# Overview

Abstract (1/10):
Summary of the summary. A short introduction with results stated clearly.

Introduction (1/10):
Introduce the topic, the problem and how the problem is being solved. The target audience are other master students.

Theory:

- Basic information about neurons and the cell membrane. (7/10)

- Basic information about different types of neurons, how they serve different purposes and have different functions. (0/10)

- Turning the neuron into a electronic circuit, explaining ion pumps and channels. (5/10)

- Explanation of action potentials and the generation of action potentials with a the hudgekin and huxley model. (1/10)

- Explanation of the principle of compartmental models with diagrams. There are many kinds. Not too long. (0/10)

- Explanation of electrodes, how they are used, what they measure, how tetrodes work. (1/10)

- Mentioning that neurons measured from the same electrode must be seperated. Cocktail party problem, source seperation. (0/10)

- Explanation of extracellular potential, the physics behind the problem, current sum to zero, how it can be calculated, etc.(4/10)

- Discussion of the difference between intracellular and extracellular spike shape. Some say the extracellular spike is the derivative. (0/10)

- Explanation of Neuron and LFPy. How they work, the principle behind them, what they are intended to calculate. (2/10)

- Explanation of the current state of cell classification. Why is it important, how is it done. Mentioning most influential work from early to current work. (0/10)

- Explanation of the Blue Brain cell database, how it was made, why is it useful, what were the focus of the models, models are public. (0/10)

Note to self, mention the most influential work that has been done on all topics mentioned in the theory. I should show I know all the basic important work that has been done in these fields.

Methods

Everything here is work which I have done myself. Show what I have done, make sure it understood as a lot.

- Explanation of the basis of differentiating spikes, how does one measure how spikes are different. Meantion spike width and ampltiude. (0/10)

- Explain different definitions of spike width and amplitude measurements. (1/10)

- Detailed explanation about the simulation environment. What does LFPyUtil solve and how does it solve it. (3/10)

- Showing a minimum working example of LFPyUtil, show what it makes easier. (0/10)

- Detailed explanation of each simulation, what are the parameters. (3/10)

- How did I use the BlueBrain models, which models are used, why are they used. (0/10)

Results:

State the results in such a way they clearly show what I want to show, but does not "jump to conclusions". State the results without bias. Include figures with text, at a glance the figures will be read seen and read first.

- Detailed explanation of the replication of Pettersen and Einevoll, show that the simulation environment can be trusted. (6/10)

- Explain any deviations from Pettersen and Einevoll in the replication. (5/10)

- Create a conclusion of the results from Pettersen and Einevoll. (0/10)

- Show which definition is best for differentiating spikes from different kinds of neurons. (0/10)

- Show that interneurons and pyramidal neurons can be classified. (0/10)

- Explain why spikes look differnt, what are the physical processes that does this? (0/10)

Discussion:

The discussion is important, make it of high quality and maybe long.

- Using spike width have already been used for differentiate neurons, show how current results backs up this statement. (0/10)

- Research has shown that thin spikes can also come from measuring near axons of pyramidal cells. (0/10)

- Argue that the models are relateable to real spikes. (0/10)

- The analysis framework can be used for future cell models. (0/10)

Note to self, when trying to explain a method first show the problem clearly then propose the solution to the problem. Engage the reader by showing the problem in such a way they can become curious for a solution.

# Abstract

The physical processes that creates electrical signals in neurons are well understood, but how the signals are processed into actions and thoughts has yet to receive a scientifically robust answer Cell type classification is of high importance because the function of different neurons is still largely a mystery.

# Contents

# 1 | Introduction

Since the conception of neuroscience the neurons function have been studied on many levels from the properties of the cell membrane to clustered networks of neurons.

There are several types of neurons and it was early noticed that different kind of neurons gave different types of signals.

This was of much interest because

Modern types of classification uses genotype?, the structure of the neuron and the electric signal.

It is useful to seperate interneurons from pyramidal neurons as pyramidal neurons are excitatory and interneurons are inhibitory.

Is it possible to seperate interneurons from pyramidal neurons soly based on the shape of the action potental.

Knowing the neuron type is important for research. While doing single cell recordings on alive subjects the researchers are recording in the dark. The electrodes only pick up on electrical signals from the brain, so the researcher does not know exactly what they cell they are recording.

Surely the different types of neurons are specialized at certain functions

In this article we show that interneurons can be seperated from pyramidal neurons based on data and models from the Blue Brain project. The program makes an easy way to do the same analysis on future models as long as the models can be loaded with LFPy.
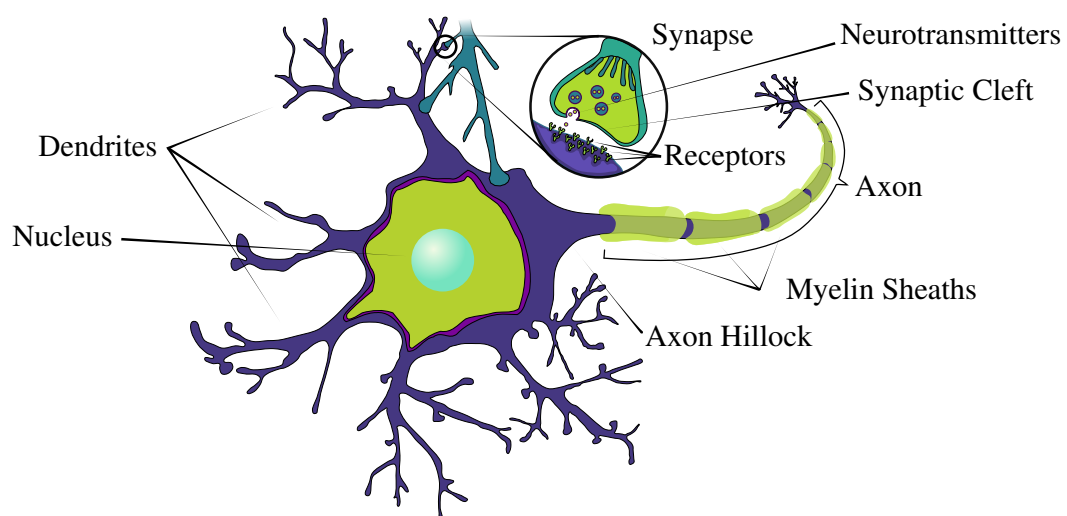
# 2 | Theory

## 2.1 The Neuron



Figure 2.1: Stuff about this neuron.

Neurons are electrically excitiable cells that are a fundamental part of all brain functions. Other names include nerve cells, neurone or more colloquially brain cells. Neurons form in big networks which process information, and in the human brain there is an estimated $10^{11}$ neurons.

