

1. Suppose there was a mutation in the voltage-gated K^+ channels of a neuron so that they opened at the same time as the Na^+ channels.

A) What effect would this have on the neuron? Explain. (6)

Weaker depolarization – instead of going to +40, it might only make it to about zero, because the potassium will go out while the sodium comes in, neutralizing its effects.

B) What if they didn't open at all during an action potential? (4)

The neuron would depolarize normally, but stay in a depolarized state. It would not return to a negative resting potential, or it would do so slowly because of leakage.

2. Consider a normal neuron at rest. Suppose there is slightly more Cl^- outside than in.

A) What will happen to the membrane potential if Cl^- channels opened? (4)

It would become more negative inside (further from threshold)

B) Under what circumstances, if any, would Cl^- not diffuse across the membrane, even if many Cl^- channels were open and there was a gradient? (4)

If the electrical potential was negative inside, and equal in energy to the concentration gradient driving Cl^- in.

C) If the Cl^- gradient was very weak, is it possible that it would diffuse against the concentration gradient? (2)

Yes, if the electrical potential pushing it back was stronger than the energy of the concentration gradient.

3. How does the refractory period keep action potentials from travelling backwards? (6)

The neuron is hyperpolarized, thus further from threshold. The current will conduct backwards, but it won't be able to reach threshold so the potential won't regenerate and keep traveling in that direction.

4. How would the following affect the likelihood of a post-synaptic neuron firing in response to stimulation from incoming action potentials? (Explain each answer)

A) having some voltage-gated Na^+ channels on the dendrites, halfway between the axon terminals and the axon hillock (4)

Depolarizations on the dendrites may cause the potential to reach threshold at these channels, letting more sodium in. This would increase the depolarization and make it more likely to reach the axon above threshold.

B) having a higher frequency of incoming action potentials (4)

This would increase the amount of sodium that entered, giving it more time to reach full depolarization. Thus, it would be more likely to be capable of conducting across the cell body and still reaching threshold at the axon.

***C) What is the advantage of this kind of complexity? (4)

Neurons can integrate output from many sources, gauge their intensity, and adjust their response. In this way they can make complex decisions.

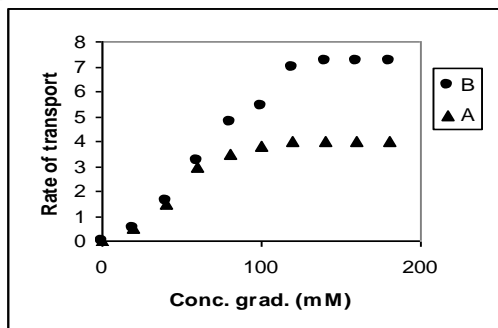
5. Consider the graph showing the rate of transport across an epithelium versus the concentration gradient.

A) What is the most likely explanation for the difference between curves A and B? (4)

B has more channels, so the rate of transport maxes out at a higher level.

B) How is the difference significant for synaptic transmission? (4)

The dendrites give a graded response because each synapse only opens a limited number of channels. There is a large sodium gradient, so it is the number of channels that limits transport. Opening more channels gives a greater depolarization. Thus the number of open channels determines the likelihood of getting a post-synaptic action potential.



6. If a neuron had very slow Na/K pumps (active transporters) how would that affect its function? (8)

It wouldn't affect action potentials at all. The biggest change would be that if it fired hundreds or thousands of action potentials in a short time, the concentration gradients would begin to weaken, and it would take longer to restore them. The concentration gradients determine the magnitude of the resting and action potentials.

7. A) Given what you know of neurons, describe a potential mechanism by which a drug can slow a patient's heart rate. (3)

It could bind to receptors on the synapse of neurons that control the heart rate and block the normal neurotransmitter that binds to that channel. If the channels were blocked from opening, this would inhibit firing.

B) Describe how another drug could act on the heart but create the opposite effect? (3)

The drug could do the same thing, except opening the channel instead of blocking it.

8. Given what you know about the chemical senses, what is the molecular basis for our ability to differentiate between different smells? (4)

Differences in the shape of the binding site of receptor proteins that bind to odor molecules.

