The Pharmacist’s Role in Advanced Cardiac Life Support (ACLS)

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I have no actual or potential conflict of interest in relation to this activity.

Pharmacist Objectives

• Review basic life support skills and step by step management of respiratory and cardiac arrest
• Explain the indication of each medication in the crash cart and their appropriate use
• Recognize life-threatening cardiac arrhythmias and identify the appropriate pharmacologic treatment
• Discuss post-resuscitation care involving medication management.

Pharmacy Technician Objectives

• Describe a patient’s condition who requires ACLS
• Review the goals of ACLS
• Recognize the common medications used in ACLS
• Describe the alternate routes of medication administration of medications found in the crash cart

Background

• 325,000 cardiac arrests/year on average
• 250,000 occur outside of hospital
• Less than 7% survival rate for out-of-hospital cardiac arrest

Pharmacists in the ER

• Respond to all medical emergencies
• Assist with implementation of protocols for ACS, CHF, Sepsis, Stroke, and DKA
• Recommend appropriate therapies when indicated
• Assist with dosing, monitoring, and titration of continuous infusions*
• Antibiotic pharmacokinetic dosing
Pharmacists in the ER

- Recommend therapy for acute toxicology cases
- Review medication profiles
- Conduct medication histories
- Patient education

Pharmacists in codes

- Recommend dosing for appropriate code medications
- Prepare meds to be given
- Keep track of timing of med doses
- Prepare critical care infusions at the bedside and assist with dosing and titration
- Provide drug information when necessary
- CPR

Cardiac Arrest

- Abrupt loss of heart function
  - Confirmed by absence of circulation
    - Pulse
    - Unresponsiveness
    - Apnea

Why do patients develop cardiac arrest?

- Coronary artery disease
- Cardiomyopathy
- Structural abnormalities
- Electrical abnormalities
- Associated Rhythms
  - Ventricular Fibrillation
  - Pulseless Ventricular Tachycardia
  - Pulseless Electrical Activity (PEA)
  - Asystole
- Other causes:
  - Trauma
  - Asthma
  - Sepsis
  - Pneumonia
  - Overdose/toxicity
  - Metabolic disorders

ACLS—Pulseless Electrical Activity

- H’s
  - Hypovolemia
  - Hypoxia
  - Hydrogen ion (acidosis)
  - Hypo/hyperkalemia
  - Hypothermia
- T’s
  - Tension pneumothorax
  - Tamponade
  - Toxins
  - Thrombosis
    - (Cardiac or Pulmonary)

Chain of Survival

- Immediate recognition of cardiac arrest and activation of the emergency response system
- Early CPR with emphasis on chest compressions
- Rapid defibrillation
- Effective advanced life support
- Integrated post cardiac arrest care
Cardiopulmonary Resuscitation

- 2010 Update
  - Emphasis on good compression
  - Allow complete chest recoil
  - Minimize interruptions in compression
  - Target 100 compressions per minute

Survival

- Increased chance of survival to hospital discharge
  - Early, High-Quality CPR
  - Rapid Defibrillation for VF/pulseless VT

- Increased rate of Return of Spontaneous Circulation (ROSC)
  - ACLS medications
  - Advanced airways
  - No placebo-controlled clinical trials demonstrate increased rate of survival to hospital discharge

Question 1

- You witness a person collapse and in cardiac arrest. After you call 911, what is the next thing that you should do?
  A.) Check for a pulse for at least 20 seconds
  B.) Give 2 rescue breaths
  C.) Start chest compressions at a rate of 100/min
  D.) Wait for EMS to arrive

Question 2

- What significantly increases chance of survival after cardiac arrest?
  A.) High dose Epinephrine drip
  B.) Rapid Sequence Intubation
  C.) Surgery
  D.) Rapid Defibrillation of VF/pulseless VT
The P wave is a record of the electrical activity through the upper heart chambers (atria).

