Collin College Cardiac Drugs Practice Test (Sample)

Study Guide



Everything you need from our exam experts!

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Questions



- 1. What is a notable point regarding the max dosage of Epinephrine in cardiac arrest?
 - A. Max dose is 2 mg
 - B. No max dose in cardiac arrest
 - C. 1 mg is the max dose
 - D. Max dosage is determined by physician
- 2. What is the onset time for Morphine?
 - A. Within 15 minutes
 - B. Within 30 minutes
 - C. Immediate
 - D. One hour
- 3. What is the primary therapeutic goal of Verapamil in the treatment of angina?
 - A. To decrease heart rate
 - B. To enhance myocardial oxygen delivery
 - C. To reduce myocardial oxygen demand
 - D. To stabilize heart rhythm
- 4. What is the primary action of Diltiazem on heart muscle cells?
 - A. Increases heart rate
 - B. Blocks sodium influx
 - C. Inhibits calcium ion influx
 - D. Stimulates adrenergic receptors
- 5. What happens if Magnesium Sulfate is administered too rapidly?
 - A. It may cause a drop in BP
 - B. It may cause an increase in heart rate
 - C. It may cause confusion
 - D. It may cause vomiting

- 6. Which condition is NOT an indication for Verapamil?
 - A. Angina
 - **B.** Hypertension (HTN)
 - C. Congestive heart failure
 - D. Second line agent for SVT
- 7. What classification does nitroglycerin fall under?
 - A. Beta-blocker
 - B. Ace inhibitor
 - C. Nitrate
 - D. Calcium channel blocker
- 8. What effect does morphine have on histamine release?
 - A. Inhibits histamine release
 - **B.** Causes histamine release
 - C. Has no effect on histamine release
 - **D.** Enhances histamine sensitivity
- 9. When administering Verapamil intravenously, what is a critical factor to monitor?
 - A. Blood glucose levels
 - B. Heart rate and blood pressure
 - C. Respiratory rate
 - D. Temperature
- 10. What are the routes of administration for Lidocaine?
 - A. IV/IM
 - B. IV/PO
 - C. IV/IO
 - D. IM/Subcutaneous

Answers



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



Explanations



1. What is a notable point regarding the max dosage of Epinephrine in cardiac arrest?

- A. Max dose is 2 mg
- B. No max dose in cardiac arrest
- C. 1 mg is the max dose
- D. Max dosage is determined by physician

In the context of cardiac arrest, the notable point regarding the maximum dosage of Epinephrine is that there is no established maximum dose. In advanced cardiac life support (ACLS) guidelines, it is recommended to administer Epinephrine every 3 to 5 minutes during cardiopulmonary resuscitation (CPR) without a predefined upper limit. The rationale behind this approach is that high doses can be used, if necessary, to achieve adequate circulation and improve the chances of resuscitation. Epinephrine is a critical drug in the management of cardiac arrest due to its vasoconstrictive properties, which can help improve coronary and cerebral perfusion pressure during CPR. The emphasis is placed on frequent dosing rather than a limit, acknowledging the unpredictable nature of cardiac arrest scenarios and the potential need for aggressive intervention. This understanding highlights the importance of clinical judgment during resuscitation, where the paramedic or physician may choose to continue administering Epinephrine as needed until return of spontaneous circulation (ROSC) is achieved, or until a different clinical decision is made.

2. What is the onset time for Morphine?

- A. Within 15 minutes
- B. Within 30 minutes
- C. Immediate
- D. One hour

Morphine is an opioid analgesic commonly used for managing severe pain. The understanding of its onset time is crucial due to its clinical implications. When administered intravenously, morphine can produce pain relief quite rapidly, often within a few minutes, which is why it is classified as having an immediate onset in this context. This immediate effect is critical for situations requiring prompt pain management, such as in emergency settings or when treating acute pain episodes. Knowing this onset is essential for healthcare providers to effectively manage patient care and anticipate necessary treatments. In contrast, the other options describe longer onset times, which do not accurately reflect morphine's pharmacodynamics when given intravenously.

