# Neuroprothetics Exercise 5
# Multicompartment Model

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## 1 Multicompartment Hodgkin–Huxley model

In this exercise, a multicompartment Hodgkin–Huxley model was implemented. A single compartment of the multicompartment model works the same as in exercise 4, which only models an idealized spherical neuron, which, in reality, fails to be an accurate assumption. Therefore, the stimulated action potential in this exercise propagates between compartments to simulate the stimulus transmission in a myelinated neuronal axon.

### 1.1 Model and Stimulus

The model consists of 100 separate compartments that are each modelled as in exercise 4. In this exercise, the compartments are connected via an axonal resistance, which was calculated to be

$$R_a = \frac{\rho_{axon} * l_{comp}}{\pi * (r_{axon})^2} = 7957\,\Omega\,, \tag{1}$$

where $\rho_{axon}$ is the electrical resistivity ($\rho_{axon} = 1\,\Omega\text{m}$), $l_{comp}$ the length of a compartment ($l_{comp} = 0.1\,\mu\text{m}$) and $r_{axon}$ the radius of the axon ($r_{axon} = 2\,\mu\text{m}$). For simplicity, these values were considered to be constant.

The model then was used to simulate the response to two different input stimuli, demonstrated in Figure 1.

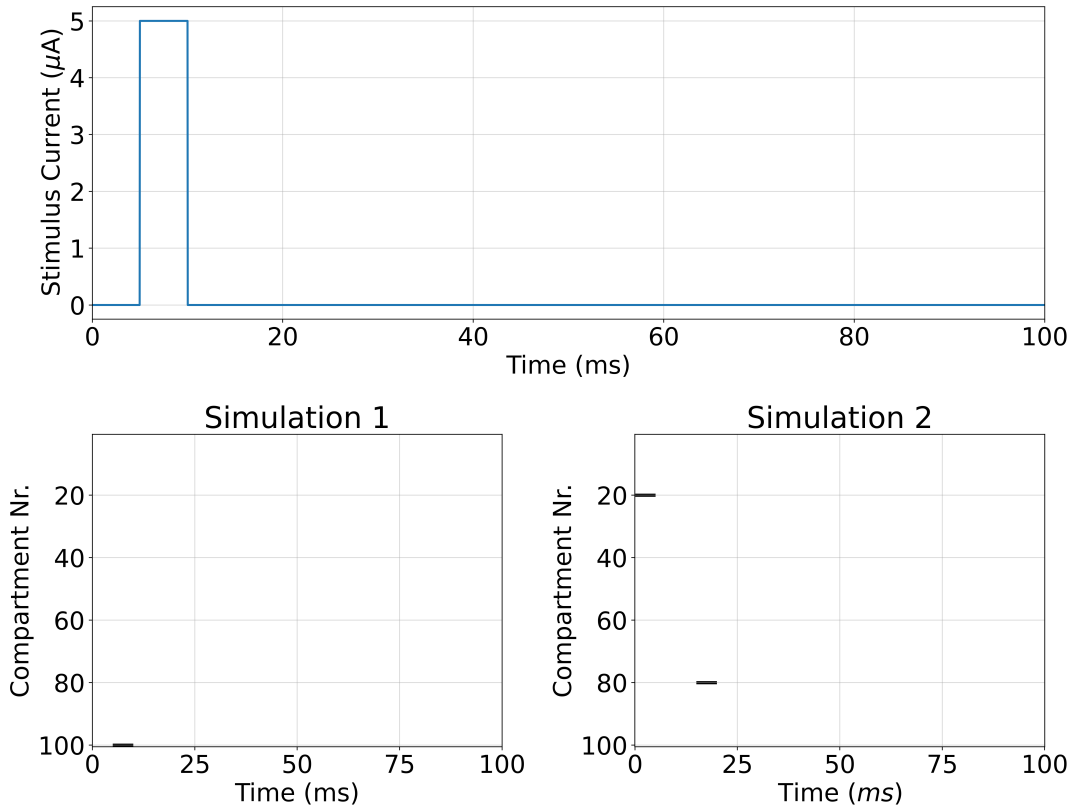


Figure 1: Visualization of input stimuli. The top plot shows the input current for one stimulated compartment in Simulation 1. The bottom plots demonstrate the stimuli per compartment and time for the corresponding simulation. In Simulation 2, the same stimulus current as in Simulation 1 was used, but at different times.