Special proteins in the cell membrane enables the neuron to fire action potentials when it is electrically excited. These action potentials are sharp voltage changes that propagates through the full structure of the neuron. The same properties that makes the neuron able to fire makes the action potential regenerative, meaning it will propagate without decay.

The body of the neuron, the soma, has dendrites and the axon attached to it. The dendrites and the axon are very thin branching structures with a width usually in the order of $1\,\mu m$. While

neurons often have many dendrites directly attached to the soma there is only one axon attached through the *axon hillock*. The axon can branch several times before it ends and usually connects to the dendrites of other neurons via synapes.

The synapes are electrically sensitive which allows information to pass between neurons. Though the majority of all synapes are axo-dendritic (axon to dendrite), other junctions are also possible. Other junctions include but are not limited to, dendrite to dendrite, axon to axon and axon to blood vessel. When an action potential reaches a synapse it will activate the synapse and pass information to the connect neuron. The information that is passed along depends on the type of synapse, and if it is of a chemical or electrical type.
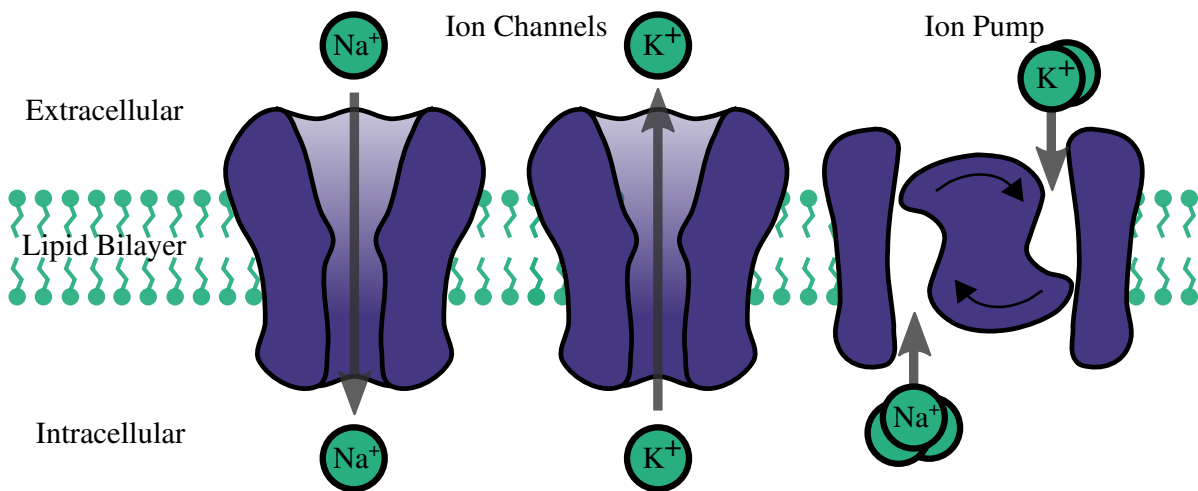
## 2.2 Electrical Activity



Figure 2.2: Something about ion pumps and channels.

The potential difference between the inside and outside the neurons are caused by different concentrations of ions in the extracellular and intracellular medium. The ions cannot pass through the cell membrane as it consists of a $5\,\mathrm{nm}$ lipid bilayer which is mostly impenetrable to ions.

In the membrane sits differnt *ion channels* and *ion pumps* which can have selective permeability to ions, this creates a potential gradient across the membrane. The most significant ions in this process are Sodium ($Na^+$), Potassium ($K^+$), Calcium ($Ca^{2+}$), Magnecium ($Mg^{2+}$) and Chloride ($Cl^-$). Ion channels are divided between passive channels and active channels where the active channels can change permeability under certain conditions while passive channels have a constant permeability.

The ion pumps differ from the channels by activly transporting certain ions through the membrane. For instance, the Sodium-Pottasium exchanger pushes two $K^+$ ions out of the cell for every three $Na^+$ it pushes into the cell. Doing this creates a net loss of charge inside the cell and the pump is therefore electrogenic. Not all pumps are electrogenic, the Sodium-Hydrogen exchanger transports $H^+$ and $Na^+$ without effecting the net charge. For each $H^+$ ion out of the cell the pump pushes one $Na^+$ into the cell.

To understand the electrical activity of neurons it is useful to view the neuron as an electronic circuit where the ion channels, ion pumps and the membrane serve as different electronic components.

Hodgkin & Huxley [5], Connor & Stevens [1], and Sterratt et al. [10]

## 2.3  Action Potential

Action potentials are sharp increases in the membrane potential followed by a less sharp decrease towards the resting potential. In the the depolarization phase the potential rises towards the peak magnitude, while in the repolarization phase the potential decreases towards the cells resting potential. When the potental is below the resting potential it reaches the afterhyperpolarization phase before it returns to its resting potential.

## 2.4  Neuron Models

There are multiple models for neurons, some of the main groups are point models and compartmental models. List many models? Multi-compartmental models can be useful to understand the processing of neurons with complex morphological structures

## 2.5  Electrodes

## 2.6  Calculating Extracellular Potential

The extracellular potential is the electric potential generated from the transmembrane currents in the neurons. When a neuron fires this can be seen from the extracellular potential which will have a spike which is similar to the intracellular spike.

By modelling the neuron as compartments and approximating each compartment as a spherical volume current source at position $\mathbf{r_0}$, the potential at at position $\mathbf{r}$ at time $t$ will be,

$$\mathbf{E}(\mathbf{r}, \mathbf{t}) = \frac{1}{4\pi\sigma} \frac{I_0(t)}{|\mathbf{r} - \mathbf{r_0}|} \tag{2.1}$$

$$\mathbf{E}(\mathbf{r}, \mathbf{t}) = \sum_{n=1}^{N} \frac{1}{4\pi\sigma} \frac{I_n(t)}{|\mathbf{r} - \mathbf{r_0}|} \tag{2.2}$$

Potential from compartments modelled as line sources.

$$\mathbf{E}(\mathbf{r}, \mathbf{t}) = \frac{1}{4\pi\sigma} \sum_{n=1}^{N} I_n(t) \frac{dr_n}{|\mathbf{r} - \mathbf{r_0}|} \tag{2.3}$$

$$= \frac{1}{4\pi\sigma} \sum_{n=1}^{N} I_n(t) \frac{1}{\Delta s_n} \log \left| \frac{\sqrt{h_n^2 + \rho_n^2} - h_n}{\sqrt{l_n^2 + \rho_n^2} - l_n} \right| \tag{2.4}$$

Taken from Lindén et al. [6]

This equation rests on two assumptions,

1. The permeability $\mu$ of the extracellular medium is the same as that of vacuum $\mu_0$.

2. The quasistatic approximation which lets the time derivatives, $\partial E/\partial t$, be ignored as source terms. See **??**

The extracellular potential can be calculated using Maxwell's equations and the continuity equation if the spatial distribution (morphology) of transmembrane currents and the extracellular conductivity is known.