3. What is the primary therapeutic goal of Verapamil in the treatment of angina?

- A. To decrease heart rate
- B. To enhance myocardial oxygen delivery
- C. To reduce myocardial oxygen demand
- D. To stabilize heart rhythm

Verapamil serves as a calcium channel blocker that primarily works by decreasing myocardial contractility and heart rate, leading to a reduction in myocardial oxygen demand. In patients with angina, the heart requires an adequate supply of oxygen to meet the demands imposed by activities or stress. When workload is decreased, as facilitated by Verapamil, the heart consumes less oxygen, thereby alleviating symptoms of angina. Decreasing myocardial oxygen demand is crucial because angina typically occurs when there is an imbalance between oxygen supply and demand. By reducing factors such as heart rate and myocardial contractility, Verapamil helps ensure that the available oxygen supply can meet the metabolic needs of the heart muscle. While enhancing myocardial oxygen delivery and stabilizing heart rhythm are important considerations in cardiac therapy, the primary therapeutic goal of Verapamil in the context of angina is specifically focused on reducing myocardial oxygen demand.

4. What is the primary action of Diltiazem on heart muscle cells?

- A. Increases heart rate
- **B.** Blocks sodium influx
- C. Inhibits calcium ion influx
- D. Stimulates adrenergic receptors

Diltiazem primarily acts by inhibiting calcium ion influx into heart muscle cells. It is a calcium channel blocker that targets L-type calcium channels primarily found in cardiac and smooth muscle tissues. By blocking these channels, Diltiazem reduces the entry of calcium ions into the cells during depolarization. This decrease in calcium availability leads to reduced contractility and heart rate, which can help manage conditions such as hypertension and certain types of angina. Calcium ions play a crucial role in the excitation-contraction coupling of cardiac muscle, as they are necessary for the contraction of muscle fibers. By inhibiting calcium influx, Diltiazem decreases the strength of heart muscle contractions and slows down the conduction through the heart, especially at the AV node. This mechanism is beneficial in reducing the workload on the heart and controlling heart rhythms, making Diltiazem effective in treating various cardiovascular conditions.

5. What happens if Magnesium Sulfate is administered too rapidly?

- A. It may cause a drop in BP
- B. It may cause an increase in heart rate
- C. It may cause confusion
- D. It may cause vomiting

Administering Magnesium Sulfate too rapidly can lead to a drop in blood pressure, also known as hypotension. This effect occurs because Magnesium acts as a smooth muscle relaxant and can cause vasodilation, resulting in decreased peripheral resistance and, consequently, lower blood pressure. When the drug is introduced quickly into the bloodstream, especially at high doses, the body may not have sufficient time to adjust, leading to significant hypotensive episodes. While other potential side effects of Magnesium Sulfate can include confusion, increased heart rate, or vomiting, these are not directly tied to the rate of administration in the same way that hypotension is. Understanding the importance of administering this medication at an appropriate rate is crucial for patient safety, allowing healthcare professionals to mitigate risks associated with rapid infusion.

6. Which condition is NOT an indication for Verapamil?

- A. Angina
- **B.** Hypertension (HTN)
- C. Congestive heart failure
- D. Second line agent for SVT

Verapamil is a calcium channel blocker primarily used to treat conditions such as angina, hypertension, and certain types of arrhythmias, including supraventricular tachycardia (SVT). The correct answer indicates that congestive heart failure (CHF) is not a typical indication for Verapamil. Verapamil works by inhibiting the influx of calcium ions into the cardiac muscle and vascular smooth muscle, leading to a decrease in cardiac contractility and relaxation of blood vessels. While it can be beneficial for managing the heart rate and blood pressure in certain patients, it may exacerbate symptoms in individuals with CHF due to its negative inotropic effects, which can further reduce the already compromised heart function in this population. In contrast, angina refers to chest pain due to reduced blood flow to the heart, and Verapamil helps to relieve this by dilating coronary arteries and decreasing myocardial oxygen demand. Hypertension is another primary indication, as Verapamil effectively lowers blood pressure by relaxing blood vessels. Additionally, it is commonly used as a second-line agent for managing SVT, helping to control the heart rate during episodes. Thus, while Verapamil is effective for various cardiac-related conditions, its use in congestive heart failure is limited