## 2 Results

The resulting signal propagation modelled by the multicompartment model is shown in Figure 2

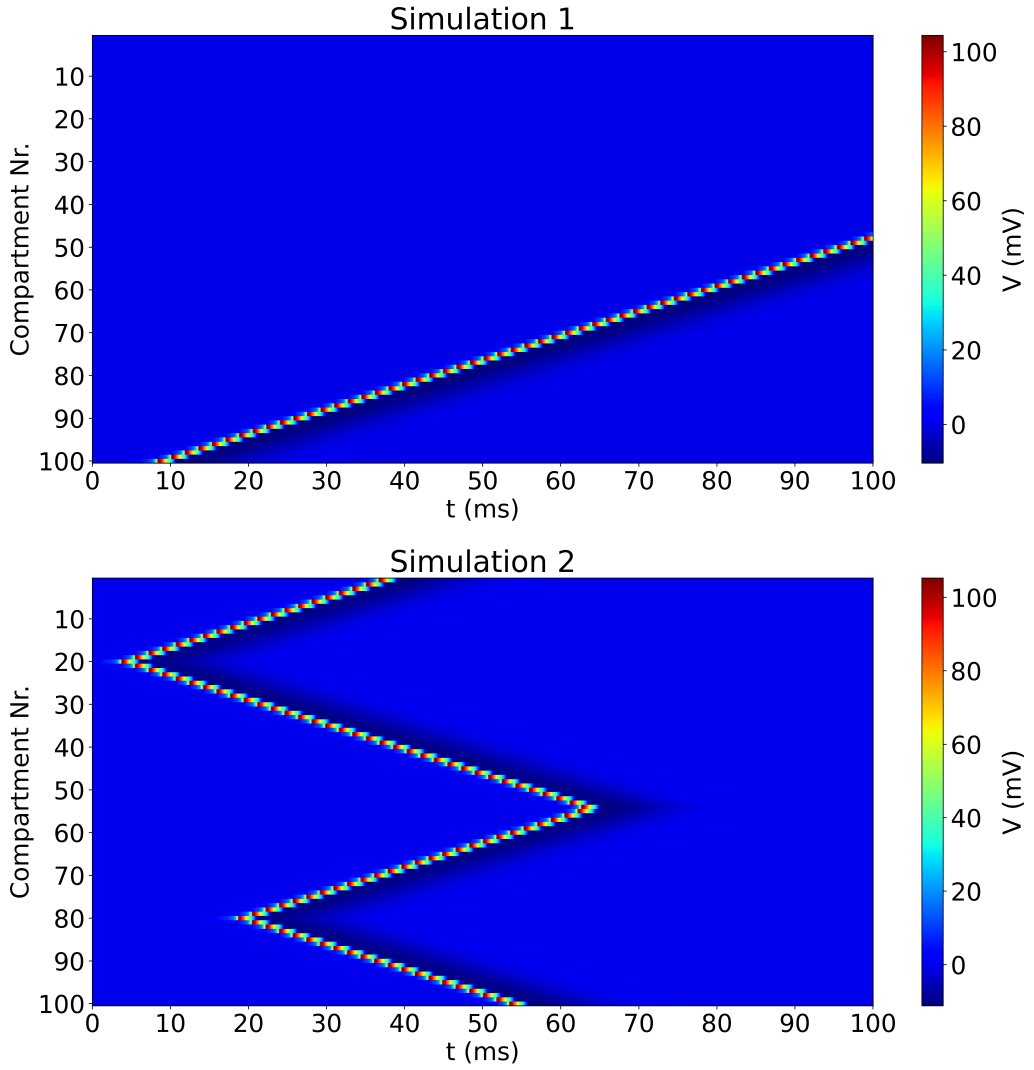


Figure 2: Multicompartment Hodgkin–Huxley model with the two stimulation as shown in Figure 1. The colour bar indicates the current membrane potential.