In the quasistatic approximation, since $\nabla \times \mathbf{E} = \mathbf{0}$, the electric field can be expressed with a scalar potential.

Forward problem = calculate the potential from the current source, inverse problem is used in magnetoenchephalography (important). The amplitude of a spike in the extracellular potential is usually in the magnintude of $< 200\mu V$. The noise of electrodes vary, but can be as much as $20\mu V$. This limits the range electrodes can record from.

The currents sum to zero, while the spike is very visible, there are many small currents in the dendrites with opposite current. ([4])

The extracellular spike width tend to increase with distance from soma because of the neuronal morphology. This article used a passive neuron model with different morphologies to show that the spike width increases with distance to soma. The spike amplitude also decreases with distance to soma and seems to follow a power law. ([9]).

The shape of extracellular spikes are mainly depedent on the membrane currents and the morphology of the cell. Some of the effects from the morphology of the cell are increased spike width and decreased amplitude from distance to soma.

Many things here from around page 245. When the conductivity $\sigma$ and the current generators are know, Maxwell's equations and the continuity equation equation can be used to calculate the electric field $E$ and magnetic field $B$. (TODO: Copied text) ([4])

## Background

Recording is usually done using electrodes, this makes recording the membrane potential more challenging than recording from the extracellular medium as the electrode has to be very close or inside the cell. At the time of writing, recording the membrane potential of a concious subject is nearly impossible, this makes understanding extracellular potentials vital for current research.

Early calculations was done by Rall 1962 investigating the interaction between action potentials and synapses using cylinders as the current source. (TODO: Read article, make more understandble.) Holt and Koch 1999 added comparmental models to reconstruct pyramidal neurons.

The information about the transmembrane current is usually difficult to obtain, as well as the morphology.

## 2.7 Neuron & LFPy

LFPy is a Python module that uses Neuron and the mentioned methods to calculate the electric field outside the neuron. [6]

## Background

# 3 | Methods

Methods mentioned here have been developed spesifically for this research.

## 3.1  Spike Width Measurement

Most extracellular spikeshapes has a minimum value greater than the maximum value, but this is not always the case when measuring far away from soma.

Width Type I - Peak-to-peak:
    Width is measured as the time from the minimum potential to the maximum. This is the time from the polarization phase to the afterhyperpolarization phase.

Width Type II - Width at Half Amplitude:
    Width is measured as the duration the spike is below half amplitude of the signal measured from the baseline at the start of the signal.

Width Type II - Width at Half Amplitude:
    Width is measured as the duration the spike is below half amplitude of the signal measured from the baseline at the start of the signal.

    There are three prevailant ways of defining spike width which has been named Type I, Type II and Type III.
    **Type I**: Width is measured from "peak-to-peak". This method can easily be implemented by measuring the width from minimum to maximum value. In the cases where the spike is flipped or is not well defined, this definition is not correct.
    As such the implementation of this definition was done by measuring the time from the the maximum absolute value to the preceding maximal absolute value on the opposing side.

## 3.2 Spike Amplitude Measurement

Amplitude is easier to calculate than the width of a spike, but there are still different ways to define the amplitude. In most cases the amplitude is defined as the distance from a baseline, in a similar manner as a sinusoid. The baseline is not always well define with spikeshapes. For the intracellular potential the baseline can for instance be set at the firing threshold, but for extracellular spikes this is not possible. Maybe more common is to set the baseline as the value during quiescence. For modelling purposes this is can provide difficulties.

## 3.3 Simulations with LFPyUtil

LFPyUtil is a python package that was created for this project with the purpose to simplify the simulation pipeline for multiple neurons and creating and easy to use interface when developing new simulations. LFPyUtil extends and uses the package LFPy to accomplish this.

The main feature of the python package LFPy is enable the calculation of extracellular potentials, but another major feature is the simplification of simulations with the simulation engine Neuron. Using Neuron requires a good understanding of the programming language hoc which creates many challanges as hoc is outdated and is no longer maintained by the developers. To use Neuron with python one has to use the python interface to Neuron which has been implemented in such a way that runs hoc code directly "under the hood". A common situation is that one must write hoc code inside strings and pass them to the python-hoc interpreter. This has the unfortunate consequence that the two programming langues are very intertwined and also does not follow common python coding conventions. This also makes it harder for users to troubleshoot errors as problems can occur either in python, hoc or between the two. To solve this the package LFPy has attempted to wrap the cell model and electrodes from the Neuron engine into python objects, such as the `LFPy.Cell` and `LFPy.StimIntElectrode` classes.

When running a simulation with Neuron there is no inherent support for running simulations simultaniously. Because LFPy is an extension to Neuron this is also lacking in LFPy. LFPyUtil attempts to solve this by starting each simulation in independent processes. This does not speed up a single simulation but rather speeds up the simulation of multiple neurons and simulations that must be run multiple times with different parameters. The major difficulties of running independent processes with Neuron is that there is no reset function which can make the state of the previous simulation effect the state of the next simulation.

In all simulations the extracellular conductivity was set to $\sigma = 0.3\,\Omega\,\text{m}$ based upon data from experimental measurements.

All stimulus electrodes uses the `LFPy.StimIntElectrode` with a custom made electrode named ISyn. With the default stimulus all transmembrane currents will be summed equal the input current, using ISyn prevents this and the currents are correctly summed to $0$.

The following items are python objects in LFPyUtil.

### 3.3.1 SphereRand

SphereRand places electrodes placed in uniformly distributied locations around the soma within a default radius of $50\,\mu m$. Spike timing is detected by thresholding the soma membrane potential. That timing is applied to all electrodes such that all electrodes measure the same part of the simulation. If the signal has several spikes the spike index must be supplied, the default setting uses the first spike.

## 3.4 Blue Brain

The Blue Brain project released XXX models based upon neurons from the hind-limb somatosensory cortex from 2-week-old Wistar Han rats.

The neuron models are based on the classication criteria set by the Blue Brain team there is only 2 classes of pyramidal neurons avaiable in L5, while the diversity in interneuron models are much greater. The number of available models were based on the variability of neurons depending on their morphological type and electrophysiological response to stimuli. As most of the encountered pyramidal neurons had a similar morphological structure and response to stimuli the team choose to only recoqnize two morphological types and one electrophysiological type, referred to as m-type and e-type.

# 4 | Results

## 4.1 Pettersen & Einevoll (2008) Reproduction

To verify that the simulation environment could be trusted some results from Pettersen & Einevoll [9] was replicated. Spesifically the spike width and amplitude dependency in relation to the distance from soma was compared to current results.

