The Cardiac Drug Box

Christina Candeloro, PharmD, BCPS
Clinical Pharmacy Specialist, Emergency Medicine, VCUHS
Assistant Clinical Professor, VCU School of Pharmacy
Richmond, VA
Objectives

- Explain the pharmacology and pharmacokinetics of the cardiac drugs you use regularly and rarely
- Review algorithms on how to appropriately use pharmacotherapy in cardiac arrests
- Discuss how to manage brady and tachyarrhythmias
- Describe the pre-hospital treatment of acute coronary syndrome
Advanced Cardiac Life Support (ACLS)

History

- **Late 1950s**
  - Mouth-to-mouth and application of electricity

- **1966**
  - NASNRC Holds first CPR conference

- **AHA and AAP**

- **2010**
  - AHA ACLS Guideline Update

*Pharmacother 2006;26:1703-1729*
Survival in Sudden Cardiac Arrest

Patients who experience out-of-hospital arrest

- 1/3 survives to hospital arrival
- 3-13% survives to discharge
- Chance for successful resuscitation/survival decreases 7-10% for each minute without adequate circulation
- After 5 minutes, 40% chance of successful revival

Pharmacother 2006;26:1703-1729
Survival in Sudden Cardiac Arrest

Four key elements, aka Chain of Survival

1. Early access
2. Early CPR
3. Early defibrillation
4. Early ACLS

A-B-C → C-A-B

Pharmacother 2006;26:1703-1729
Survival in Sudden Cardiac Arrest

Automatic External Defibrillators are everywhere!
CPR Quality
- Push hard 5-6 inches [5 cm] and fast 100-120/min and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Petals compressor every 2 minutes
- If no advanced airway, 30:2 compression:ventilation ratio
- Quantitative waveform capnography
  - If PetCO₂ <10 mm Hg, attempt to improve CPR quality
- Intra-aortic pressure
  - If relaxation phase (diastolic pressure) <20 mm Hg, attempt to improve CPR quality

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Absent sustained increase in PetCO₂ typically >10 mm Hg
- Spontaneous arterial pressure waves with intra-aortic monitoring

Shock Energy
- Dynamic: Manufacturer recommendation (e.g., initial dose of 150-200 J). If unknown, use maximum available. Second and subsequent doses should be equal, and higher doses may be considered.
- Monophasic: 360 J

Drug Therapy
- Epinephrine IV/IO Dose: 1 mg every 3-5 minutes
- Vasopressin IV/IO Dose: 40 units can replace first or second dose of epinephrine
- Amiodarone IV/IO Dose: First dose: 300 mg bolus. Second dose, 150 mg
- Advanced Airway
  - Supraglottic advanced airway or endotracheal intubation
  - Waveform capnography to confirm endotracheal tube placement
  - 8-12 breaths per minute with continuous chest compressions

Reversible Causes
- Hypoxia
- Hypotension
- Hypothermia
- Hypo-hyperkalemia
- Hypovolemia
- Tension pneumothorax
- Tamponade, cardiac
- Tachyarrhythmias
- Pulmonary disease
- Myocarditis
- Thrombosis, pulmonary
- Thrombosis, coronary

Figure 1. ACLS Cardiac Arrest Algorithm.
ACLS

Adult Cardiac Arrest

Shout for Help/Activate Emergency Response

Start CPR
- Give oxygen
- Attach monitor/defibrillator

Return of Spontaneous Circulation (ROSC)

Check Rhythm

If VF/VT
Shock

Continuous CPR

Consider Advanced Airway
Quantitative waveform capnography

Drug Therapy
IV/IO access
Epinephrine every 3-5 minutes
Amiodarone for refractory VF/VT

Treat Reversible Causes

Monitor CPR Quality

CPR Quality
- Push hard (≥2 inches [5 cm]) and fast (≥100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 30:2 compression-ventilation ratio
- Quantitative waveform capnography
  - If PETCO₂ <10 mm Hg, attempt to improve CPR quality
  - Intra-arterial pressure
  - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Abrupt sustained increase in PETCO₂ (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Shock Energy
- Biphasic: Manufacturer recommendation (e.g., initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- Monophasic: 360 J

Drug Therapy
- Epinephrine IV/IO Dose: 1 mg every 3-5 minutes
- Vasopressin IV/IO Dose: 40 units can replace first or second dose of epinephrine
- Amiodarone IV/IO Dose: First dose: 300 mg bolus. Second dose: 150 mg.

