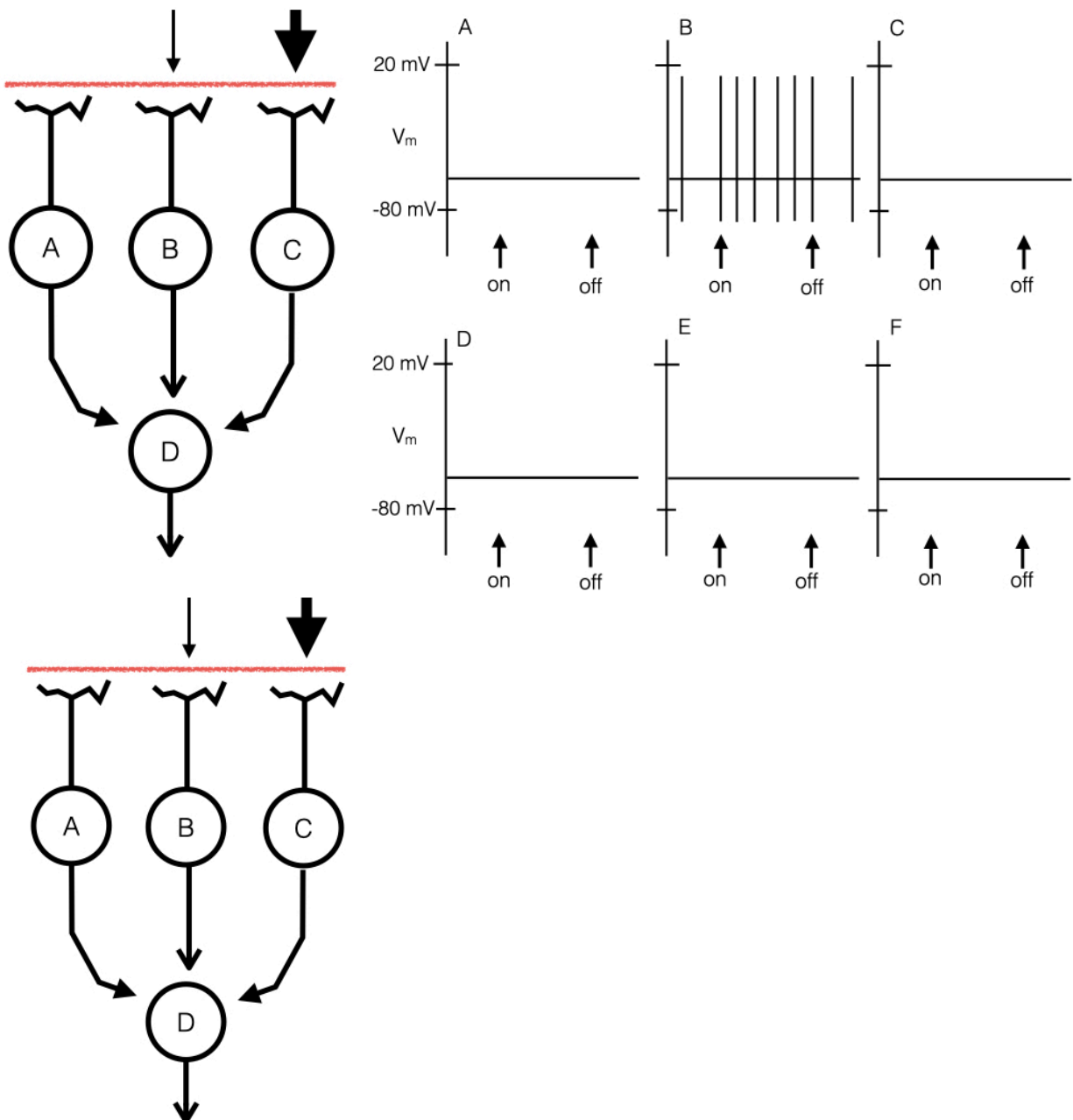


**Question 1 (18 marks).** Consider the two different arrangements of sensory systems in the images below. The top system depicts first- and second-order sensory neurons that use lateral inhibition. The lower system depicts first- and second-order sensory neurons that use convergence. In both cases, a stimulus is applied that is suprathreshold for cells “B” and “C” but not A. The intensity is greatest for cell “C” (indicated by the size of the arrows). Assume that all neurons in both arrangements have the same action potential size and firing frequency when they are at rest (ie: when there is no stimulus present).

The graphs on the right represent the action potentials (vertical lines) produced by the primary and secondary neurons. Only cell “B” has been completed. Note: the small arrows under the x-axis represent when the stimulus was applied (“on”) and removed (“off”).