### 4.1.1 Simulation

**Cell:** The Mainen & Sejnowski [7] morphology was used with a passive model, which is the same model used in Pettersen & Einevoll [9]. The cell was rotated using PCA (principal component analysis) on the compartment positions. This calculates three orthogonal vectors such that the positions of the compartments has the greatest variance along the first principal component, second highest along the second and third most along the third. The first principal component was made paralell to the y-axis which puts the apical dendrites along this axis
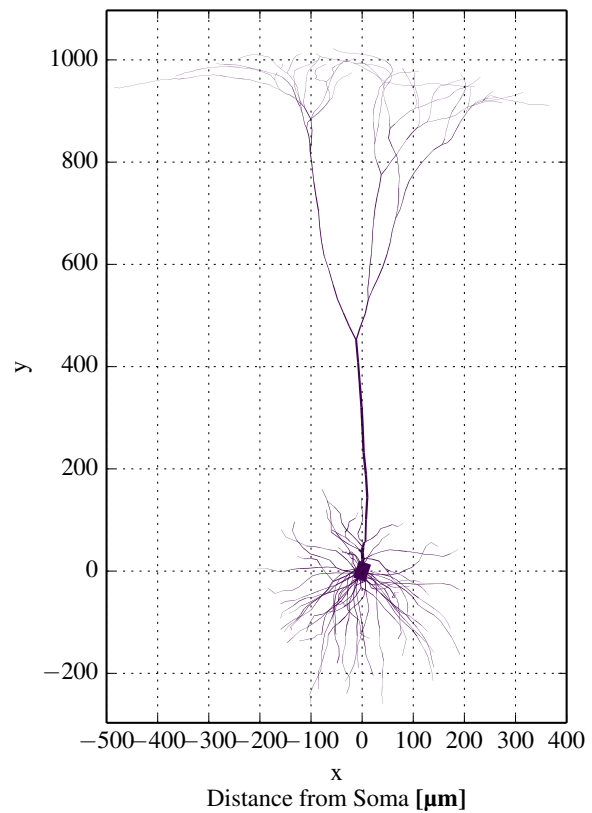


Figure 4.1: Morphology of Mainen & Sejnowski [7] cell. The apical dendrites are located along the y-axis after rotation with PCA.
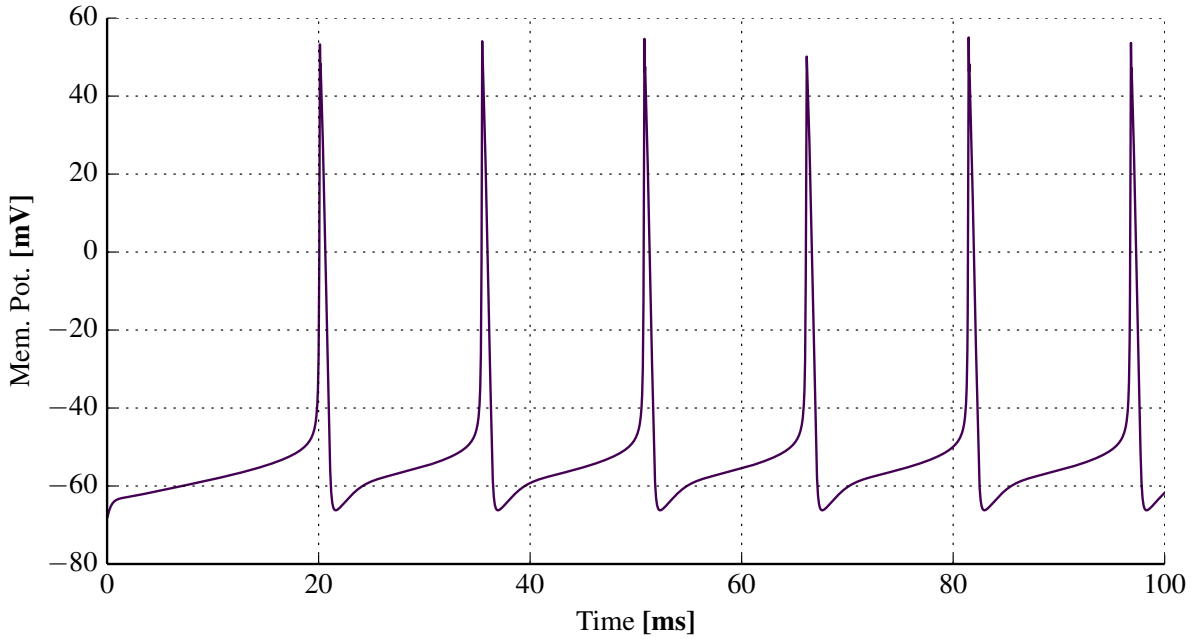
(fig. 4.1).



Figure 4.2: Simulation of the Connor-Stevens model using parameters from Dayan & Abbott [3]. A similar graph is shown in fig. 6.1 (B) in their book.

**Spike Generation:** An action potential was generated using the Connor-Stevens model (Connor & Stevens [1] and Connor et al. [2]) using the same parameters as Dayan & Abbott [3]. In fig. 4.2 the Connor-Stevens simulation is shown where the second spike was used for further analysis. This spike had an amplitude of $119.49\,\mathrm{mV}$ from baseline. The baseline was estimated to $-53.26\,\mathrm{mV}$ and the peak at $53.26\,\mathrm{mV}$. These values matches Dayan & Abbott [3], but not the spike used in Pettersen & Einevoll [9] which had an amplitude of $83\,\mathrm{mV}$ from baseline. Pettersen & Einevoll [9] does not go into further detail about the creation of the action potential other than stating that the action potential used were similar to Dayan & Abbott [3]. The difference can be explained by the fact that action potentials from pyramidal neurons often peaks at $20\,\mathrm{mV}$, and that this was achived by scaling the original signal from the Connor-Stevens model. To compensate for the difference the action potential used in further simulations were scaled to $83\,\mathrm{mV}$ (fig. 4.3).

The input current was set to $12.6\,\mathrm{\mu A\,cm^{-2}}$. and was very carefully adjusted to make the magnitude spectrum (fig. 4.5) similar to Pettersen & Einevoll [9] figure 3. Without adjustment the magnitude spectrum tended to have a different inital value, from 6 to $8\,\mathrm{mV}$, and was not as smooth.

