Q1. Which feature best explains why sympathetic stimulation produces more widespread and longer-lasting effects than parasympathetic stimulation?

A. Sympathetic postganglionic axons release acetylcholine which is rapidly inactivated.
B. Sympathetic postganglionic axons diverge extensively and adrenal medullary hormones circulate systemically.
C. Parasympathetic preganglionic neurons synapse directly on skeletal muscle.
D. Parasympathetic stimulation opens voltage-gated K■ channels faster than sympathetic stimulation.

Q2. A cholinergic neuron in the autonomic nervous system could be which of the following?

A. Most sympathetic postganglionic neurons except those innervating sweat glands.
B. All parasympathetic postganglionic neurons and all preganglionic autonomic neurons.
C. Adrenal chromaffin cells secreting norepinephrine.
D. Somatic motor neurons that secrete norepinephrine at the neuromuscular junction.

Q3. Activating an α1 adrenergic receptor on a visceral effector most commonly causes:

A. Inhibition via opening K■ channels.
B. Excitation such as smooth muscle contraction or vasoconstriction.
C. Degradation of norepinephrine in the synaptic cleft.
D. Direct release of acetylcholine from postganglionic neurons.

Q4. Which statement correctly contrasts nicotinic and muscarinic receptors?

A. Nicotinic receptors sometimes hyperpolarize cells; muscarinic receptors always depolarize.
B. Nicotinic receptors are ionotropic and cause depolarization; muscarinic receptors are metabotropic and can excite or inhibit depending on the subtype.
C. Both receptor types are rapidly inactivated by acetylcholinesterase at the receptor site.
D. Muscarinic receptors are found only on skeletal muscle fibers.

Q5. Why do effects of cholinergic neurons tend to be briefer than adrenergic effects?

A. Acetylcholine is quickly broken down by acetylcholinesterase, while norepinephrine lingers in the cleft.
B. Cholinergic neurons release less neurotransmitter per vesicle than adrenergic neurons.
C. Nicotinic receptors internalize faster than adrenergic receptors.
D. Adrenergic neurons are myelinated while cholinergic neurons are not.

Q6. Which of these best describes an agonist?

A. A substance that blocks a receptor and prevents any ligand binding.
B. A molecule that binds to and activates a receptor, mimicking the natural neurotransmitter.
C. A drug that accelerates enzymatic breakdown of neurotransmitter.
D. A transporter that removes neurotransmitter from the synaptic cleft.

Q7. During a single action potential in a typical neuron, the membrane potential moves approximately from:

A. –90 mV → 0 mV → +100 mV.
B. –70 mV → –55 mV → –40 mV.
C. –70 mV → +30 mV → –70 mV (with possible after-hyperpolarization to ~ –90 mV).
D. 0 mV → +30 mV → +70 mV.

Q8. Which factor contributes the least to the resting membrane potential of a typical neuron?

A. Unequal distribution of ions between ECF and cytosol.
B. Inability of many anions to leave the cell.
C. Electrogenic action of the Na■–K■ ATPase pump.
D. Rapid cycling of voltage-gated Na■ channels at rest.

Q9. The graded potential differs from an action potential because a graded potential:

A. Is all-or-none and nondecremental.
B. Can sum with other graded potentials and is decremental over distance.
C. Is propagated without regeneration along the membrane.
D. Always triggers neurotransmitter release at axon terminals.

Q10. If a ligand-gated Na■ channel opens on a dendrite creating a small depolarization, that event is called:

A. An action potential.
B. A graded (postsynaptic) potential that may contribute to threshold at the axon hillock.
C. A refractory potential.
D. An electrogenic potential created by the Na■–K■ pump.

Q11. Which channel type is correctly paired with its primary physiological trigger?

A. Mechanically-gated — opens in response to neurotransmitter binding.
B. Ligand-gated — opens in response to changes in membrane voltage.
C. Voltage-gated — opens in response to changes in membrane potential.
D. Leak channel — opens only in response to mechanical stretch.

Q12. Why can an action potential not travel backward toward the soma after it is initiated?

A. Because the membrane behind it is still in the refractory period and cannot be reactivated.
B. Because axons are insulating cables that only allow forward current flow.
C. Because neurotransmitter release prevents backward conduction.
D. Because voltage-gated Na■ channels exist only ahead of the impulse.