Advanced Airway
- Supraglottic advanced airway or endotracheal intubation
- Waveform capnography to confirm and monitor ET tube placement
- 8-10 breaths per minute with continuous chest compressions

Reversible Causes
- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo-/hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

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Figure 2. ACLS Cardiac Arrest Circular Algorithm.

Circulation 2010;122:S729-S767
Cardiac Arrest

Role of Drug Therapy

- Basic CPR more effective in improving survival to hospital discharge than drug therapy
- **Primary goal:** facilitate restoration and maintenance of a perfusing spontaneous rhythm
- Not the most important thing in ACLS
- Adjuncts that enhance the likelihood of ROSC
- Must be coordinated with nonpharmacologic treatments

Pharmacother 2006;26:1703-1729
Circulation 2010;122:S729-S767
ACLS – Pharmacology 101

- **Agonist**
  - A chemical substance that upon binding to a specific receptor, activates it and produces a response

- **Antagonist**
  - A chemical substance that upon binding to a specific receptor, blocks the binding of agonists and opposes response on that receptor

http://www.uic.edu/classes/bios/bios100/f05pm/art_agonist.gif
ACLS – the important equations

Blood Pressure = Cardiac Output x Systemic Vascular Resistance

- Stroke Volume x Heart Rate
- Preload (volume)

Pharmacother 2006;26:1703-1729
### ACLS – the important receptors

<table>
<thead>
<tr>
<th>Receptor</th>
<th>Location</th>
<th>Resultant Effects</th>
<th>Drugs that Agonize</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \alpha_1 )</td>
<td>Peripheral Vasculature</td>
<td>( \uparrow ) Systemic Vascular Resistance (SVR) Vasoconstricts</td>
<td>Epinephrine Dopamine ( (\uparrow \text{ dose}) )</td>
</tr>
<tr>
<td>( \beta_1 )</td>
<td>Heart</td>
<td>( \uparrow ) Inotropy and Chronotropy ( \uparrow ) Force of contraction &amp; HR ( \uparrow ) Cardiac Output</td>
<td>Epinephrine DPA ( (\text{med}/\uparrow \text{ dose}) )</td>
</tr>
<tr>
<td>( V_1 )</td>
<td>Peripheral Vasculature</td>
<td>( \uparrow ) Systemic Vascular Resistance (SVR) Vasoconstricts</td>
<td>Vasopressin</td>
</tr>
</tbody>
</table>

*Pharmacother 2006;26:1703-1729*
ACLS – the important equations

Blood Pressure = Cardiac Output x Systemic Vascular Resistance

Stroke Volume x Heart Rate

Preload

Epinephrine

Pharmacother 2006;26:1703-1729
ACLS – the important equations

Blood Pressure = Cardiac Output x Systemic Vascular Resistance

Stroke Volume x Heart Rate

Preload

Vasopressin

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Cardiac Arrest - What is important?

- ROSC!!!
- But before that...?
  - ORGAN PERFUSION
    - Enhancing cerebral perfusion pressures
    - Enhancing coronary perfusion pressures

*Pharmacother* 2006;26:1703-1729
Cardiac Arrest – Role of Drug Therapy

Vasoactive Agents – why?

- No trials show that vasopressors increase neurologically intact survival to hospital discharge
- Evidence suggests that catecholamines improve chance of initial ROSC
- In the absence of adequate circulation vasoconstricting drugs may enhance organ perfusion resulting in ↑ cerebral and coronary perfusion pressures while ↓ blood flow to visceral and muscle tissues
- ↑ coronary perfusion pressure and myocardial blood flow are associated with success of defibrillation and ROSC

*Pharmacother* 2006;26:1703-1729
*Circulation* 2010;122:S729-S767
Cardiac Arrest – Epinephrine

- Preferred initial catecholamine in ACLS for PEA, asystole, VF, pulseless VT
- MOA: $\alpha_1$, $\beta_1$ agonist – used for vasopressor properties
  - $\uparrow$ SVR and CO
  - $\uparrow$ coronary and cerebral perfusion pressures
- Dose 1 mg every 3-5 minutes (IV/IO) ASAP
  - In VF/pulseless VT give after the first shock is attempted if rhythm persists $\rightarrow$ if shock restores rhythm, a pressor may be detrimental
- $t1/2 \approx 2$ minutes
- Can be given via ETT (not preferred)
  - 2-2.5 mg diluted in 10 ml