**Parameters:** Parameters for the Neuron simulation were the same as used in Pettersen & Einevoll [9] and the action potential used as a boundary condition in the soma is the one speci-fied above. Membrane resistance $R_m = 30\,\mathrm{k\Omega\,cm^{-2}}$, membrane capacitance $C_m = 1\,\mathrm{\mu F\,cm^{-2}}$, axial resistance $R_a = 150\,\Omega\,\mathrm{cm^{-2}}$, time resolution $dt = 2^{-5}\,\mathrm{ms}$. The reversal potential was set to zero. The action potential was imposed in all soma sections using the "play" vector function
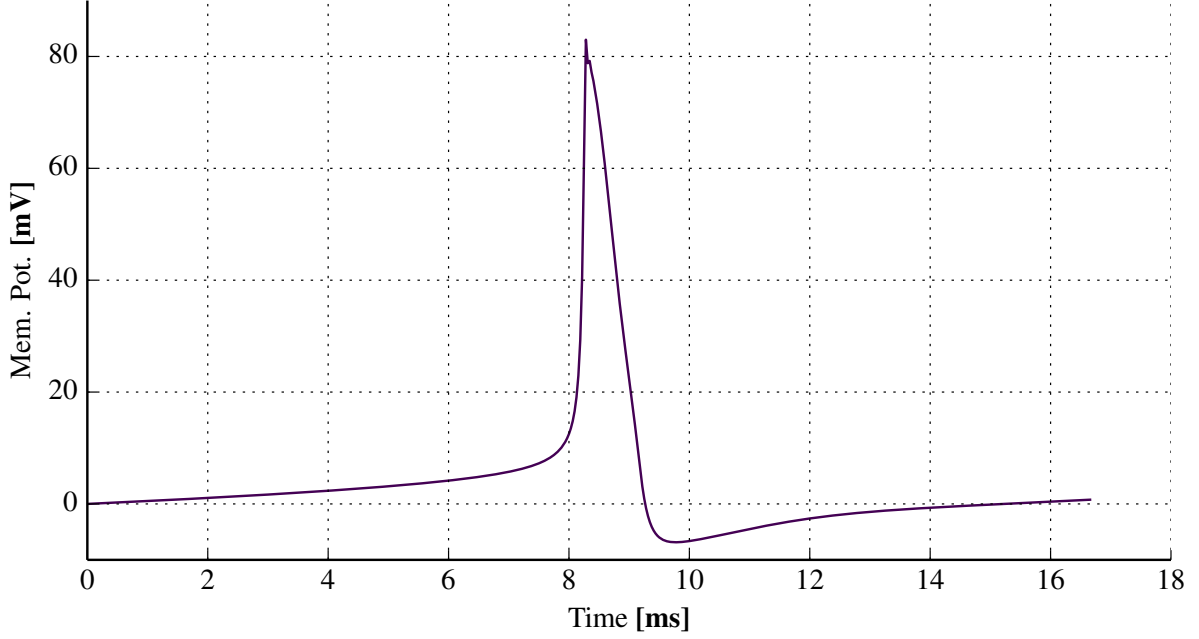
Figure 4.3: The second spike in fig. 4.2 scaled to $83\,\mathrm{mV}$ to match the action potential used in Pettersen & Einevoll [9].

in Neuron.

**Electrode Positions:** Recording sites were placed in the xz plane at 11 linearly spaced positions along 36 lines with equal angular spacing (fig. 4.4). Pettersen & Einevoll [9] states the recording positions were in the plane perpendicular to the apical dendrites, this is ensured by the rotation done with PCA and putting the electrodes in the xz-plane.

**Spike Width & Amplitude:** A baseline was set as the value at the start of the signal. Amplitude was calculated as the difference between the maximum value and the baseline. The spike width was calculated at as the width at half maximum value.

At $dt = 2^{-5}\,\mathrm{ms} = 0.031\,25\,\mathrm{ms}$, the spike width from the Connor-Steven model was $0.5625\,\mathrm{mV}$. This is similar to the reported spike width from Pettersen & Einevoll [9] at $0.55\,\mathrm{ms}$ which must have been rounded to the nearest $0.05\,\mathrm{ms}$.
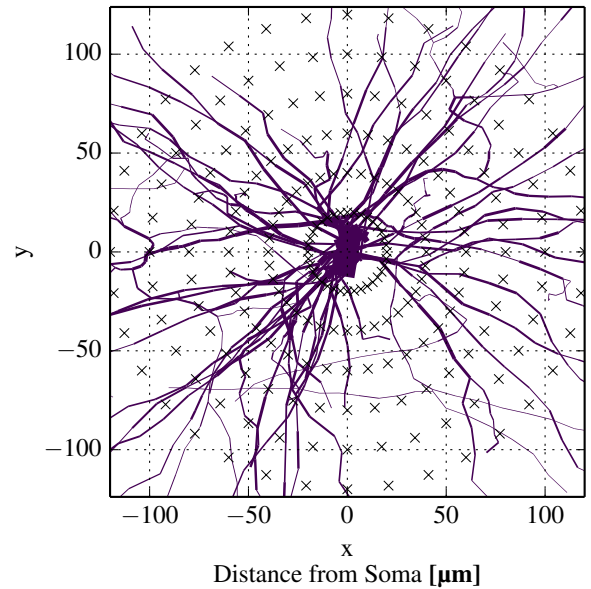


Figure 4.4: Electrode positions placed in a plane around soma perpendicular to the axis along the apical dendrites.

### 4.1.2 Results

The action potental that was used in Pettersen & Einevoll [9] is similar to the one used here. The amplitude of the fourier transform is displayed in fig. 4.5, which is in close resemblance to the action potential in fig. 3 in the paper.
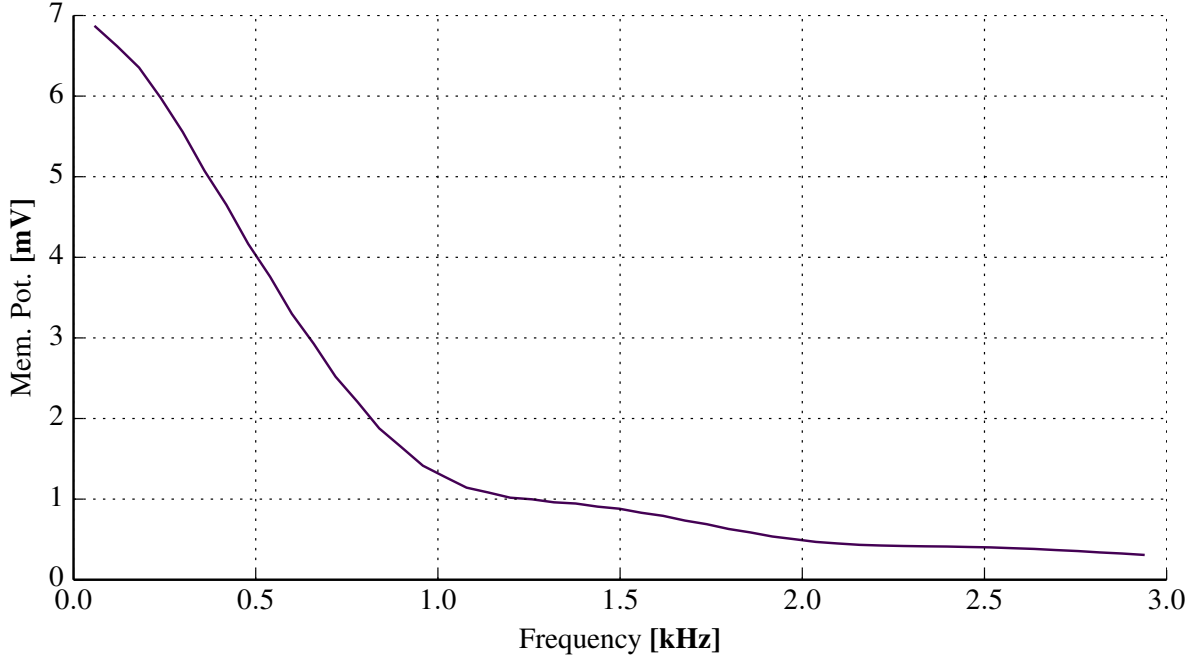


