**Introduction**

Cardiac arrest is a leading cause of death across the United States, with over 300,000 cases of cardiac arrest occurring outside of hospitals each year. Rapid initiation of basic and advanced cardiovascular life support is imperative to improve patient outcomes. This chapter provides an overview of current best practices in basic life support (BLS) and advanced cardiovascular life support (ACLS) for healthcare providers managing adult cardiac arrest.

Pharmacists play a vital role in recognizing the signs and symptoms of cardiac arrest, understanding the underlying causes, and implementing appropriate pharmacological interventions. By staying updated with the latest advancements and controversies in the field, pharmacists can contribute to the optimal management of cardiac arrest cases.

In this section, we will begin by discussing the basics of cardiac arrest, including its definition, classification, and pathophysiology. We will then explore the H's and T's, which represent the common causes of cardiac arrest, and their pharmacological implications. Next, we will delve into the ACLS protocols, outlining the pharmacist's role in each step. Pharmacotherapy will be thoroughly discussed, covering first-line and alternative therapies, mechanisms of action, dosing, side effects, contraindications, and monitoring parameters. Post-cardiac arrest care, special considerations, and ethical aspects will also be addressed. The subtopic will conclude with clinical scenarios to enhance practical understanding, followed by essential exam preparation tips.

By the end of this subtopic, clinical pharmacists will have a comprehensive understanding of cardiac arrest management, enabling them to contribute effectively to interdisciplinary teams and provide optimal care to patients in cardiac arrest situations.

**Cardiac Arrest Basics**

Cardiac arrest is a life-threatening condition characterized by the sudden cessation of cardiac activity, resulting in the absence of a palpable pulse and loss of consciousness. It can be classified into different types, including ventricular fibrillation (VF), pulseless ventricular tachycardia (pVT), asystole, and pulseless electrical activity (PEA).

Understanding the basics of cardiac arrest is crucial for clinical pharmacists involved in its management. Here are the key points to consider:

1. Definition and Classification:
   * Cardiac arrest is the sudden cessation of cardiac activity, leading to the absence of a palpable pulse and loss of consciousness.
   * VF is a rapid and disorganized electrical activity in the ventricles.
   * pVT is a regular but excessively rapid ventricular rhythm without a pulse.
   * Asystole is the absence of any electrical activity in the heart.
   * PEA refers to the presence of electrical activity without a palpable pulse.

1. Pathophysiology:
   * Cardiac arrest often occurs due to underlying cardiovascular diseases such as coronary artery disease, heart failure, or arrhythmias.
   * Ischemia, electrolyte imbalances, toxins, and other factors can disrupt the normal electrical conduction system of the heart, leading to arrhythmias and cardiac arrest.

1. Role of the Pharmacist:
   * Clinical pharmacists play a vital role in recognizing the signs and symptoms of cardiac arrest, assisting in immediate management, and optimizing pharmacotherapy.
   * Pharmacists provide drug information, ensure appropriate medication selection, dosing, administration, and monitoring during resuscitation efforts.
   * They collaborate with the healthcare team to prevent medication errors, manage drug interactions, and address specific pharmacological considerations in cardiac arrest management.

**H's and T's**

The H's and T's represent the common causes of cardiac arrest. Understanding these causes and their pharmacological implications is crucial for clinical pharmacists. Here are the key points to consider:

H's - The H's represent the primary causes of cardiac arrest:

1. Hypoxia: Inadequate oxygen supply to tissues and organs.
2. Hypovolemia: Reduced circulating blood volume.
3. Hydrogen ion (acidosis): Imbalance in the body's acid-base status.
4. Hyper-/Hypokalemia: Abnormal potassium levels in the blood.
5. Hypothermia: Abnormally low body temperature.

T's - The T's represent the secondary causes of cardiac arrest:

1. Toxins: Exposure to drugs, medications, or toxins that can disrupt cardiac function.
2. Tamponade (Cardiac): Accumulation of fluid or blood in the pericardial sac, compressing the heart.
3. Tension pneumothorax: Accumulation of air in the pleural space, causing lung collapse and compression of the heart.
4. Thrombosis (Coronary or Pulmonary): Formation of a blood clot that obstructs blood flow to the coronary arteries or pulmonary vasculature.
5. Trauma: Significant injury or trauma to the chest or heart.

