

Instructions:

Please do not open the exam until told to do so.

Write your name at the top of all 7 pages.

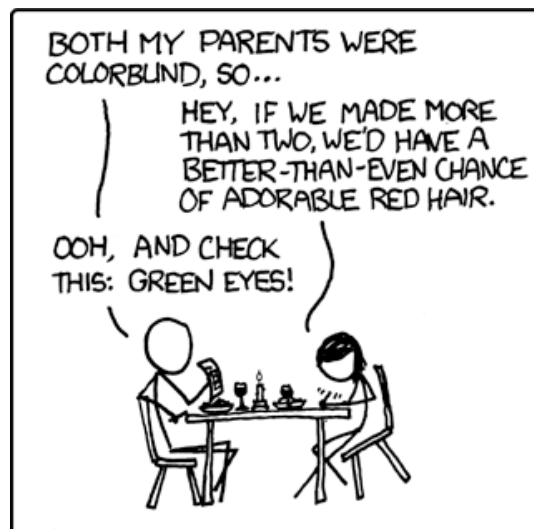
Choose the best answer for the multiple choice questions, and be BRIEF in the short answers questions.

$$\begin{aligned} a + b &= c \\ 4a - 3a + 4b - 3b &= 4c - 3c \\ 4a + 4b - 4c &= 3a + 3b - 3c \\ 4(a + b - c) &= 3(a + b - c) \\ 4 &= 3 \end{aligned}$$

Two women are selling apples. The first sells 30 apples at 2 for \$1, earning \$15. The second sells 30 apples at 3 for \$1, earning \$10. So between them they've sold 60 apples for \$25.

The next day they set the same goal but work together. They sell 60 apples at 5 for \$2, but they're puzzled to find that they've made only \$24.

What became of the other dollar?



TRIVIA: 30% OF BIOLOGIST FIRST DATES DISINTEGRATE INTO MAKING PUNNETT SQUARES.

Relax.

Don't think about how this exam might affect the rest of your life.

Don't worry about not getting into veterinary, pharmacy, or medical school.

Don't ponder how that would affect your capacity earn an income, which would prevent you from buying a Porsche and

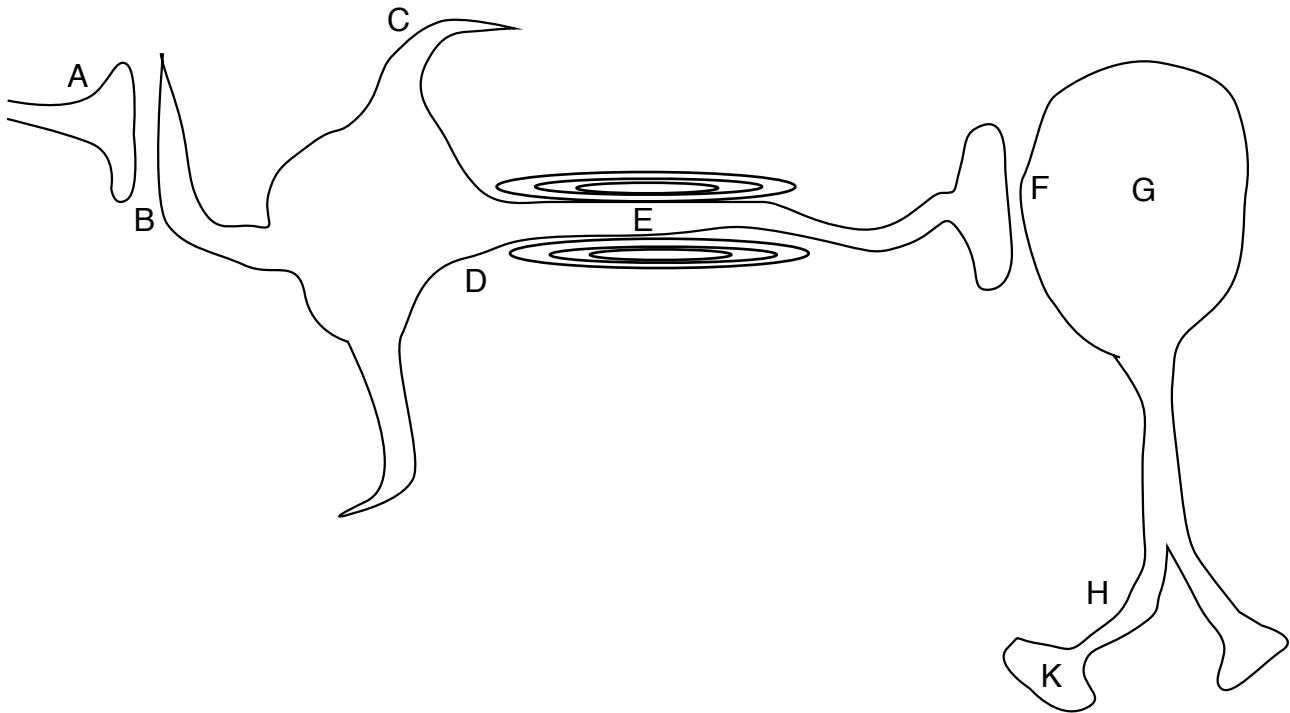
then would determine whether you are a selectable mate and might then cause you to live alone for the rest of your days in a nowhere job watching reruns all night.

