80/80



Cognitive Science 107B—Winter 2007 Print Name: Key

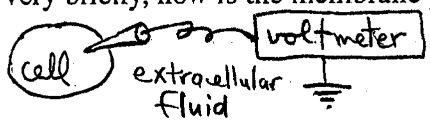
**Systems Neurobiology**

First exam (VersionA)—8 questions, equally weighted (7-8 min/question)

**1.** For questions below, assume: *(i)* a channel only conducts a hypothetical *negative* ion *(ii)* the Nernst potential for this ion is *-100 mV (iii)* a cell with these channels in its membrane is currently *at rest* (defined as -70 mV), and, *(iv)* the channel is *closed.*

(a) Very briefly, how is the membrane potential of a cell measured?

(3)




or

– a voltmeter is used to determine voltage difference between inside and outside of cell using intracellular electrode

(b) Which *direction* will the ions flow (in/out) if the channel is briefly opened?

(2) – in    Nernst is more negative than present (resting) potential

(c) What would happen to the membrane potential if you added some of these ions to the fluid *outside* the cell?

(2)
– make it more negative if channel open
or
– leave it unchanged if channel closed

closed vs. opened: Ambiguous!

[assumes added negative ions accompanied by positive, so net charge added is zero]

(d) Why is the Nernst potential sometimes called the *reversal potential*?

(3)
– the Nernst potential is a "target" membrane potential:
[ if present potential above Nernst, opening channel lowers it,
if present potential below Nernst, opening channel raises it

**2.** Axonal action potentials result from the opening and closing of voltage-gated sodium and potassium channels.

(a) Since both the sodium and potassium channels are opened by depolarization and since they have opposite effects on the membrane potential, why don't they cancel each other out? (why does a large positive spike in membrane potential occur?)

(4)
– the sodium channels open more quickly than the potassium channels
[depolarizing inward sodium current is followed by hyperpolarizing outward potassium current]

(b) If two spikes going in opposite directions along an axon collide, both will die out. Why?

(3)
– each spike is followed by a wave of inactivated sodium channels; when the waves meet, there is no longer any depolarizing sodium current

(c) Axons often branch and an action potential may split and propagate down both the main and side branch. Since axonal sodium channels open when depolarized, but *only* if the depolarization ***happens quickly enough,*** would it be better to ***reduce*** or ***increase*** the ***capacitance*** of a side branch in order to make sure that the spike went down it, too? Very briefly, why?

(3)
– better to reduce capacitance of side branch
– less capacitance means voltage will be able to change more quickly, reducing chance of conduction failure due to not changing voltage quickly enough

**3.** We discussed a simple, layered network model with Hebbian synapses, maps, and a Gaussian probability of connection between layers. The change in connection strength was proportional to the correlation of activity between input and output at each connection.

(a) Give an example of 5 *pairs* of *input values* that are strongly ***negatively correlated***

(2) (2, -3) (-5, 4) (3, -2) (-1, 1) (-7, 3) ← – mostly opposite sign

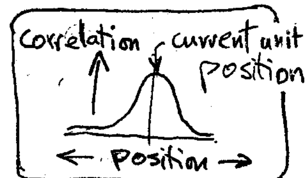
(b) If the input values in (a) were applied to a two-inputs-one-output version of the Hebbian model with two positive weights, would those weights go up or down? Why?

(3) – down $\Delta w \approx I_i \, [I_{other} \, w_{other}]$ → since $I_i I_{other}$ is negative and w is positive, $\Delta w$ is negative

– $\Delta w$ proportional to correlation

(c) What does it mean for a layer to have a Gaussian two-point correlation?

(2) – for a given unit, the correlation with neighboring units falls of as a Gaussian as a function of distance




(d) In this model, a 4th layer unit starts off with random input weights. Hebbian learning driven by noise in the input layer causes the unit to develop receptive fields that have all-maximum and all-minimum weights coming from parallel rows of 3rd layer units. Describe the ***specific biological fact*** that this ***progression*** was supposed to be a model of.

(3) – the origin of orientation selectivity in cortical neurons (V1) before eye opening (and without genetic specification)

**4.** NMDA channels are found in the post-synaptic membrane of some synapses and seem to be involved in "Hebbian" (correlation-dependent) changes in synaptic strength.

(a) Briefly, how does an NMDA channel detect *correlation* between *input* and *output*?

(3) NMDA channel opening requires
1) glutamate indicating input activated
2) depolarization indicating output activated

(b) What is the *evidence* that the *induction* of LTP *requires* NMDA channel currents?

