BAMC Telemetry Practice Test (Sample)

Study Guide

BY EXAMZIFY

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Questions

1. **What is the threshold level for depolarization in cardiac cells?**
   - **A. About -70 mV**
   - **B. About -60 mV**
   - **C. About -80 mV**
   - **D. About -90 mV**

2. **What EKG change can indicate myocardial injury or infarction?**
   - **A. Normal ST segment**
   - **B. ST segment depression**
   - **C. ST segment elevation**
   - **D. Q wave inversion**

3. **Where do the Purkinje fibers transmit impulses in the heart?**
   - **A. To the atria from the SA node**
   - **B. Into subendocardial layers of both ventricles**
   - **C. From the bundle branches to the atrioventricular node**
   - **D. To the left anterior fascicle**

4. **What distinguishes permanent pacing?**
   - **A. Pacer releases a stimulus at a fixed rate**
   - **B. It is not dependent on intrinsic heart rhythms**
   - **C. Control device and wires are implanted into the chest**
   - **D. It stimulates the heart only during emergency situations**

5. **What is the inherent rate of automaticity for the AV node?**
   - **A. 20-40 bpm**
   - **B. 40-60 bpm**
   - **C. 60-100 bpm**
   - **D. 80-100 bpm**

6. What does failure to capture typically indicate?

- **A. The pacemaker is firing too frequently**
- **B. The pacer spike does not result in a heart contraction**
- **C. The pacemaker is sensing intrinsic rhythms**
- **D. The heart rate is within normal limits**

7. How is the heart rate characterized in ventricular tachycardia?

- **A. 60-100 bpm**
- **B. 100-250 bpm**
- **C. Below 60 bpm**
- **D. Over 300 bpm**

8. What is the common arrhythmia resulting in sudden cardiac death?

- **A. Atrial flutter**
- **B. Ventricular fibrillation**
- **C. Sinus bradycardia**
- **D. Supraventricular tachycardia**

9. During which period can a stronger than normal impulse stimulate depolarization?

- **A. Absolute refractory period**
- **B. Relative refractory period**
- **C. Normal excitability period**
- **D. Depolarization period**

10. What pharmacologic options can be utilized to decrease heart rate in supraventricular tachycardia?

- **A. Beta blockers and calcium channel blockers**
- **B. ACE inhibitors and diuretics**
- **C. Antibiotics and analgesics**
- **D. Anticoagulants and antiplatelet agents**

Answers

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

Explanations

1. What is the threshold level for depolarization in cardiac cells?

A. About -70 mV

B. About -60 mV

C. About -80 mV

D. About -90 mV

The threshold level for depolarization in cardiac cells is approximately -60 mV. This value is significant because it represents the point at which the membrane potential becomes sufficiently positive to activate voltage-gated sodium channels. When these channels open, sodium ions rush into the cell, causing a rapid depolarization that leads to the generation of an action potential. This process is crucial for the proper functioning of the heart, allowing for coordinated contractions and rhythm of the cardiac muscle. Understanding why this threshold is set around -60 mV involves knowing that cardiac action potentials differ from those in other types of excitable tissues, like neurons. Cardiac cells maintain a resting membrane potential more negative than this threshold, and reaching -60 mV is essential for triggering the heart's electrical activity. Thus, this specific threshold is vital for initiating the sequence of depolarization, followed by repolarization, which is necessary for each heartbeat.

2. What EKG change can indicate myocardial injury or infarction?

A. Normal ST segment

B. ST segment depression

C. ST segment elevation

D. Q wave inversion

ST segment elevation is a significant indicator of myocardial injury or infarction, particularly in the setting of acute coronary syndrome. When there is damage to the heart muscle due to a lack of blood supply, the electrical activity recorded by an electrocardiogram (EKG or ECG) can show characteristic changes. During an acute myocardial infarction, particularly an ST-Elevation Myocardial Infarction (STEMI), the ischemic conditions lead to a shift in the normal baseline of the ST segment. This elevation above the baseline can be attributed to the injured myocardial tissue and represents the active process of injury. It reflects the increased intracellular ion concentrations and changes in the cellular membrane potential in the affected area, indicating that the myocardium is in distress. In contrast, while other changes like ST segment depression, normal ST segments, or Q wave inversions can indicate various forms of cardiac stress or prior infarctions, they do not specifically signify the acute stage of myocardial injury. ST segment depression, for instance, typically indicates ischemia rather than a full-blown infarction. Hence, the presence of ST segment elevation is a critical finding that prompts immediate medical intervention to restore blood flow and minimize heart damage.