9. How can you detect the direction from which a sound comes? (6)

By comparing the intensity of sound at both ears. The ear on the far side of the sound is in the "shadow" of the head, and thus hears a lower intensity.

10. Why is low frequency light not detected by the eye? (4)

It doesn't have the right amount of energy to be absorbed by retinal, and it doesn't have enough energy to cause a shape change in that molecule in any case.

11. Suppose you found a region on the skin of a snail that you thought may be an ear. How would you test this hypothesis? (There are several possible answers) (6)

Look for hair cells or other mechanoreceptors under the skin and see if they fire action potentials in response to sounds played at the snail.

12. What is the role of cyclic nucleotides (cAMP and cGMP) in sensory perception? (6)

These nucleotides are created or broken down by enzymes that are activated by a stimulus. When they are present they open ion channels and depolarize the cell.

13. Consider two muscles with different-sized thick filaments. One of the muscles (A) had the myosin heads clustered near the tip of the thick filament. The other muscle (B) had thick filaments with twice as many myosin heads, but they were spread out along the length of the filament. Sketch a graph showing how the tension produced by the muscle varies with extension in each case (draw them on the same graph). Explain your graph. (8)

When the myosin is clustered near the tip, the force will be constant over a long range of extensions, because the myosin will all bind easily to actin at most extensions. It would take a lot of extension to eliminate actin/myosin-head overlap.

When the myosin is spread out, any extension is likely to reduce the number of myosin that can grab onto actin, so the force will decrease. If there are twice as many myosin heads, the force will be 2x greater than the other muscle fiber at optimal extension. In both cases, shortening beyond the optimal extension will weaken the force similarly (possibly worse with the myosin spread out, due to greater interference from the actin from the other side).

14. Some muscles, such as finger muscles, have fine control of the force they produce. Other muscles, such as the hamstrings, have cruder control-- they are incapable of producing a wide variation of gentle forces. What could account for this? (6)

Finer control by having a large number of small motor units. Cruder control from only a few, large motor units. In the latter case, you can only increase the force in a few large increments rather than many fine increments.

1. A) Suppose the resting potential of one animal's neurons is -60 mv (inside relative to outside) and a very different species has a resting potential of -90 mv. Describe one possible cause for this difference. (4)

The latter species has a stronger potassium gradient.

B) The membrane potentials also differ at the peak of their action potentials (+60 mv for one, +50 mv for the other). Describe one possible cause for this difference. (4)

The latter species has a weaker sodium gradient.

C) The duration of their action potentials also differs (0.75 ms for one, 2 ms for the other). What cellular event must be different to create an action potential that lasts longer? (4)

The duration that the sodium channels stays open is longer in the latter case.

D) What type of experiment would you do to test the mechanism you propose in C? (you don't have to describe the experiment). (2)

Voltage clamping.

2. Some animals are sensitive to magnetic fields. These animals often contain crystals of magnetite. Magnetite is a mineral that orients in a magnetic field so that it always points north (like a compass needle). Describe a possible mechanism by which changes in the animal's orientation relative to a magnetic field could trigger an action potential. You can make any assumptions you want about where the magnetite is found, and in what kind of arrangement it is found. (8)

One option: The magnetite is next to a mechanoreceptor (for ex. A hair cell), such that movement of the magnetite pulls open sodium channels, depolarizing the cell. Another option: It is next to a membrane bound protein, movement of which activates a G-protein linked signal transduction pathway that creates a cyclic nucleotide that binds to and opens sodium channels.

4. In chemoreceptors, what is the function of G-protein linked enzymes? (8)

They create cyclic nucleotides that open sodium channels, depolarizing the cells. This occurs in some cells in response to a chemical binding to a G-protein-linked receptor that activates the enzyme.

3. A) Why is a single action potential reaching a synapse unlikely to trigger an action potential in a post-synaptic neuron? Explain in detail. (10)

It may only open a few sodium channels. This may not depolarize the neuron all the way to the equilibrium potential for sodium because the rate of transport is limiting. The relatively smaller depolarization may not be able to conduct all the way across the cell body and still be above threshold at the axon.