The QRS complex is a record of the movement of electrical impulses through the lower heart chambers (ventricles).

The ST segment shows when the ventricle is contracting but no electricity is flowing through it. The ST segment usually appears as a straight, level line between the QRS complex and the T wave.

The T wave shows when the lower heart chambers are resetting electrically and preparing for their next muscle contraction.

NORMAL EKG

PEA/Asystole

• Epinephrine
  – Potent alpha and beta receptor agonist
  – Increased coronary and cerebral vascular perfusion pressure
  – Increased heart rate and myocardial contractility
  – 1mg IV/IO/ET q3-5min (No Max)

PEA/Asystole

• Vasopressin
  – Hormone released from pituitary gland
  – Non-adrenergic vasopressor
  – Peripheral vasoconstriction through V1 receptor in endothelium
  – No increase in O2 demand
  – Similar outcomes compared to epi
  – 40 units IV x 1 (may replace first or second epi)

• Atropine
  – No longer recommended
  – “Unlikely to have therapeutic benefit”

Any organized rhythm without detectable pulse is “PEA”
**VF/Pulseless VT**

- **Ventricular Fibrillation**
- **Ventricular Tachycardia**

**VF/Pulseless VT**
- Defibrillate
  - Shock patient with 200 Joules or max available
- Give vasopressor if rhythm persists after 1 shock and 2min CPR
  - Epinephrine or Vasopressin

**Refractory VF/Pulseless VT**
- **Amiodarone**
  - Class III antiarrhythmic
  - Blocks sodium, calcium, and potassium channels and beta receptors
  - Prolongs action potential and refractory period
  - Increased short-term survival compared to lido
  - Amiodarone 300mg IV followed by 1 dose of 150mg IV after 5 minutes

**Refractory VF/Pulseless VT**
- **Lidocaine**
  - Class 1b antiarrhythmic
  - Increases electrical stimulation threshold of ventricle and decreases neuronal permeability of sodium ions resulting in inhibition of depolarization and blockade of conduction
  - No proven short or long term efficacy
  - Alternative to amiodarone (2nd line)
  - Lidocaine 1.5mg/kg (100mg) IV, then 0.5mg/kg q5 min. up to 3mg/kg

**Polymorphic VT**
- Polyomorphous ventricular tachycardia: QRS complexes display multiple morphologies (“polymorphic”)
- Torsade de Pointes

**Polymorphic VT**
- Requires Immediate Defibrillation
- Prolonged QT (Torsade de Pointes)
  - Magnesium sulfate 1-2gm diluted in 10ml D5W IV push
    - Negative inotrope that slows electrical signals in AV node
    - May be drug induced
- Absence of prolonged QT interval
  - Commonly caused by myocardial infarction
  - Amiodarone may reduce recurrence until Cath
Drugs That Prolong QT Interval

- Antiarrhythmics
  - Dofetilide, procainamide, quinidine, sotalol, amiodarone, flecanide
- Antipsychotics
  - Haloperidol, thioridazine, pimozide, ziprasidone, clozapine, quetiapine, risperidone
- Narcotics
  - Methadone
- Antimicrobials
  - Clarithromycin, erythromycin, levofloxacin, moxifloxacin, fluconazole, voriconazole
- Antiemetics
  - Droperidol, chlorpromazine, ondansetron

Patient Case RM

- CC/HPI: 80 yo female found unresponsive and pulseless at nursing home. EMS arrived and started CPR right away. On arrival to ER, pt has been in asystole for 15 minutes and has received 2 rounds of epinephrine.
- PMH: HTN, DM, HL, CABG
- Vitals: Asystole

Question 3

- Patient is still in cardiac arrest and monitor shows asystole. What is the drug of choice at this point?
  - A.) Atropine 1mg IV
  - B.) Epinephrine 1mg IV
  - C.) Calcium Chloride 1gm IV
  - D.) Amiodarone 150mg IV