Don't think about these things.

Relax.

Take a deep breath. Have some chocolate.

Despite all evidence to the contrary, these are drawings of neurons.



Write the letter from the reference points above that most accurately match the description below. You may use each letter only once. [2 pts each]

- 1) The soma: _____ **G** _____
- 2) A dendrite: _____ **C** _____
- 3) The axon hillock: _____ **D** _____
- 4) Location of VGLUTS: _____ **K** _____
- 5) Location of VGCCs: _____ **A** _____
- 6) Region of high membrane resistance: _____ **E** _____
- 7) A synapse: _____ **B** _____
- 8) A collateral: _____ **H** _____
- 9) Likely location of NMDA receptor: _____ **F** _____

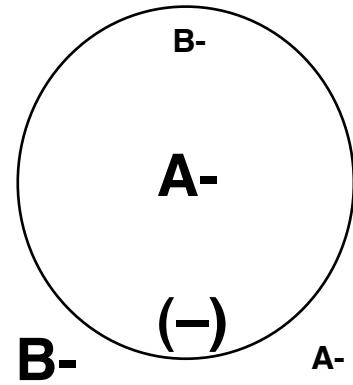
10) In a simple spinal reflex circuit, there is an excitatory path and a reciprocal inhibitory path. One muscle is flexed, while the other is relaxed.

a) What is the neurotransmitter found at the neuromuscular junction of the flexed muscle?
 [2 pts] **ACh**

b) Name a neurotransmitter that is likely found at the neuromuscular junction of the relaxed muscle?
 [2 pts] **ACh**

c) Why does there need to be an inhibitory pathway? Why bother to have inhibition at all?
 [3 pts]
Because the opposing muscle will have a reflex of its own in response to the initial reflex.

11) To the right is a yankee cell. The concentrations of the ions are represented by the size of the letter. A high concentration is denoted by a large letter. The cell has a negative membrane potential.



a) Which ion is most likely permeable at rest and why?

[5 pts]

B

At rest, B will have two gradients acting on it in opposition. Concentration driving it in and electrical driving it out.

Or you can use the Nerst equation to estimate the Equilibrium potential.

b) What would cause a depolarisation of this cell? In other words, which ion current(s) would likely change and how?

[5 pts]

Increased permeability of A or decreased permeability of B.

12) Name three sources of the sodium current of the cell at rest.

[5 pts]

***There is a transient current through voltage-gated sodium channels
There is a current of sodium through symports with neurotransmitters
There is a current of sodium through antiports with calcium***

13) In some neurons that have a strong potassium leak current at rest and a fairly strong current through voltage-gated channels with depolarisation, it is possible to drive the cell to a potential more positive than the equilibrium of sodium. How would this be possible?

[5 pts]

Some cells depolarise with Calcium influx. This can drive the cell past the equilibrium of sodium.

14) The reversal potential for a certain membrane that is permeable to potassium and sodium is at the midway point between the equilibrium potentials for the two ions. However, the cell can change the channels in that membrane remaining permeable to both ions but now the reversal potential has become more positive.

What specifically has changed to cause this?

[5 pts]

The new channels are more permeable to sodium than potassium.