7. What classification does nitroglycerin fall under?

- A. Beta-blocker
- B. Ace inhibitor
- C. Nitrate
- D. Calcium channel blocker

Nitroglycerin is classified as a nitrate, which is essential for understanding its mechanism of action and therapeutic effects in treating conditions like angina pectoris and acute coronary syndromes. Nitrates like nitroglycerin work by relaxing and dilating blood vessels, which decreases the workload on the heart and improves blood flow to the myocardium. When administered, nitroglycerin is converted to nitric oxide in the body, leading to smooth muscle relaxation. This vasodilation effect helps relieve chest pain by reducing the heart's oxygen demand while simultaneously increasing oxygen supply. Understanding this classification is crucial for healthcare professionals in effectively managing cardiac-related conditions and knowing how to use this medication in conjunction with other treatments. In contrast, beta-blockers, ACE inhibitors, and calcium channel blockers have different mechanisms of action that do not primarily focus on vasodilation via nitrate activity. Beta-blockers mainly reduce heart rate and myocardial contractility, ACE inhibitors block the conversion of angiotensin I to angiotensin II to lower blood pressure and decrease fluid retention, and calcium channel blockers primarily prevent calcium from entering heart and blood vessel muscle cells, decreasing contractility and causing vasodilation in a different manner. Each of these classes serves distinct purposes in cardiac care but does

8. What effect does morphine have on histamine release?

- A. Inhibits histamine release
- **B.** Causes histamine release
- C. Has no effect on histamine release
- D. Enhances histamine sensitivity

Morphine is known to cause the release of histamine from mast cells. This is an important pharmacological effect because histamine release can lead to various physiological responses, including vasodilation and potential hypotension. The mechanism behind this involves morphine interacting with the opioid receptors, which can stimulate mast cells to degranulate and subsequently release histamine. Understanding this effect is crucial for healthcare providers, particularly when administering morphine, as they may need to monitor for signs of allergic reactions or histamine-related side effects, such as itching, flushing, or changes in blood pressure. Hence, knowing that morphine causes histamine release helps in preparing for and managing these potential complications effectively.

9. When administering Verapamil intravenously, what is a critical factor to monitor?

- A. Blood glucose levels
- **B.** Heart rate and blood pressure
- C. Respiratory rate
- D. Temperature

When administering Verapamil intravenously, it is essential to monitor heart rate and blood pressure because Verapamil is a calcium channel blocker that affects the cardiovascular system. It specifically helps to reduce heart rate and diminish the force of cardiac contractions, thereby lowering blood pressure. Achieving the right balance is crucial to avoid complications such as bradycardia (an excessively slow heart rate) or hypotension (low blood pressure). These effects are particularly important in patients with existing cardiac conditions, which could be exacerbated by changes in heart rate and blood pressure. Regular monitoring ensures that the patient's vital signs remain within a safe range throughout the administration of the medication.

10. What are the routes of administration for Lidocaine?

- A. IV/IM
- B. IV/PO
- C. IV/IO
- D. IM/Subcutaneous

Lidocaine can be administered through several routes, with intravenous (IV) and intraosseous (IO) being particularly important for its therapeutic effects. The IV route allows for rapid onset of action, making it ideal for emergency situations, such as the treatment of arrhythmias or during cardiopulmonary resuscitation. The IO route is also significant in emergencies, especially when IV access is challenging or unavailable; it enables the drug to be delivered directly into the bone marrow and into systemic circulation quickly. While Lidocaine can be absorbed through other routes, such as intramuscular (IM), the key therapeutic applications in acute care emphasize IV and IO as more effective routes for delivering the drug swiftly, which is why this combination is the correct answer. Other potential routes like subcutaneous or oral (PO) are not as commonly used in critical situations where rapid action is required, making them less relevant for the primary uses of Lidocaine in emergency medicine.