

# CS346 HW3 Writeup

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## Exercise One:

### Voltage over time:

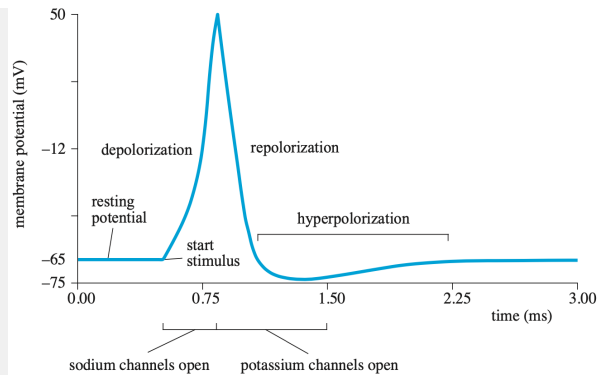
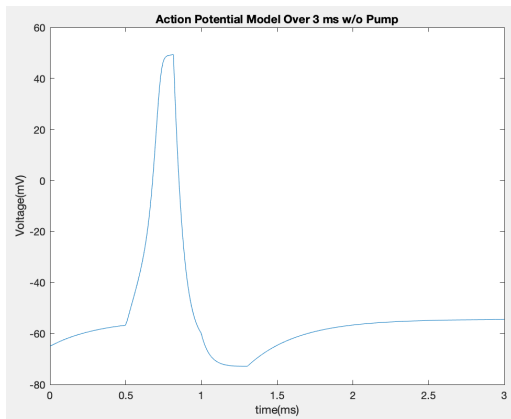


Figure 7.9.2 Action potential with membrane potential (mV) versus time (ms)

Without the  $\text{Na}^+\text{-K}^+\text{-ATPase}$  pump, the voltage  $V$  slowly increased from equilibrium, rather than being canceled by the pump, from time 0 to time 0.5 where the stimulus current was added. This slow increase continued through hyperpolarization, causing the voltage to rise past the equilibrium point.

$n$ ,  $m$ , and  $h$ -values over time:

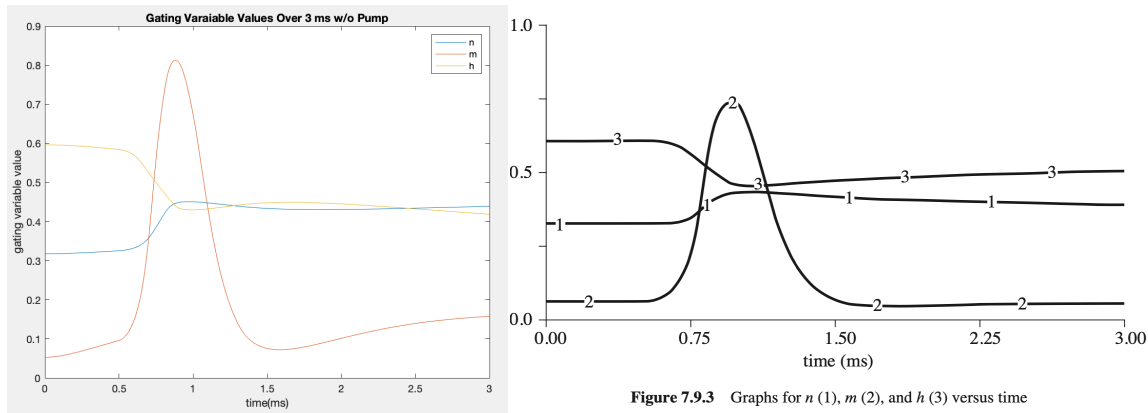


Figure 7.9.3 Graphs for  $n$  (1),  $m$  (2), and  $h$  (3) versus time

The value of potassium gating value  $n$  was equal to the value of potassium gating value  $h$  around time .8. This is distinct from the textbook, where these two gating values never overlap but similar to the addition of the Na<sup>+</sup>-K<sup>+</sup>-ATPase pump. Unlike the simulation with the Na<sup>+</sup>-K<sup>+</sup>-ATPase pump, the potassium gating value  $h$  and potassium gating value  $n$  continued to weave back and forth, overlapping continuously. In the Na<sup>+</sup>-K<sup>+</sup>-ATPase pump simulation, the two values never intersect after the first two overlaps.