Question 4

- After 2 more rounds of Epi, the patient goes into V.Fib. The patient is shocked and CPR is continued. After 2 minutes patient is still in V.Fib, so is shocked again and another round of epi is given. If patient stays in V.Fib, what is the most appropriate treatment at this point?
  - A.) Give double the dose of Epinephrine
  - B.) Amiodarone 150mg IV bolus
  - C.) Amiodarone 300mg IV bolus
  - D.) Lidocaine 100mg IV bolus

Tachycardia (with pulse)

- Narrow QRS complex tachycardias (QRS <0.12 seconds)
  - Reentrant tachycardias (SVT)
    - Abnormal rhythm circuit that allows a wave of depolarization to repeatedly travel in a circle in cardiac tissue
    - Sinus tach
    - Atrial Fibrillation
    - Atrial Flutter
    - Multifocal atrial tachycardia (MAT)
    - Junctional tachycardia
- Wide QRS complex tachycardias (QRS > 0.12 seconds)
  - V-tach and V-fib
  - Pre-excited tachycardias (WPW syndrome)

Tachycardia (with pulse)

- Synchronized Cardioversion if Unstable
  - Supraventricular tachycardia (SVT), A-fib, A-flutter, monomorphic (regular) VT
  - Timed with QRS complex to avoid shock delivery during refractory period (V-fib)
  - 120 Joules
- Adenosine
  - Stable paroxysmal SVT (narrow-complex), diagnostic
  - Slows conduction time through AV node
  - 6mg rapid IV push followed by 20ml saline flush
  - Repeat 12mg IV after 1-2 min. x 2
Tachycardia (with pulse)

- **Verapamil**
  - Stable Narrow-complex SVT if not converted by adenosine
  - Decrease AV Node conduction
  - 5mg IV bolus q15min

- Other options include:
  - Diltiazem, metoprolol, esmolol

Stable Wide-Complex Tachycardia (VT)

- **Procainamide**
  - Class Ia antiarrhythmic
  - Increased electrical stimulation threshold of ventricle
  - Increased refractory period and reduction in conduction velocity
  - 50mg/min (max bolus: 17mg/kg), then 1-4mg min.
  - Avoid in prolonged QT and CHF

- **Amiodarone**
  - 150mg over 10 mins., then 1mg/min x 6 hrs followed by 0.5mg/min

- **Lidocaine**
  - Second line

Bradycardia (with pulse)

- **Atropine**
  - 0.5mg IV q3-5min (max: 3mg)
  - Not effective for type II second-degree or third degree AV block

- **Transcutaneous/Transvenous Pacing**
  - May be first-line if unstable
  - No response to atropine

- **Dopamine**
  - 5-10mcg/kg/min (max: 20mcg/kg/min)

- **Epinephrine**
  - 1-2mcg/min (max: 10mcg/min)

Sodium Bicarbonate

- **Not recommended for routine use**

- May see benefit in the following:
  - Preexisting metabolic acidosis
  - Hyperkalemia
  - TCA overdose

  - 1mEq/kg IV (1syringe of 8.4%=50mEq)

Calcium Chloride

- **Not used routinely**

- Calcium Channel Blocker overdose

- Hyperkalemia/Hypermagnesemia
  - Stabilize myocardial cell membrane

  - 1000mg (1vial) IV push over 2-5min

Patient Case DP

- **CC/HPI:** 65yo Male presents to the ER with sudden onset of severe SOB while mowing his lawn. Pt also reported pain and swelling in his right calf that started a few days ago.

- **PMH:** None

- **Social Hx:** 50 pack per year smoker.
Patient Case DP

• Vitals
  – BP 80/50, HR 140, RR 40, 80% on room air
• Pt was put on a non-rebreather mask and sat increased to 95%
• Shortly after, patient became pale and diaphoretic and pressure dropped to 60/40 despite fluid resuscitation.
• Then patient went unresponsive and a code was called.

