Problem Set II – Please write all answers on this sheet.

1. A squid axon 1 mm in diameter has a resting membrane potential of -75 mV. A microelectrode is inserted into the axon near the middle. It is used to inject sufficient current to depolarize the membrane at that point to -70 mV. A second (recording) microelectrode is inserted into the membrane 6 mm away from the first. It records a membrane voltage of -72.5 mV.

   a. What is the space constant, \( \lambda \), of the axon?

   b. The axon (horizontally extended) is then submerged in heavy, nonconductive fluorocarbon oil to half its diameter (see figure below). What is the new space constant?

   ![](image.png)

   c. What biological phenomenon resembles the fluorocarbon experiment above? In what way?
2. You are given a frog sartorium neuromuscular junction as diagrammed. With this preparation, you can stimulate the presynaptic nerve fiber and record the endplate potential from the muscle at M. The initial bath solution is Ringer’s solution (i.e. a salt solution with \( \text{Na}^+, \text{K}^+, \text{Ca}^{2+}, \) and \( \text{Cl}^- \) concentrations adjusted to match those in frog blood).

Quantal size, \( v_1 \) (measured in mV), and mean quantal content, \( m \) (in dimensionless whole numbers) have been extensively discussed in connection with synaptic physiology.

a. What changes would you have to make in your preparation or recording circuitry before you could do quantal analysis on synaptic (sometimes termed junctional) transmissions?

b. What effect would you expect the following drugs applied to the superfusion solution to have on \( v_1 \) and \( m \). For each drug indicate whether you expect an increase (\( \uparrow \)), decrease (\( \downarrow \)), or no effect (NE), for both quantal size and mean quantal content. If your answer is incorrect, indicating what you know about the drug’s mode of action gets partial credit.

<table>
<thead>
<tr>
<th>DRUG</th>
<th>( v_1 )</th>
<th>( m )</th>
<th>Mode of action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cobalt</td>
<td>NE</td>
<td>( \downarrow )</td>
<td>blocks voltage-gated ( \text{Ca}^{2+} ) channels, decreasing presynaptic ( \text{Ca}^{2+} ) influx &amp; vesicle fusion</td>
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<tr>
<td>Curare (( \beta )-tubocurarine)</td>
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<td>( \alpha )-bungarotoxin (Btx)</td>
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<td>Organophosphorus Insecticides</td>
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<tr>
<td>Nifedipine</td>
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<tr>
<td>Tetraethylammonium (TEA)</td>
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<tr>
<td>Tetanus Toxin</td>
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<td>Phytostigmine</td>
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<tr>
<td>Acetylcholinesterase</td>
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</table>
3. A neuron in the human CNS has:

\[
\begin{align*}
E_K &= -60 \text{ mV} \\
E_{Na} &= +40 \text{ mV} \\
E_{Cl} &= -55 \text{ mV}
\end{align*}
\]
\[
\begin{align*}
V_{\text{rest}} &= -55 \text{ mV} \\
\text{threshold} &= -40 \text{ mV}
\end{align*}
\]

a. Synaptic channels in this neuron, which are opened by the neurotransmitter GABA, are selectively permeable to chloride ions. Would you expect GABA synapses to be \textbf{excitatory} or \textbf{inhibitory}? (Circle one answer here and in parts b and c) Why?

b. Other synapses on this neuron, which are responsive to glutamate, are equally conductive to sodium or potassium \((g_K = g_{Na})\). What is the reversal potential of such a glutamate responsive synapse on this cell? Is it \textbf{excitatory} or \textbf{inhibitory}?

c. Sodium and potassium ions are conducted by this neuron’s glycine-gated channels. However, these channels are six times as conductive to potassium ions as to sodium ions. Would you expect glycine-responsive synapses on this neuron to be \textbf{excitatory} or \textbf{inhibitory}? Why?