_Pharmacother 2006;26:1703-1729_  
_Circulation 2010;122:S729-S767_
Cardiac Arrest – ETT Epinephrine

Treatment

- Epinephrine – the math

1:1000 = 1g/1000mL

1g/1000mL = 1000mg/1000mL

1000mg/1000mL = 1 mg/1ml

1 mg/1ml = 2 mg/2 ml

1:10,000 = 1g/10,000mL

1g/10,000mL = 1000mg/10,000mL

1000mg/10,000mL = 0.1 mg/ml

0.1 mg/1ml = 2 mg/20 ml
ACLS – Epinephrine – the optimal dose

- Controversial; Original guidelines: 0.5 – 1 mg every 5 mins
- Late 1980s: ↓ survival rates, anecdotal observations of success with higher doses
- Further data 5 mg vs 1 mg in five in and out of hospital trials
  - Slight increase in rates of resuscitation in some
  - Higher post-resuscitation complications
    - Cardiac dysfunction/arrhythmias
    - Hypoxia
    - ↑ frequency of adverse neurologic outcomes/ ↑ hospital LOS
- Current (limited) data (and guidelines) favors 1 mg dose
- Higher dose if: CCB/BB OD

Pharmacother 2006;26:1703-1729
ACLS – Epinephrine – the optimal interval

- Current guidelines recommend every 3-5 minutes
- Whether or not this is optimal is unclear
- In practice, intervals frequently exceed 5 minutes
  - Mean 6.8 mins out-of-hospital vs 5.6 mins in-hospital
- Give drug with every other pulse check

Pharmacother 2006;26:1703-1729
ACLS – Vasopressin

- Initial catecholamine in ACLS for PEA, asystole, VF, pulseless VT
- MOA: V₁ agonist
  - ↑ SVR ; ↑ coronary and cerebral perfusion pressures
- Dose 40 units every 3-5 minutes (IV/IO) to replace first or second dose of epinephrine ASAP
  - In VF/pulseless VT give after the first shock is attempted if rhythm persists → if shock restores rhythm, a pressor may be detrimental
- Can be given via ETT (not preferred) - Consider 2x the dose
- Potentiates the effects of catecholamines
- Half-life: 10-20 minutes
- Works in acidemic patient

Pharmacother 2006;26:1703-1729
Circulation 2010;122:S729-S767
# ACLS – Vasopressin – the data

## Table 3. Data on Outcomes in All 1186 Patients and on Cerebral Performance in 115 Patients at Hospital Discharge.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Vasopressin Group (N=589)</th>
<th>Epinephrine Group (N=597)</th>
<th>P Value</th>
<th>Odds Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>All patients</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spontaneous circulation restored with study drugs</td>
<td>145/589 (24.6)</td>
<td>167/597 (28.0)</td>
<td>0.19</td>
<td>1.2 (0.9–1.5)</td>
</tr>
<tr>
<td>Hospital admission</td>
<td>214/589 (36.3)</td>
<td>186/597 (31.2)</td>
<td>0.06</td>
<td>0.8 (0.6–1.0)</td>
</tr>
<tr>
<td>Hospital discharge</td>
<td>57/578 (9.9)</td>
<td>58/588 (9.9)</td>
<td>0.99</td>
<td>1.0 (0.7–1.5)</td>
</tr>
<tr>
<td><strong>Ventricular fibrillation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spontaneous circulation restored with study drugs</td>
<td>82/223 (36.8)</td>
<td>106/249 (42.6)</td>
<td>0.20</td>
<td>1.3 (0.9–1.8)</td>
</tr>
<tr>
<td>Hospital admission</td>
<td>103/223 (46.2)</td>
<td>107/249 (43.0)</td>
<td>0.48</td>
<td>0.9 (0.6–1.3)</td>
</tr>
<tr>
<td>Hospital discharge</td>
<td>39/219 (17.8)</td>
<td>47/245 (19.2)</td>
<td>0.70</td>
<td>1.1 (0.7–1.8)</td>
</tr>
<tr>
<td><strong>Pulseless electrical activity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spontaneous circulation restored with study drugs</td>
<td>21/104 (20.2)</td>
<td>17/82 (20.7)</td>
<td>0.93</td>
<td>1.0 (0.5–2.1)</td>
</tr>
<tr>
<td>Hospital admission</td>
<td>35/104 (33.7)</td>
<td>25/82 (30.5)</td>
<td>0.65</td>
<td>0.8 (0.5–1.6)</td>
</tr>
<tr>
<td>Hospital discharge</td>
<td>6/102 (5.9)</td>
<td>7/81 (8.6)</td>
<td>0.47</td>
<td>1.4 (0.5–4.7)</td>
</tr>
<tr>
<td><strong>Asystole</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spontaneous circulation restored with study drugs</td>
<td>42/262 (16.0)</td>
<td>44/266 (16.5)</td>
<td>0.87</td>
<td>1.0 (0.7–1.6)</td>
</tr>
<tr>
<td>Hospital admission</td>
<td>76/262 (29.0)</td>
<td>54/266 (20.3)</td>
<td>0.02</td>
<td>0.6 (0.4–0.9)</td>
</tr>
<tr>
<td>Hospital discharge</td>
<td>12/257 (4.7)</td>
<td>4/262 (1.5)</td>
<td>0.04</td>
<td>0.3 (0.1–1.0)</td>
</tr>
<tr>
<td>Cerebral performance among all patients who survived to discharge</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Good cerebral performance</td>
<td>15/46 (32.6)</td>
<td>16/46 (34.8)</td>
<td>0.99</td>
<td></td>
</tr>
<tr>
<td>Moderate cerebral disability</td>
<td>7/46 (15.2)</td>
<td>12/46 (26.1)</td>
<td>0.30</td>
<td></td>
</tr>
<tr>
<td>Severe cerebral disability</td>
<td>9/46 (19.6)</td>
<td>7/46 (15.2)</td>
<td>0.78</td>
<td></td>
</tr>
<tr>
<td>Coma or vegetative state</td>
<td>15/46 (32.6)</td>
<td>11/46 (23.9)</td>
<td>0.49</td>
<td></td>
</tr>
</tbody>
</table>

