Neurobiology and Human/Animal Behaviour
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problem set #1

The Nernst Equation:

$$E = \frac{RT}{zF} \ln\left(\frac{Ko}{Ki}\right)$$

For a cation of unit valence, at room temperature,

$$E = 58\text{mV} \log\left(\frac{Ko}{Ki}\right)$$

The Goldman (a.k.a. Hodgkin-Huxley) Equation:

$$Vm = \frac{RT}{F} \ln\left(\frac{pK\,Ko + pNa\,Nao + pCl\,Cli}{pK\,Ki + pNa\,Nai + pCl\,Clo}\right)$$

At room temperature,

$$Vm = 58 \log\left(\frac{Ko + (pNa/pK)\,Nao + (pCl/pK)\,Cli}{Ki + (pNa/pK)\,Nai + (pCl/pK)\,Clo}\right)$$

**Relevant reading:** Nichols, Martin, & Wallace, *From Neuron to Brain*, chapters five through ten.

**QUESTIONS FROM LECTURE #1** (Don't worry so much about these; you'll get it again in more detail at the end of the term.)

1. Place the following brain subdivisions in order from rostral to caudal:
(a) mesencephalon, prosencephalon, rhombencephalon
(b) diencephalon, metencephalon, telencephalon, myelencephalon, mesencephalon

2. Early in embryogenesis, cells have differentiated into endoderm, mesoderm, and ectoderm. Which of these three tissues gives rise to the nervous system? Is the notochord derived from this same tissue? What signalling event triggers the differentiation of neural tissue, and how is the notochord involved in it?

3. In the developed animal, oculomotor nerves from the hindbrain innervate the eyes. Why is the cephalic flexure during embryogenesis critical to the establishment of this innervation?

4. What is the internal rostral-to-caudal structure of the embryonic rhombencephalon, and *in general* what sort of chemical signalling produces this structure?

## QUESTIONS FROM LECTURE #2

1. Explain why the side of the membrane having the higher concentration of the permeant cation (*e.g.* $K^+$) is electrically negative relative to the other side.

2. Using the following ion concentrations from frog muscle tissue:

| | external | internal |
|---|---|---|
| $Na^+$ | 108mM | 13.0mM |
| $K^+$ | 2.5mM | 138mM |

calculate the resting potential at room temperature of a membrane permeable only to sodium and potassium, when:
(a) permeability to $K^+$ is 100 times the permeability to $Na^+$
(b) permeabiluty to $Na^+$ is 100 times the permeability to $K^+$
(c) $K^+$ and $Na^+$ permeabilities are equal

3. What can metabotropic (*e.g.* G-protein coupled) receptors do that ionotropic (directly coupled) receptors can't do? What can ionotropic receptors do that metabotropic receptors can't do? (Why do you think retinal photoreceptors, for instance, evolved to use G-protein coupling instead of direct coupling?) What are the molecular bases of these properties?

4. What can chemical synapses do that gap junctions (*i.e.*, electrical synapses) can't? What can gap junctions do that chemical synapses can't? Why do you think heart muscle, for instance, has evolved to be rich in gap junctions?

5. What special property does the NMDA glutamate receptor have, and what is the ionic basis of this property?

## QUESTIONS FOR LECTURE #3

1. What chemical properties of the ion channel keep it embedded in the cell membrane?

2. Suppose you're given the amino acid sequence of a receptor molecule. Without knowing its three-dimensional structure, can you predict whether it's directly coupled or G-protein coupled? (For purposes of simplifcation, we leave aside the possibility of a non-G-protein-coupled metabotropic receptor.) If so, how, and if not, why not?

3. What kinds of experiments established the role of calcium in exocytosis?

4. Sketch a typical plot of membrane potential versus time during an action potential. Label each phase of the action potential and describe the electrochemical mechanisms that contribute to it. Describe (or draw) how the picture would change under each of the following circumstances:
(a) tetrodotoxin (TTX) blocks sodium channels
(b) voltage-gated potassium channels take longer than normal to open after a depolarisation
(c) the chain in the ball-and-chain structure at the end of the sodium channel protein becomes much shorter
(d) a mutation in the code for the voltage-gated sodium channel allows sodium and potassium ions to pass equally well through the pore

5. Define 'absolute refractory period' and 'relative refractory period', and relate these concepts to inactivation of sodium and potassium channels.

6. Define shunting inhibition and describe its relation to dendritic structure.

7. Define synaptic facilitation, synaptic depression, and post-tetanic potentiation, and explain briefly the relationships between these phenomena and calcium influx.

8. What is a node of Ranvier? How would the function of a myelinated axon be altered if it had no nodes of Ranvier? Why is this the case?

**QUESTIONS FOR LECTURE #4**

1. Where is the choroid plexus located, and what does it do?

2. What is an anastomosis? What major arterial structure forms an anastomosis delivering blood to the cerebrum, and of what arteries is it composed?

3. Anastomoses in large arteries, and overlap of perfusion territories in capillary systems, can be useful in cases of localised blockages but very damaging in cases of ischaemia due to a large-scale drop in blood pressure. Why?

4. Deoxyhaemoglobin is paramagnetic and therefore spoils the nuclear magnetic resonance signal from tissue that surrounds capillaries – that is, the MR image from tissue that contains a lot of deoxyhaemoglobin looks darker. Oxyhaemoglobin, on the other hand, is not paramagnetic and does not spoil

the MR signal. Why then do you think that active brain regions – *i.e.*, regions that are *using up* a lot of oxygen – look brighter on MR images? (Think about autoregulation.)

**ESSAY QUESTION:**

How may the activity of one neuron influence the activity of another? Your explanation should include mechanisms of signal propagation within neurons and between neurons.