# 3 Discussion

## 3.1 Propagation profiles

In simulation 1, we can see a clear and intuitive propagation of the Signal along the compartments with time (Figure 2 top plot). Each stimulated compartment is able to raise the potential of the adjacent one above the threshold to induce an action potential.

In simulation 2, however, we observe that signals that approach the same compartment from different directions vanish as soon they "collide" (Figure 2 bottom plot). This phenomenon is due to the refractory period of the compartments. After the compartment where the signals met, each signal faces a bunch of compartments that had recently exhibited an action potential due to the opposing signal and, therefore, are in a refractory period where they cannot be stimulated again. This results in the signal coming to a stop. The (relative) refractory period, where only a strong stimulus could induce an action potential, can be seen in the dark blue, shadow-like regions in Figure 2, where the membrane potential is $V_m < 0$. Between threshold-crossing and $V_m = 0$, there is the absolute refractory period, which is responsible for the signal stopping, as just described.

## 3.2 Influence of membrane capacity and axonal resistance on model

Each compartment represents a node of Ranvier. It can be modelled as an RC element where $R$ is the membrane resistance and $C = C_m$ is the membrane capacity. Each compartment is connected to the previous and next one by the axonal resistance $R_a$.

### 3.2.1 Membrane capacity

The influence of the membrane capacitance can be understood by considering the RC properties of the compartments. The membrane capacity acts like a buffer for the ionic current, which becomes larger when the capacity increases. Therefore, the membrane potential rises much slower, which can eventually lead to the capacitor's potential never reaching the firing threshold since a certain current can always "leak" through $R_a$. In the attachment (Figure 3), the effect of a membrane potential that is twice as big as the initial one is displayed. There you can also see that in Simulation 1, a firing potential is reached in the very last compartment where the stimulation took place, but not in the second compartment or in the compartments in the second simulation. This is due to the last compartment only having one adjacent $R_a$ where the current can leak. In the other cases, though, current can leak through two different $R_a$'s, essentially doubling the leakage current, resulting in the current integration in the capacitor being too slow to reach a membrane potential that could trigger an action potential. Vice versa, cutting the membrane capacitance in half leads to a faster rise of the membrane potential and, therefore, also to a faster firing and faster transmission to the next compartment, as shown in the attachment (Figure 4).

### 3.2.2 Axonal resistance

The axonal resistance limits the current that is transmitted from one compartment to another. As long as the current is still high enough to induce an action potential, the next compartment will be triggered to initiate an action potential. In the attachment (Figure 5), you can see the model's performance with an axonal resistance twice as high as the original. Due to the mitigated current, the signal takes longer to induce an action potential. After the third compartment, the current isn't high enough to induce another action potential in the fourth compartment. On the other hand, halving the axonal resistance just leads to a faster propagation of the signal since the firing threshold will always be reached but faster, as demonstrated in the attachment (Figure 6).

## 3.3 Effects of myelinization and change in axonal diameter

The effects of myelinization of the axons, as well as a change in the axonal diameter, can be related to the previously described properties of membrane capacitance and axonal resistance.

The myelinization of an axon can be thought of as adding multiple RC elements in series to the model between nodes of Ranvier. That decreases the total membrane capacitance and increases the membrane resistance (but not axonal resistance!). This ensures small membrane currents in the myelinated part of the axon but doesn't affect the resistance along the axon. As explained before, this leads to a quicker surpassing of the threshold potential in the adjacent node of Ranvier and, eventually, to a faster propagation of the action potential. On the other hand, a not sufficiently myelinated axon might lose the action potential completely due to the leaking current.