Figure 4.5: Magnitude specter of simulated somatic membrane potential.

The spike width increases with the distance from soma as seen in fig. 4.6. These results are lower than the widths reported in Pettersen & Einevoll [9]. (Use more time on editing the Connor-Stevens model to come closer to an max.amplitude on 20mV?).

Sudden changes in spike width was experienced with increased distance from soma. Above $200\mu V$ the spikes shapes are not well defined. This was also reported in Pettersen & Einevoll [9].

Pettersen & Einevoll [9] reports a spike amplitude above $150\mu V$ at $20\mu m$, this does not match current findings. fig. 4.7 shows spike amplitude with logarithmic axes. (TODO: Is numbers on the power law decays necessaryy?) Although the data does not match Pettersen & Einevoll [9], it is comparable with what is expected in the near and far limit field of a ball and stick neuron. In the near field the the expectation is a $1/r$ decay and in the far field it is $1/r^2$ or $1/r^3$ depending on distance. (TODO: Clearify this, put reference back to theory chapter.)

### 4.1.3 Discussion

## 4.2 Optimal Width Definition

Many different definitions of spike width has been used to differentiate neurons, but to date it is not clear if some definitions are more suited for neuron classification than others. This
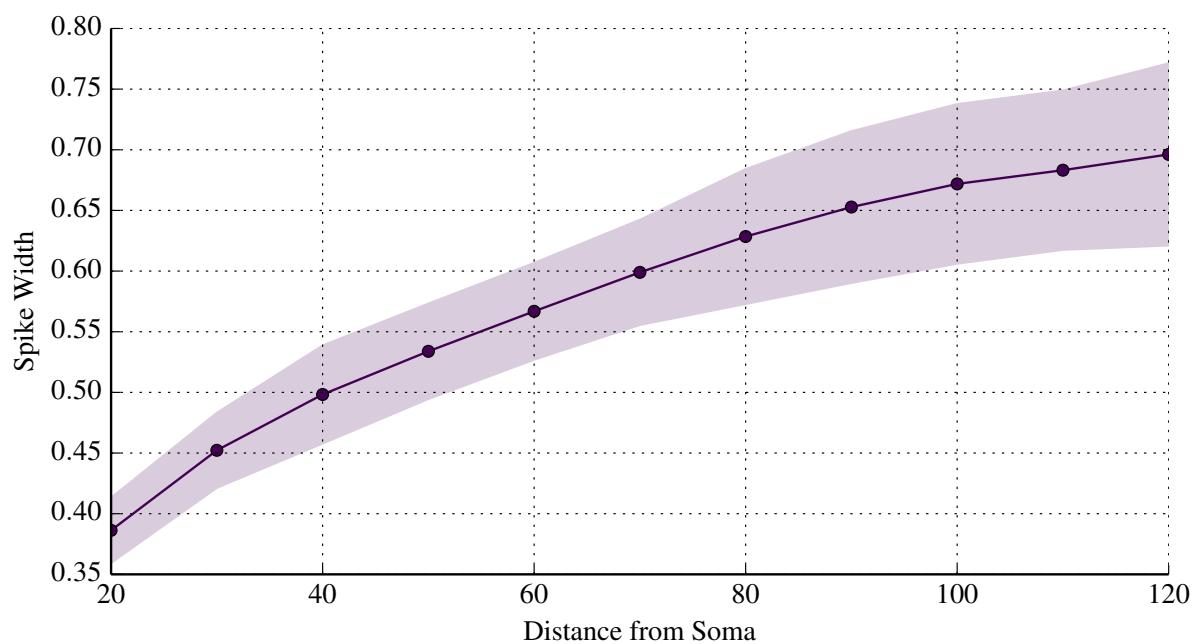
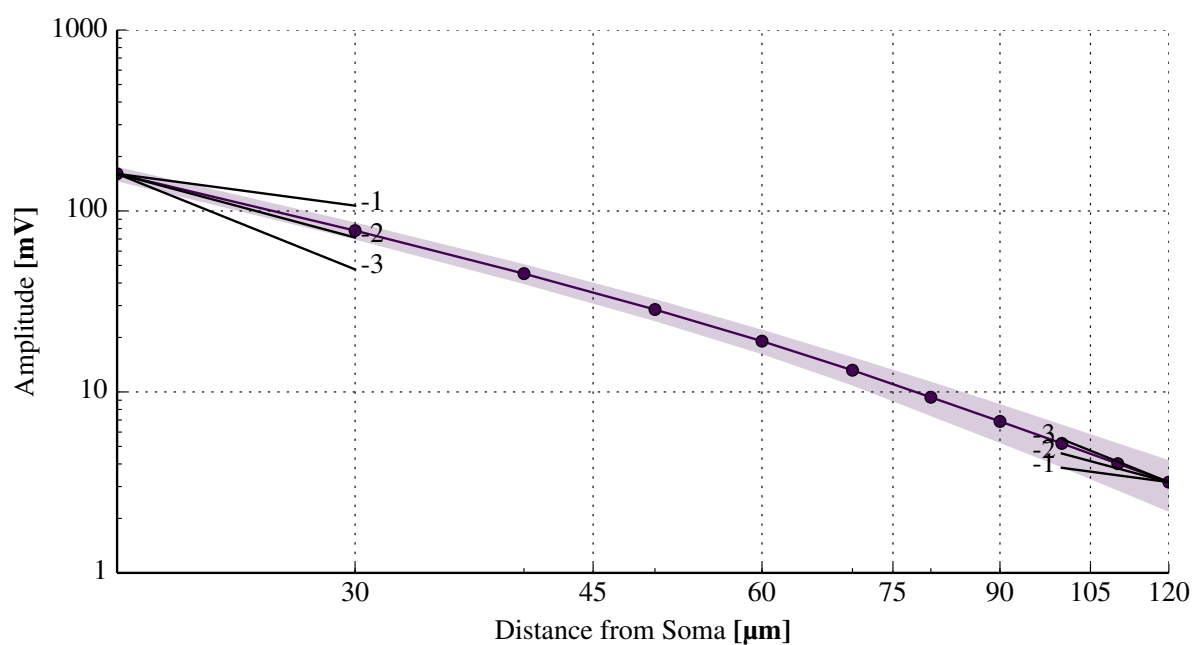Figure 4.6: Spike width over distance. Mean +/- 1 std.



