

Virtual Lab Cardiovascular Physiology Practice Test (Sample)

Study Guide



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SAMPLE

Questions

SAMPLE

- 1. The parasympathetic nervous system primarily influences heart rate by releasing what neurotransmitter?**
 - A. Norepinephrine**
 - B. Dopamine**
 - C. Serotonin**
 - D. Acetylcholine**
- 2. What function does the vagus nerve primarily serve in relation to the heart?**
 - A. It increases the heart rate**
 - B. It provides oxygen to the heart**
 - C. It carries signals that decrease the heart rate**
 - D. It aids in the contraction of heart muscles**
- 3. What type of contraction allows the ventricles to pump blood efficiently?**
 - A. Tonic contraction.**
 - B. Phasic contraction.**
 - C. Voluntary contraction.**
 - D. Myogenic contraction.**
- 4. Which effect does Verapamil primarily produce?**
 - A. Positive chronotropic and positive inotropic**
 - B. Negative chronotropic and negative inotropic**
 - C. Positive chronotropic and negative inotropic**
 - D. Negative chronotropic and positive inotropic**
- 5. Why is the resting heart rate lower than the intrinsic firing rate of the SA node?**
 - A. The sympathetic nervous system has control over the heart rate**
 - B. The parasympathetic nervous system has more control over heart rate**
 - C. There are no external factors affecting heart rate**
 - D. The body requires less blood flow at rest**

- 6. What is the effect on heart contractions when resting potential is decreased?**
- A. Increases strength of contractions**
 - B. No effect on contractions**
 - C. Decreases strength of contractions**
 - D. Stops contractions**
- 7. In a 5°C Ringer's solution, what is the observed heart rate behavior in a frog's heart?**
- A. Beats faster than baseline**
 - B. Beats slower than baseline**
 - C. Remains at baseline**
 - D. Irregular heartbeat pattern**
- 8. What does vagal escape imply about the heart's physiology?**
- A. It is solely controlled by the vagus nerve**
 - B. It can recover from vagal inhibition through other mechanisms**
 - C. It indicates permanent damage to heart function**
 - D. It shows a lack of responsiveness to stimulation**
- 9. What physiological response occurs when atropine is administered?**
- A. Decreased heart rate**
 - B. Increased heart rate**
 - C. Stable vascular resistance**
 - D. No change in heart rhythm**
- 10. Why does the amplitude of the ventricular systole remain unchanged with increased stimulation?**
- A. New contractions begin earlier**
 - B. Relaxation phase is prolonged**
 - C. A new contraction cannot begin until relaxation occurs**
 - D. Stimulation causes fatigue**

Answers

SAMPLE

1. D
2. C
3. D
4. B
5. B
6. C
7. B
8. B
9. B
10. C

SAMPLE

Explanations

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1. The parasympathetic nervous system primarily influences heart rate by releasing what neurotransmitter?

- A. Norepinephrine**
- B. Dopamine**
- C. Serotonin**
- D. Acetylcholine**

The parasympathetic nervous system primarily influences heart rate through the release of acetylcholine. This neurotransmitter acts on the heart's pacemaker cells located in the sinoatrial (SA) node, which is responsible for initiating the heartbeat. When acetylcholine is released, it binds to specific receptors in the heart, leading to a decrease in heart rate, known as negative chronotropic effect. This is part of the body's broader response to promote relaxation and conserve energy, countering the effects of the sympathetic nervous system, which usually increases heart rate and blood pressure during stress or physical activity. Understanding this mechanism is crucial in comprehending how the autonomic nervous system regulates heart function.

2. What function does the vagus nerve primarily serve in relation to the heart?

- A. It increases the heart rate**
- B. It provides oxygen to the heart**
- C. It carries signals that decrease the heart rate**
- D. It aids in the contraction of heart muscles**

The vagus nerve primarily serves to carry signals that decrease the heart rate, which is a crucial aspect of the body's autonomic nervous system regulation. This nerve is part of the parasympathetic nervous system and exerts a calming influence on the heart. When stimulated, the vagus nerve releases neurotransmitters such as acetylcholine, which act on the heart's pacemaker cells in the sinoatrial (SA) node. This action reduces the firing rate of these pacemaker cells, leading to a decrease in heart rate. Understanding the role of the vagus nerve is vital as it contrasts with the sympathetic nervous system, which promotes an increase in heart rate and force of contraction in response to stress or physical activity. The balance between these two systems allows for the modulation of heart function according to the body's current needs, demonstrating the importance of the vagus nerve in maintaining homeostasis within the cardiovascular system.