The change of the axonal diameter, however, changes the axonal resistance inversely quadratically since $R_a \propto \frac{1}{d^2}$. Therefore, doubling the axon's diameter will result in a $R_a$, which is four times smaller. The effect of a change in the axonal resistance to the propagation of the action potential was already described in section 3.2.2. However, it still needs to be considered that a larger axonal diameter also increases the surface of the membrane, which leads to a larger membrane capacity. Although the membrane capacity only scales linearly with the diameter, making the axon thicker would also lower the membrane resistance, effectively increasing the leaking current. That's why evolution settled for myelinization rather than thicker axons.

# Attachments

## Increased membrane capacitance

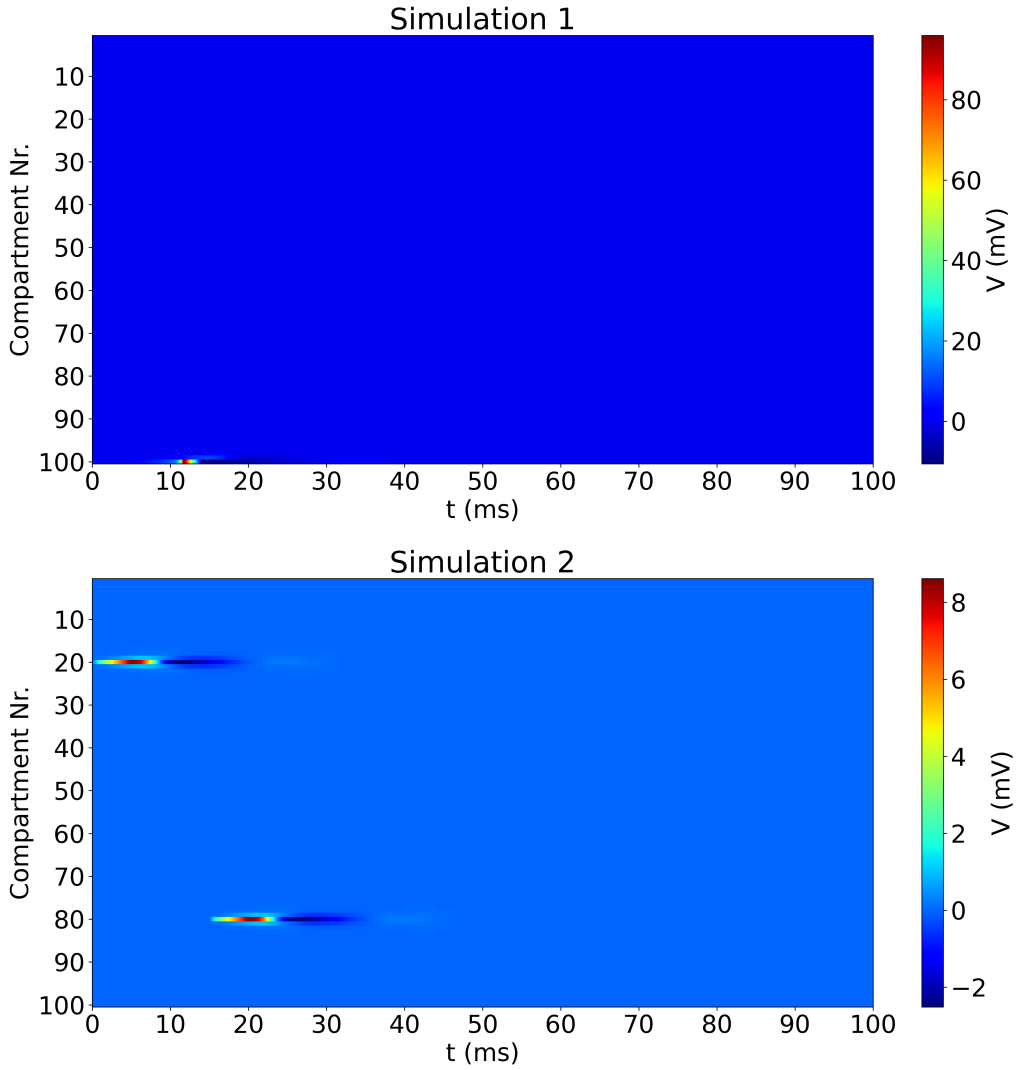


Figure 3: Multicompartment Hodgkin–Huxley model with $C_{m,new} = 2 * C_m$. Note the different colour bars. In simulation 2, no voltage spike above 9 mV is reached.