(2) – blocking NMDA channels (e.g., hi $Mg^{++}$) prevents LTP

(c) What is the *evidence* that the *maintenance* of LTP *does not* require NMDA channel currents?

(2) – blocking NMDA channels after LTP induced does not affect LTP

(d) If a spike invades a single NMDA+AMPA synapse releasing glutamate, the resulting post-synaptic depolarization will not be enough to release the magnesium block on the NMDA channels. This differs from our simple Hebbian learning model. How would we have to change the *model* weight update rule to simulate this?

(3) – in the model, any positive or negative correlation, no matter how small affects weight

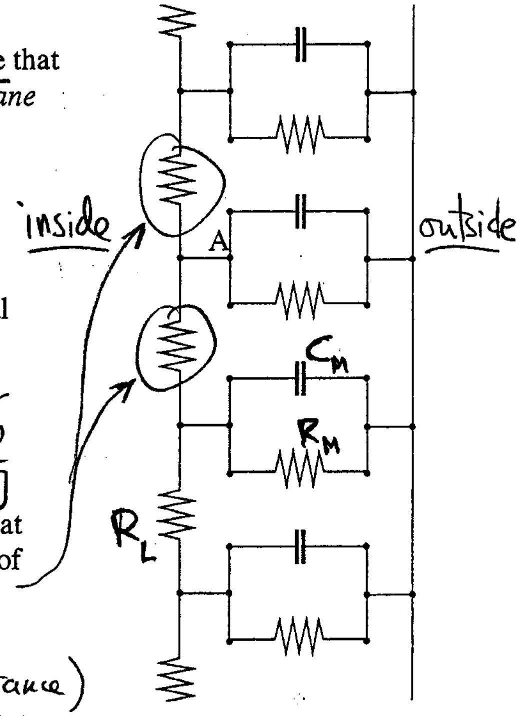
– simulate by having correlation threshold below which, no change

$$\Delta w = \begin{cases} \text{corr} & \text{if corr} > \text{thresh} \\ 0 & \text{if corr} \le \text{thresh} \end{cases}$$

**5.** Voltage changes in wires are propagated at nearly the speed of light (very fast). In cells, by contrast, a *current pulse* injected into the end of a dendrite can take a number of milliseconds (a long time) to strongly affect the cell body because of capacitance.

4 (a) Label the parts of the equivalent electrical circuit for a dendrite that correspond to: *inside, outside, membrane resistance, membrane capacitance, longitudinal resistance*

1 point → $C_M$, $R_M$, → $R_L$




2 (b) Which is greater per unit length of dendrite: ***membrane*** resistance or ***longitudinal (=internal)*** resistance?

$R_M \gg R_L$

2 (c) If we ***decrease*** the diameter of a dendrite, will the longitudinal resistance be ***increased*** or ***decreased***?

increased [for given size resistor, a smaller number of resistors in parallel have higher resistance]

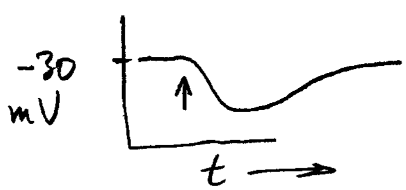
2 (d) Assume we inject a constant amount of current into our model at point A for a long time (e.g., 10 sec). ***Circle*** the ***part or parts*** of the circuit through which the ***most*** current will be flowing at the end of 10 sec (don't circle any of the wires!)

— two longitudinal resistors (low resistance) nearest injection site, follows from (a)

**6.** The retina contains photoreceptors, bipolar cells, and ganglion cells (among other cell types).

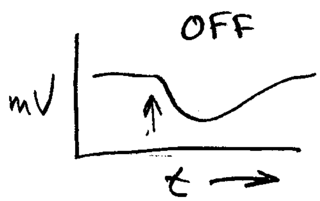
(a) Draw a graph of how the ***membrane potential*** of a photoreceptor ***changes*** when you shine a light on it. Label both axes and indicate when the light was turned on with an arrow.

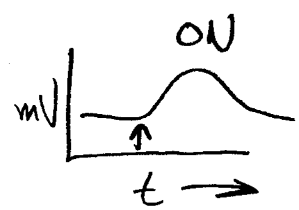
3




2 (b) How does the membrane potential ***response to light*** of ON and OFF bipolar cells differ?

— it moves in opposite directions




3 (c) Many cells in the retina *do not* spike. Why is this?