B) Each neuron in the brain may receive input from hundreds of neurons. The effect of the input from each may differ. Specifically, a single action potential from one specific neuron may bring the neuron closer to firing than a single action potential from another neuron. Describe one possible difference between neurons that could give rise to this difference in effect. (6)

Differences in the amount of neurotransmitter released could give more or less depolarization. (another answer could be if the synapse is closer to the axon, it has less distance to conduct so the depolarization would not weaken as much before reaching the voltage-gated channels of the axon.

5. How does the myelin sheath speed the transmission of action potentials? (8)

It slows the leakage of charge so it can conduct further down the axon without needing to regenerate. Conduction is relatively instantaneous, and thus faster than regenerating more often.

7. Suppose the stone in a balance receptor was larger and heavier (and thus harder to move).

A) Would this have any effect on the sense of static equilibrium (sense of orientation)? Explain. (6)
No – or possibly improve it because it will more reliably be on the bottom. Wherever the rock sits will be interpreted as down.

B) Would it affect dynamic equilibrium (sense of movement)? Explain. (6)
Possibly, if it was harder to move, any slight movement you made might not shift it. Thus you would be insensitive to slight movements.

6. Color blindness most likely involves a defect in what type of protein? Explain. (6)

The opsin protein surrounding retinal. Normally this controls what wavelengths of light can reach and activate retinal. A defect in one of these may give you less ability to distinguish that particular color from others.

8. If you stimulate a frog's sciatic nerve with increasing stimulus strengths, the force produced increases.

A) Why does force increase? (4)

You recruit more motor units.

B) Sometimes when the stimulus increases by a certain amount, the force increases only a small amount, but sometimes it increases by a large amount. Why may there be a difference? (4)

The motor units are different sizes (different number of fibers per unit for ex.)

C) Why does the force level off at some point and not get any stronger no matter how high the stimulus goes? (4)

You've recruited all the motor units.

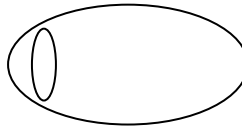
10) Muscles fatigue when lactic acid builds up. The high concentration of hydrogen ions (H⁺) affects the cellular machinery in the muscle. Specifically, hydrogen ions can bind to proteins and cause shape changes in the protein, rendering them less effective. Give three specific proteins that could be inhibited by hydrogen ions, and describe the effect on the muscle if that protein is impaired. (12)

Troponin – if it can't bind calcium well, fewer myosin binding sites would be exposed – force would decrease per contraction.

Myosin – if it can't ratchet as strongly, as fast, or if it can't bind well to actin – would change performance correspondingly.

Ca pumps of SR – if they slow their reuptake, muscle will relax more slowly.

11) A common cause of poor vision is if the eyes aren't perfectly circular. If it was shaped as in the exaggerated drawing below, how would this affect vision?



The image would tend to be focused in front of the retina, and thus out of focus at the retina.

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Fall 2005, Animal Physiology Exam 1

2. One way of controlling neural activity is pre-synaptic inhibition. In this situation, an axon terminal from one nerve sends a signal to the axon terminal of another nerve, reducing the amount of neurotransmitter it releases per action potential. If, for example, half as much neurotransmitter was released per action potential, how would that affect the likelihood of generating a post-synaptic action potential, and the strength and/or duration of any resulting post-synaptic action potential? Explain in detail. (10)

Fewer sodium channels opened, so weaker depolarization. This would be further from threshold at the V-gated channels of the axon, so it would be less likely to fire an AP. If it did fire an AP, the strength and duration would be the same because it is all-or-nothing.

3. Suppose you inactivated a neuron's Na/K exchange pumps (the active transporters), then immediately stimulated the neuron.

A) Would the action potential fail to occur or be different from usual? Explain briefly. (4)

No, the pumps are not involved in action potentials. Depolarization only depends on sodium channels.

B) Would repolarization fail to occur or be different from usual? Explain briefly. (4)

No, that only depends on K⁺ channel opening.

C) Would the cell's ability to respond to a second stimulus be changed? Explain briefly. (4)

With inactive Na/K pumps the cell would not be able to maintain the gradients, but it would take many action potentials to run down the gradients significantly.