Q13. Which description best explains saltatory conduction in myelinated axons?

A. Continuous opening of voltage-gated channels along the entire axon produces faster conduction.
B. Action potentials regenerate only at nodes of Ranvier, allowing the impulse to “jump” and increase conduction speed.
C. Myelin increases capacitance and slows current flow so nodes are required.
D. Oligodendrocytes actively pump Na■ across internodal regions to sustain conduction.

Q14. The inactivation gate and activation gate of a voltage-gated Na■ channel are important because:

A. They ensure Na■ can flow inward indefinitely during a single action potential.
B. Activation gate opens at threshold while the inactivation gate closes shortly after, creating a transient Na■ influx that contributes to the depolarizing phase.
C. The inactivation gate opens only after K■ channels close.
D. Both gates are controlled by extracellular ligand binding, not voltage.

Q15. Which explanation correctly links receptor subtype to typical physiological effect?

A. $\beta 2$ receptor activation typically causes excitation of visceral smooth muscle (contraction).
B. $\alpha 2$ receptor activation usually leads to inhibition, often by decreasing neurotransmitter release or cellular activity.
C. $\beta 1$ receptor activation generally causes smooth muscle relaxation in bronchioles.
D. $\alpha 1$ receptor activation typically promotes lipolysis in adipose tissue.

Q16. How does the adrenal medulla contribute to the sympathetic response differently than sympathetic postganglionic axons?

A. Chromaffin cells secrete epinephrine and norepinephrine into the bloodstream, producing systemic effects that last longer.
B. The adrenal medulla releases acetylcholine directly onto skeletal muscle.
C. Adrenal hormones are inactivated faster than neurotransmitters in synaptic clefts.
D. Chromaffin cells are parasympathetic postganglionic neurons located in autonomic ganglia.

Q17. Which best describes the refractory period?

A. A time when graded potentials cannot sum.
B. The interval after an action potential when a normal threshold stimulus cannot produce another action potential (absolute refractory), followed by a relative refractory when a larger-than-normal stimulus can.
C. A period wherein action potentials travel faster due to increased availability of Na■ channels.
D. A phase exclusive to cardiac muscle and not present in neurons.

Q18. Why does norepinephrine generally produce longer-lasting effects than acetylcholine when released at autonomic synapses?

A. Norepinephrine is degraded faster in the synaptic cleft than acetylcholine.
B. Acetylcholine binds to adrenergic receptors that internalize quickly.
C. NE lingers in the synaptic cleft and blood-borne catecholamines circulate, while ACh is rapidly hydrolyzed by acetylcholinesterase.
D. Adrenergic receptors are only found on postganglionic neurons, not effectors.

Q19. Which of the following is NOT part of SLUDD typically associated with parasympathetic activity?

A. Lacrimation.
B. Salivation.
C. Defecation.
D. Increased heart rate.

Q20. A pharmacologic antagonist that selectively blocks β1 receptors would most directly reduce:

A. Bronchial smooth muscle relaxation.
B. Heart rate and force of contraction.
C. Pupillary dilation.
D. Salivary secretion.

Q21. What is the primary immediate ionic basis for the rapid depolarizing phase of the neuronal action potential?

A. Massive outflow of K■ through leak channels.
B. Inward movement of Na■ through voltage-gated Na■ channels.
C. Opening of voltage-gated Ca■ channels in dendrites.
D. Electrogenic action of Na■–K■ ATPase reversing polarity.

Q22. The “all-or-none” principle of action potentials means:

A. Any graded depolarization produces an action potential of variable amplitude.
B. An action potential either occurs fully if threshold is reached, or not at all; its amplitude does not depend on stimulus strength (above threshold).
C. Action potentials diminish in amplitude with distance.
D. Subthreshold stimuli produce smaller amplitude action potentials.

Q23. Which is the best functional reason smooth muscle and glands can continue to function some after autonomic nerve supply is lost?

A. They have myelinated somatic motor input.
B. They possess intrinsic pacemaker activity or baseline activity independent of ANS input.
C. They do not require neurotransmitter signaling for any function.
D. Autonomic ganglia become skeletal muscle cells in denervation.