- a) Using the action potentials made by cell B as a guide, complete the action potential graphs above by drawing in the action potentials for cells A, C, D, E and F in the top system. I mean, actually draw the action potentials onto the graphs, please. Assume the cells all have the same firing properties (ie: are tonic or phasic), and that they all have the same magnitude of action potentials at rest (ie: comparable to those drawn in B) (5 marks).

*A: less than B, but NOT ZERO*

*C: Higher than B*

*D: less than F, and either less than or equal to E (both E and D could be zero)*

*E: must be less than F*

*F: must be more than D and E.*

All action potentials should be roughly the same size.

- b) Explain your drawing for cell E in the top system (ie: with lateral inhibition). Be sure to include how lateral inhibition caused this effect. (2 marks)

*The lower # of APs (or none at all!) are a result of less (or no!) NT release from the presynaptic terminal of cell.*

*Less NT = fewer receptors activated = fewer APs generated*

- c) Lateral inhibition allows your sensory system to do what? (1 mark)

*Locate the stimulus with great accuracy.*

- d) Where in the body might you expect to find a sensory system with lateral inhibition (like the top image above) compared to a system with a large amount of convergence onto a second-order neuron like the second image below? (1 mark)

*Hands/fingers/lips*

- e) Which cell D - from the lateral inhibition system, or the convergence system - has a larger receptive field? Why? (2 marks)

*convergence*

*b'c it includes the receptive fields of cells A, B and C, while that of the top system includes the receptive field of sensory neuron A only.*

- f) Which cell D - from the lateral inhibition system, or the convergence system - has better sensory discrimination? Why? (2 marks)

*lateral inhibition*

*b/c it includes only information from cell A, AND the surrounding cells (eg: E) are 'silenced' (or inhibited) by lateral inhibition*

- g) In the lower system (ie: with convergence), what would be the perception of the person if we applied two stimuli of equal magnitude to cells A and B? Explain, and be specific. (2 marks)

*It would be of a single stimulus*

*It would be (roughly) double the intensity of each individual stimulus.*

- h) Propose a **mechanism** by which cell C “laterally inhibits” cell B in the top arrangement (with lateral inhibition). (3 marks)

*Anything reasonable:*

*Activate a  $K^+$  conductance*

*Close  $Ca^{2+}$  channels*

*Open  $Cl^-$  channels.*

*Each of these reduces the amount of  $Ca^{2+}$  coming into the terminal, reducing NT release.*

- Describe how the nervous system encodes stimulus intensity and duration
- Predict how changes in the intensity and duration of a stimulus are encoded by the nervous system.
- Describe how convergence of sensory neurons affects the size of receptive fields, and how receptive field size affects stimulus discrimination.
- Explain how lateral inhibition increases contrast of incoming stimuli.

**Question 2 (15 marks).**

- a) Fill in the blanks for the following (short-form is fine! Eg: nAChR for nicotinic): (2 marks)
- The neurotransmitter released by the preganglionic neuron of the prevertebral ganglion is:       ACh      .
  - The receptor on the smooth muscle of the stomach receiving input from the parasympathetic nervous system is:       muscarinic AChRs      .
- b) What would happen to the following organs if we had damage to all of the paravertebral ganglia (and only the paravertebral ganglia)? **One to five words** should suffice. (4 marks)

bronchioles of the lungs	constrict; narrow; unable to dilate
heart (rate)	decrease; or unable to increase
smooth muscle of stomach	nothing
smooth muscle of blood vessels leading to skeletal muscles of legs	nothing

- c) Someone shows up to a clinic and they have ingested a drug, but they don't know what drug it was. They have the following symptoms: difficulty breathing (due to constriction of bronchioles); diarrhea (due to contraction of the intestines); very low heart rate. The drug was either a blocker of receptors or an activator of receptors. Based solely on the symptoms above, tell us 1) what receptors the drug targeted, and 2) whether it was a blocker or activator. You do not need to explain WHY at this point. [note: there may be multiple possible answers. I only want ONE]. (1 mark)

*Muscarinic agonist.*

*OR*

*Antagonist of all beta receptors. [would have to be ALL receptors explicitly ... both 1 and 2]*

- d) Tell us WHY you selected the response to the preceding question. You should refer to the symptoms in this explanation. (3 marks)

*Full points for a drug that would cause ALL of the symptoms noted.*

To treat this person, you have the following drugs available that have the following mechanism of action:

Drug	action
A	activates alpha 1 receptors

B	blocks all beta receptors
C	<b>blocks all muscarinic receptors</b>
D	activates beta 1 receptors only
E	activates beta 2 receptors only

e) Which drug will you use to treat this person and why? You only need to pick ONE! (2 marks)

*Drug A will lead to constriction of blood vessels leading to the skin and GI tract; not helping in this scenario.*

*Drug B will produce the effects observed. Not helpful here*

*Drug C: will remove parasympathetic tone pretty much everywhere, which would alleviate the symptoms observed.*