## Decreased membrane capacitance

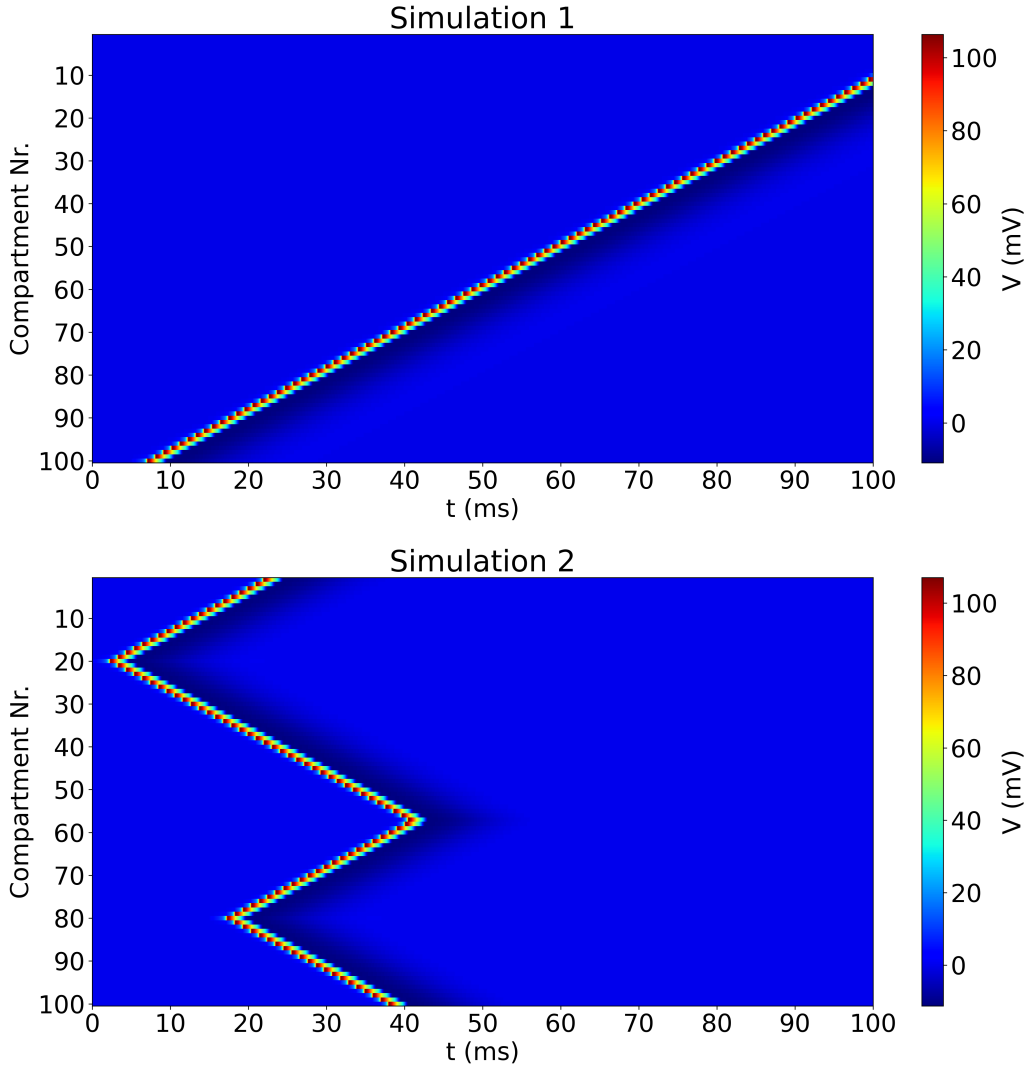


Figure 4: Multicompartment Hodgkin–Huxley model with $C_{m,new} = \frac{1}{2} * C_m$.