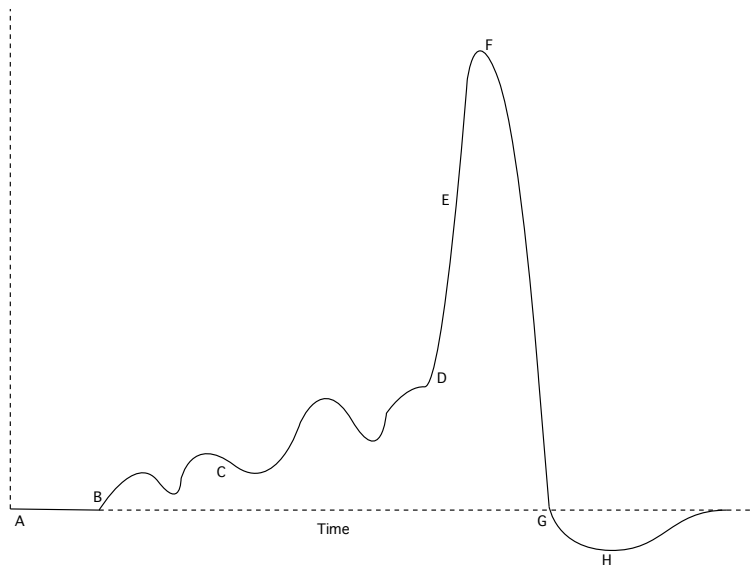
15) I am fairly certain that my ex-girlfriend is from Pluto. On that dwarf planet, a cell at rest is only permeable to Zn⁺⁺. Just like on earth, potassium is 20 times more concentrated inside the cell than out. Sodium is 20 times more concentrated outside than in and zinc is one hundred times more concentrated inside than outside. Chloride is not present. All other aspects of Plutonium cells are the same as here on earth.

Write the proper equation that would describe the resting potential of the cell.

[4 pts]

Since only Zinc is permeable, it would drive the membrane to E_{Zn}

$$V_m = (58/2) \log 1/100 \rightarrow (58/2) 2 \rightarrow 58$$



16) Write the letter from the voltage trace above that best matches the conditions below:
[3 pts per]

 H $58 \log \frac{[K]_o + 100[Cl]_i + [Na]_o}{[K]_i + 100[Cl]_o + [Na]_i}$

 F/G $58 \log \frac{900[K]_o + 20[Cl]_i + [Na]_o}{900[K]_i + 20[Cl]_o + [Na]_i}$

 E $58 \log \frac{400[K]_o + 10[Cl]_i + 1000[Na]_o}{400[K]_i + 10[Cl]_o + 1000[Na]_i}$

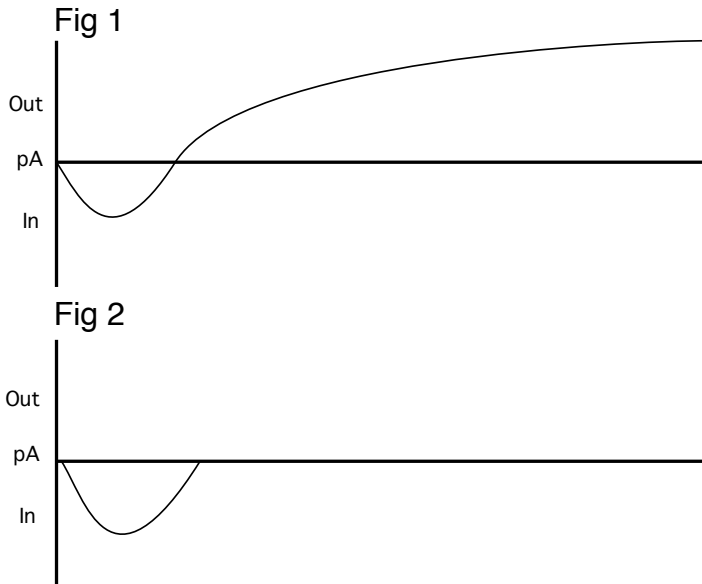
 A/G $58 \log \frac{100[K]_o + 10[Cl]_i + [Na]_o}{100[K]_i + 10[Cl]_o + [Na]_i}$

 C $58 \log \frac{150[K]_o + 10[Cl]_i + [Na]_o}{150[K]_i + 10[Cl]_o + [Na]_i}$

 D $58 \log \frac{300[K]_o + 10[Cl]_i + 350[Na]_o}{300[K]_i + 10[Cl]_o + 350[Na]_i}$

17) Why do we almost never reach the equilibrium potential of sodium in a motor neuron?
[3 pts]

The driving force on potassium is very large as the cell nears the E_{Na}, while the driving force on sodium is very small.



