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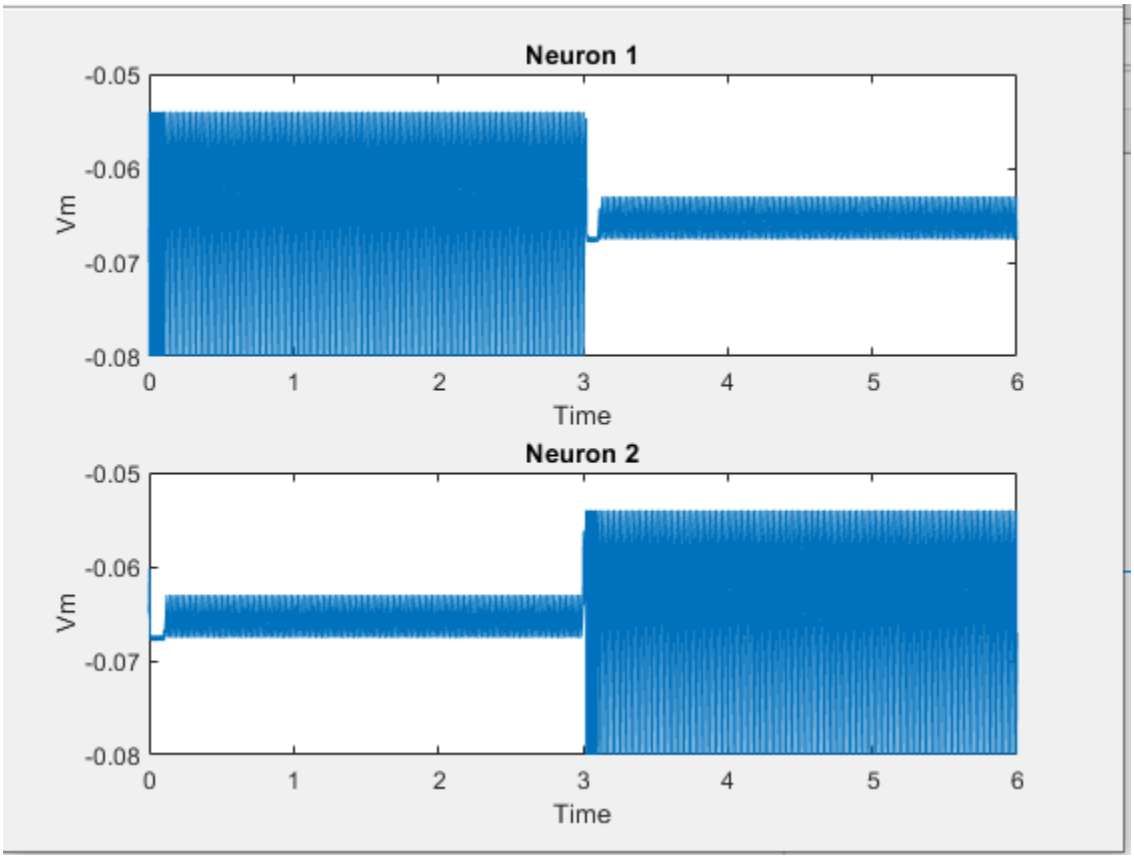