3. Where do the Purkinje fibers transmit impulses in the heart?

A. To the atria from the SA node

B. Into subendocardial layers of both ventricles

C. From the bundle branches to the atrioventricular node

D. To the left anterior fascicle

The Purkinje fibers play a crucial role in the heart's electrical conduction system by transmitting impulses throughout the ventricles. Their primary function is to ensure the rapid and coordinated contraction of the ventricular myocardium, which is essential for effective pumping of blood. When an electrical impulse is generated, it travels through the bundle of His and then into the right and left bundle branches. The Purkinje fibers branch off from these bundle branches and extend into the subendocardial layers of both ventricles. From there, they execute the conduction of the impulse to myocytes, leading to synchronized contraction of the ventricular muscles. This process is key for maintaining a strong and consistent heartbeat, as it allows the ventricles to contract almost simultaneously, optimizing the heart's efficiency in blood circulation. Therefore, the correct choice accurately reflects the specific pathway and function of the Purkinje fibers in the heart's conduction system.

4. What distinguishes permanent pacing?

A. Pacer releases a stimulus at a fixed rate

B. It is not dependent on intrinsic heart rhythms

C. Control device and wires are implanted into the chest

D. It stimulates the heart only during emergency situations

Permanent pacing is characterized by the implantation of a control device, often referred to as a pacemaker, along with leads (wires) that are placed into the heart. This setup is specifically designed for long-term management of patients with bradycardia or other types of cardiac arrhythmias that require constant electrical stimulation to maintain an adequate heart rate. The control device continuously monitors the heart's activity and provides electrical impulses when necessary, ensuring that the heart beats at a normal rate regardless of the heart's intrinsic ability to do so. This is what makes permanent pacing a long-term solution rather than an emergency response or a temporary measure. In contrast, the other choices describe aspects of pacing that either do not pertain exclusively to permanent pacing or refer to temporary pacing methods. For instance, while it is true that permanent pacing is not dependent on intrinsic heart rhythms and may provide stimuli at a fixed rate, these characteristics can also apply to temporary pacing, thereby not exclusively defining permanent pacing. Thus, the implantation of a control device and leads into the chest is the key feature that highlights the nature of permanent pacing.

5. What is the inherent rate of automaticity for the AV node?

A. 20-40 bpm

B. 40-60 bpm

C. 60-100 bpm

D. 80-100 bpm

The inherent rate of automaticity for the AV node is typically between 40 to 60 beats per minute (bpm). This intrinsic pacing ability comes from specialized cells within the AV node that can generate electrical impulses independently when they are not receiving signals from the SA node, which is the primary pacemaker of the heart. Understanding the function of the AV node is crucial in the context of cardiac rhythm and the hierarchy of pacemaker activity in the heart. When the primary pacemaker (SA node) fails or when there's a conduction block, the AV node can take over, providing a backup pacing mechanism to maintain a basic cardiac rhythm. Therefore, the range of 40-60 bpm reflects its role in sustaining heart activity under such conditions, making it the correct answer in this scenario.

6. What does failure to capture typically indicate?

A. The pacemaker is firing too frequently

B. The pacer spike does not result in a heart contraction

C. The pacemaker is sensing intrinsic rhythms

D. The heart rate is within normal limits

Failure to capture in the context of a pacemaker means that the electrical impulse generated by the pacemaker does not result in a corresponding contraction of the heart muscle. This typically occurs when the pacing stimulus is either too weak to depolarize the myocardium or if there is an issue with the lead placement or function. When capture fails, the heart does not respond appropriately to the pacing, which could lead to bradycardia or other cardiac problems. Understanding this concept is critical for healthcare providers, as it directly impacts patient management. Recognizing failure to capture is essential for determining whether adjustments to the pacing settings, lead placement, or further diagnostic evaluations are necessary. Options related to the pacemaker firing too frequently or sensing intrinsic rhythms may relate to the pacing function but do not accurately define the situation where there is a failure to elicit a heart contraction. The heart rate being within normal limits is not a direct indication of capture status, since failure to capture can occur regardless of heart rate. Thus, the indication that the pacer spike does not result in a heart contraction is key to identifying this specific pacemaker malfunction.