Cardiac Arrest – Role of Drug Therapy

Antiarrhythmics—why?

- Have not shown to improve to survival to hospital discharge
- Has shown to improve the rate of ROSC and hospital admission in adults with refractory VF/pulseless VT
- Potential to normalize abnormally depolarizing/conducting myocardial cells
- Combination of defibrillation + antiarrhythmic may restore a sustainable rhythm

Pharmacother 2006;26:1703-1729
Circulation 2010;122:S729-S767
Cardiac Arrest – Amiodarone

- Initial antiarrhythmic in VF/pulseless VT
- Class III antiarrhythmic
- MOA: Na, K, Ca channel antagonist aka “King of Dirty”
  - Prolongs action potential and refractory period in myocardial tissue
  - Decreases AV conduction and sinus node function
- Dose 300 mg (IV/IO) x1 followed by 150 mg IV/IO in 3-5 minutes if no response and still in VF/pulseless VT
  - Consider when VF/VT is unresponsive to CPR, defibrillation, and vasopressor therapy
- ETT administration NOT recommended because of local irritating effects

*Pharmacother* 2006;26:1703-1729
*Circulation* 2010;122:S729-S767
ARREST TRIAL

- Out-of-hospital VF/Pulseless VT (n=504)
- After three shocks → amiodarone 300 mg IV vs placebo

ACLS – Amiodarone - administration

- Lots of warnings
- In large trials, diluted with 20-30 ml volume
- Undiluted amiodarone can cause:
  - Bradycardia
  - Hypotension (polysorbate 80)
  - Phlebitis
- Recommendation: to dilute and give slowly
- However, in cardiac arrest situation with a pulseless patient any delay should be avoided

Pharmacother 2006;26:1703-1729
Helsinki EMS service
Undiluted amiodarone vs placebo