Q24. If an interneuron receives many small excitatory postsynaptic potentials (EPSPs) that overlap in time, causing the membrane at the axon hillock to reach threshold, this is called:

A. Temporal summation (multiple inputs over time at the same synapse) or spatial summation (inputs from multiple synapses); both allow graded potentials to add up to reach threshold.
B. Refractory summation.
C. Action potential averaging.
D. Electrogenic summation produced by Na■–K■ pumps.

Q25. Which statement about leak channels is correct?

A. They open only when mechanically stimulated.
B. They randomly alternate between open and closed and are more numerous for K■ than for Na■, contributing to resting potential.
C. Leak channels are the same as voltage-gated channels.
D. They are absent from neuronal membranes.

Q26. In the context of autonomic innervation, which statement is accurate?

A. All sympathetic postganglionic neurons are adrenergic and release norepinephrine at their effectors.
B. All parasympathetic preganglionic neurons release norepinephrine.
C. Some sympathetic postganglionic neurons that innervate sweat glands are cholinergic and release acetylcholine.
D. Somatic motor neurons are part of the ANS and release norepinephrine.

Q27. During the repolarizing phase of an action potential, which events occur?

A. Voltage-gated Na■ channels open more; K■ channels close immediately.
B. Voltage-gated Na■ channels inactivate and slow Na■ influx while voltage-gated K■ channels open increasing K■ efflux, driving the membrane back toward resting potential.
C. Na■–K■ pumps invert and cause depolarization.
D. Ligand-gated C■ channels open to repolarize the membrane.

Q28. Which statement correctly relates adrenergic receptor binding to ligand specificity?

A. Norepinephrine stimulates alpha receptors more strongly than beta receptors, while epinephrine stimulates both alpha and beta receptors potently.
B. Epinephrine binds only to muscarinic receptors.
C. Norepinephrine is unable to bind any beta receptor subtypes.
D. Acetylcholine binds α and β adrenergic receptors with highest affinity.

Q29. Why does the Na■–K■ ATPase contribute a small but measurable electrogenic effect on resting membrane potential?

A. Because it transports equal numbers of Na■ and K■ out of the cell, creating no net charge difference.
B. Because it moves three Na■ out for every two K■ brought in, producing a small net outward positive current that slightly hyperpolarizes the cell (≈ -3 mV).
C. Because it allows Na■ to leak back in without ATP.
D. Because it directly opens K■ leak channels when active.

Q30. Which of the following best explains why graded potentials are limited to short-distance communication?

A. They trigger conduction by saltatory jumping.
B. They are decremental — their magnitude decreases with distance due to current leakage and membrane resistance.
C. They are actively regenerated at nodes of Ranvier.
D. They always produce action potentials that propagate long distances.

Q31. Which of the following pairs is mismatched?

A. Parasympathetic — “rest-and-digest,” increases SLUDD functions.
B. Sympathetic — “fight-or-flight,” increases heart rate and dilates pupils.
C. Cholinergic neuron — releases norepinephrine.
D. Adrenergic neuron — releases norepinephrine (noradrenaline).

Q32. A drug that blocks acetylcholinesterase (AChE) at parasympathetic synapses would most likely cause:

A. Shortened and reduced parasympathetic effects.
B. Prolonged activation of muscarinic and nicotinic receptors, leading to enhanced SLUDD responses and potential bradycardia.
C. Immediate activation of α and β adrenergic receptors.
D. Inability of preganglionic neurons to release acetylcholine.

Q33. (True/False) The membrane potential becoming more negative than the usual resting level after an action potential is called after-hyperpolarization; it often reaches about −90 mV.

Q34. (True/False) Graded potentials occur only in axons and are regenerated repeatedly so they do not decrease with distance.

Q35. (True/False) Activation of $\beta 2$ adrenergic receptors typically causes bronchodilation and relaxation of some smooth muscles.

Q36. (True/False) The sympathetic division predominates during quiet resting digestion, promoting increased salivation and intestinal motility.

Answer Key

Answer Key

1. B
2. B
3. B
4. B
5. A
6. B
7. C
8. D
9. B
10. B
11. C
12. A
13. B
14. B
15. B
16. A
17. B
18. C
19. D
20. B
21. B
22. B
23. B
24. A
25. B
26. C
27. B
28. A
29. B
30. B
31. C
32. B
33. True
34. False
35. True
36. False