**ACLS Protocols**

Basics of BLS Sequence

The foundation for all resuscitation efforts begins with prompt, high-quality BLS. Key steps include:

* Initial assessment of responsiveness
* Activation of emergency response team
* Checking pulse and breathing
* Beginning chest compressions if no normal breathing/pulse detected
* Establishing advanced airway (bag-valve mask, OPA/NPA)
* Application of AED when available and delivery of shock if advised
* Continuing CPR cycles of 30 compressions and 2 breaths
* Ensuring compressions are hard and fast at 100-120/min with full chest recoil
* Minimizing interruptions in compressions and avoiding hyperventilation
* Utilizing teamwork with compressor rotation every 2 minutes

The BLS sequence establishes circulation through continual chest compressions and provides oxygenation through assisted ventilations. High-quality CPR is the foundation for all further ACLS interventions.

ACLS Algorithms Overview

While BLS focuses on immediate CPR and defibrillation, ACLS protocols build on these efforts by addressing the specific underlying cardiac rhythm and providing pharmacologic support. ACLS algorithms guide the response team through treatment decisions in a stepwise approach. Rhythms are categorized as shockable (ventricular fibrillation, pulseless ventricular tachycardia) versus non-shockable (asystole, pulseless electrical activity). The pharmacist plays a key role in preparing and administering ACLS medications per protocol for each rhythm.

**Ventricular Fibrillation/Pulseless Ventricular Tachycardia**

Ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT) are chaotic heart rhythms originating from the ventricles. They are identified on ECG by irregular undulations or rapid spikes without coordinated cardiac activity. These rhythms are treated with immediate defibrillation.

 Key ACLS interventions for VF/VT arrest include:

* Performing continuous chest compressions during arrest
* Charging defibrillator to maximum energy dose per manufacturer guidelines; deliver shock ASAP
* Resuming compressions for 2 minutes before rechecking rhythm
* Giving EPINEPHrine 1 mg IV/IO push every 3-5 minutes
* Considering antiarrhythmic if shocks unsuccessful: amiodarone 300 mg IV bolus or lidocaine 1-1.5 mg/kg IV/IO bolus
* Identifying and treating reversible causes ("Hs and Ts")
* Re-shocking every 2 minutes if shockable rhythm persists

Electrical shocks are the mainstay of therapy for VF/VT, along with high-quality CPR. Medications are supplementary and should never delay defibrillation. Epinephrine maintains cardiac perfusion, while antiarrhythmics help stabilize the myocardial membrane. Return of spontaneous circulation depends on restoring an organized rhythm.

**Pulseless Electrical Activity**

In pulseless electrical activity (PEA), ECG shows coordinated electrical activity but no palpable pulses. Causes can be remembered using the "Hs and Ts" mnemonic:

* H's: Hypoxia, Hydrogen ion (acidosis), Hypokalemia/hyperkalemia, Hypoglycemia, Hypovolemia
* T's: Toxins, Tamponade, Tension pneumothorax, Thrombosis (cardiac or pulmonary)

PEA treatment focuses on performing continuous CPR while identifying and correcting any underlying issues:

* Start high-quality chest compressions; give EPINEPHrine 1 mg IV/IO every 3-5 minutes
* Give fluid bolus if hypovolemia suspected
* Consider sodium bicarbonate for severe acidosis
* Give glucose for hypoglycemia; may correct potassium if hyper/hypokalemia
* Consider naloxone for opioid overdose
* Decompress tension pneumothorax
* Consider ultrasound to identify cardiac tamponade
* Administer antidotes for toxicologic causes
* Use fibrinolytics cautiously if massive PE suspected

Restoring adequate oxygen and circulation through medications and interventions for reversible causes provides the best chance of rhythm improvement and ROSC with PEA.

**Asystole**

Asystole represents complete absence of cardiac electrical activity on ECG. Like PEA, asystole is a non-shockable rhythm. Asystole often occurs after prolonged VF/VT or PEA arrest when the heart becomes exhausted. Management of asystole arrest focuses on high-quality CPR and IV epinephrine:

* Begin chest compressions immediately
* Give 1 mg epinephrine IV/IO push every 3-5 minutes
* Identify and treat reversible causes
* Continue resuscitation efforts for at least 20 minutes before considering termination of efforts

With no electrical activity to work with, high-quality compressions become even more critical to circulate epinephrine and provide some coronary perfusion in hopes of stimulating an organized rhythm. Epi every 3-5 minutes provides vasopressor support. Reversible causes should be addressed aggressively.