## Exercise Two:

\*Formatting note: the graphs are in order of the current model, previous exercise's model, and textbook's output.

### Voltage over time:

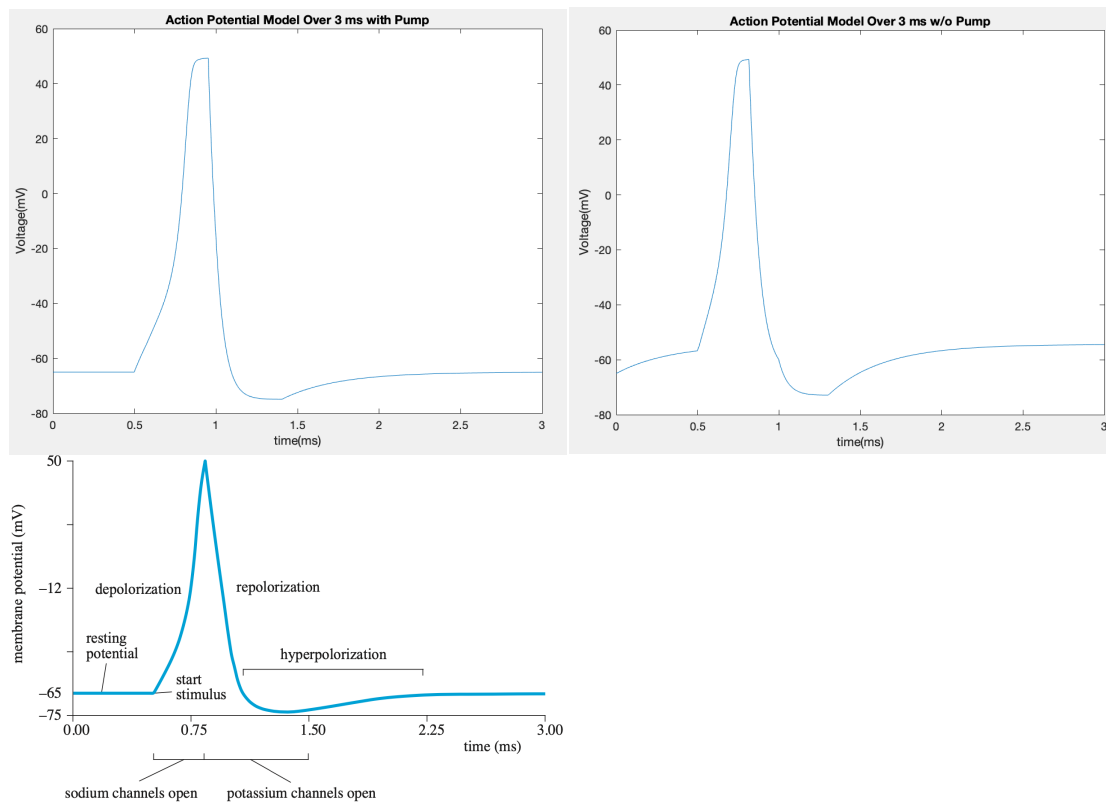


Figure 7.9.2 Action potential with membrane potential (mV) versus time (ms)

Unlike exercise one without the  $\text{Na}^+-\text{K}^+-\text{ATPase}$  pump, the voltage  $V$  remains constant until the stimulus current is introduced. Similarly, this equilibrium is restored after the hyperpolarization occurs. This difference exists because the  $\text{Na}^+-\text{K}^+-\text{ATPase}$  pump accounts for the leakage that takes place during equilibrium.

n, m, and h-values over time:

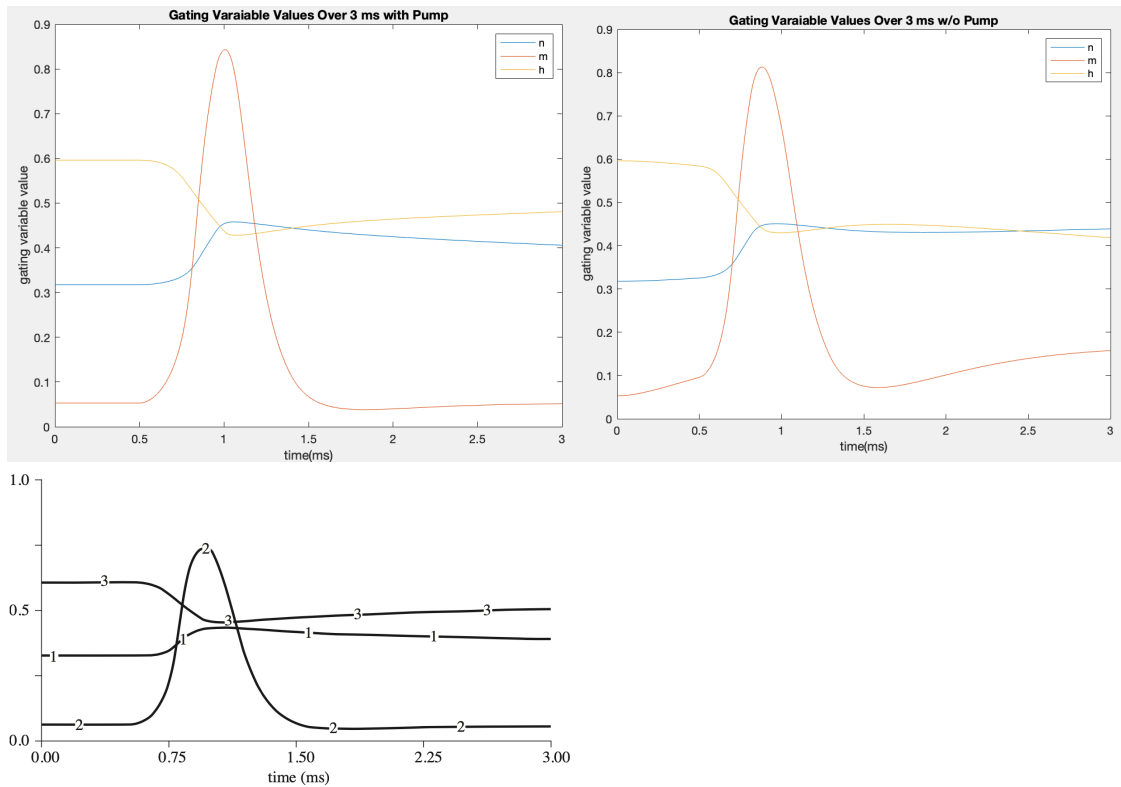


Figure 7.9.3 Graphs for  $n$  (1),  $m$  (2), and  $h$  (3) versus time

With the addition of the  $\text{Na}^+\text{-K}^+\text{-ATPase}$  pump, we now see the potassium gating value  $n$  and potassium gating value  $h$  in equilibrium after the hyperpolarization. This is similar to the textbook's output. It happens here rather than exercise one because of the  $\text{Na}^+\text{-K}^+\text{-ATPase}$  pump's ability to maintain voltage equilibrium. However, it still exists where the potassium gating value  $n$  exceeds the potassium gating value  $h$  during hyperpolarization. This never happens in the textbook's output. Furthermore, the sodium gate value  $m$  is also shown to be in equilibrium apart from during the action potential. This is because the  $\text{Na}^+\text{-K}^+\text{-ATPase}$  pump is maintaining it.

## Exercise Three:

\*Formatting note: the graphs are in order of the current model, previous exercise's model, and textbook's output.