Post-return of spontaneous circulation blood pressure levels, vasopressor treatment and transcutaneous pacing among patients resuscitated from out-of-hospital VF/VT indexed according to whether or not they received amiodarone.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Amiodarone (n = 46)</th>
<th>No amiodarone (n = 70)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Highest measured SAP (available in 87%)</td>
<td>140 (128–164)</td>
<td>130 (120–151)</td>
<td>NS</td>
</tr>
<tr>
<td>Lowest measured SAP (available in 71%)</td>
<td>97 (84–120)</td>
<td>100 (90–119)</td>
<td>NS</td>
</tr>
<tr>
<td>Proportion with SAP &lt;90 prehospitaly</td>
<td>26%</td>
<td>22%</td>
<td>NS</td>
</tr>
<tr>
<td>Proportion with SAP &lt;120 prehospitaly</td>
<td>46%</td>
<td>59%</td>
<td>NS</td>
</tr>
<tr>
<td>Highest measured DAP (82%)</td>
<td>90 (79–98)</td>
<td>80 (63–91)</td>
<td>0.012</td>
</tr>
<tr>
<td>Lowest measured DAP (62%)</td>
<td>66 (59–77)</td>
<td>63 (59–80)</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Highest measured heart rate min⁻¹ (78%)</td>
<td>90 (72–107)</td>
<td>98 (80–115)</td>
<td>NS</td>
</tr>
<tr>
<td>Lowest measured heart rate min⁻¹ (58%)</td>
<td>60 (52–77)</td>
<td>75 (64–90)</td>
<td>0.010</td>
</tr>
<tr>
<td>Vasopressor treatment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dopamine infusion</td>
<td>41%</td>
<td>33%</td>
<td>NS</td>
</tr>
<tr>
<td>adrenaline infusion</td>
<td>2%</td>
<td>0%</td>
<td>NS</td>
</tr>
<tr>
<td>maximum dose of dopamine used (µg kg⁻¹ min⁻¹)</td>
<td>10 (10–15)</td>
<td>10 (8–10)</td>
<td>NS</td>
</tr>
<tr>
<td>Treatment of bradycardia with atropin</td>
<td>2%</td>
<td>1%</td>
<td>NS</td>
</tr>
<tr>
<td>Transcutaneous pacing</td>
<td>2%</td>
<td>3%</td>
<td>NS</td>
</tr>
</tbody>
</table>

### Amiodarone - Compatibility Info

#### Compatibility Chart

- **G**: Indicates compatibility for this method.
- **U**: Uncertain or variable for this method.
- **I**: Indicates incompatibility for this method.
- **X**: No data for any method.

#### Drugs

<table>
<thead>
<tr>
<th>Drug</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone hydrochloride</td>
</tr>
</tbody>
</table>

#### Solutions

<table>
<thead>
<tr>
<th>Solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium chloride 0.9% - See Normal saline</td>
</tr>
</tbody>
</table>
# Amiodarone - Compatibility Info

<table>
<thead>
<tr>
<th>Administration Method:</th>
<th>Solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drugs:</td>
<td>Amiodarone hydrochloride</td>
</tr>
<tr>
<td>Vehicles:</td>
<td>none</td>
</tr>
<tr>
<td>Solutions:</td>
<td>Normal saline- Sodium chloride 0.9%</td>
</tr>
</tbody>
</table>

Results: 4 Compatible and 1 Incompatible and 1 undetermined study result(s). Click on a study to view details.

<table>
<thead>
<tr>
<th>Study</th>
<th>Drug 1</th>
<th>Vehicle 1</th>
<th>Drug 2</th>
<th>Vehicle 2</th>
<th>Solution</th>
<th>Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study 1</td>
<td>Amiodarone hydrochloride 2 mg/mL</td>
<td></td>
<td></td>
<td></td>
<td>Normal saline-Sodium chloride 0.9%</td>
<td>Compatible</td>
</tr>
<tr>
<td>Study 2</td>
<td>Amiodarone hydrochloride 2 mg/mL</td>
<td></td>
<td></td>
<td></td>
<td>Normal saline-Sodium chloride 0.9%</td>
<td>Compatible</td>
</tr>
<tr>
<td>Study 3</td>
<td>Amiodarone hydrochloride 1.8 mg/mL</td>
<td></td>
<td></td>
<td></td>
<td>Normal saline-Sodium chloride 0.9%</td>
<td>Compatible</td>
</tr>
<tr>
<td>Study 4</td>
<td>Amiodarone hydrochloride 1.8 mg/mL</td>
<td></td>
<td></td>
<td></td>
<td>Normal saline-Sodium chloride 0.9%</td>
<td>Compatible</td>
</tr>
<tr>
<td>Study 5</td>
<td>Amiodarone hydrochloride 0.84 mg/mL</td>
<td></td>
<td></td>
<td></td>
<td>Normal saline-Sodium chloride 0.9%</td>
<td>Uncertain or Variable</td>
</tr>
<tr>
<td>Study 6</td>
<td>Amiodarone hydrochloride 0.6 mg/mL</td>
<td></td>
<td></td>
<td></td>
<td>Normal saline-Sodium chloride 0.9%</td>
<td>Incompatible</td>
</tr>
</tbody>
</table>
ACLS – Lidocaine