**Pharmacist Role in ACLS**

Pharmacists serve an indispensable role during the organized chaos of an adult code blue. Key responsibilities include:

* Ensuring ACLS medication tray/cart is fully stocked and immediately accessible
* Preparing all code medications accurately and efficiently
* Providing medication recommendations and ACLS protocol guidance
* Promoting proper drug administration technique and routes
* Assisting with identifying and treating reversible causes
* Communicating drug choices, doses, timing clearly throughout resuscitation
* Documenting all medications given and patient response/outcomes
* Participating in post-event debriefing to improve future performance

In particular, the pharmacist focuses on preparing vasopressor drips, pushing emergency drugs per algorithm, assisting with code team decisions, and suggesting antidotes or reversal agents where applicable. Smooth teamwork and communication facilitates appropriate medication delivery during a highly stressful event.

**Pharmacotherapy**

**First-Line Therapies:**

1. Epinephrine (Adrenaline):
   * Mechanism of Action: Epinephrine acts as a potent alpha-1 adrenergic agonist, increasing systemic vascular resistance and improving coronary and cerebral perfusion pressures. It also stimulates beta-1 adrenergic receptors, enhancing myocardial contractility and heart rate.

* Rationale:
  + Epinephrine is considered first-line vasopressor therapy for cardiac arrest. It improves coronary perfusion pressure, increases rate of return of spontaneous circulation, and improves likelihood of hospital admission.
* Dosing:
  + Adult: The initial dose is 1 mg (1:10,000 concentration) IV/IO every 3-5 minutes during cardiac arrest. The dose can be administered via an endotracheal tube as 2 to 2.5 mg (1:1,000 concentration), followed by a flush of 10 mL of normal saline.
  + Pediatric: The dose is 0.01 mg/kg (1:10,000 concentration) IV/IO every 3-5 minutes during cardiac arrest. If endotracheal administration is necessary, the dose is 0.1 mg/kg (1:1,000 concentration), followed by a flush of 5 mL of normal saline.
* Side Effects: Epinephrine can cause increased heart rate, increased myocardial oxygen demand, vasoconstriction, hypertension, and arrhythmias.
* Monitoring Parameters: Blood pressure, heart rate, ECG monitoring, signs of perfusion improvement.
* Compatibility/Interactions: Epinephrine is incompatible with sodium bicarbonate

1. Vasopressin
   * Mechanism: Vasopressin is an endogenous peptide hormone that causes vasoconstriction via V1 receptor agonism on vascular smooth muscle. This increases systemic vascular resistance and coronary perfusion pressure.
   * Rationale: Vasopressin can be used as an alternative to epinephrine in cardiac arrest resuscitation. Thought to not be impacted by acidosis as much as epinephrine and has a longer duration of action.
   * Dosing: Typical dosing is 40 units IV/IO bolus, which can be repeated once in lieu of the first two epinephrine doses.
   * Precautions: Vasopressin can induce cardiac, mesenteric, and limb ischemia at high doses. Use caution in patients at risk for ischemia.
   * Compatibility/Interactions: Vasopressin is compatible with epinephrine when administered via separate IV site. No major interactions with other ACLS medications.
2. Amiodarone:
   * Mechanism of Action: Amiodarone is a class III antiarrhythmic agent with multiple electrophysiological effects. It prolongs repolarization and action potential duration, inhibits adrenergic stimulation, and blocks multiple ion channels, including potassium, sodium, and calcium channels.
   * Dosing:
     + Adult: The initial dose is 300 mg IV/IO push, followed by a second dose of 150 mg IV/IO if needed. The second dose can be repeated if necessary.
     + Pediatric: The dose is 5 mg/kg IV/IO push, followed by a second dose of 5 mg/kg if needed. The second dose can be repeated if necessary.
   * Side Effects: Amiodarone can cause hypotension, bradycardia, QT interval prolongation, pulmonary toxicity, hepatotoxicity, and thyroid dysfunction.
3. Lidocaine:

* Mechanism of Action: Lidocaine is a class Ib antiarrhythmic agent that stabilizes the myocardial cell membrane by blocking sodium channels, reducing the rate of rise of the action potential and suppressing ventricular arrhythmias.
* Dosing:
  + Adult: The initial dose is 1 to 1.5 mg/kg IV/IO, followed by a maintenance infusion of 1 to 4 mg/min.
  + Pediatric: The dose is 1 mg/kg IV/IO bolus, followed by a maintenance infusion of 20 to 50 mcg/kg/min.
* Side Effects: Lidocaine can cause central nervous system effects, such as dizziness, confusion, and seizures, as well as cardiovascular effects likehypotension, bradycardia, and heart block.