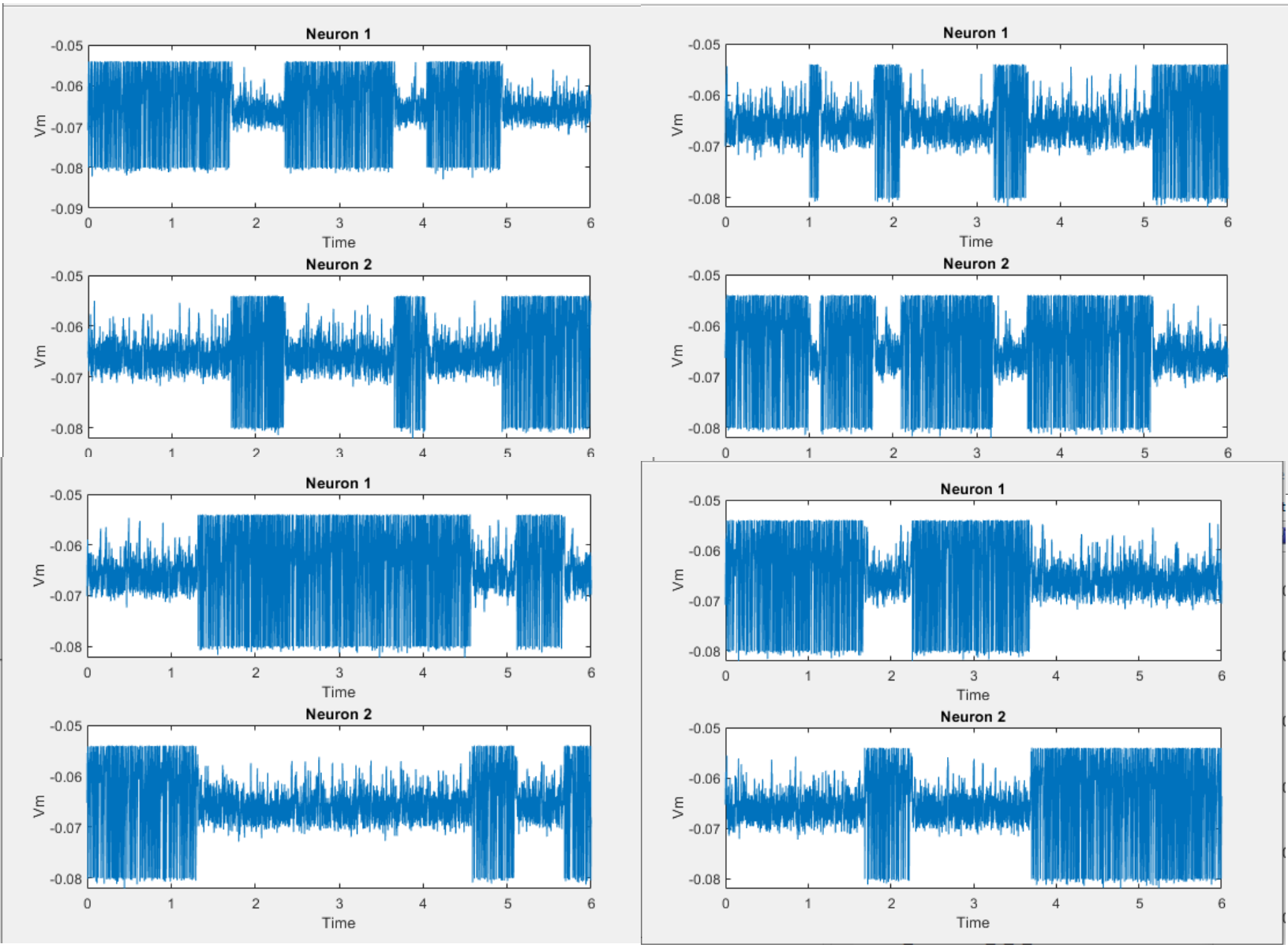
Tutorial 5.3