**Increased axonal resistance**

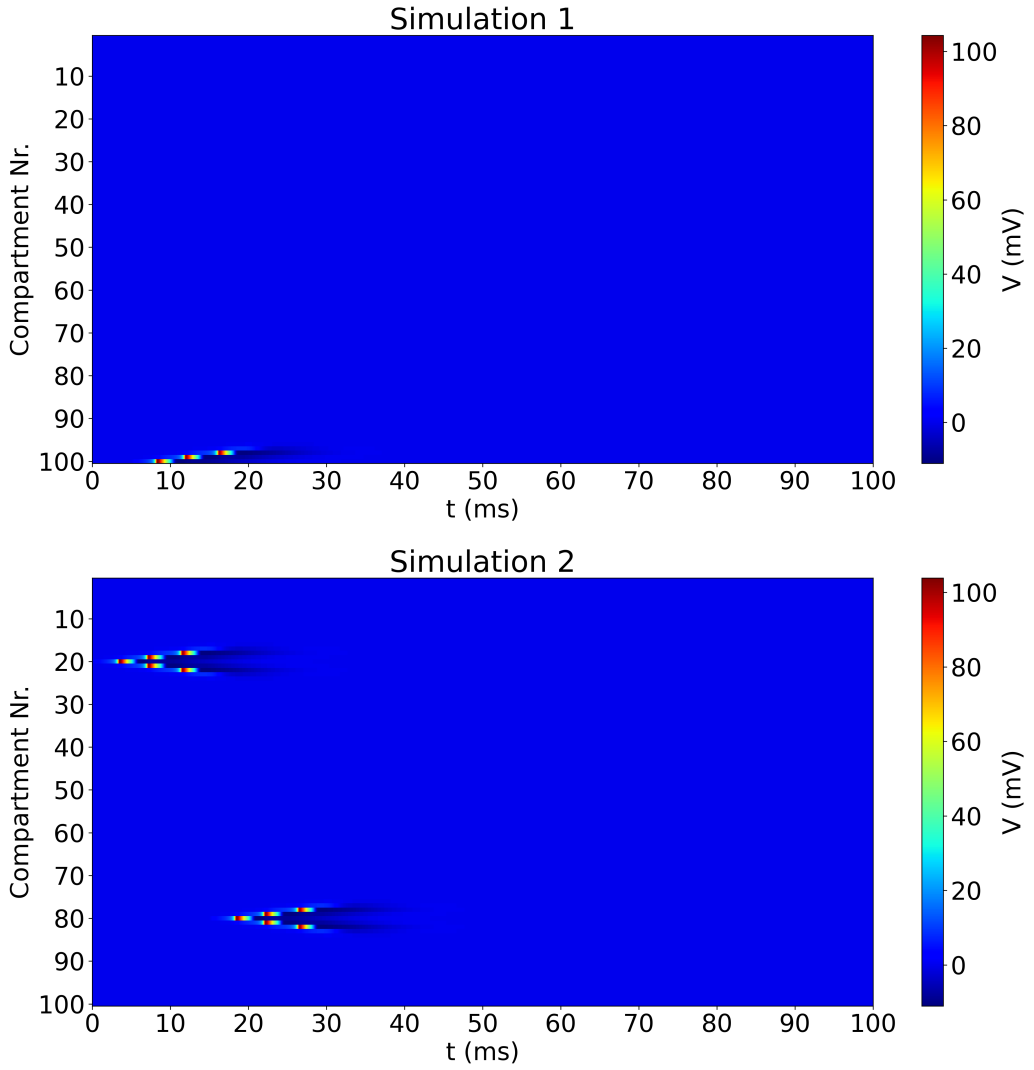


Figure 5: Multicompartment Hodgkin–Huxley model with $R_{a,new} = 2 * R_a$. Note that only the axonal resistance was changed, although a change in diameter would also affect the membrane capacity, as explained in section 3.3.