- Second-line antiarrhythmic in VF/pulseless VT
  - If amiodarone not available
- Class Ib antiarrhythmic
- MOA: Na channel antagonist
  - Suppresses automaticity of conduction tissue
- Dose 1-1.5 mg (IV/IO), can repeat 0.5-0.75 mg/kg every 5-10 mins (maximum dose 3 mg/kg)
- Can be given via ETT (not preferred)
  - 2-4 mg/kg
- Data are lacking

Pharmacother 2006;26:1703-1729
Cardiac Arrest – Lidocaine – the data

ALIVE TRIAL

- Out-of-hospital shock-resistant VF/Pulseless VT (n=504)
- Amiodarone 5 mg/kg (2.5 mg/kg repeat) vs Lidocaine 1.5 mg/kg (1.5 mg/kg repeat dose)

Cardiac Arrest – Role of Drug Therapy

Magnesium – why?

- Hypomagnesemia can inhibit conductance through myocardial K channels → prolonged action potential during ventricular repolarization → QT prolongation
- Giving magnesium may improve K transport through myocardial K channels and shorten action potential
- Used when there is suspicion of torsade de pointes, even in the absence of hypomagnesemia

Pharmacother 2006;26:1703-1729
ACLS – Torsades de Pointes (TdP)
ACLS – TdP – Causative Agents

- Antiarrhythmics – Class I
  - Quinidine, disopyramide, dofetilide, ibutilide, sotalol

- Antimicrobials
  - Macrolides, fluoroquinolones, azoles, antimalarials

- Antidepressants
  - Amitriptylline, fluoxetine

- Antipsychotics
  - Haloperidol, thioridazine

- Miscellaneous
  - Methadone

PSAP VI. 39-55.
## ACLS – TdP – Causative Agents

### Table 1
Twenty most commonly reported drugs associated with torsades de pointes (TdP) between 1983 and 1999

<table>
<thead>
<tr>
<th>Drug</th>
<th>TdP (n)</th>
<th>Fatal (n)</th>
<th>Total (n)</th>
<th>TdP/total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sotalol</td>
<td>130</td>
<td>1</td>
<td>2758</td>
<td>4.71</td>
</tr>
<tr>
<td>Cisapride</td>
<td>97</td>
<td>6</td>
<td>6489</td>
<td>1.49</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>47</td>
<td>1</td>
<td>13725</td>
<td>0.34</td>
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<tr>
<td>Erythromycin</td>
<td>44</td>
<td>2</td>
<td>24776</td>
<td>0.18</td>
</tr>
<tr>
<td>Ibutidine</td>
<td>43</td>
<td>1</td>
<td>173</td>
<td>24.86</td>
</tr>
<tr>
<td>Terfenadine</td>
<td>41</td>
<td>1</td>
<td>10047</td>
<td>0.41</td>
</tr>
<tr>
<td>Quinidine</td>
<td>33</td>
<td>2</td>
<td>7353</td>
<td>0.45</td>
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<tr>
<td>Clarithromycin</td>
<td>33</td>
<td>0</td>
<td>17448</td>
<td>0.19</td>
</tr>
<tr>
<td>Haloperidol</td>
<td>21</td>
<td>6</td>
<td>15431</td>
<td>0.14</td>
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<tr>
<td>Fluoxetine</td>
<td>20</td>
<td>1</td>
<td>70929</td>
<td>0.03</td>
</tr>
<tr>
<td>Digoxin</td>
<td>19</td>
<td>0</td>
<td>18925</td>
<td>0.10</td>
</tr>
<tr>
<td>Procaínamide</td>
<td>19</td>
<td>0</td>
<td>5867</td>
<td>0.32</td>
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<tr>
<td>Terodiline</td>
<td>19</td>
<td>0</td>
<td>2248</td>
<td>0.85</td>
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<tr>
<td>Fluconazole</td>
<td>17</td>
<td>0</td>
<td>5613</td>
<td>0.30</td>
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<tr>
<td>Disopyramide</td>
<td>16</td>
<td>1</td>
<td>3378</td>
<td>0.47</td>
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<tr>
<td>Bepridil</td>
<td>15</td>
<td>0</td>
<td>384</td>
<td>3.91</td>
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<tr>
<td>Furosemide</td>
<td>15</td>
<td>0</td>
<td>15119</td>
<td>0.10</td>
</tr>
<tr>
<td>Thioridazine</td>
<td>12</td>
<td>0</td>
<td>6565</td>
<td>0.18</td>
</tr>
<tr>
<td>Flecaínide</td>
<td>11</td>
<td>2</td>
<td>3747</td>
<td>0.29</td>
</tr>
<tr>
<td>Loratidin</td>
<td>11</td>
<td>1</td>
<td>5452</td>
<td>0.20</td>
</tr>
</tbody>
</table>
ACLS – Magnesium