# Aii. )



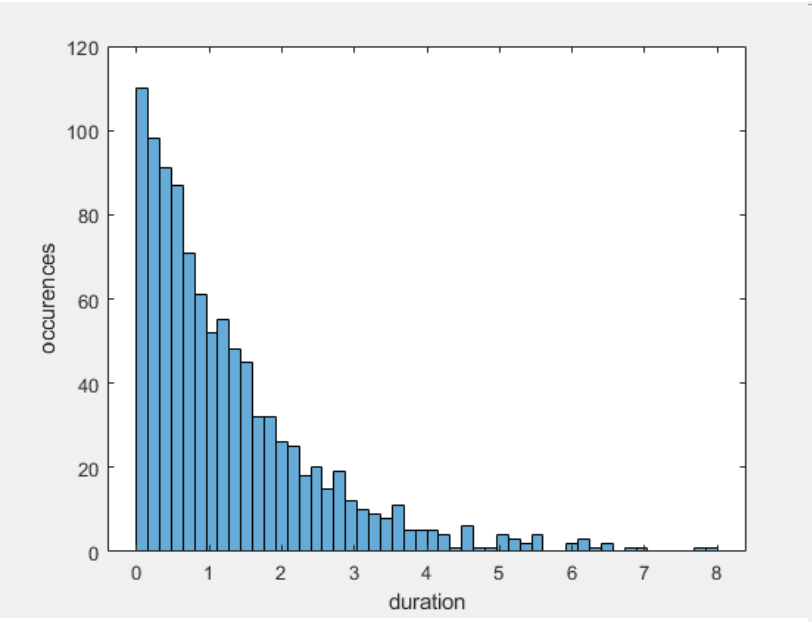

In the above trial, two LIF neurons connected together through inhibitory synapses are simulated without noise or synaptic depression. As one might imagine, a LIF neuron without such added characteristics in attempt to simulate something slightly closer to biological results in a static, deterministic behavior. Neuron 1 is provided with excitatory input for the first 100ms of the simulation, making it active. When neuron 1 fires it provides inhibitory input to neuron 2 through a step increase in the synaptic gating variable. This causes neuron 2 to become less likely to fire. Given that each neuron has a stable state of high firing rate with the baseline input, one neuron will continue to fire and suppress the other until the system is interrupted. Which neuron is active can be swapped by providing sufficient excitatory input to the inactive neuron, as seen above at the 3 second mark. When neuron 2 is provided excitatory input powerful enough to overcome neuron 1's inhibition, it spikes, inhibiting neuron 1 and preventing it from further spiking. In this way the system is bistable, as either neuron can be in an "active" or "inactive" state depending on the opposite neuron.

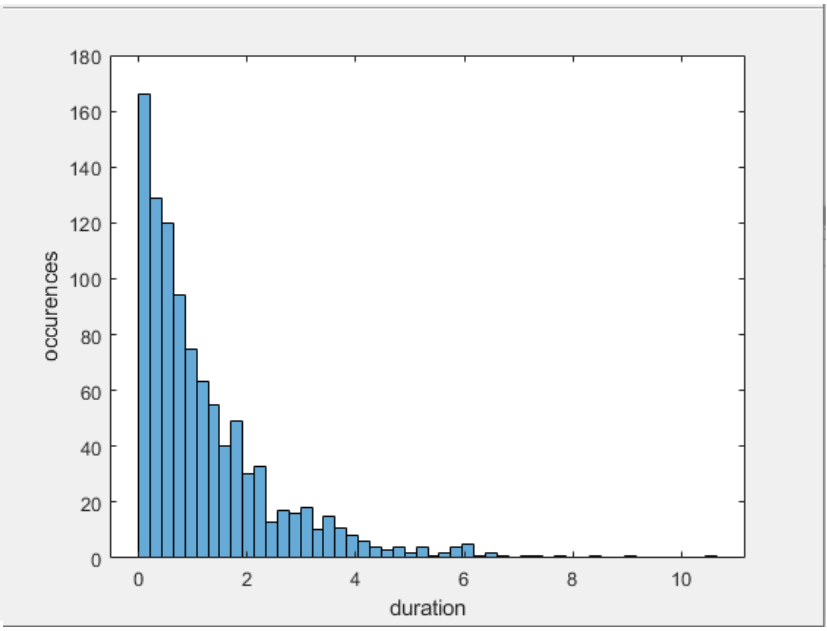
# Aiii.)




The above are the results from 4 separate trails of the LIF neurons with inhibitory connection, this time with an added noise term instead of charge introduced at discrete timepoints. Each neuron when provided noise has some amount of fluctuations in its membrane potential in either state, but at any time point there is still one neuron which is "active" and one that is being suppressed. This is illustrated by the clear decrease in activity of one neuron while the other is spiking. When a neuron is provided sufficient excitatory noise to overcome the inhibition it is receiving from the currently spiking neuron, their roles switch. The paradigm tend towards one of two states because of the inhibitory connections, making it significantly less likely for the inhibited neuron to spike, and therefore more likely that the currently active neuron will continue spiking.