3. What type of contraction allows the ventricles to pump blood efficiently?

- A. Tonic contraction.
- B. Phasic contraction.
- C. Voluntary contraction.
- D. Myogenic contraction.**

The ventricles of the heart pump blood efficiently primarily due to myogenic contraction. This type of contraction is intrinsic to the cardiac muscle tissue, which means that it is initiated by the muscle fibers themselves rather than by external nervous stimulation. Myogenic contraction occurs because cardiac muscle cells possess specialized properties, including the ability to generate action potentials that lead to muscle contraction. The pacemaker cells in the sinoatrial (SA) node spontaneously depolarize and create electrical signals that cause a coordinated contraction of the ventricular muscles. This ensures that when the ventricles contract, they do so in a synchronized manner, effectively pushing blood out into the pulmonary artery and aorta with each heartbeat. The efficiency of ventricular pumping is further enhanced by the elastic properties of the heart muscle, allowing for maximum stroke volume—the amount of blood ejected with each beat—during each contraction. This contrasts with other forms of contraction, which are not intrinsic to cardiac physiology and do not facilitate the same level of coordination and efficiency as myogenic contraction.

4. Which effect does Verapamil primarily produce?

- A. Positive chronotropic and positive inotropic
- B. Negative chronotropic and negative inotropic**
- C. Positive chronotropic and negative inotropic
- D. Negative chronotropic and positive inotropic

Verapamil primarily produces a negative chronotropic and negative inotropic effect on the heart. This means that it decreases both the heart rate (chronotropy) and the force of contraction (inotropy). As a calcium channel blocker, Verapamil inhibits calcium influx into cardiac and smooth muscle cells. In the heart, this leads to a reduction in the excitability of the sinoatrial (SA) node, which is responsible for initiating the heartbeat. Therefore, the negative chronotropic effect reflects a decrease in heart rate, which is particularly beneficial in conditions such as tachycardia where the heart is beating too fast. In terms of inotropic effects, Verapamil lowers the force of cardiac contraction. This is significant because reduced contractility can help prevent excessive workload on the heart, especially in conditions such as hypertension or heart failure, where managing the heart's workload is crucial for patient health. Overall, these effects support Verapamil's use in treating arrhythmias and other cardiovascular conditions, making it important for managing heart rate and contractility effectively.

5. Why is the resting heart rate lower than the intrinsic firing rate of the SA node?

A. The sympathetic nervous system has control over the heart rate

B. The parasympathetic nervous system has more control over heart rate

C. There are no external factors affecting heart rate

D. The body requires less blood flow at rest

The resting heart rate is indeed lower than the intrinsic firing rate of the SA node primarily because the parasympathetic nervous system has a dominant influence over heart rate regulation at rest. The SA node, which is the natural pacemaker of the heart, has an intrinsic firing rate of about 60 to 100 beats per minute when it is not influenced by any neural inputs. However, under typical resting conditions, the parasympathetic system, particularly through the vagus nerve, exerts a strong inhibitory effect on the heart rate. When the vagus nerve is activated, it releases acetylcholine, which acts on the heart to decrease the firing rate of the SA node. This action lowers the overall heart rate to a range more typical of a resting state, generally around 60 to 80 beats per minute. This modulation allows the body to conserve energy and maintain a state of homeostasis when high levels of physical activity or stress are not present. Understanding this interaction is critical because it illustrates how the autonomic nervous system plays a key role in fine-tuning various physiological functions, including heart rate, to meet the body's demands in different states of activity and rest.

6. What is the effect on heart contractions when resting potential is decreased?

A. Increases strength of contractions

B. No effect on contractions

C. Decreases strength of contractions

D. Stops contractions

When the resting potential of cardiac muscle cells decreases, it affects the threshold for depolarization, which is crucial for initiating heart contractions. The resting potential is the baseline electrical charge across the cardiac cell membrane when the cell is not electrically stimulated. A decrease in resting potential means that the membrane is less polarized, which makes the cells more positive compared to their normal resting state. This change can lead to an inability to generate the proper action potentials required for muscle contraction effectively. The heart relies on coordinated electrical impulses to trigger contractions, and if the resting potential is less negative, it may hinder the cells' ability to reach that threshold or alter the strength and timing of muscle contraction. As a result, the strength of contractions is compromised because the heart muscle cells may not depolarize as effectively or as synchronously as they should, leading to weaker contractions. Therefore, a decrease in resting potential directly correlates with a decrease in the strength of heart contractions.