- Treatment of choice for TdP
- Routine use in cardiac arrest is not recommended
- MOA: unknown
  - May improve K transport through myocardial K channels and shorten action potential
- Dose 1-2 g IV
  - In patients arresting, can give IVP diluted in 10ml
  - In patient in TdP with a pulse, dilute in 100ml D5W and give over 15-20 mins to avoid hypotension
- AE: flushing, hypotension
Cardiac Arrest – Role of Drug Therapy

Atropine – why?

- Inhibits cholinergic responses that diminish HR and SVR
- Supporting data are limited and unclear in terms of the effectiveness for asystole/PEA

Pharmacother 2006;26:1703-1729
Cardiac Arrest – Atropine

- Routine use of atropine is **NOT** recommended
- **Removed** from PEA/asystole algorithms for lack of data
- Was never in VF/Pulseless VT algorithms
- MOA: blocks acetylcholine at parasympathetic sites
  - ↑ HR
- Dilates pupils for a significant time period

*Pharmacother 2006;26:1703-1729*
*Circulation 2010;122:S729-S767*
Cardiac Arrest – Role of Drug Therapy

Sodium Bicarbonate – why?

- Acidosis occurs in patients with circulatory collapse and respiratory failure due to accumulation of H\(^+\) ions and CO\(_2\)
  - At ↓ pH (acidosis) there is reduced binding of O\(_2\) to hemoglobin and therefore ↓ oxygen delivery
- Sodium bicarbonate is an alkalizing agent

*Pharmacother 2006;26:1703-1729*
THE CONCERNS

- Administration of NaHCO$_3$ has not shown beneficial effects on survival; may worsen chance of survival
  - pH values > 7.55 at 10 mins during arrest were associated with ↓ survival
  - Alterations in defibrillation thresholds
  - Compromised coronary perfusion pressures
  - Creation of hyperosmolar state
  - Hypernatremia
  - Inactivation of catecholamines
  - Central venous acidosis by producing excess CO$_2$

Pharmacother 2006;26:1703-1729
### The Concerns

**Table 2.** \( \text{PCO}_2 \) in Aortic, Mixed Venous, and Great Cardiac Vein Blood Before and After Infusion of Buffer Agents and Saline Control

<table>
<thead>
<tr>
<th>Site</th>
<th>Solution</th>
<th>Control –2 Min</th>
<th>Preinfusion</th>
<th>Precordial compression during ventricular fibrillation</th>
<th>Postinfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>5 Min</td>
<td>9 Min</td>
<td>Postresuscitation +2 Min</td>
</tr>
<tr>
<td>Aorta</td>
<td>( \text{NaHCO}_3 )</td>
<td>41.1±1.0</td>
<td>40.4±2.6</td>
<td>53.0±3.0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Carbicarb</td>
<td>41.2±1.7</td>
<td>37.6±3.3</td>
<td>31.2±6.0†</td>
<td>62.8±3.7</td>
</tr>
<tr>
<td></td>
<td>( \text{NaCl} )</td>
<td>45.5±1.8</td>
<td>37.5±2.8</td>
<td>38.5±3.7</td>
<td>67.6±6.1</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>( \text{NaHCO}_3 )</td>
<td>51.2±2.3</td>
<td>66.3±5.0</td>
<td>77.1±4.6§</td>
<td>93.6±8.9</td>
</tr>
<tr>
<td></td>
<td>Carbicarb</td>
<td>49.2±2.9</td>
<td>64.3±7.2</td>
<td>54.5±5.9†§</td>
<td>79.5±6.8</td>
</tr>
<tr>
<td></td>
<td>( \text{NaCl} )</td>
<td>53.5±2.2</td>
<td>66.5±3.7</td>
<td>60.5±4.4</td>
<td>79.4±5.4</td>
</tr>
<tr>
<td>Great cardiac vein</td>
<td>( \text{NaHCO}_3 )</td>
<td>52.5±1.5</td>
<td>99.6±7.4</td>
<td>135.7±16.7§</td>
<td>82.4±5.6</td>
</tr>
<tr>
<td></td>
<td>Carbicarb</td>
<td>53.0±2.5</td>
<td>135.6±20.7</td>
<td>121.4±22.6</td>
<td>71.2±4.3</td>
</tr>
<tr>
<td></td>
<td>( \text{NaCl} )</td>
<td>55.0±2.1</td>
<td>120.4±19.5</td>
<td>101.1±13.4</td>
<td>70.3±4.7</td>
</tr>
</tbody>
</table>