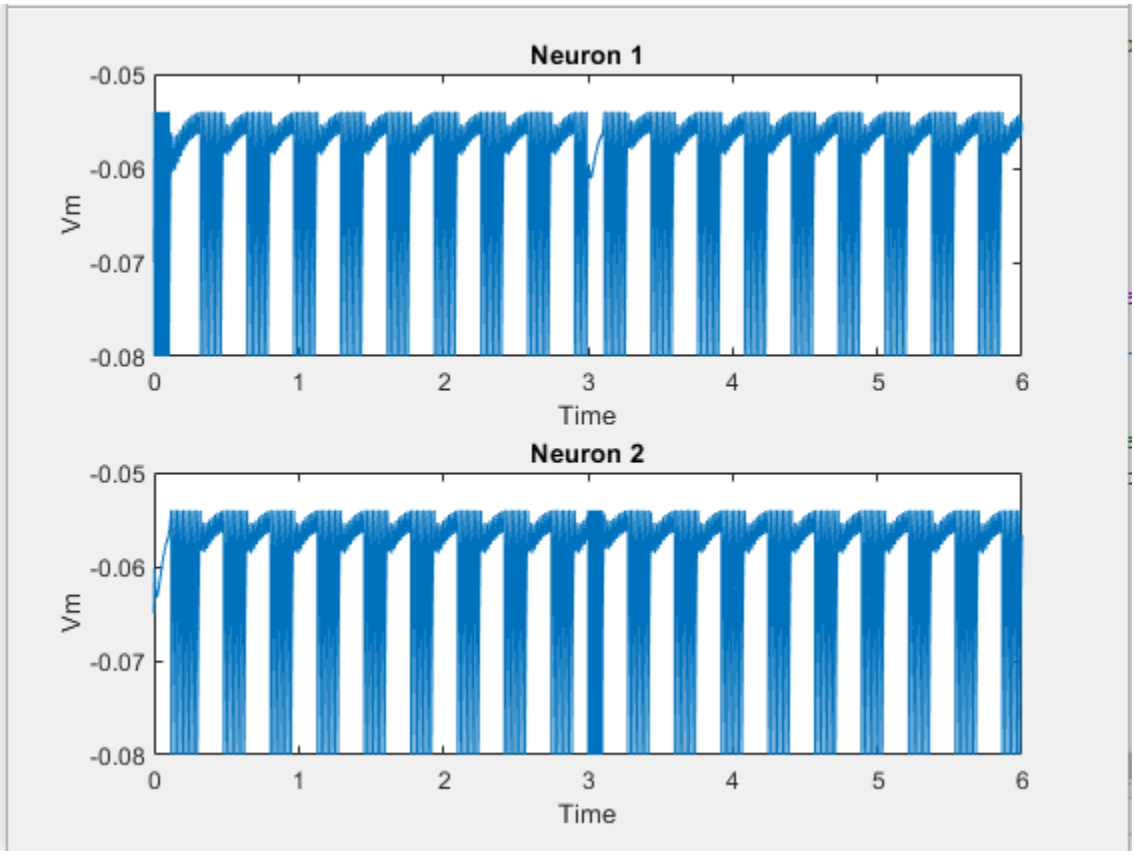
# Aiv.)




The above are histograms of the duration of each state in a 1000+ second simulation of the same parameters described in Aiii. Fitting a curve to the histogram would reveal an exponentially decaying gradient. In the case where random noise is applied to both neurons such that they are equally likely to be provided enough input to promote or discourage the continuation of the current state, a vast majority of states last less than 1 second. The exponentially decreasing gradient makes intuitive sense, as the less states that last beyond 1 second, the less are able to exist in bins outside of this range. To put it more succinctly, the underrepresentation of longer states isn't necessarily due to some factor keeping the states from crossing a threshold, rather the randomness of noise encourages a swap with a given likelihood at any small window of time, making it decreasingly likely that a state lasted long enough to reach the 5 second + mark.