Figure 4.7: Spike amplitude over distance. Mean +/- 1 std. The power law decays $1/r$, $1/r^2$ and $1/r^3$ are shown at the leftmost and rightmost data points.

section covers an analysis of two different commonly used spike definitions that have been used to classify interneurons and pyramidal neurons.

The two definitions are the peak-to-peak spike definition (Type I) and width at half amplitude from baseline (Type II). To investigate which of the two width definitions is more optimal for differentiating interneurons from pyramidal neurons, three classes of the most abundant neurons were selected from the blue brain models. Two classes of interneurons and one class of pyramidal neurons. There were two classes of pyramidal models available but as the one class had identical dendrites it was not analyzed. The number of inhibitory neuron classes were much greater, the two classes were selected by being the most abundant inhibitory neurons in the L5 area. In addition to these classes, one addition group was analyzed named "All Inter." which included all available interneuron models in L5. In this group many of the classes represent only a very small portion of the number of neurons in L5. The findings from Markram et al. [8] suggest that overall the ratio between excitatory and inhibitory neurons is around $87\% \pm 1\%$. Of these, $50\%$ were classified as baskets cells (LBC and NBC). The interneurons from L5 were seperated into 9 m-types and 10 e-types which gave a total number of 48 uniquie models. As such some small classes of interneurons are overrepresented in the grouped called "All Inter.".

The classes of neurons were Thick Thufted Pyramidal Cell with an early bifurcating apical thuft (TTPC2), Large Basket Cell (LBC) and Nest Basket Cell (NBC). Each of the three class had 5 seperate models where each model had different m-type but identical e-type. The e-type of TTPC2 class was continuous adapting (cAD), LBC was delayed stuttering (dSTUT) and NBC was continuous non-accommodating (cNAC) (Markram et al. [8, p. 463]).

Simulations were ran using using the SphereRand (section 3.3.1) simulation . A good definition is recoqnized by having a better seperation between inhibitory and excitatory neurons. The results of the simulations can be seen in fig. 4.8. For both deifinitions the spike width of interneurons are smaller than the width of pyramidal neurons. These findings are in line with previously established findings. With the Type I definition the seperation between the two classes are greater in both absolute and relative value for all distances from soma. The seperation between the mean of pyramidal and interneurons for Type I were $0.40\,\mathrm{ms}$ at $30\,\mathrm{\mu m}$ and $0.60\,\mathrm{ms}$ at $100\,\mathrm{\mu m}$. For Type II the seperation was $0.15\,\mathrm{ms}$ at $30\,\mathrm{\mu m}$ and $0.35\,\mathrm{ms}$ at $100\,\mathrm{\mu m}$. These results suggsts that using a type I deifinition of the spike width increases the chance of correctly classifying the neuron class.

A further distinguishment can be seen using the coefficient of variation $c_v$ (also known as signal to noise ratio, SNR) of the two width definitions (fig. 4.9).

$$c_v = \frac{\sigma}{\mu} \tag{4.1}$$

In many cases the lower $c_v$ value will be more robust to noise.

## 4.3   Optimal Amplitude Definition

The two amplitude definitions investigated here are the amplitude from baseline (Type I) and the peak-to-peak amplitude definition (Type II). The simulations were ran using the same neurons and simulations as the previous section.
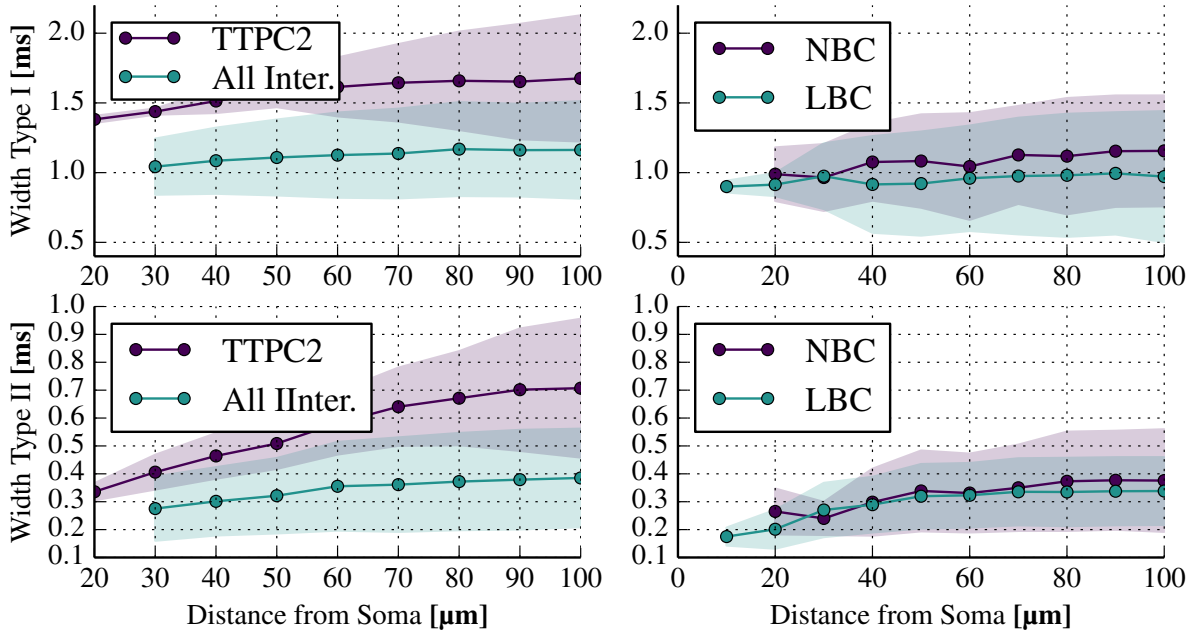
Figure 4.10 shows the

Figure 4.8: Nothing.

## 4.4  Classifying Inter and Pyramidal Neurons

Blue brain reconstruced neurons and indentified 55 types of morphological structures.

Excitatory neurons represented above $87\%$ of all neurons in L5.

50 % of all inhibitory interneurons are baskets cells (LBC and NBC), these types have been simulated.