The top trace (Fig.1) is a whole cell recording of a neuron voltage clamped at +5 mV. The bottom trace (Fig.2) is the same cell after treatment with a drug.

18) Answer the questions below with one or only a **few** words:

a) What do these traces represent (what are we measuring)? [3 pts] **current**

b) What drug could produce the trace in Figure 2? [3 pts] **TEA**

c) The inward portion of the trace in Figure 2 is larger than it is in Figure 1. Why? [4 pts]

The current in Figure 1 is the sum of two opposing currents.

d) In reality, this is not representative of a true cell. If both sodium and potassium channels were blocked, what would we expect to see in Figure 1?

[4 pts]

The chloride current entering the cell remains. This would be equal to a positive current leaving the cell (attempting to drive the cell to E_{Cl})

19) Why does Botox paralyze while tetanospasmin cause muscle rigor when both are cleaving the snare complex and preventing vesicular release?

[5 pts]

Botox specifically cleaves snares of ACh axon terminals.

Tetanospasmin specifically cleaves the snares of inhibitory (GABA) terminals

20) Name two V-snares:

[6 pts]

Synaptobrevin and synaptotagmin

21) Schnookums has poisoned you with curare dart to the backside.

a) What kind of toxin is curare? In other words, what is its interaction with the receptors?

[3 pts]

it is a competitive (reversible) non-depolarizing blocker.

b) Why would smoking cigarettes save you from slow suffocation?

[3 pts]

The nicotine has a higher affinity for the binding site and is an agonist.

This would out-compete curare for the binding site.

c) Neostigmine is an acetylcholine esterase inhibitor. Would this help or hurt in the case above? Why? [3 pts]

It could help by increasing the residence time (and concentration) of ACh in the synapse. The increased concentration could compete with the curare and dislodge it from the binding site.

In the second case, it would not help since nicotine outcompetes ACh.

d) GABA and succinylcholine have similar post-synaptic effects. What are they?

[3 pts]

They are both depolarising inhibitors.

e) How are GABA and succinylcholine different in their post-synaptic effects?

[3 pts]

They bind to different receptors. One increases the permeability of Cl while the other increases the permeability of Na and K.

22) Briefly describe how SSRIs work to potentiate neurotransmitter effects at the synapse.

[4 pts]

These bind and block the re-uptake receptors on the presynaptic cell. This increases the residence time of serotonin in the synapse.

23) To the right is a dosage response curve for a drug.

The other two lines (dashed and grey) represent the dosage curve of the drug in the presence of another drug.

Choose from this list to answer the questions below.

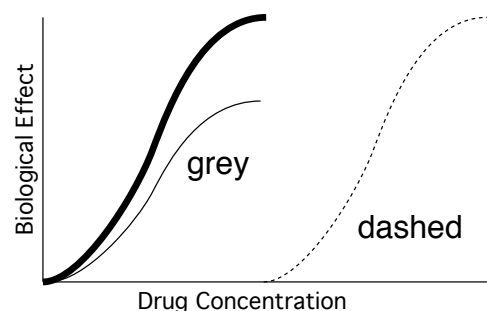
[3 pts each]

competitive agonist

competitive antagonist

non-competitive agonist

non-competitive antagonist



a) The grey line is the drug with a: **noncompetitive antagonist**
reluctantly accepted competitive agonist

b) The dashed line is the drug with a: **competitive antagonist**

24) As you grow from infancy through puberty, your axons lengthen. For a time, the distance between the nodes is longer than normal.

a) Why is this a potential problem?

[10 pts]

This can lead to back-propagation in the axon since the prior node has time to return to rest with the increased distance between nodes.

Also accepted the notion that the signal would weaken and not make it to the terminus. (although, we said that this would not happen)

b) Without changing the amount of myelination, only using potassium channels, what changes could you make to accommodate the longer distance between the nodes?

[10 pts]

Reducing the number of late-opening potassium channels will lengthen the refractory period.

Changing the suite of potassium channels to lower conductance channels.

25) Briefly, why are MAO inhibitors not an effective treatment for diseases that result in low levels of Dopamine?

[5 pts]

MAOs are involved in the entire anabolic pathway of all the catecholamines. Inhibiting these will increase the amount of norepinephrine and epinephrine also. There is no specificity to MOAs acting on dopamine.

There is also a lack of specificity to those neurons that are releasing NT. All of the neurons will have an increase in the amount of NT.

The uncontrolled levels of DA have led to schizophrenic episodes.