

*Q about mistake in flashCube file, concerning the basilar membrane in the cochlea: apical and basal are turned around.*

The correct phrasing is as follows: "The basilar membrane vibrates most at its **basal** end (nearest the tympanic membrane) with high frequency sounds. It vibrates most at its **apical** end (farthest from the tympanic membrane) with low frequency sounds."

*Q: You talked about the corpus striatum and neocortex and evolution. How does that tie in with our ability to multi-task?*

Sensory information reaches both corpus striatum and neocortex from thalamus. This was true early in evolution. Later in evolution, the main route for sensory information to reach the corpus striatum was through the neocortex. The neocortex influences movement not only *via* the striatum but by various routes that don't involve the striatum. For example, there are transcortical connections that reach the motor cortex, and there are projections from various areas to the midbrain's superior colliculus. These pathways operate simultaneously with the pathways that go to the corpus striatum. Thus, we can follow learned habits using striatal mechanisms, and simultaneously we can use the same information to make plans, using prefrontal neocortex.

*Q: What does psychedelic mean in the context of hallucinogens?*

It means "clear mind" and mind-expanding. Hallucinogens can cause vivid sensory effects and alterations of the apparent meaning of sensations. They are "clear" in the sense of being very strong and influential, but those states do not correspond well to the real world. Furthermore, the drugs can damage the brain.

*Q: Is directional specificity in amacrine cells and ganglion cells caused by two types of neurotransmitters being released, or is it because of IPSP? (I was confused because the diagram showing directional specificity didn't show bipolar cells synapsing on ganglion cells, but I thought they did).*

Bipolar cells do synapse on ganglion cells, but they also synapse on amacrine cells. The amacrine cells synapse on ganglion cells and also on each other. In the model, the directional specificity results from asymmetric excitatory and inhibitory connections by the amacrine cells that are activated by the bipolars. When the stimulus moves in the null direction, amacrine cells cause inhibition before the excitation from the next amacrine cell can be effective. When the stimulus moves in the preferred direction, the inhibition arrives too late, after the excitation. Inspect the picture and you can see this.

*Q: Can you explain again the test of how they found the refractory periods of drive and reward?*

If you stimulate axons with pairs of pulses, each pair will act like a single pulse if the second in the pair occurs when the axon is within the refractory period. If the separation between the pulses in a pair is greater than the refractory period of the axons, then each pulse pair will stimulate

action potentials twice. The measures of drive (e.g., speed of running to get to the place where they were stimulated) showed increases at one inter-pulse interval, and the measures of reward (learning to make one choice rather than another) showed increases at another interpulse interval. Therefore, the two effects depended on different axons, with different refractory periods.

*Q: What is the reason behind the different effects observed when *cerveaux isole* and *encephale isole*? (I mean which connections are being damaged when the cortex becomes permanently asleep but the rest of the brain still has normal sleep/wake cycles, and why are they preserved in the other lesion?)*

In the *cerveaux isole* cat, the hindbrain is behind the cut and the forebrain is in front of the cut. Since the forebrain is always sleeping and the spinal and hindbrain mechanisms controlling the muscles show the signs of sleeping and waking (a 24 hour cycle with periodic relaxation of muscles as seen in REM sleep during one part of the cycle), we know that the mechanisms controlling sleep-waking cycles must be behind the cut. If the cut is made just behind the hindbrain (*encephale isole*), then the forebrain shows sleep-waking cycles but the body does not. The conclusion is that the basic control of the sleep-waking cycle comes from the hindbrain. (This does not contradict the evidence that sleep and waking are also regulated by hypothalamic mechanisms.)

*Q: The nucleus laminarius has to be have spatial summation from opposite sides to be excited. Where do these inputs come from? Also I think you mentioned that this was similar to the superior olive in the human brain but didn't go into the details. Can you explain how the the superior olive works in a similar way?*

The inputs from the opposite side are from decussating branches of axons from the cochlear nucleus (the part called *nucleus magnocellularis* in the bird). There is evidence that the medial nucleus of the superior olive works in the same way, but the best evidence comes from the studies of the chicken. [See below for more on this.]

From the review:

- *question #3 (if the input from lungs is necessary for the breathing cycle)*

Breathing can occur without the input from the lungs. Study the model presented in class 23.

- *question #18: meaning of multiple parallel pathways and some examples*

For the whole organism: the inputs coming in through different modalities at the same time. In the visual system, the retina projects to multiple subcortical structures, so information is being processed simultaneously in each of those structures, serving different functions. Within the pathway going just to the lateral geniculate body there is a separation between the pathways from the small retinal ganglion cells and the large retinal ganglion cells. They are concerned with different kinds of information. There are many other examples.

- *question #24 (what do you mean by functional correlates of anatomical plasticity?)*

An example of such plasticity after brain damage in baby hamsters: After unilateral SC ablation, fibers will grow over the damaged tissue, and some of them will cross to the wrong side of the brain. The functional effect is turning in the wrong direction for stimuli in parts of the visual field.

- *question #26 (examples of experiments showing that total blindness of visually guided movements still allows for response to light)*

Cut the optic tract below the lateral geniculate nuclei, eliminating inputs to the midbrain and thalamus, and the animal will be blind in all tests of visually guided behavior (e.g., orienting movements, visual discrimination tests, tests of reactions to predators) but the animal can still have its circadian activity rhythm entrained by the light-dark cycle. This is because it still has the connection from the retina to the suprachiasmatic nucleus.

- *question #41 (why does the labeled line theory fail to explain pain perception?)*

It cannot easily explain why pain comes back after cutting the spinothalamic tract, since that is supposed to be the pathway for pain. By itself, it cannot explain pain that sometimes occurs after very long latencies, as in causalgia cases. Something else is going on other than simply stimulation of one specific pathway to the brain.