1. Magnesium Sulfate:
   * Rationale: Magnesium is indicated for torsades de pointes or suspected hypomagnesemia. It is not used routinely in pulseless arrest.
   * Mechanism of Action: Magnesium stabilizes cell membranes and antagonizes calcium, resulting in vasodilation and antiarrhythmic effects.
   * Dosing:
     + Adult: The dose is 1 to 2 g IV/IO over 5 to 20 minutes.
     + Pediatric: The dose is 25 to 50 mg/kg IV/IO over 5 to 20 minutes, up to a maximum of 2 g.
   * Side Effects: Magnesium sulfate can cause flushing, hypotension, respiratory depression, and loss of deep tendon reflexes.
2. Calcium
   * Indication: Calcium reverses cardiotoxic effects of hyperkalemia, hypocalcemia, calcium channel blocker overdose, and magnesium toxicity. It also provides membrane stabilization in hypocalcemia.
   * Dosing: Give 1-2 grams IV push over 5-10 minutes, typically as calcium chloride or gluconate.
3. Sodium Bicarbonate
   * Indication: Sodium bicarbonate corrects metabolic acidosis which can improve efficacy of resuscitative drugs and antiarrhythmics. Also indicated for hyperkalemia and tricyclic antidepressant overdose.
   * Dosing: Give 1 mEq/kg IV push initially during cardiac arrest. Can repeat every 10 minutes as needed based on arterial blood gas results.
4. Dextrose
   * Indication: Dextrose is used to reverse hypoglycemia during cardiac arrest.
   * Dosing: Give 25-50 grams IV push (50 mL of D50W or 100 mL of D25W) as initial dose. Recheck glucose after 5 minutes and re-treat as needed.
5. Alteplase
   * Indication: Alteplase breaks down fibrin clots via thrombolysis. It is used for suspected massive pulmonary embolism and STEMI during cardiac arrest when capacity for percutaneous intervention is limited.
   * Dosing: Administer 50-100 mg IV infusion over 2 minutes for pulmonary embolism induced arrest.
6. Esmolol
   * Indication: Esmolol is an ultra-short acting beta-blocker used in refractory ventricular fibrillation when wide QRS duration is suspected as the perpetuating issue.
   * Dosing: Give a 500-1000 mcg/kg IV loading dose can consider following with an infusion of 50-300 mcg/kg/min titrated to effect.
7. Naloxone

* Indication: Naloxone reverses opioid-induced respiratory depression and hypotension. It is indicated for opioid overdose.
* Dosing: Give 0.4-2 mg IV, IM, IO, or intranasally. May repeat every 2-3 minutes as needed to reverse respiratory depression.

**Medications Via ET Tube**

* Dose: 2-2.5 times the IV dose diluted in 10 mL normal saline and administered via ET tube.
* Medications (NAVEL)
  + Naloxone
  + Atropine
  + Vasopressin
  + Epinephrine
  + Lidocaine

**Differences in Treatment Approach:**

The treatment approach in ACLS may vary depending on the underlying cause and rhythm of cardiac arrest. Here are some key differences:

1. Ventricular Fibrillation (VF) and Pulseless Ventricular Tachycardia (pVT):
   * Defibrillation is the primary treatment for shockable rhythms, with immediate CPR and rapid defibrillation as the main focus.
   * Epinephrine, amiodarone, or lidocaine may be administered to support circulation and attempt to restore a perfusing rhythm.
2. Asystole and Pulseless Electrical Activity (PEA):
   * Epinephrine is the mainstay of treatment, with emphasis on high-quality CPR, airway management, and addressing potential reversible causes (H's and T's).

By understanding these differences, clinical pharmacists can tailor their interventions based on the specific rhythm and underlying cause of cardiac arrest, optimizing patient care during resuscitation.