4. Suppose you've been hired by a drug company to design a therapy for a particular disease. It's known to be caused by excessive firing of neurons in the area of the brain controlling thirst. Describe an approach to correcting this disease that you think would be fruitful. (8)

Identify the normal neurotransmitter and its receptor in these neurons. Find a drug that binds to this receptor and blocks it. (several related possibilities)

5. What determines what frequencies we can hear, and what frequencies are too high? Your answer should make clear that you know the basic mechanism by which we sense sound. (8)

The cochlear membrane's mechanics change along its length so that one end vibrates only with high frequencies and the other end with low frequencies. Thus, you only hear frequencies that are capable of vibrating some part of this membrane. These frequencies will activate hair cells under the vibrating part of the membrane.

6. List the major similarities in the way an olfactory chemoreceptor cell and a light receptor cell operate. (8)

Both use membrane-bound receptor proteins

Both use a signal transduction pathway usually involving a G-protein and linked enzyme

The enzyme creates/degrades a cyclic nucleotide

The end result is the opening or closing of sodium channels, changing the polarization of the membrane.

7. Suppose there are ATP-sensitive potassium channels on the muscle cell membrane. ATP is a ligand that holds these channels closed. How would these channels affect a muscle's response to fatigue? (10)

If ATP started to decrease due to fatigue, there would be less ATP holding these channels closed, they would open, letting potassium out, hyperpolarizing the membrane. Thus, the fiber would be less likely to be depolarized -- less likely to contract.

8. Identify two specific processes in a muscle twitch that require ATP. Be specific. (8)

Myosin head activation

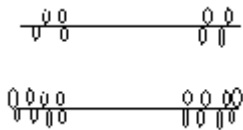
Myosin head release from actin

(also removal of Ca^{++} by SR)

9. What are three adaptations an organism could have that could increase the endurance of a muscle? (6)

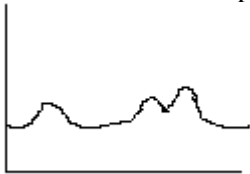
More mitochondria per fiber, increased capillary density, more myoglobin etc...

10. Consider two muscles, one whose thick filaments are shown above (with fewer heads per myosin molecule), and the other whose thick filaments are shown below (with more heads per myosin). For each muscle, graph what you would expect a single twitch to look like. Assume there is no load on the muscle. Put both graphs on the same figure, for easier comparison. The graph should show tension over time. (6)



More force with more myosin, same velocity (slope of rising force).

11. Consider the following recording of a muscle stimulated three times. Explain what is happening at the cellular level to make the third peak taller than the other two. (6)



The stimuli come fast enough that the muscle is still active when the second stimulus comes. That means that it may be fully active when the calcium is maximal. In the earlier peaks, the peak force comes after a lot of calcium has been put away so there are fewer myosin-binding sites available.

12. A) How does the width of a muscle affect the unloaded velocity? (4)

No effect. The unloaded velocity depends mostly on the intrinsic cycling rate of myosin heads.

B) How does the width of a muscle affect the loaded velocity? (4)

More tension, thus load/tension ratio is less and velocity is higher.

1. A) Explain why the action potential is an all-or-nothing event. Your answer should explain why it is either on or off, and why the magnitude is always the same. (6)

Either you hit threshold or don't, and if you do, all the sodium channels open and you reach the equilibrium potential for sodium.

B) Explain why the depolarization on the dendrites caused by neurotransmitter crossing a synapse is not all-or-nothing. (6)

Because the amount of open channels depends on the amount of neurotransmitter released. The amount of sodium entering is limited by the number of channels.

C) What is the advantage to the fact that synaptic depolarizations on the dendrites are not all-or-nothing? (6)

The depolarization can vary over a wide range, adding up many inputs to "decide" whether or not to fire an action potential.

2. A chemosensory receptor cell has ligand-gated and voltage-gated sodium channels. Explain the role of each in creating an action potential. (10)

The ligand gated channels bind to the stimulus (odor molecules), and depolarize the cell. If there is enough stimulus, this can bring the cell to threshold. This will open voltage gated channels on the axon and trigger a full action potential.

