Instructor's Answer Key

Chapter 10: Sensory Physiology

Answers to Test Your Understanding of Concepts and Principles

- 1. *Lateral inhibition* is the diminution of incoming sensory information from receptive fields that are peripheral to the one that is stimulated to the greatest degree. This results in sharpening of sensation by enhancing the contrast between the sensory fields that is most stimulated and the surrounding sensory fields. Lateral inhibition occurs in the relaying of tactile information in the skin, in the cochlea where it enhances pitch discrimination, in the relaying of impulses from the retina, where antagonistic center and surround visual fields help to heighten visual contrast of edges, and in olfaction where it helps the brain more clearly distinguish closely related odors. [Note: This question is also answered in the Student Study Guide.]
- 2. The generator (receptor) potential is a graded depolarization that is produced by sensory nerve endings (receptors) and is analogous in its characteristics to an EPSP. The greater the stimulus intensity, the greater the amplitude of the generator potential. Once threshold depolarization has been reached, the greater the amplitude of the generator potential, the greater will be the frequency of action potentials that the receptor stimulates in the sensory axon. Acting through changes in action potential frequency, tonic receptors thus provide information about the relative intensity of a stimulus.
- 3. The phenomenon of phantom limb pain occurs in people with an amputated limb. One explanation of phantom limbs is that the nerves remaining in the stump can grow into nodules called neuromas, which generate nerve impulses that are transmitted to the brain and are interpreted as arising from the missing limbs. Other theories propose that the source of the phantom pain may arise in the reorganization activity of several brain regions whose usual activity is somehow changed by the absence of the sensations that would normally arise from the missing limb.

- 4. The senses of smell and taste are closely related. The receptors for taste and olfaction respond to molecules that are dissolved in fluid, and are thus classified as chemoreceptors. These chemoreceptors are further classified as exteroceptors, which respond to chemical changes in the external environment. Taste receptors are specialized epithelial cells with microvilli. These receptors are not neurons, but can depolarize under appropriate stimulation and to release chemical transmitters that stimulate associated sensory neurons. Salty and sour tastes perception is initiated by ion movement through membrane channels. Sweet, bitter, and umami taste responses, however, are produced by activating specific membrane receptor proteins coupled to G-proteins (gustducins). Taste perception involves the action of secondmessenger systems, including cAMP. Olfactory receptors are bipolar sensory neurons with nonmotile cilia. Odorant molecules bind to receptor proteins, cause the release of different Gprotein subunits that activate adenylate cyclase to produce cAMP molecules in the receptor. Increased cAMP concentrations open ion channels allowing the influx of Na^+ and Ca^{2+} producing a graded depolarization that can lead to action potentials. Information from olfactory receptors is transmitted directly to the cerebral cortex instead of being relayed through the thalamus.
- 5. As the head accelerates in one direction, inertia causes structures in the vestibular apparatus to lag behind. These structures are the perilymph within the semicircular canals (rotational) and the calcified stones of the otolith membrane (linear). As a result of the inertia of perilymph, the cupula within the semicircular canal bends, deforming hair cells and stimulating the production of action potentials. Similarly, the inertia of the otolith membrane within the utricle and the saccule also causes bending of hair cell processes, depolarization and the production of action potentials.
- 6. Accommodation refers to the ability of the eyes to maintain a clear focus on the retina when the distance from the object seen to the eyes is varied. This is accomplished by changes in the curvature of the lens as a result of contraction and relaxation of the ciliary muscles. When the object is farther than twenty feet from the eyes, the ciliary muscles are relaxed, and the lens is pulled to its thinnest, least convex form. When the object is closer than twenty feet, the ciliary muscles contract, taking slack out of the suspensory ligaments and allowing the lens to become thicker and more convex. Also, as an object is brought closer to the face the extrinsic eye muscles contract causing medial movement of the eyes. This ciliary and extrinsic muscle contraction explains why it is more of a strain to look at near objects than at far ones.
- 7. Rod and cone photoreceptors are activated when light produces a chemical change in molecules of pigment contained within the membranous discs of the outer segments of the receptor cells. In the dark, the photoreceptors release an inhibitory neurotransmitter that hyperpolarizes the bipolar neurons. Thus inhibited, the bipolar neurons do not release excitatory neurotransmitter to the ganglion cells. Light inhibits the photoreceptors from releasing their inhibitory neurotransmitter and by this means stimulates the bipolar cells, and thus the ganglion cells that transmit action potentials to the brain.