7. How is the heart rate characterized in ventricular tachycardia?

A. 60-100 bpm

B. 100-250 bpm

C. Below 60 bpm

D. Over 300 bpm

In ventricular tachycardia, the heart rate is characterized by being between 100 and 250 beats per minute (bpm). This rapid heart rate originates from the ventricles of the heart and is a serious condition that can be life-threatening. The characteristics of ventricular tachycardia include a regular rhythm and wide QRS complexes on an electrocardiogram (ECG). The elevated heart rate can result in inadequate blood flow to the body and may lead to symptoms like dizziness, palpitations, and loss of consciousness. Understanding the typical heart rates for various cardiac conditions is vital for effective diagnosis and treatment. Options that suggest heart rates below the normal range for ventricular tachycardia do not align with the established definitions and clinical criteria associated with this arrhythmia.

8. What is the common arrhythmia resulting in sudden cardiac death?

A. Atrial flutter

B. Ventricular fibrillation

C. Sinus bradycardia

D. Supraventricular tachycardia

Ventricular fibrillation is a critical and life-threatening arrhythmia that can lead to sudden cardiac death. In this condition, the heart's ventricles quiver ineffectively instead of contracting normally, resulting in an inability to pump blood effectively. This chaotic electrical activity in the heart prevents it from maintaining a viable cardiac output, leading to loss of consciousness and, if uncorrected, death within minutes. The abrupt onset of ventricular fibrillation often occurs in individuals with underlying heart disease, but it can also happen in those without prior symptoms. Immediate defibrillation is required to restore normal heart rhythm, making early recognition and treatment of this arrhythmia crucial for survival. In contrast, atrial flutter, sinus bradycardia, and supraventricular tachycardia do not typically result in sudden cardiac death and are usually more manageable with appropriate medical intervention. While they can cause complications, they do not have the same immediate fatal consequences as ventricular fibrillation.

9. During which period can a stronger than normal impulse stimulate depolarization?

A. Absolute refractory period

B. Relative refractory period

C. Normal excitability period

D. Depolarization period

The relative refractory period is the correct choice because it is the phase following the absolute refractory period during which a stronger-than-normal stimulus can lead to depolarization of the cardiac cells. This period occurs because, while some sodium channels are still inactive, others are transitioning back to a state where they can be activated. Therefore, if the stimulus is sufficiently strong, it can cause depolarization by triggering these channels. During the absolute refractory period, the cardiac muscle cells cannot be depolarized regardless of the strength of the stimulus, as the sodium channels remain closed and inactive. The normal excitability period, while it indicates a state of readiness to respond to stimuli, does not specifically denote a point where a stronger impulse can cause depolarization. The depolarization period is the phase when depolarization is actively occurring, and it does not correspond to the ability of a stronger stimulus to induce further depolarization, as this is typically already ongoing.

10. What pharmacologic options can be utilized to decrease heart rate in supraventricular tachycardia?

A. Beta blockers and calcium channel blockers

B. ACE inhibitors and diuretics

C. Antibiotics and analgesics

D. Anticoagulants and antiplatelet agents

The correct answer is centered on the role of beta blockers and calcium channel blockers in managing supraventricular tachycardia (SVT). Both of these classes of medications are effective at decreasing heart rate by influencing the electrical conduction system of the heart. Beta blockers work by antagonizing the effects of catecholamines, such as adrenaline, which can increase heart rate and myocardial contractility. This reduction in sympathetic stimulation helps to slow down the heart rate, making them particularly useful in conditions like SVT. Calcium channel blockers also contribute to heart rate control by inhibiting the influx of calcium into cardiac muscle cells. This action leads to reduced automaticity in the atrial tissue, and slower conduction through the AV node, both of which are vital in managing SVT. Other choices, such as ACE inhibitors and diuretics, primarily target blood pressure control and fluid management rather than directly influencing heart rate in the context of SVT. Antibiotics and analgesics are not relevant, as they are geared toward treating infections and pain, respectively, not arrhythmias. Anticoagulants and antiplatelet agents are used to prevent thromboembolic events, which can occur as a complication of certain arrhythmias but do not affect heart rate