3. A) Imagine there is a toxin that blocks voltage-gated calcium channels (there probably is). How would this affect synaptic transmission? Explain. (4)

Block the release of neurotransmitter (which depends on the influx of calcium on pre-synaptic axon terminals).

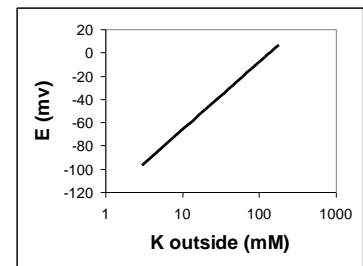
B) How is it possible to have a drug that specifically increases alertness, while another specifically acts on the pleasure centers of the brain? (6)

Each circuit has different neurotransmitters and thus different receptor proteins. You can have a drug that mimics a specific NT and binds to receptors in one circuit and not others.

4. Suppose you are studying the neurons of a garden snail. You measure the resting potential across the membrane while manipulating the concentration of potassium outside the neuron. You get the results shown in the following graph. From this you can tell what the intracellular concentration of potassium is.

A) Roughly what is the intracellular concentration of potassium? Explain how you know. (6)

A little over 100 mM – when the extracellular conc. equals this, the electrical potential is 0 V. No gradient = no electrical potential.



B) You find that the resting potential before you start messing with the potassium is -80mv. From this, what is the typical extracellular potassium concentration? (6)

Around 7 mM.

C) What would happen to the resting potential if you changed the sodium concentration outside the neuron? Explain. (6)

Not much – the resting potential depends mostly on potassium. (it is true that a stronger sodium gradient can lead to slightly more sodium leakage at rest, pushing the resting potential a little more positive).

5. Explain how our depth perception works. (8)

Each eye sees a slightly different view. The difference is greater for closer objects. The brain compares the difference between what each eye sees – larger differences imply the object is closer.

6. A) High frequency sound attenuates rapidly (the sound is scattered and weakened as it travels). What consequences does this have for determining the location of a sound source? (5)

There is a larger difference between the ears with high frequency because the head scatters more of the sound so that less reaches the far side.

B) One drawback to echolocation is that animals like bats have to call very loudly. Why does this complicate echolocation? Explain. (5)

They are often calling rapidly, and yet listening for very faint echoes between shrieks.

7. What is the function of the photopigment rhodopsin (opsin + retinal) in vision? (8)
It absorbs light, changing shape in the process and thus activating the signal transduction pathway that closes sodium channels.

8. A) If you stretch a vertebrate cross-striated muscle to 170% of its resting length, it will be unable to produce force. Why? (8)

There will be no more overlap between actin and myosin. Thus, no cross-bridges to produce force.

9. A) Why do muscles become rigid if no ATP is present? (5)

The myosin needs to bind to ATP to let go of actin. If it stays bound to actin, the muscle cannot change length.

B) What is the function of calcium in muscles? (5)

It triggers contraction by binding to troponin. This causes a conformation change which pulls tropomyosin off the myosin-binding sites. This allows myosin can bind.

Name: _____

Exam I, Fall 06

1. People who suffer from salt imbalances (for ex. through dehydration or some pathological condition) often experience overexcitable nerves that fire too often (causing cramps, random twitches, or difficulty thinking clearly). If the intracellular concentration of potassium was unusually low for example, nerves may begin to fire action potentials more often than they are supposed to. Why would this happen? (10)

The resting potential is determined by the electrical potential that balances the potassium gradient. Weaker potassium gradient leads to weaker resting potential. Thus closer to threshold.

2. At a synapse, opening one sodium channel (for the normal duration before closing) causes a small depolarization. Opening a handful of sodium channels may cause a larger depolarization. Opening more channels may lead to an even larger depolarization, up to a point. Then the depolarization would be the same regardless of how many other sodium channels opened. Explain why the number of open channels increases the depolarization, and why it doesn't matter beyond a certain point. (10)

The channels are limiting the rate of transport – there is a strong gradient and each channel is transporting as fast as it can, so adding more channels increases the sodium influx. At some point, though, you reach the equilibrium potential for sodium where the electrical potential is just strong enough to match the concentration gradient, so no more diffusion occurs regardless of how many channels are open.