### Decreased axonal resistance

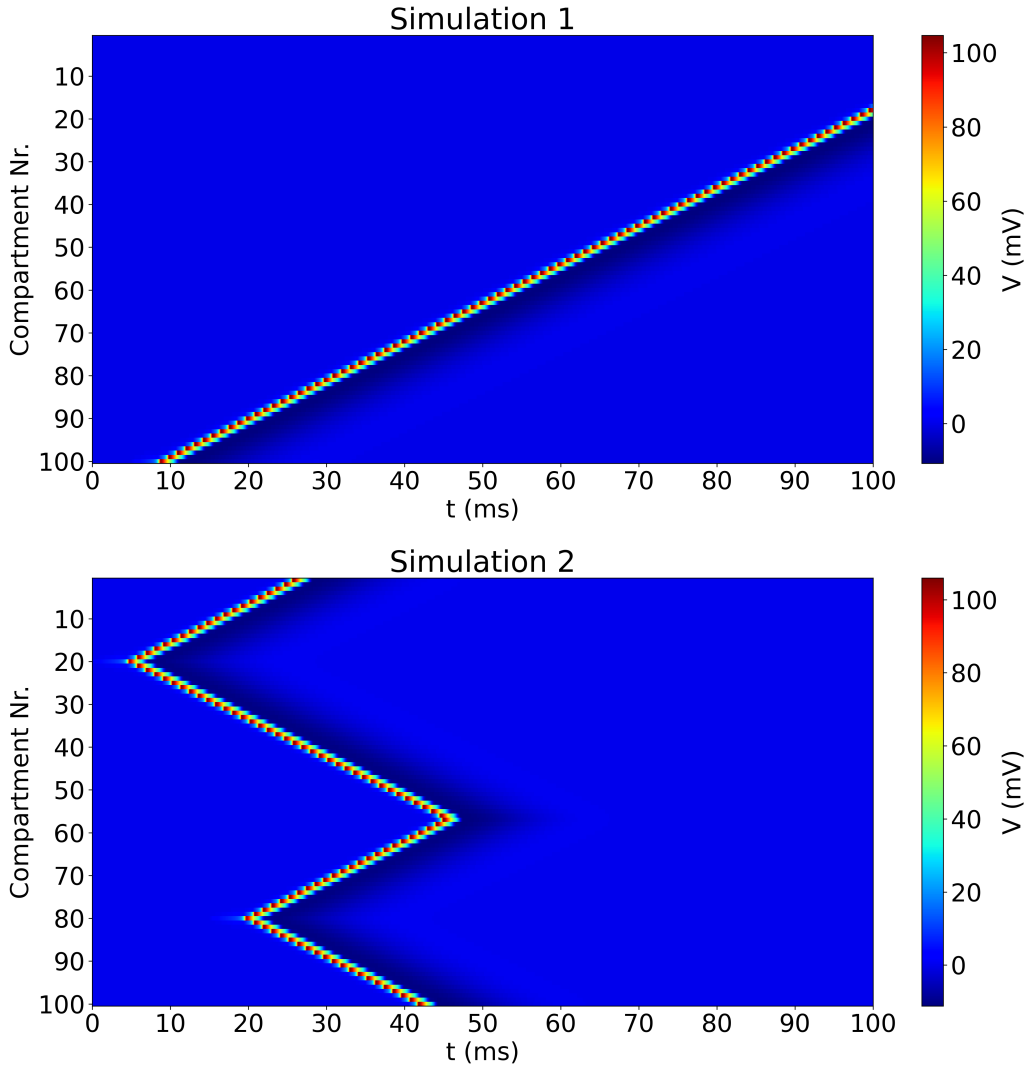


Figure 6: Multicompartment Hodgkin–Huxley model with $R_{a,new} = \frac{1}{2} * R_a$. Note that only the axonal resistance was changed, although a change in diameter would also affect the membrane capacity, as explained in section 3.3.