— they are close enough to each other that passive dendritic potentials propagate sufficiently to cause vesicle fusion and synaptic transmission

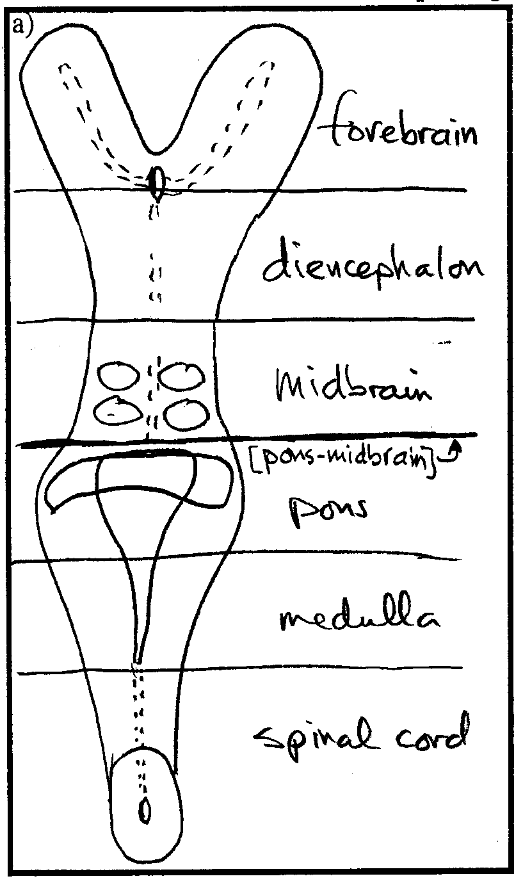
2 (d) Some cells in the retina *do* spike. Which ones are these, and why do they spike?

— retinal ganglion cells (and one amacrine cell type)
— they spike because their targets (e.g. superior colliculus, dLGN) are many mm away (passive propagation would die out)

**7.** Make a diagram of the neural tube below *left* and *label* its 6 main caudal-to-rostral segments. Include the ***cerebellum*** and the ***ventricle openings***, and use ***dotted lines*** to show where the ventricle is ***not*** open.

6

a)



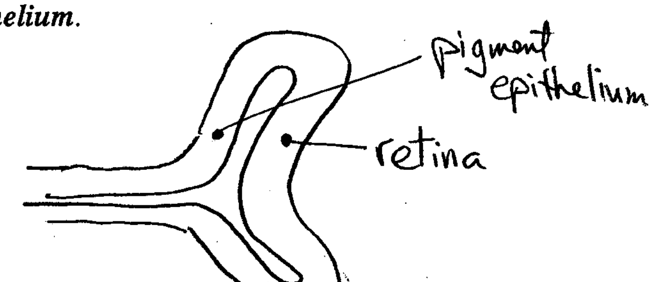

(b) State a general rule for whether the connection between two ***brain structures*** will be crossed or uncrossed.

2

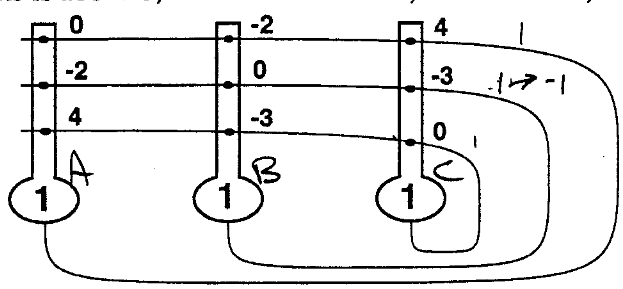
- if two structures on same side of pons/midbrain junction then uncrossed
- if on opposite sides, crossed

(c) Draw a crude picture of the ***optic cup*** (an outpocketing of the neural tube) and indicate which part of it will form the ***retina*** and which part will form the ***pigment epithelium***.

2




**8.** The recurrent network below has 3 units (***initial*** activations are inside) and 9 weights. The update rule is: if weighted sum of inputs is above 0, unit activation => 1, if below zero, unit => -1.




3

(a) Update each unit *once*, ***left to right*** (***show calculations***, don't forget to ***propagate updates***!)

A: $(1 \times 0) + (1 \times -2) + (1 \times 4) = 2$ which is $> 0$ so 1 no change

B: $(1 \times -2) + (1 \times 0) + (1 \times -3) = -5$ which is $\leq 0$ so -1 change

C: $(1 \times 4) + (-1 \times -3) + (1 \times 0) = 7$ which is $> 0$ so 1 no change

3

(b) Was the network in a stable state before you updated it? (why or why not)?

no, unit B changed

2

(c) Is the network in a stable state *after* you updated all three units? (why or why not)?

yes, updating any unit causes no further change

2

(d) Is this weight matrix *symmetric* (say why it is or isn't)?

yes, $w_{ij} = w_{ji}$ [coming = going] (self connections = 0 irrelevant to question!)