

Answers to student questions, 2:

*Q: Phenomena of anatomical plasticity not involving brain lesions: Could it involve changes in cortical maps after sensory deprivation?*

Yes, that could be a good answer. Changes in the width of ocular dominance columns in the striate cortex after early monocular deprivation is the best known phenomenon.

*Q Describe the properties of receptive fields for two neocortical cells, one in the "primary" visual cortex (striate cortex = area 17) and one in an extrastriate area.*

The chapter on vision in the textbook describes such receptive fields pretty well.

*Q: What are: "cascade specification" and "magnification"?*

Cascade specification was described most explicitly in the discussion of the retina (Werblin and Dowling experiments with mudpuppy), but also when I showed some unit recording results from auditory cortex. It means that in successive stages along a sensory pathway, receptive field properties become more and more selective because outputs of neurons at one stage are combined by convergent connections with neurons at the next stage.

Magnification is a term used to describe topographic maps of sensory surfaces. It refers to how much brain area or distance along a surface is devoted to a given area of the sensory surface or distance along that surface. For example, in the visual cortex, magnification can be expressed in terms of millimeters of cortex per degree of visual angle.

*Q: In which lectures did we discuss the Mishkin experiment (was it explicitly stated as his experiment - I can't seem to be able to find it)*

It was in lecture 31 on ablation studies of the visual system: See the slide entitled "Breaking the connection between striate cortex and inferotemporal cortex: the lesion sequence"

*Q: Could you clarify if the following is correct?*

*Superior colliculus: "where" system, for orienting toward visual stimulus*

*Striate cortex: "what" system, pattern discrimination*

*So the above is the double-dissociation discovered from Syrian hamster studies?*

A: The "double dissociation" means that for lesions of the SC, the animals lose their ability to orient towards a stimulus but don't lose their ability to discriminate patterns, whereas for lesions of the visual cortex, it is the opposite: they lose pattern discrimination ability but not orienting ability.

*Q: But then there are these dorsal and ventral streams that correspond to magnocellular and parvocellular systems? Do the dorsal and ventral streams separate from striate cortex?*

*Dorsal (magnocellular system): where*

*Ventral (parvocellular system): what*

A: The magnocellular and parvocellular systems stay separated beginning at the retina, through the lateral geniculate nucleus and to the striate cortex, and some separation is maintained after that because the small receptive fields and the color responsiveness are more important for object vision, the major function of the ventral stream going into the inferotemporal cortex. The dorsal stream is more concerned with spatial location, for which color is not important, whereas movement is often important. But the separation of stimulus properties is not complete. Both magno- and parvocellular systems contribute to the ventral stream, and both contribute to the dorsal stream as well, but they differ in their relative contributions in the way that you are thinking.

*Q: The only receptive fields I know about are of the ganglion cells: the midget cells (small receptive field) of the parvocellular system and parasol cells (large receptive field) of the magnocellular system.*

*What threw things off is that on one of the review packets the TAs gave [2002], it says "IT cortex cells have large receptive fields." But from the above, IT is part of the ventral stream, which has small ganglion cells. Is that completely different from the receptive fields of cells in the striate/extrastriate areas?*

A: First, the ventral stream has properties which are not determined only by the small ganglion cells of the retina, but inputs originating in those cells are very important. The receptive field of a cell can be described as not only a location in the sensory space, and a size, but also by the specific properties of the stimulus that are most important for exciting the cell.

Neville described how receptive fields are built up in the mudpuppy retina (class 29), but for the neocortex, I expected you to be reading the textbook, and I covered few details. But later, I described some receptive fields of neurons in the auditory cortex. In that case, the properties were defined in terms of temporal patterns of sounds, like tones rising or falling in frequency.

*Q: What are the lateral and medial forebrain bundles and their importances?*

The medial forebrain bundle connects the limbic endbrain structures with the hypothalamus and the limbic midbrain areas. Axons go in both directions within it. Thus, it is of major importance for limbic system functions like control of motivational states and expression of emotion.

The lateral forebrain bundle consists of all the axons connecting neocortex and corpus striatum with brainstem and spinal cord. Thus, it is critical for both sensory and motor system functions.

*Q: What's the general conclusion of the Lawrence and Kuypers expt?*

Separate control of 1) axial muscles and whole body movements, and 2) distal muscles and fractionated movements. #1: Medial hindbrain pathways. #2: Lateral hindbrain pathways, especially the axons coming from the red nucleus of the midbrain.

*Q: What's the point of Bizzi's expt?*

They discovered a basic plan of propriospinal neuron organization for control of the limbs. Separate pools of interneurons were found to control movements towards different spatial locations. By combining the activation of different spinal modules, higher systems can produce more complex movements, by a kind of vector summation.

*Q: What's the difference between Long-term Potentiation and Post-tetanic Potentiation?*

Post-tetanic potentiation occurs in reflex connections to motor neurons in the cord; the "tetanus" required to get the potentiation of the connection is not physiological -- it is an artificially fast electrical stimulus of sensory fibers.

LTP occurs with physiologically possible inputs to cells in various places believed to be important in natural learning, such as in the hippocampus and the neocortex.

*Q: What's the difference between habituation and adaptation?*

Adaptation is a decreased responsiveness of primary sensory neurons; it is not a CNS phenomenon, but occurs at the level of receptors. Habituation occurs in the CNS.

*Q: What causes the splitting of the activity cycle in regards to the per gene?*

The splitting of the activity cycle described in the text (fig. 14.9) indicates more than one biological clock mechanism. Studies of expression of several "per" genes have shown a possible correlate of this: These genes can be expressed in different phases in the left and right suprachiasmatic nuclei, and different per genes may not be in synch with each other.

*Q: What's optokinetic nystagmus?*

If all or large parts of the visual world move across the retina together, the eyes follow reflexly, and then when they reach an extreme position, they jump back to a centered position before continuing to follow.

*Q: What's the role of the olivocochlear bundle?*

One role is the suppression of lower threshold fibers in the auditory nerve in the presence of noise. This improves discriminability of sounds when there is a lot of background noise.