would look like:

$$\hat{x}_i = \begin{cases} 0 & \sum_{j=0}^n w_{ji}\hat{x}_j + b_i \leq 0 \\ \sum_{j=0}^n w_{ji}\hat{x}_j + b_i & \text{else} \end{cases}$$

Other activation layers, such as Sigmoid, map the output the neuron to some logistic function of its inputs. One reason why ReLU is effective is because it similarly captures the dynamic and non-linear activation of biological neurons when presented some stimulus. One characteristic of biological neurons that it fails to capture, however, is the firing-rate limit of a neuron. This will be explored in the next section.

2.5.2 Biological Neurons

Biological neurons can be modeled in a variety of ways – through the highly-detailed Hodgkin-Huxley model of neurotransmitter interactions, through the significantly less complex Spiking ReLU model, or through the nice middle-ground of the Leaky Integrate-and-Fire model. One thing more characteristic about biological neuron simulation is the dependency on time – neuron voltage accumulates over time, and the resultant firing is based on the approximations of firing rates subject to some key parameters.

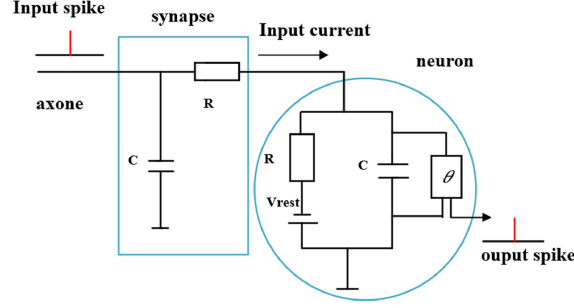


Figure 7: LIF Circuit Equivalence Diagram [7]

Biological neurons tend to be focused along some stimulus. For some areas in the secondary visual cortex, we will see firing at varying rates for bars of light moved around the visual field. The intensity of this stimulus for some neuron can be defined as its input – typically measured as a current in biological studies. Spiking Leaky Integrate-and-Fire (LIF) neurons suppose a firing rate that has the shape of an inverse logarithm – this is fairly consistent with the Hodgkin-Huxley model’s results and allows us to use it as a good estimator without the more significant computational cost of Hodgkin-Huxley neurons. The LIF neurons operate on a few key principles:

1. Action potential in the neuron’s soma is stored similar to that of a capacitor
2. The capacitor leaks – without some stimulus, it will return to a natural resting state
3. Once the neuron reaches some threshold voltage, it will trigger a spike
4. Successive spikes are bound by an absolute refractory time period during which they cannot spike again

Due to the variety in cell biology, biological neurons each typically have some bias, gain, and maximum firing rate that cannot be modified through learning processes – it is the weighting of the connections between them that can be modified. A computationally simplified version of LIF neuron firing rate estimation is shown below:

$$G(J) = \begin{cases} \frac{1}{\tau_{REF} - \tau_{RC} \log\left(1 - \frac{V_{th}}{R * J}\right)} & 1 - \frac{V_{th}}{R * J} > 0 \\ 0 & \text{else} \end{cases}$$

A circuit representation of the LIF neuron and its interactions can be developed to reflect the behaviour of the biological interactions relating to current and voltage between and within neurons. Figure 7 on page 10 illustrates the circuit equivalence from which we can derive the above approximation.

The parameters of the simplification are the RC time constant of the LIF circuit – τ_{RC} – equal to the product of the soma’s internal resistance and capacitance. τ_{REF} is the

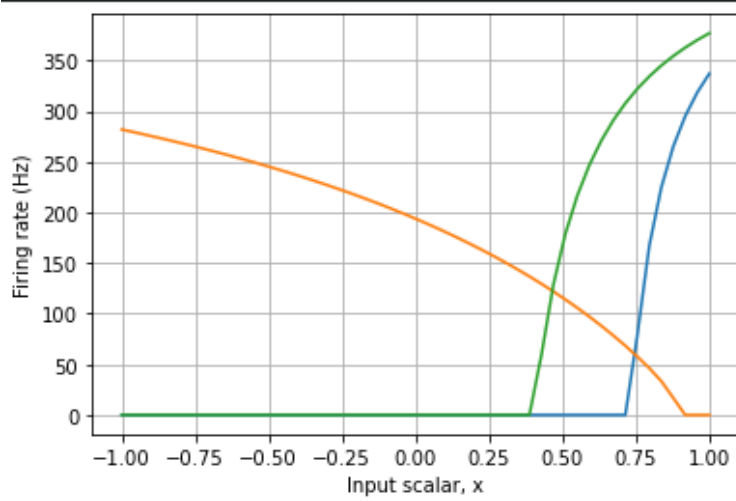


Figure 8: Nengo Tuning Curve for Three Neurons with Varying Parameters

absolute refractory time – a sort of hyperparameter. V_{th} is the threshold of soma voltage to trigger a spike. J is the provided stimulus that is computed by taking the product of the neuron’s gain, α , the neuron’s encoder, e , and the normalized stimulus value, x , plus some internal current bias, J_{bias} . Gain correlates with how fast a neuron’s firing rate with increase as an encoder-aligned stimulus increases in magnitude. Neurons’ encoders modify the intensity of the neuron’s response to stimuli in multiple dimensions. Encoders are typically unit vectors sampled from (I think) the unit hypersphere. Given randomly sampled parameters for simulation, a neuron tuning plot can be produced. One is shown in Figure 8 on page 11 for three neurons simulated in Nengo.

2.5.3 Population Connections

Similar to conventional ANNs, the connections between neuron populations in SNNs are linear transformations of the population’s overall state. The weights of the different connections between populations are called decoder values. These decoder values can be optimally calculated supposing some input and ideal output data exist, but they can also be trained using supervised or unsupervised learning techniques – similar to ANNs. The non-linear encoding and activity of neurons as well as the linear transformations between populations enable neural networks in general to act as universal function estimators.

2.6 Neural Engineering Framework

The Neural Engineering Framework is the framework by which biological neuron systems are constructed – it posits three main principles [8]:

1. *Neural representations are defined by the combination of nonlinear encoding (exemplified by neuron tuning curves) and weighted linear decoding.*
2. *Transformations of neural representations are functions of variables that are represented by neural populations. Transformations are determined using an alternately weighted linear decoding (i.e., the transformational decoding as opposed to the representational decoding).*
3. *Neural dynamics are characterized by considering neural representations as control theoretic state variables. Thus, the dynamics of neurobiological systems can be analyzed using control theory.*

This provides a methodology by which we can approach designing and implementing biological neuron models – aka SNNs. For further reading, see CNRG @ UWaterloo — NEF Framework.

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