All models from blue brain from L5 was ran using the SphereRand simulation and were divided between interneurons and pyramidal neurons.
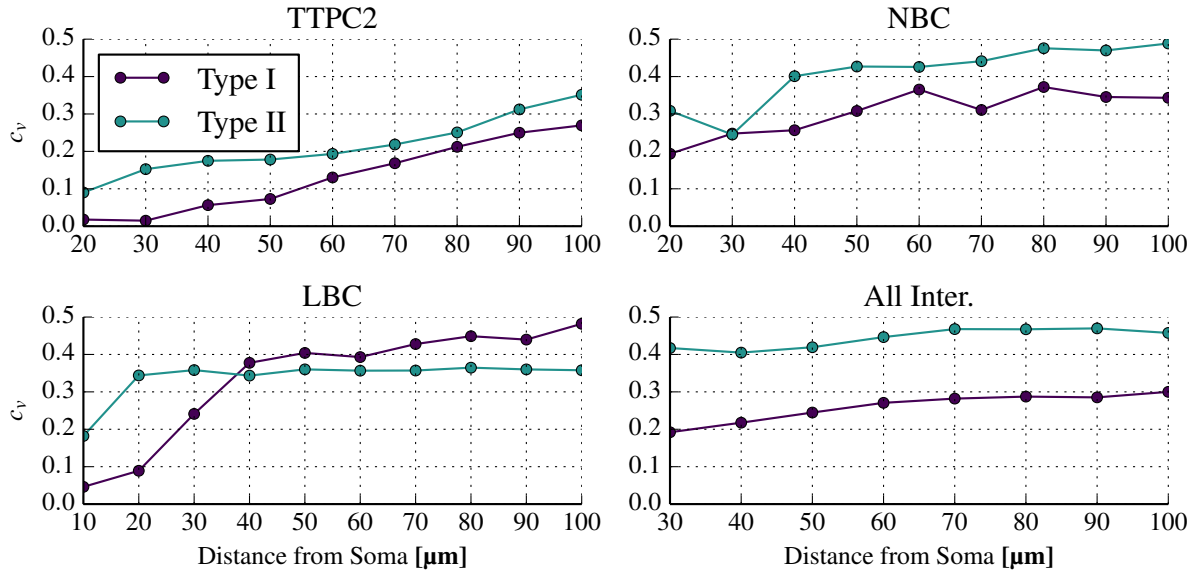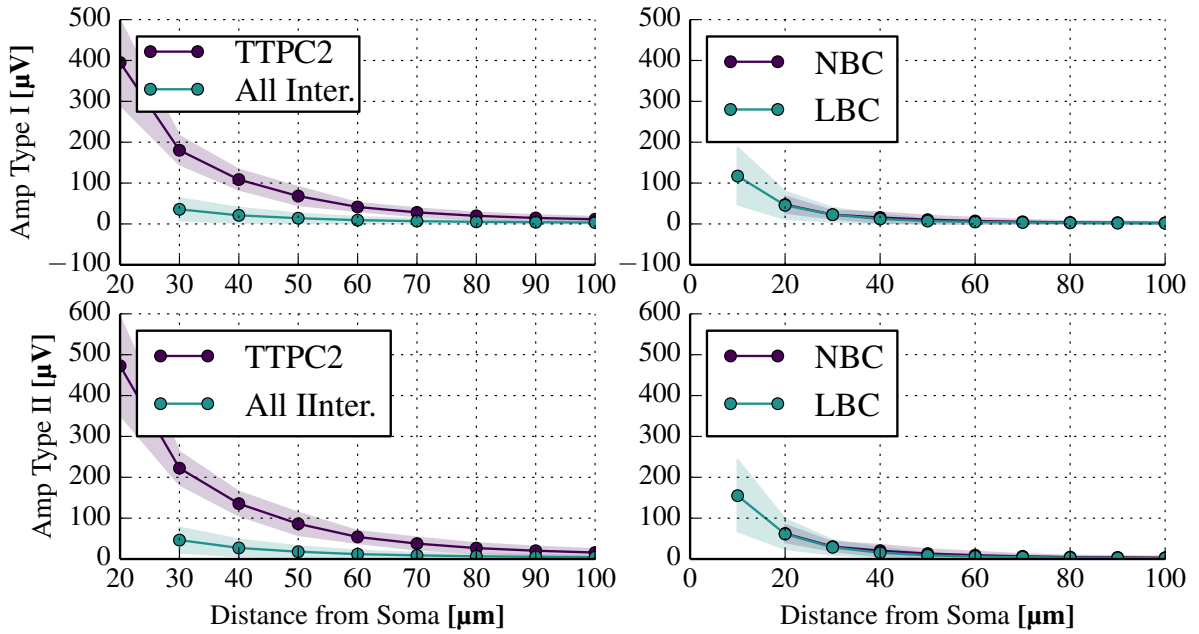
Figure 4.9: Nothing.



Figure 4.10: Nothing.

# 5 | Discussion

# Bibliography

[1] J. A. Connor & C. F. Stevens. "Prediction of repetitive firing behaviour from voltage clamp data on an isolated neurone soma." In: *The Journal of Physiology* 213.1 (1971), pp. 31–53.

[2] J. A. Connor, D. Walter, & R. McKown. "Neural repetitive firing: modifications of the Hodgkin-Huxley axon suggested by experimental results from crustacean axons." In: *Biophysical Journal* 18.1 (1977), pp. 81–102. ISSN: 0006-3495. (Visited on 11/13/2015).

[3] Peter Dayan & Laurence F. Abbott. *Theoretical neuroscience*. Vol. 806. Cambridge, MA: MIT Press, 2001. (Visited on 11/12/2015).

[4] Matti Hämäläinen et al. "Magnetoencephalography- theory, instrumentation, and applications to noninvasive studies of the working human brain." In: *Reviews of modern Physics* 65.2 (1993), p. 413.

[5] Alan L. Hodgkin & Andrew F. Huxley. "A quantitative description of membrane current and its application to conduction and excitation in nerve." In: *The Journal of physiology* 117.4 (1952), pp. 500–544.

[6] Henrik Lindén et al. "LFPy: a tool for biophysical simulation of extracellular potentials generated by detailed model neurons." In: *Frontiers in neuroinformatics* 7 (2013).

[7] Zachary F. Mainen & Terrence J. Sejnowski. "Influence of dendritic structure on firing pattern in model neocortical neurons." In: *Nature* 382.6589 (1996), pp. 363–366. (Visited on 11/13/2015).

[8] Henry Markram et al. "Reconstruction and simulation of neocortical microcircuitry." In: *Cell* 163.2 (2015), pp. 456–492.

[9] Klas H. Pettersen & Gaute T. Einevoll. "Amplitude variability and extracellular low-pass filtering of neuronal spikes." In: *Biophysical journal* 94.3 (2008), pp. 784–802.

[10] David Sterratt et al. *Principles of computational modelling in neuroscience*. 2011.