3. A. Axons that are larger in diameter transmit signals faster. This is because the rate of charge leakage is slower than a skinnier axon (note that this refers to the general leakage of ions across the membrane, not movements controlled by specific channels). How does slower leakage lead to increased signal velocity down the axon? (5)

The charge can conduct farther and still be above threshold. Conduction is basically instantaneous, so it jumps further down the axon before needing to regenerate.

B. If ion leakage from the dendrites was **increased** due to greater general leakiness, how would this affect the firing of the neuron in response to action potentials coming in from a pre-synaptic neuron? (5)

The depolarizations would be weaker by the time they reached the axon of the post-synaptic neuron. Thus they would be further from threshold and less likely to trigger an AP.

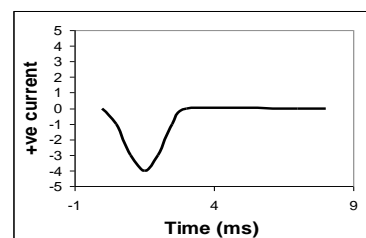
4. Consider the following recording from a voltage clamp experiment. A drug was used to block a channel.

A) Which channel was blocked? (4)

Potassium, because there is no outflow of positive current after the inflow.

B) On a separate chart, draw what the action potential (voltage vs. time) during this recording would look like. Make sure that events line up on the x-axis where you would expect based on the voltage clamp data. (6)

During the inflow of positive charge, the voltage would rise to about +40. Then at about 3 ms, there would be little further change as no more current flows (potential stays at around 40).



5. Imagine a drug that blocked reuptake of an excitatory neurotransmitter such as epinephrine by pre-synaptic cells.
A. Explain how this might affect nerve cell firing. (4)

The neurotransmitter would stay in the synapse, constantly stimulating the cell. (note that this is a sympathetic system transmitter so heart would speed etc...)

B. Would this affect all nerve cells? Explain. (4)

No, only those that used that neurotransmitter.

7. Some animals have excellent night vision. There are a number of things that would provide good night vision.

A. Describe how the fovea (the area in the center of the retina) could be changed, and also possibly the connections to the optic nerve, in order to improve night vision. (6)

More rod cells in the fovea, with large numbers wired together to a single ganglion cell (summing their output).

B. Describe how changes in the activity of proteins or enzymes on the vesicle membrane of photoreceptor cells could improve night vision. (6)

More active enzymes could break down more cGMP per activated rhodopsin, thus closing a larger number of channels per photon hitting rhodopsin.

8. Do balance receptor cells depend on sodium gradients? Explain briefly. (6)

Yes. They are mechanoreceptors, and the mechanical stimulus opens sodium channels to depolarize the cell.

9. In some non-striated muscle cells, like those lining the intestine, calcium levels in the cytoplasm never drop to zero. They stay constant at a low level, and may rise and fall slowly. How would this affect the way the muscle behaved? Explain. (8)

There would always be some open myosin binding sites, and thus some crossbridges (with the number varying with the calcium concentration). The muscle would always be producing a force, depending on how many crossbridges were active.

10. Most muscles are neither 100% oxidative, nor 100% glycolytic – they are a mixture of these fiber types. All the fibers in a single motor unit, though, are the same (either glycolytic or oxidative). Explain what this means for the control of muscle performance. (8)

When you increase force, you do it by recruiting more motor units. You can recruit either a fast, low endurance unit or a slower, high endurance unit.

11. Squid tentacles are extremely fast. Two adaptations that they have are the following: they have many more sarcomeres attached end-to-end in each muscle fiber than is typical, and they have very short distances from the sarcoplasmic reticulum to the actin and myosin filaments.

A. Explain how each of these would affect the speed of the tentacle strike. (6)

More sarcomeres end-to-end -- velocity in series is additive.

Very short SR to myofilament distances – shorter diffusion time for calcium, quicker activation.

B. Name one other adaptation that would increase the speed of the strike. (4)

More glycolytic muscle

(or) Stronger muscle, lighter tentacle to improve load/tension ratio