

# Instructor's Answer Key

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## Chapter 9: Autonomic Nervous System

### Answers to Test Your Understanding of Concepts and Principles

1. Most sympathetic ganglia are located parallel to the vertebral ganglia in a paravertebral chain. In addition, there are collateral or prevertebral sympathetic ganglia within the abdominal cavity (celiac, superior and inferior mesenteric ganglia). Sympathetic neurons are thoracolumbar in origin – emerge as thoracic (T1-12) to the lumbar (L2) spinal nerves; whereas parasympathetic neurons are craniosacral – emerge from the brain (cranial nerves) or descend within the spinal cord and emerge from the sacral region. Parasympathetic ganglia are located either next to or actually within the organ innervated by fibers from these ganglia. [Note: This question is also answered in the Student Study Guide.]
2. The inner part of each adrenal, called the adrenal medulla, is derived from the same embryonic tissue that gives rise to sympathetic ganglia. Like sympathetic ganglia, each adrenal medulla is innervated by preganglionic sympathetic nerve fibers. Thus, whenever the sympathetic nervous system is activated the adrenal medulla is stimulated by these preganglionic fibers to secrete the hormone epinephrine (80%) and norepinephrine (20%). Epinephrine is delivered by the blood to target organs where it acts much like the neurotransmitter, norepinephrine that is released by the nerve endings of postganglionic sympathetic nerve fibers in response to fight-or-flight. The epinephrine secreted by the adrenal medulla thus augments and complements the actions of norepinephrine and sympathetic nerves.
3. Adrenergic stimulation (by epinephrine and norepinephrine) results in increased heart rate, increased strength of the heart's contraction, and either constriction or dilation of blood vessels. The alpha-adrenergic effect (vasoconstriction) is the dominant effect compared to the beta-adrenergic effect (vasodilation). Adrenergic stimulation reflects sympathetic stimulation or mass activation and in this way stimulates the heart and helps to raise blood pressure. Cholinergic effects of parasympathetic nerves by contrast, act to slow the cardiac rate and cause vasodilation in some blood vessels. The secretions and motility of the digestive tract, and blood flow through the digestive tract, are increased by cholinergic effects and decreased slightly by adrenergic effects.
4. Changes in the level of sympathetic nerve activity can have opposing effects on a target organ. The smooth muscles in the wall of most blood vessels, for example, receive only sympathetic innervation. At a particular level of sympathetic activity these smooth muscles may maintain a state of contraction that is associated with vasoconstriction. In this case, a decrease in sympathetic nerve activity will cause more relaxation of these smooth muscles and result in vasodilation. The adrenal medulla (and its release of the hormone epinephrine), the arrector pili muscles in the skin, and sweat glands in the skin are the other examples of effectors that receive only sympathetic innervation.

5. There are two major types of adrenergic receptors, called alpha and beta. Each of these, in turn, is divisible into two subtypes. Stimulation of  $\alpha_1$  receptors causes smooth muscle contraction that lead to vasoconstriction, for example. Stimulation of  $\alpha_2$  receptors in the brain decreases the level of activity of the sympathetic nervous system. When activated,  $\beta_1$  receptors in the heart stimulate the pumping activity of the heart. Stimulation of the  $\beta_2$  receptors causes muscle relaxation, as in the bronchioles of the lungs. Drugs are used clinically to selectively stimulate or block each of these receptors, and thus, for example, to control blood pressure or treat asthma.
6. Drugs that function as  $\alpha_1$  agonists, such as phenylephrine, are often part of nasal sprays because they promote vasoconstriction in the nasal mucosa. Atenolol is a drug that blocks  $\beta_1$  receptors (antagonist) located on the heart resulting in a lowered cardiac rate and blood pressure. Terbutaline is an effective  $\beta_2$  agonist used in the relief of bronchoconstriction in asthmatics. Clonidine is a drug that selectively stimulates  $\beta_2$  receptors located on neurons in the brain. Clonidine reduces the activation of the sympathoadrenal system and thereby helps to lower the blood pressure.
7. Cholinergic receptors are divided into nicotinic and muscarinic subtypes. Like the drug muscarine, ACh stimulates the muscarinic cholinergic receptors in visceral organs such as the heart, digestive system, and other target organs of postganglionic parasympathetic nerve fibers. These axons must thus exert their effects on the target organs by stimulating the muscarinic subtype of cholinergic receptors. By contrast, the drug nicotine specifically stimulates cholinergic receptor proteins in autonomic ganglia or at the neuromuscular junction of skeletal muscle fibers. ACh released by somatic motor neurons and by preganglionic autonomic ganglia thus stimulates nicotinic subtypes of ACh receptors.
8. The drug curare (tubocurarine), used clinically to cause skeletal muscle relaxation, specifically blocks nicotinic receptors that are stimulated by nicotine. The drug atropine, which specifically blocks the muscarinic receptors, is used clinically to dilate pupils during eye examinations, to dry mucous membranes of the respiratory tract prior to general anesthetic, and to inhibit spasmodic contractions of the lower digestive tract. Muscarine, derived from some poisonous mushrooms, stimulates muscarinic ACh receptors.