# Bv.)

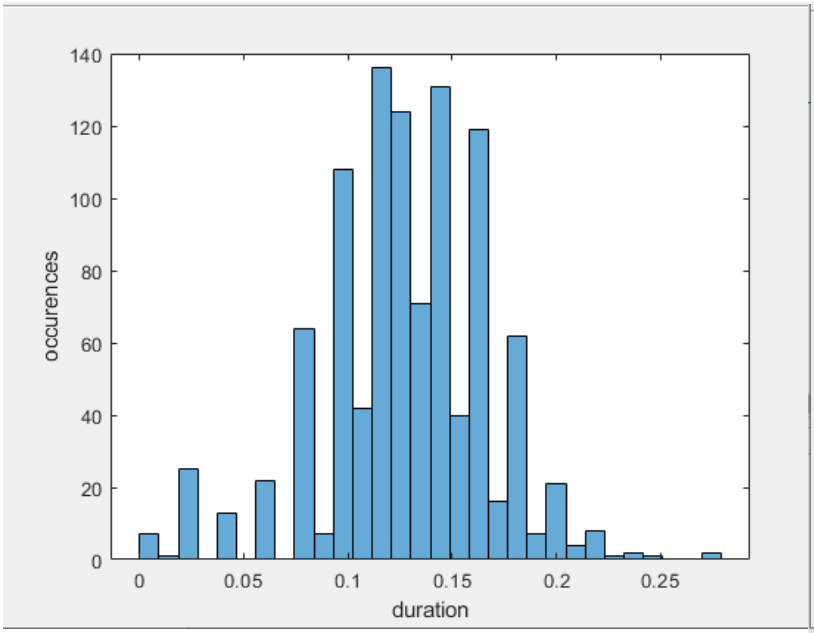


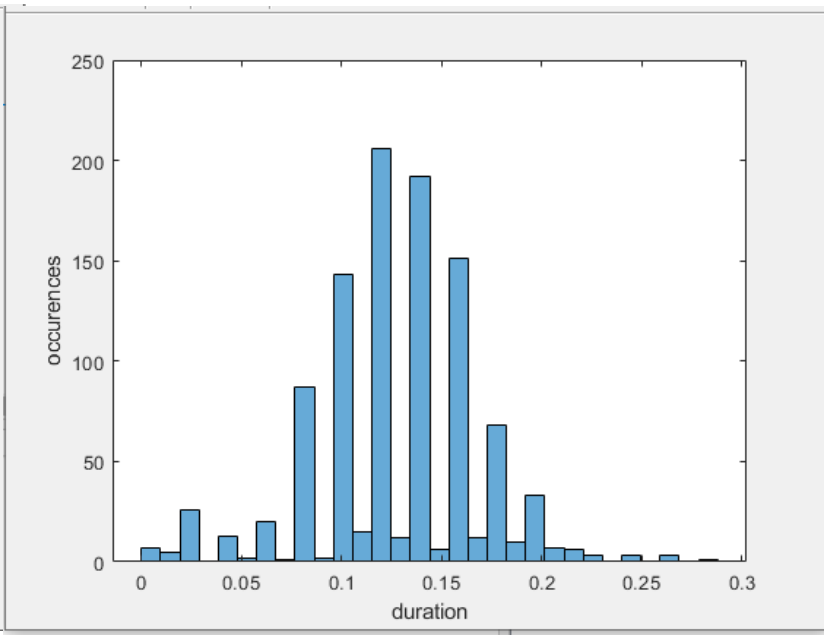

Above is a simulation of the same model as Aii, except with simulated dynamics of synaptic depression. Synaptic depression changes the model activity from an unchanging one of two bistable states to oscillatory. This is clearly illustrated by the absence of spiking activity of neuron 2 when neuron 1 is spiking and vice versa. The cycle length should depend on the probability of vesicle release and depression time constant (for reasons detailed below). Indeed, the active vs inactive state of each neuron seems to swap about 5 times each second, which is consistent with the depression time constant and probability of release values used in simulation, both of which being 0.2.

When a neuron spikes, its depression variable is updated according to the probability of vesicle release. This is because synaptic depression is essentially a measure of how many vesicles are ready for release at the time of a spike, and as this number decreases, so too does the relative impact of a spike. The depression variable then begins a growth back to its initial value of 1 (representing 100% vesicle readiness) over the order of the depression time constant. After an oscillatory cycle, one neuron typically has a depression variable near 0, and the other near 1. As time goes on, the neuron with a near 0 depression variable slowly grows towards 100% vesicle readiness, a process that takes an amount of time according to the depression time constant. The opposite neuron gradually decreases in vesicle readiness as it

spikes, eventually making its spikes have next to no impact on the other neuron. At this time, the role's reverse, as the previously active neuron is no longer inhibiting the previously suppressed neuron, and the previously suppressed neuron has regenerated its release ready vesicles.

# Bvi.)




The above is histograms from 2 trials with both synaptic depression and small amounts of noise. There are many differences in distribution of time in states from Aiv caused by the inclusion of synaptic depression. Firstly, synaptic depression eliminates the previously only remote chance of larger state durations (one the order of 1 second +). This is because synaptic depression by definition puts a limit on how long a high firing rate neuron can effectively impact another neuron with which it is connected. After about 0.2 seconds, the synaptic depression variable of the active neuron has decreased enough to the point that it can no longer effectively inhibit the opposite neuron, making the opposite neuron require significantly less noise to begin spiking.

Additionally, the time bins above seem to have oscillatory number of occurrences, rather, there are adjacent time bins that have drastically different number of occurrences. This is made more clear when one decreases the size of the time bins (see below). Something is causing a disproportionately high number of occurrences at every 0.02 second mark (ish). My guess is that this is due to how exactly synaptic depression is updated and calculated in our model. Given that both probability of release is 0.2 and updating depression is always just

multiplying current depression value by (1-prob of release), it seems plausible that simply scaling the depression value down by multiplying by 0.8, and synaptic depression change being calculated as a difference between 1 and the previous depression value / 0.2 (depression time constant), are contributing to a reduction in likelihood of specific state durations.