Question 5

• Nurse couldn’t feel a pulse so she started chest compressions. After 2 minutes of compressions and 1 round of epi, monitor showed asystole. What therapy should be considered in this patient?
  A.) Defibrillation
  B.) Intracardiac Epinephrine 5mg IV
  C.) Heparin drip
  D.) Alteplase 50mg IV push

Question 6

• The pt is suspected to have a PE, so Alteplase 50mg IV push is given. After 5 more minutes of chest compressions, the pt gets a pulse back. What should the pharmacist be concerned with at this point?
  A.) Blood pressure management
  B.) Continuing the remainder of the alteplase
  C.) Timing of heparin drip
  D.) All of the above

Fibrinolytics

• Coronary and Pulmonary thrombosis are most common causes of cardiac arrest
• Insufficient evidence to advocate routine use
  – 2 large studies showed no benefit with tpa or tenecteplase (increased ICH)
  – Consider use if suspected AMI or PE
• If Pulmonary Embolism suspected or known
  – Mortality 95%
  – Fibrinolytics may improve survival to discharge and long-term neurologic function
  – Alteplase 50mg IV and repeat after 15 min if no ROSC

Alternate Routes

• Intraosseous (IO)
  – Safe and effective for fluid resuscitation and drug administration
  – Same dose as IV
• Endotracheal (ET) Tube administration
  – Naloxone
  – Atropine
  – Vasopressin
  – Epinephrine
  – Lidocaine
  Dilute dose in 5-10ml sterile water

Post-Cardiac Arrest Care

• Optimize cardiopulmonary function and organ perfusion
• Identify and treat underlying cause and prevent recurrent arrest
• Optimize Survival and Neurologic recovery
  – Determined by extent of brain injury and cardiovascular instability
Vasopressors

Goal: Maintain SBP≥90 or MAP≥65

- Norepinephrine
  - 5mcg/min, increase by 5mcg/min q5 min (MAX: 200mcg/min)
- Epinephrine
  - 1-2mcg/min, increase by 1mcg/min q5 min (MAX: 10mcg/min)
- Phenylephrine
  - 100-200mcg/min, increase by 25-50mcg/min q10 min (MAX: 300mcg/min)
- Vasopressin
  - 0.03 units/min
- Dopamine
  - 10mcg/kg/min, increase by 5mcg/kg/min q5min (MAX: 20mcg/kg/min)

Anti-Arrhythmics

- Amiodarone
  - 450mg/250ml D5W
  - 1mg/min x6hrs, then 0.5mg/min
- Procainamide
  - 17mg/kg loading dose (MAX: 1000mg)
  - 2-4mg/min
- Lidocaine
  - 1mg/kg loading dose
  - 1-2mg/min (MAX: 4mg/min)

Therapeutic Hypothermia

- Use of mild hypothermic techniques and interventions, designed to suppress many chemical reactions associated with reperfusion injury in order to prevent or reduce cerebral injury in cardiac arrest survivors
- Post cardiac arrest brain injury
  - Excitotoxicity
  - Disrupted calcium homeostasis
  - Free radical formation
  - Pathological protease cascade
  - Cell death signaling pathway

Therapeutic Hypothermia

- Induction
  - Rapid cooling within 4-8 hours to 32-34°C
  - Cold saline infusion
  - Ice packs
  - Cooling blankets
  - Sedation and paralysis
- Maintenance
  - Temp of 32-34°C for 12-24 hours
- Rewarming
  - Warm by 0.5-1°C per hour

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Question 7

- What intervention, post ROSC, has shown to improve neurologic function in comatose patients at discharge?
  A.) Vasopressor support with Dopamine
  B.) Continuous Amiodarone drip for V. Fib
  C.) Cooling patient to 30° C for 24 hours
  D.) Cooling patient to 33° C for 24 hours

Goal Objectives

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- Discuss post-resuscitation care involving medication management.
References


- https://www.baylorhearthospital.com/afibHeartConductionSystem.html