## Answers to Test Your Ability to Analyze and Apply Your Knowledge

1. Spinal cord damage at the cervical level (spinal shock) would negatively affect autonomic motor effector impulses that emerge below the neck injury. Since the sympathetic division of the autonomic nervous system emerges from the spinal cord along the T1 to L2 portion of the vertebral column, sympathetic innervation of smooth muscle, cardiac muscle, and glands would be blocked. Therefore, postganglionic sympathetic stimulation of smooth muscle in blood vessels would be inhibited leading to vessel relaxation and circulatory slowdown. This subsequent decrease in blood flow to the brain would affect consciousness and possibly cause shock. Also, low blood flow results because this injury removes any normal autonomic reflex stimulation of the heart pacemaker region to increase heart rate or any enhancement of cardiac contractility that would increase stroke volume and cardiac output. Finally, the postganglionic sympathetic interruption in impulses to endocrine glands that modulate the “fight-or-flight” response would include the adrenal glands (sympathoadrenal), whose normal release of catecholamine hormones epinephrine and norepinephrine would be compromised. Consequently, the drop in the levels of circulating catecholamine hormones would further reduce the patient’s ability to maintain brain circulation and could result in circulatory shock.
2. Although both sympathetic and parasympathetic nerves innervate most organs, some – including the adrenal medulla, arrector pili muscles, sweat glands, and most blood vessels – receive only sympathetic innervation. Activation of the sympathetic “fight-or-flight” response to shock in this person would constrict most blood vessels resulting in pale, cold, skin, while stimulation of the heart sinoatrial node would cause a reflex increase in heart rate. Sympathetic nerve activity also stimulates sweat glands in the trunk causing the release of a watery fluid that in addition to its cooling effect will also cause clammy skin. The weak pulse is partly due to sympathetic relaxation of skeletal muscle blood vessels that causes blood pooling in the periphery with very low venous blood return producing a very inadequate stroke volume. As the venous return and cardiac output fall the heart contractility becomes weaker and weaker. Drug treatments of choice would be sympathetic agonist such as epinephrine which would generate peripheral vasoconstriction, increase venous return, stimulate the heart to beat faster with stronger beats, and improve circulation of blood to all areas, including the skin.
3. At the starting block, higher brain centers have started mass activation of the sympathoadrenal “fight-or-flight” response. Visceral responses such as the gastrointestinal tract or the reproductive system for example that is primarily under parasympathetic control would be inhibited while all systems under sympathetic regulation would be energized. Typical organ responses for example, would include the pupils of the eyes would dilate, the heart would accelerate and increase its force of contraction, vascular smooth muscle to viscera would close in favor of widespread vasodilation of skeletal muscle (cholinergic) blood vessels, bronchiolar smooth muscle would relax facilitating breathing, and the sweat glands of the skin would secrete a watery fluid to help in thermoregulation.

4. Beta-blocking drugs such as propranolol or atenolol, block beta<sub>1</sub> receptors that are located in the heart and thereby lowers the diastolic blood pressure by lowering the heart rate at the SA node. Propranolol, however also blocks beta<sub>2</sub> receptors located in the smooth muscle of lung bronchioles. This opposes the beta<sub>2</sub> bronchodilation effect caused by epinephrine, resulting in bronchoconstriction and asthma in susceptible people. Atenolol, however, does not have this effect on the breathing. All subtypes of adrenergic beta-receptors produce their effects by releasing G-proteins that result in stimulation of cyclic AMP production within the target cells. Caffeine in coffee and other derivatives such as theophylline (in tea) raise the intracellular concentrations of cyclic AMP by inhibiting the enzyme responsible for degrading the second messenger. By inhibiting this enzyme, phosphodiesterase, caffeine promotes the action of beta stimulation and facilitates breathing by relaxing bronchiolar smooth muscle.
5. Cold medications may contain an *alpha*<sub>1</sub>-adrenergic agonist that would promote contraction of smooth muscle, particularly in blood vessels and help relieve the “runny nose” of the common cold. Because vasoconstriction in the periphery also raises blood pressure, people with hypertension are warned on the label to use certain cold medications with caution. Use of an *alpha*<sub>2</sub>-adrenergic agonist such as clonidine is more complex since it similarly raises blood pressure by peripheral vasoconstriction yet is prescribed to lower blood pressure by somehow acting in the brain to reduce the activity of the entire sympathoadrenal system. Atropine (belladonna) specifically blocks the muscarinic cholinergic (ACh) receptors, primarily in the visceral organs. Atropine is therefore used to inhibit glandular secretions in the respiratory tract that may accompany the unpleasant symptoms of a cold. Atropine may also be given to a patient with gastritis to inhibit the stomach acid secretion and hence, lessen the inflammation. However, by reducing stomach secretion of acid, the secretion of pepsin enzymes and other components of gastric juice would also be compromised. In this way, atropine would directly interfere with the ability to digest and absorb foods, particularly those foods high in protein.