*Drug D: will increase heart rate (and force of contraction)*

*Drug E: would fix the airway and GI issues, but not the cardiac issues.*

f) A colleague chooses to administer a drug that increases cAMP levels in all smooth muscle and cardiac pacemaking cells. What will this do to the following? circle your answer. (1 mark)

Heart rate:      INCREASE                      NOT CHANGE IT                      DECREASE  
*increase it.*

smooth muscle of the stomach:  
                    INCREASE                      NOT CHANGE IT                      DECREASE  
*this would decrease contraction of the smooth muscle, decreasing GI motility*

*[note to IAs: if they only say “increase” or “decrease”, that is sufficient.*

Would your colleague’s administration of the cAMP-increasing drug alleviate the symptoms of the patient from question c)? Why or why not. (2 marks)

*It would work!*

*Because this would cause the smooth muscles’ of the bronchioles and GI tract to relax, alleviating symptoms.*

*It would also increase the heart rate, alleviating that symptom too.*

- Identify the location of neuron cell bodies in sympathetic and parasympathetic nervous systems.
- Identify the neurotransmitters involved in both sympathetic and parasympathetic nervous systems, and which receptors are involved in mediating their actions.

- Predict the effect of increasing/decreasing sympathetic/parasympathetic action on target organs.
- Predict the effect of damage to the following to the function of the sympathetic NS: paravertebral ganglia, prevertebral ganglia, and adrenal medulla.
- Predict the effect of damage to the following to the function of the parasympathetic NS: cranial nerves III, VII, IX, and X, and the sacral segments of the parasympathetic system.
- Compare and contrast different adrenergic receptor types (eg:  $\alpha_1$ ,  $\beta_1$ ,  $\beta_2$ ) with regards to their location, role, and pathway (which GPCR pathway they use).

**Question 3 (12 marks).**

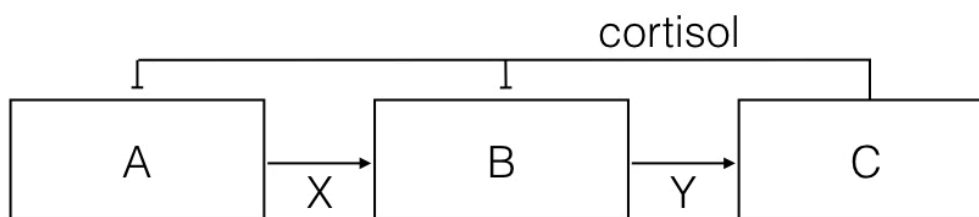
- a) Which of the following classes of hormone have at least one hormone that can bind to receptors in the cytoplasm? (circle all that apply) (1 mark)

*STEROID*

*PEPTIDE*

*AMINE*

Consider the endocrine pathway below when answering the remaining questions.



- b) Identify the following: (2 marks)

Structure B: ant. pit.

Hormone X: CRH

Structure C: adrenal cortex

Hormone Y: ACTH

Two people go to their doctor because they have excess (too much) cortisol secretion. Both patients are given a drug that suppresses the secretion of Hormone Y. Person A's cortisol levels return to normal after receiving the drug, while Person B's cortisol levels remain elevated.

- c) What anatomical location is most likely the source of the problem for Person A? Explain how you know, or why you cannot tell. (2 marks)

*Either hypothalamus or anterior pituitary.*

*The adrenal glands are working fine, because restoring hormone Y to normal fixed the problem. So the elevated level of Y was either caused directly by the ant. pit., or by excess Hormone X (CRH) from the hypothalamus.*

- d) What anatomical location is most likely the source of the problem for Person B? Explain how you know, or why you cannot tell. (2 marks)

*Adrenal cortex.*

*Since giving them a drug that suppresses Y has not fixed the problem, the adrenal cortex is not responding to Y appropriately.*

- e) What were the circulating levels of Hormone X, Y in person A **before** the drug treatment? Explain how you know, or why you cannot tell. (2 marks)

Hormone X:

*we don't know. High levels of X would have caused the high Y (and high cortisol) levels; but if the problem was the anterior pituitary, we'd expect low levels of hormone X.*

[NOTE TO IAs: PLEASE BE CONSISTENT WITH WHAT STUDENTS WROTE IN Qs C) and D)]

Hormones Y:

*they were high. Suppressing their production fixed the problem, so they must have been too high to begin with.*

- f) What were the circulating levels of Hormone X, Y in person B **before** the drug treatment? Explain how you know, or why you cannot tell. (2 marks)

Hormone X:

*low*

*Cortisol exerts a 'negative feedback' onto hypothalamus, so production of Hormone X would go down.*

Hormones Y:

*low*

*Cortisol exerts a 'negative feedback' onto hypothalamus, so production of Hormone X would go down.*

Consider a completely new situation separate from the Person 1 and Person 2 scenario above. In our new case, a patient has a complete blockage in the portal veins leading into the pituitary from the hypothalamus. Vessels leaving the pituitary are fine (ie: not blocked).