- 8. The cones in the fovea centralis synapse with bipolar cells in a ratio of 1:1 (no convergence). Each bipolar cell here thus receives input from only one cone, and "sees" only the portion of the retina served by that cone. This allows great resolution of the image when compared to the periphery where cones are more scattered and interspersed with rods. In more peripheral parts of the retina many rods synapse with a single bipolar cell, so that the receptive field of the bipolar cells is greater at the periphery than at the fovea. The convergence of rods on bipolar cells at the periphery of the retina is used when an image is seen from the "corners of the eyes." Rod convergence allows summation of light stimuli to occur and thus provides greater sensitivity at low levels of illumination.
- 9. All rods have thousands of molecules of the same visual pigment, rhodopsin. This pigment appears purple combined red and blue since it reflects these colors and maximally absorbs wavelengths at about 500 nm (blue-green light). Thus an image produced by stimulation of rod photoreceptors will be seen in only one color of different intensities, or black and white. It is the presence in the retina of cone photoreceptors that have different visual pigments present in the three types of cones that permits color vision. Color blindness is caused by a congenital lack of one or more types of cones, usually the absence of either the L (red) or M (green) cones (are dichromats). Similarly, people who have only one of the three types of cones (are monochromats) lack selected color vision and therefore see only in black and white.
- 10. Light in the green region of the spectrum (500 nm) is absorbed best by rhodopsin, and thus is most effective in stimulating rods and is more easily seen. Rhodopsin does not absorb red light well. A red light in a darkroom will not cause bleaching of rhodopsin, and thus will not hinder dark-adaptation. A person can see more clearly in a darkroom with red light because the red light specifically stimulates one of the three types of cones and the rods will still be functional (dark-adapted).
- 11. The receptive fields of ganglion cells are roughly circular with an antagonistic center and surround. If light that shines in the center (on-center fields) of the field stimulates the ganglion cell, light that shines in the surround of the field (off-center fields) will inhibit the ganglion cell, and vice versa. The antagonism of center and surround of the visual field helps to sharpen the difference in effect of light intensity shining on different parts of the retina and thus helps to sharpen the image produced.
- 12. Three genes code for the photopsins involved in color vision. The gene for the S cones (short wavelengths) is located on chromosome number 7, whereas the genes for the M (medium wavelengths) and L (long wavelengths) cones are located on the long arm of the X chromosome. The number of genes coding for the sense of taste is complex and still unknown. On the other hand a family of genes, perhaps a many as a thousand, code for the olfactory receptor proteins involved in smell. As a result, the brain is required to process the information from many more inputs in olfaction (and taste) as compared to vision. This would suggest that the level of integration is more complex, and consequently the pathways to the regions of the brain interpreting olfactory information.

Answers to Test Your Ability to Analyze and Apply Your Knowledge

- Hearing requires compression and rarefaction waves of particles entering the external auditory meatus and subsequent mechanical displacement of the tympanic membrane, ossicles, and so on. In outer space where a vacuum exists, the absence of particle waves would preclude hearing. The receptors for hearing (hair cells) therefore are responsive to mechanical stimulation – particle waves, ossicles amplification, oval window compression, with subsequent fluid waves in the endolymph fluids surrounding and stimulating the hair cell receptors. Visual receptors by contrast, are photoreceptors (rods and cones) that are stimulated by electromagnetic waves in the visual portion of the light spectrum. Light changes the biochemical structures of visual pigment molecules present in the photoreceptor cells resulting in electrochemical stimulation of neighboring cells of the retina and ultimately generating action potentials that travel to the brain for interpretation.
- 2. People with conduction deafness can't hear because the transmission of sound waves from the air to the cochlea of the inner ear for transduction into action potentials has been interrupted by disease, damage, or blockage along the conduction pathway. The hair cell receptors in the cochlea however are unaffected and can respond normally. Consequently, the conduction deaf person's speaking volume, which is heard from inside the head and thus bypasses the blockage, resembles that of someone with normal hearing. People with sensorineural deafness, by contrast, cannot hear his/her own voice volume normally because the hair cells in his cochlea are *not* responding to the sound vibrations resonating in his head and therefore tend to speak in louder tones.
- 3. The fact that opioid drugs have little effect on the initial sharp pain of a pinprick implies that opioid drugs do not inhibit firing of sensory pain fibers along rapidly conducting myelinated axons. Instead, opioid drugs inactivate the slower conducting unmyelinated axons that synapse in the spinal cord using substance P and glutamate as neurotransmitters, thereby relieving the sensation of dull, persistent pain. Aspirin works as an analgesic in the relief of this latter type of pain by inhibiting the enzyme responsible for the synthesis of the specific prostaglandin (PG) subtype that causes pain.
- 4. The G-proteins involved in taste perception are called *gustducins* whereas those associated with the photoreceptors of the eye are called *transducins*. Both result in the activation of second-messenger systems that lead to firing of the respective sensory neurons and the transmission of impulses to the brain for interpretation. Sensory modulation and control seems to be the advantages of having G-proteins mediate the effect of a stimulus on a receptor cell. In response to stimuli, G-proteins in the receptor cells via dissociation of their subunit complex combinations can respond in a variety of ways by activating different second-messenger systems. For example, sweet taste receptors act by producing cyclic AMP second messengers whereas the artificial sweeteners aspartame, saccharin, and cyclamate may activate others, such as inositol triphosphate (IP₃) and diacylglycerol (DAG) second-messenger systems.

5. The vestibular apparatus sensory structures provide us with our orientation of the head with respect to gravity and to movement in any direction. Inertia provides us with our reference position sensation. The sensory hair cells with their extensions (kinocilium and stereocilia) are upright and unbent when the head is at rest or not moving. A normal background pattern of action potentials is continuously sent by sensory neurons to the brain recording the lack of head movement. Movement of the head in any direction (acceleration/deceleration) causes endolymph fluids surrounding the hair cells to move. Depending on the direction of movement, the hair cells extensions bend and either increase or decrease their frequency of firing impulses, notifying the brain of not only the direction of the movement but also the intensity. Once an airplane reaches cruising speed there is no longer any acceleration or deceleration, the hair cell extensions return to their upright position and the firing pattern of impulses returns to normal. At this constant velocity there is no sensation of movement.