### Voltage over time:

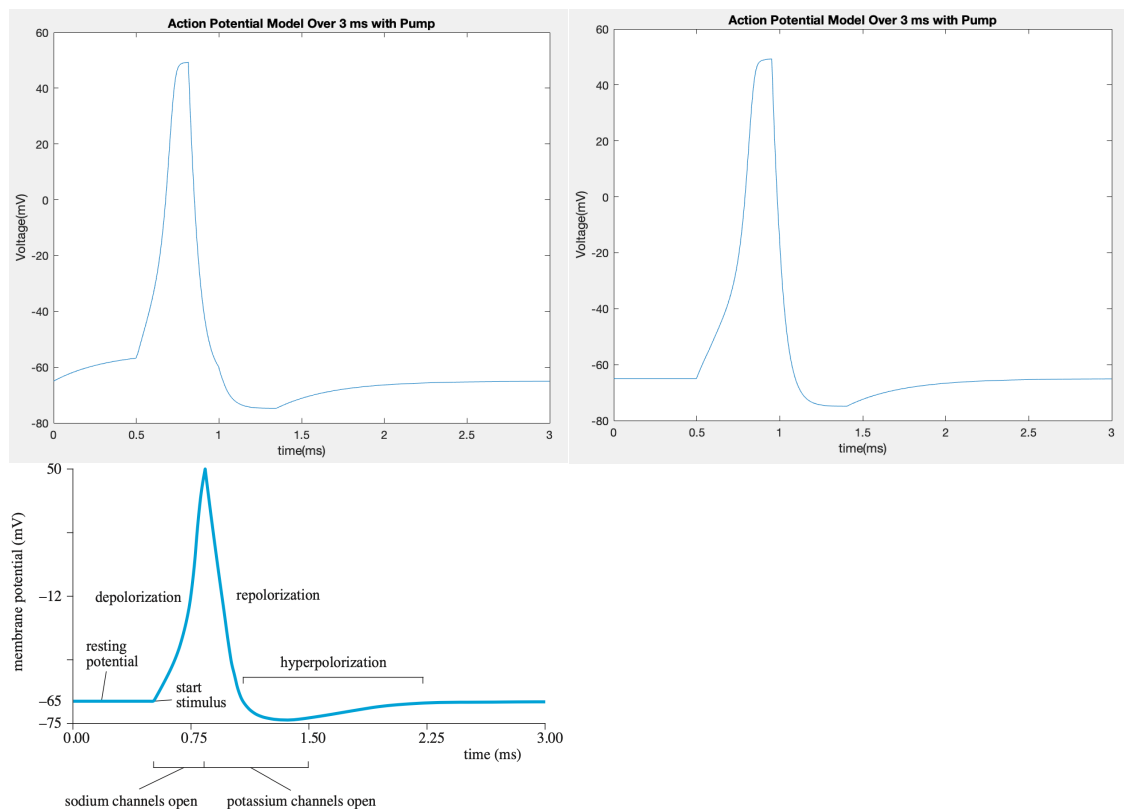


Figure 7.9.2 Action potential with membrane potential (mV) versus time (ms)

Here we find the model from exercise three deviating from both the textbook output and the model from exercise two. In exercise two, we had the  $\text{Na}^+\text{-K}^+\text{-ATPase}$  pump running during initialization. However, in this exercise, we set a condition where in addition to the potassium gate being closed, the neuron must be in the hyperpolarization phase. This means that the  $\text{Na}^+\text{-K}^+\text{-ATPase}$  pump would maintain equilibrium and prevent leakage only after the action potential has taken place. This leads to the curved beginning to the model.

$n$ ,  $m$ , and  $h$ -values over time:

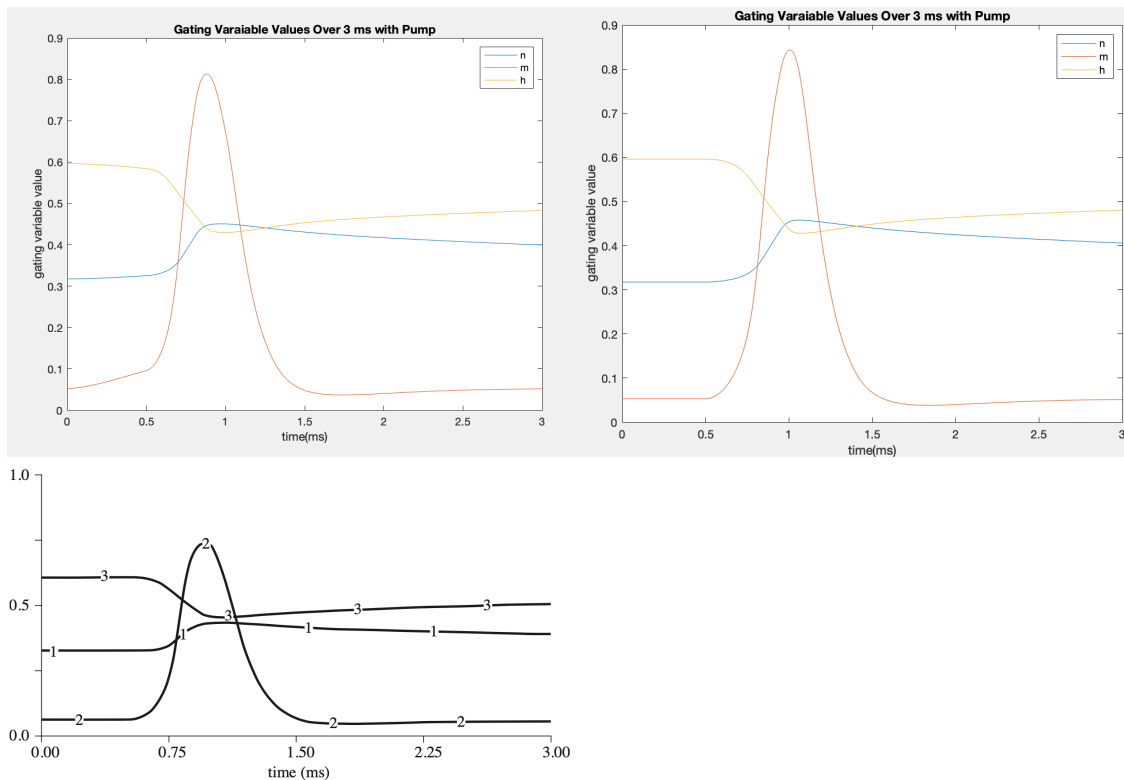
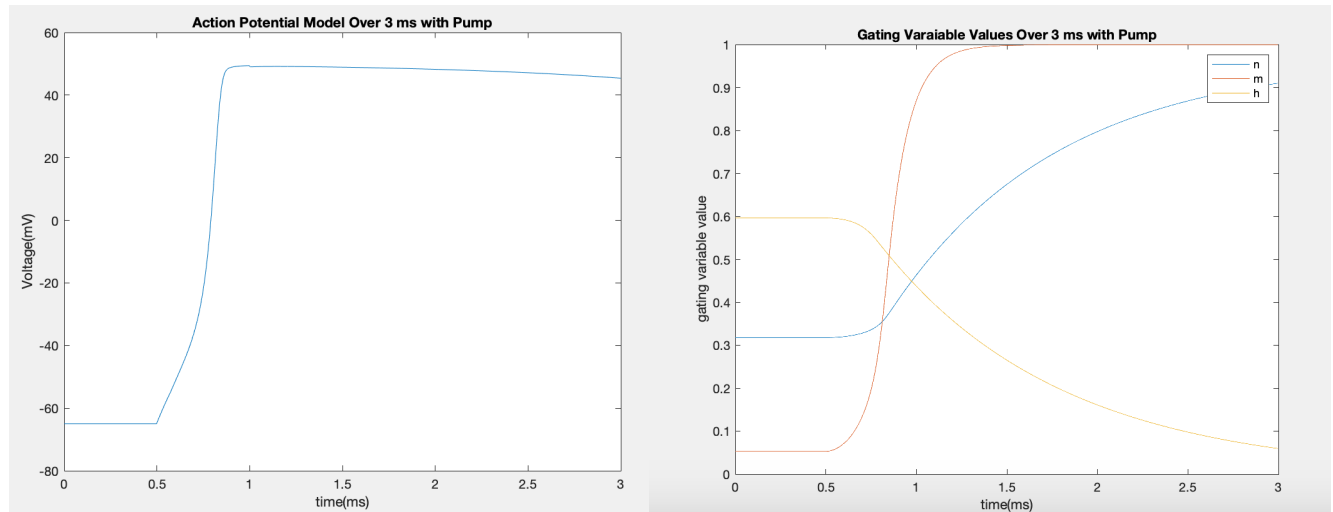


Figure 7.9.3 Graphs for  $n$  (1),  $m$  (2), and  $h$  (3) versus time

Similar to the voltage graph, exercise three's model deviates from both the textbook's output and exercise two's model. Since the  $\text{Na}^+\text{-K}^+\text{-ATPase}$  pump isn't active at initialization, we can see the sodium gating value  $m$  slowly increase in value. This results in a lower spike in the sodium gating value  $m$  as well. This is because the voltage has already increased due to leakage, so the sodium gate doesn't need to be as activated. Because the conditions are met after the action potential, we find that the sodium gate  $m$  values remain the same in both models after the action potential. The potassium gating value  $n$  and potassium gating values  $h$  remains unchanged between the three models.