- g) What would this do to circulating levels of the following: (circle your choice) (1 mark)

ACTH:            INCREASE THEM            *NO CHANGE*            DECREASE THEM

Oxytocin:       INCREASE THEM            NO CHANGE            *DECREASE THEM*

- Predict the outcome(s) of disrupting different parts of a feedback loop.
- Compare and contrast steroid, amine and peptide hormones, including site of receptors and consequences of receptor activation.
- Know the anatomy of the hypothalamic/pituitary pathway.
- List hormones secreted by the posterior pituitary, and the effects they have on target organs.
- For all the anterior pituitary hormones, know: the hormones that caused their release; their targets; the hormones their targets release; the impact of the target's hormones on the body.





**Question 4 (10 marks).**

- a) At rest, the length of the A-band is  $4.0\text{ }\mu\text{m}$ . The H-zone is  $0.5\text{ }\mu\text{m}$ . The thin filament (from z-line to filament tip) is  $2.0\text{ }\mu\text{m}$ . What is the length of the sarcomere at rest? (1 mark)

$$2 + 2 + 0.5 = 4.5\text{ }\mu\text{m}$$

- b) Compared to a normal muscle cell, what will happen to the number of cross-bridges under each of the following circumstances. For each, state WHY you think so. (2 marks each = 4 marks)

- (i) We remove all physical connections from Ryanodine receptors (RyRs) and all other receptors, then stimulate the muscle cell.

*Decreased cross bridges*

*The DHP receptors can no longer open RyRs, so the  $\text{Ca}^{2+}$  stays in the SR.*

- (ii) We add thapsigargin, a drug that stops the SERCA pumps, then stimulate the muscle cell.

*We'd never remove  $\text{Ca}^{2+}$  after a stimulation, so the  $\text{Ca}^{2+}$  would stick around and you'd have more crossbridges, lasting for longer.*

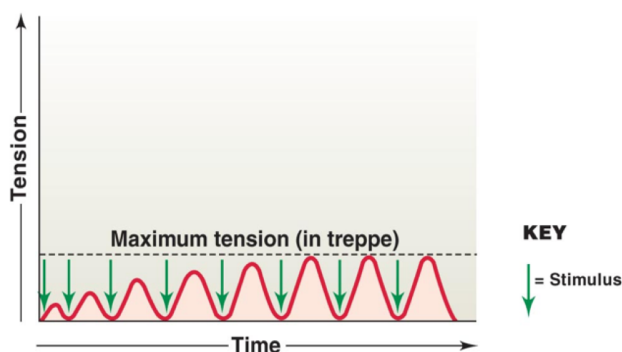
*[note: they might say something like "a single stimulus would produce the same # of cross bridges, but for a longer time. That would be correct, and I'd give them points for that. But they should make it explicitly clear]*

- c) If you stimulate all motor units of a muscle with a single stimulus, is this muscle maximally stimulated? Why or why not? (2 marks)

*No; you could still increase the frequency of stimuli to get tetanus in all motor units.*

- d) In a condition called "treppe", the amount of tension in a single muscle cell increases with subsequent stimulation, but the cell is allowed to *fully relax* each time (see figure below). Can you predict what is happening to allow the muscle to increase tension with subsequent stimulation while being allowed to relax fully between each stimulation? Your answer should include a discussion of how tension is generated. (3 marks)

**Treppe**



*If the muscle is relaxing fully, all the  $\text{Ca}^{2+}$  is coming off troponin, and the # of cross-bridges is zero.*

*Somehow, subsequent stimulation produces more tension, so we're getting more cross bridges presumably due to increased  $\text{Ca}^{2+}$ .*

*There are several possibilities to explain this, including:*

*before all the  $\text{Ca}^{2+}$  is sequestered into the SR (but after it has left the troponin), the second stimulation arrives, so we add newly released  $\text{Ca}^{2+}$  to the amount of  $\text{Ca}^{2+}$  that was left-over from the previous stimulation.*

*$\text{Ca}^{2+}$  actually potentiates RyRs, leading to greater  $\text{Ca}^{2+}$  release.*

- Know the anatomy of the neuromuscular junction including pre- and post-synaptic components, and ligand receptor types.
- Outline the sliding filament theory, and identify which region(s) (ie: I, A, or H "bands") get shorter during contraction.
- Outline the sequence of events between AP reaching the axon terminal of the motor nerve and the increase of cytosolic  $[\text{Ca}^{2+}]$ , leading to contraction of the muscle (excitation-contraction coupling).
- Specifically, identify the roles of L-type calcium channels, ryanodine receptors, and SERCA pumps.
- Describe the processes underlying summation of skeletal muscle.
- Describe the functional differences between summation and tetanus.