**Special Resuscitation Situations**

While ACLS provides standardized algorithms for most arrest scenarios, certain variables require modifying the typical resuscitation approach:

* Wide complex tachycardia with pulses - Consider calcium channel blockers in addition to usual ACLS meds
* Dialysis patient - Risk of hyperkalemia; have calcium ready
* Toxicologic arrest - Give antidotes early; consult poison control
* Pregnancy - Provide manual uterine displacement during CPR
* Hypothermia - Discontinue at 30°C; consider avoiding epinephrine/antiarrhythmics

**ACLS Medication Administration Tips**

The pharmacist is responsible for preparing ACLS medications during a code. Rapid and proper drug delivery is crucial. Key techniques include:

* Always clarify dose, dilution, route for each medication
* Use pre-filled syringes when possible (e.g. epi, lidocaine, amiodarone)
* Dilute pressor drips in hospital concentration (norepinephrine in D5W or NS)
* Piggyback vasopressors into IVF or saline lock – avoid T-connectors
* Give IV push drugs rapidly with flush
* ET epinephrine dose is 2-2.5 times IV dose (dilute in 5 mL NS)
* IO access works well if IVs difficult; use pressure bag
* Bring backup meds in case extra doses needed
* Use code cart/tray for organized storage and timely retrieval

**Post-Cardiac Arrest Care**

Post-cardiac arrest care plays a crucial role in improving patient outcomes following successful resuscitation. As a clinical pharmacist, understanding the principles and pharmacological considerations in post-cardiac arrest care is essential.

Here are the key points to consider:

1. Goals of Post-Cardiac Arrest Care:
   * Restore and optimize organ perfusion and function.
   * Prevent further cardiac events or re-arrest.
   * Minimize the extent of neurological injury.
   * Identify and treat underlying causes of cardiac arrest.
2. Strategies in Post-Cardiac Arrest Care:
   * Targeted Temperature Management (TTM): Inducing therapeutic hypothermia or targeted normothermia to preserve neurological function.
   * Hemodynamic Optimization: Maintaining adequate perfusion pressure, optimizing fluid resuscitation, and using vasoactive medications if needed.
   * Coronary Reperfusion: Identifying and treating underlying coronary artery disease with percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG), if indicated.
   * Metabolic Optimization: Correcting electrolyte imbalances, managing glucose levels, and addressing acid-base disturbances
3. Key Pharmacological Considerations:
   * Sedation and Analgesia: Ensuring patient comfort and minimizing pain during post-resuscitation care using appropriate sedatives and analgesics.
   * Neuromuscular Blockade: Consideration of neuromuscular blockade for targeted temperature management or refractory shivering.
   * Antiarrhythmic Medications: Continuation of antiarrhythmic therapies initiated during cardiac arrest, such as amiodarone or lidocaine, if indicated.
   * Neuroprotective Agents: Limited evidence supports the use of specific neuroprotective agents, such as magnesium, to mitigate neurological injury post-cardiac arrest.

As a clinical pharmacist, your role in post-cardiac arrest care involves medication management, monitoring for potential adverse effects, ensuring appropriate sedation and analgesia, and contributing to the optimization of pharmacotherapy based on individual patient needs.

By understanding the goals, strategies, and pharmacological considerations in post-cardiac arrest care, you can actively contribute to the interdisciplinary team and help improve patient outcomes.

**Clinical Scenarios**

To enhance the practical understanding of managing cardiac arrest, let's explore a few detailed clinical scenarios. Each scenario will highlight a key learning point or common pitfall in cardiac arrest management. Here are the scenarios:

1. Scenario 1: Pediatric Cardiac Arrest
   * Description: A 6-year-old child presents with sudden collapse and is unresponsive. Bystanders initiate CPR.
   * Learning Points:
     + Importance of weight-based dosing and age-appropriate medications in pediatric resuscitation.
     + Recognition of pediatric-specific resuscitation algorithms and appropriate defibrillation energy levels.
     + Role of pediatric advanced life support (PALS) medications and their administration.
2. Scenario 2: Cardiac Arrest in Pregnancy
   * Description: A pregnant patient at 32 weeks gestation develops sudden cardiac arrest. Resuscitation efforts are initiated.
   * Learning Points:
     + Considerations for maternal perfusion, oxygenation, and medication selection in managing cardiac arrest during pregnancy.
     + Involvement of obstetric and neonatal specialists in resuscitation efforts.
     + Balancing the potential impact of medications on both the mother and the fetus.
3. Scenario 3: Drug Overdose-Related Cardiac Arrest
   * Description: A patient presents with cardiac arrest due to an opioid overdose. Naloxone is administered, and resuscitation is initiated.
   * Learning Points:
     + Importance of identifying the causative agent in drug overdose-related cardiac arrest.
     + Prompt administration of appropriate antidotes or reversal agents.
     + Monitoring for potential adverse effects or drug interactions during resuscitation.