## Exercise Four:



The threshold of 50mV is never reached, so the potassium channel never opens, the sodium channel never closes and repolarization never takes place. This idea is supported by the value of the gating variable  $m$ . It reaches 1 as a value supporting the likelihood that the sodium channel remains active. Instead, leakage remains at a maximum, slowly reducing voltage.

## Exercise Five:

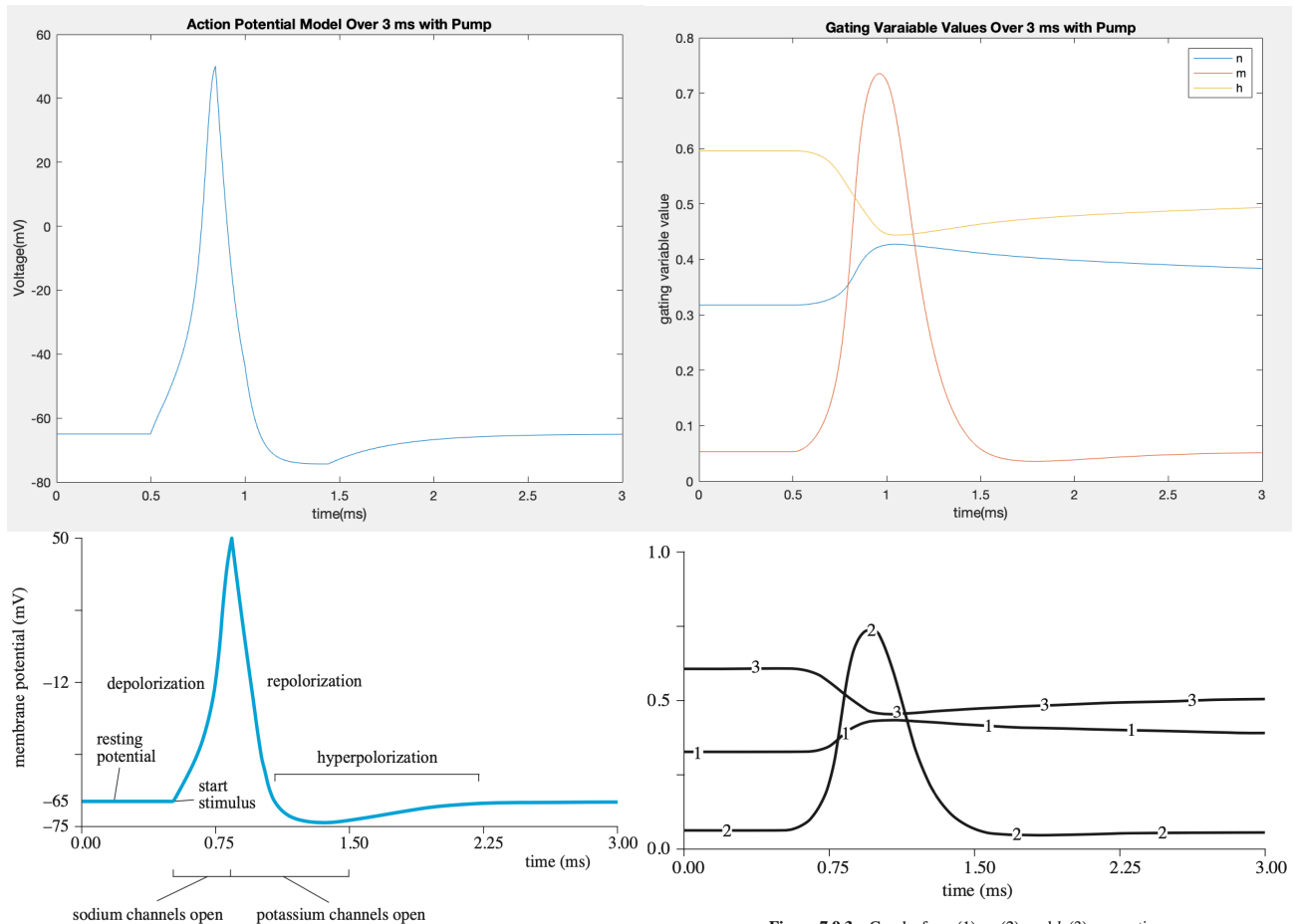


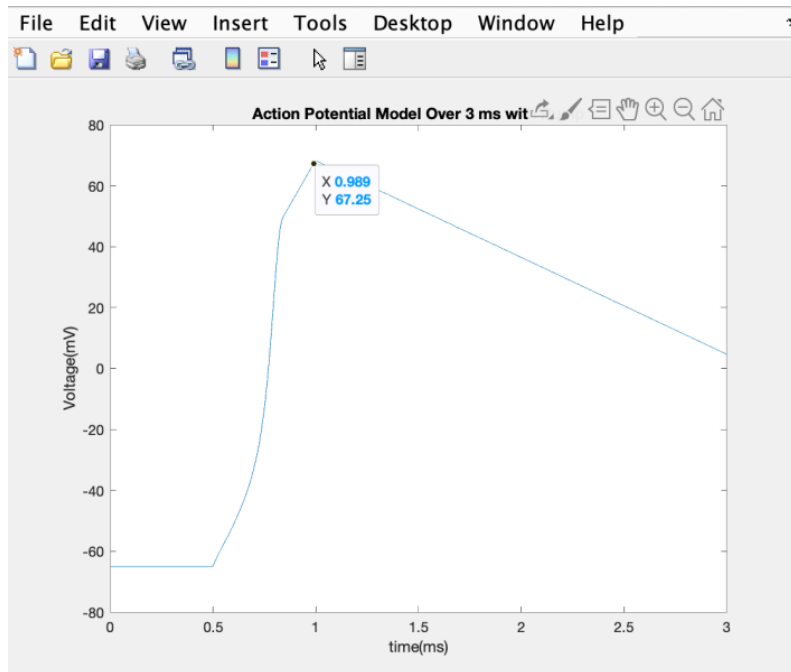
Figure 7.9.2 Action potential with membrane potential (mV) versus time (ms)

Figure 7.9.3 Graphs for  $n$  (1),  $m$  (2), and  $h$  (3) versus time

In this exercise we set a voltage threshold for the gating of the leakage channel, meaning it was only open when the value of voltage was less than -54.4 mV. The result of this produces a model seen above as almost identical to the given examples of HH in the textbook. These models appear to make sense as at the beginning of the model, voltage is at its resting value implying leakage is open and counteracting the  $\text{Na}^+/\text{K}^+$ -ATPase pump. The channel then closes and the current becomes 0 when the applied current is introduced at 0.5 ms. The lack of this leakage channel increases the active effect on voltage for the Sodium channel resulting in a sharper and greater increase in overall voltage. Then the system works as expected as potassium channels open and sodium channels close, causing the voltage to plummet and enter hyperpolarization. At this point, the leakage channel is open again, allowing for a steady return back to near resting potential.



## Exercise Six:



Our first idea for getting the action potential to a maximum higher value was to decrease the value of the potassium conductance from 36 millisiemens per centimeters squared to 2 millisiemens per centimeters squared. This, in turn, resulted in the maximum voltage of the action potential reaching a peak of around 67.25 Mv, increasing from around 50 Mv. This makes sense because it the potassium that helps to hyperpolarize the neuron, lower the voltage, so by decreasing its conductance it can not transmit as much voltage out of the neuron

Our other idea focused around the inclusion of more channels for different ions. After some investigation, we learned that the inclusion of Calcium channels, which in an actual neuron open towards the peak voltage of the action potential, act as almost another stimulus causing the voltage to momentarily jump to a higher apex. This, however, is beyond the scope of our current model.