7. In a 5°C Ringer's solution, what is the observed heart rate behavior in a frog's heart?

- A. Beats faster than baseline**
- B. Beats slower than baseline**
- C. Remains at baseline**
- D. Irregular heartbeat pattern**

In a 5°C Ringer's solution, the heart rate of a frog's heart typically decreases compared to baseline conditions. This response is primarily due to the effects of temperature on physiological processes. Lower temperatures generally slow down metabolic reactions and neurotransmission, impacting the heart's pacemaker activity, specifically in the sinoatrial (SA) node, which regulates heartbeat. As the temperature drops, the electrical activity and contractility of cardiac myocytes are reduced, leading to a decreased heart rate. This phenomenon is consistent with the principle that colder temperatures inhibit the function of enzymes and cellular processes required for the generation of action potentials, which are crucial for maintaining a normal heart rhythm. In simpler terms, as the temperature of the solution lowers, the frog's heart will exhibit a slower pace than it would under normal, warmer conditions, supporting the observation that the heart beats slower than baseline in cooler environments.

8. What does vagal escape imply about the heart's physiology?

- A. It is solely controlled by the vagus nerve**
- B. It can recover from vagal inhibition through other mechanisms**
- C. It indicates permanent damage to heart function**
- D. It shows a lack of responsiveness to stimulation**

Vagal escape is a phenomenon that demonstrates the heart's ability to recover its function after being inhibited by the vagus nerve, which is part of the parasympathetic nervous system. When the vagus nerve exerts its influence, it typically decreases heart rate and can lead to bradycardia. However, after a period of this vagal stimulation, the heart may find ways to escape this inhibition, allowing for a return to a more normal heart rate. This recovery suggests that there are other mechanisms and intrinsic pathways in the heart that can counteract the effects of vagal stimulation. For example, tissues in the heart can increase their excitability or other autonomic influences, stemming from the sympathetic nervous system, can kick in to raise heart rate or increase contractility. Consequently, vagal escape indicates a certain level of resilience and adaptability in cardiovascular physiology, showcasing that the heart can maintain or restore function even in the face of significant vagal tone.

9. What physiological response occurs when atropine is administered?

- A. Decreased heart rate
- B. Increased heart rate**
- C. Stable vascular resistance
- D. No change in heart rhythm

When atropine is administered, it acts as an anticholinergic agent, which means it blocks the effects of the neurotransmitter acetylcholine at muscarinic receptors in the heart. Normally, acetylcholine, released by the vagus nerve, slows down the heart rate by exerting a parasympathetic influence. By inhibiting this action, atropine effectively reduces the vagal tone, leading to an increase in heart rate. This increase occurs as the heart becomes less influenced by the calming effects of parasympathetic activity, allowing the intrinsic firing rate of the sinoatrial (SA) node to prevail. In the case of the other choices, the administration of atropine does not lead to a decreased heart rate, stable vascular resistance, or no change in heart rhythm, as these outcomes would be indicative of either continued parasympathetic activity or neutral effects, which is contrary to the mechanism of action of atropine. Thus, the physiological response to atropine is marked by an increased heart rate due to the decrease in parasympathetic influence on the heart.

10. Why does the amplitude of the ventricular systole remain unchanged with increased stimulation?

- A. New contractions begin earlier
- B. Relaxation phase is prolonged
- C. A new contraction cannot begin until relaxation occurs**
- D. Stimulation causes fatigue

In the context of cardiac physiology, the amplitude of ventricular systole remaining unchanged despite increased stimulation can be understood through the mechanics of cardiac contraction and relaxation. The heart has a refractory period during which it cannot be stimulated again until it has relaxed sufficiently. This physiological limitation ensures that the heart contracts in a controlled manner, allowing for effective blood ejection and preventing excessive strain on the cardiac muscle. When stimulation is increased, the heart cannot initiate a new contraction until the previous contraction has fully relaxed. Therefore, even if the frequency of stimulation increases, the fact that a new contraction cannot begin until the myocardium has undergone sufficient relaxation leads to the amplitude of the contraction staying consistent. This mechanism is crucial for maintaining proper cardiac output and preventing concurrent contractions that could arise from overlapping stimuli, which might otherwise impair effective blood pumping. In this scenario, options that suggest earlier contractions or prolonged relaxation phase don't accurately depict the physiological constraints imposed by the heart's cycle. Thus, the most fitting explanation relates to the necessity of the relaxation phase before a new contraction can occur, highlighting the importance of timing in cardiac contractions.