**Exam Preparation**

Here are three essential tips to help learners answer board certification exam questions correctly regarding ACLS:

1. Familiarize yourself with the ACLS algorithms: Understanding the step-by-step approach of ACLS algorithms, such as VF/pVT, asystole/PEA, and bradycardia/tachycardia, is crucial. Review the specific interventions, medication administration, and electrical therapies associated with each algorithm to confidently answer related exam questions.
2. Know the dosing and indications of key ACLS medications: Be well-versed in the dosing, mechanisms of action, indications, and potential side effects of key ACLS medications like epinephrine, amiodarone, and vasopressin. Focus on the differences between first-line therapies and alternative options, as well as specific considerations for pediatric and pregnant patients.
3. Understand the pharmacotherapy for special circumstances: Pay attention to the pharmacological considerations in special situations such as pediatric cardiac arrest, cardiac arrest in pregnancy, and drug overdose-related cardiac arrest. Be knowledgeable about weight-based dosing, age-appropriate medications, and specific antidotes or reversal agents for overdose scenarios.

**Key Guidelines and Evidence**

Key Guidelines in ACLS:

1. American Heart Association (AHA) Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: These guidelines provide comprehensive recommendations for the management of cardiac arrest and other cardiovascular emergencies. They cover topics such as basic life support, advanced life support, post-cardiac arrest care, and special resuscitation situations.

Landmark Trials in ACLS:

1. ARREST Trial (Amiodarone in the Out-of-Hospital Resuscitation of Refractory Sustained Ventricular Tachycardia): This trial compared the use of amiodarone versus placebo in patients with out-of-hospital cardiac arrest caused by refractory ventricular tachycardia. It showed that amiodarone administration improved survival to hospital admission compared to placebo.
2. ALIVE Trial (Amiodarone Versus Lidocaine in Prehospital Ventricular Fibrillation Evaluation): This trial compared the effectiveness of amiodarone and lidocaine in patients with refractory ventricular fibrillation. It found that amiodarone and lidocaine both improved survival to hospital admission compared to placebo, with no significant difference between the two drugs.
3. ROC-ALPS Trial (Resuscitation Outcomes Consortium–Amiodarone, Lidocaine, or Placebo Study): This large, multicenter trial compared the efficacy of amiodarone, lidocaine, and placebo in patients with out-of-hospital ventricular fibrillation or pulseless ventricular tachycardia. The study did not find a significant difference in survival with good neurological outcome or survival to hospital discharge between the three groups.
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   1. Randomized controlled trial comparing IV epinephrine vs. placebo in out-of-hospital cardiac arrest
   2. Primary outcome: Survival at 30 days
   3. Results: Improved 30-day survival with epinephrine (3.2% vs. 2.4%), but more severe neurologic impairment (31% vs. 17.8%)
5. Kudenchuk PJ, Brown SP, Daya M, et al. Amiodarone, lidocaine, or placebo in out-of-hospital cardiac arrest. N Engl J Med. 2016;374(18):1711-1722.
   1. Compared amiodarone, lidocaine, and placebo for out-of-hospital shock-refractory VF/VT
   2. Primary outcome: Survival to hospital admission
   3. Results: No difference between amiodarone vs. lidocaine vs. placebo on survival to admission
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   1. Systematic review and meta-analysis of epinephrine vs. placebo in cardiac arrest
   2. Primary outcome: ROSC, survival to discharge
   3. Results: Epinephrine consistently improved ROSC but no definitive benefit on survival
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   1. Evaluated early vs. late epinephrine administration during in-hospital cardiac arrest
   2. Primary outcome: Favorable functional survival
   3. Results: Favorable survival more likely with epinephrine given within first 10 minutes

**Subtopic Summary**

In summary, this subtopic on ACLS and the pharmacotherapy of cardiac arrest has provided learners with a comprehensive understanding of managing cardiac arrest situations. Key learning points include the recognition and classification of cardiac arrest, the pharmacist's role in ACLS protocols, and the pharmacotherapy utilized during resuscitation efforts. Learners have gained insights into the first-line and alternative therapies, including their mechanisms of action, dosing, side effects, and monitoring parameters. Additionally, special considerations in pediatric, pregnant, and drug overdose-related cardiac arrest cases have been discussed. Key takeaways include the importance of following ACLS algorithms, being familiar with medication dosing and indications, and understanding the unique aspects of managing cardiac arrest in special circumstances. By applying these learnings, learners can confidently contribute to resuscitation efforts and optimize pharmacotherapy to improve patient outcomes in cardiac arrest situations.

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