\( p<0.05 \) vs. \( \text{NaHCO}_3 \) and \( \text{NaCl} \) by one-way analysis of variance and a least-significant difference test; \( || p<0.01 \) vs. preinfusion by the Wilcoxon’s pair test; \( $ p<0.05 \) vs. preinfusion.
Cardiac Arrest – Sodium Bicarbonate

- **Routine use of NaHCO₃ is NOT recommended**

- MOA: dissociates to provide bicarbonate ion (HCO₃⁻) which neutralizes H⁺ ion
  - ↑ blood/urinary pH

- **When should I use it?** Cardiac arrest associated with:
  - Hyperkalemia
  - TCA/phenobarbital overdose
  - Severe metabolic acidosis after adequate ventilation

- **Dose 1 mEq/kg IV/IO**
  - Very irritating to the vein, central administration preferred

*Pharmacother* 2006;26:1703-1729
*Circulation* 2010;122:S729-S767
Cardiac Arrest – Role of Drug Therapy

Calcium – why?

- Ca is a critical ion necessary for muscle contraction and cardiac conduction, enzymatic reactions, platelet aggregation, and receptor activation
- Moderates the effects of K alterations at the cell membrane
- At low calcium concentrations, smooth muscle contraction may not be sufficient to maintain adequate pressures

Pharmacother 2006;26:1703-1729
Cardiac Arrest – Calcium Chloride

- Routine use of Ca is **NOT** recommended

- When should I use it? Cardiac arrest associated with:
  - Hyperkalemia
  - Hypocalcemia
  - Calcium channel blocker overdose

- Dose 1000 mg IV/IO
  - Very irritating to the vein, central administration preferred

1g Ca Chloride $= 13.4$ mEq $\text{Ca}^{2+}$
1g Ca Gluconate $= 4.3$ mEq $\text{Ca}^{2+}$

*Pharmacothen 2006;26:1703-1729*
*Circulation 2010;122:S729-S767*
# PEA/Asystole

Reversible Causes of Arrest: H’s and T’s

<table>
<thead>
<tr>
<th>Cause</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>H</strong> Hypovolemia</td>
<td>Crystalloids/Blood</td>
</tr>
<tr>
<td><strong>H</strong> Hypoxia</td>
<td>Oxygen/intubation</td>
</tr>
<tr>
<td><strong>H</strong> Hydrogen ions</td>
<td>Increase respiratory rate on vent/bicarbonate</td>
</tr>
<tr>
<td>(acidosis)</td>
<td></td>
</tr>
<tr>
<td><strong>H</strong> Hypo-/hyperkalemia</td>
<td>K or Calcium/Sodium Bicarbonate</td>
</tr>
<tr>
<td><strong>H</strong> Hypothermia</td>
<td>Warmth</td>
</tr>
<tr>
<td><strong>H</strong> Hypoglycemia</td>
<td>Dextrose</td>
</tr>
</tbody>
</table>

*Pharmacother 2006;26:1703-1729  
Circulation 2010;122:S729-S767*
# PEA/Asystole

Reversible Causes of Arrest: H’s and T’s

<table>
<thead>
<tr>
<th>Cause</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tension PTX</td>
<td>Needle decompression</td>
</tr>
<tr>
<td>Tamponade, cardiac</td>
<td>Pericardiocentesis</td>
</tr>
<tr>
<td>Toxins</td>
<td>Treat per toxin – e.g. naloxone, sodium bicarbonate</td>
</tr>
<tr>
<td>Thrombosis, PE</td>
<td>Thrombolytics</td>
</tr>
<tr>
<td>Thrombosis, heart</td>
<td>Reperfusion Strategies</td>
</tr>
</tbody>
</table>

*Pharmacotherapy* 2006;26:1703-1729  
*Circulation* 2010;122:S729-S767
ACLS – Alternate Administration Sites

INTRAOSSEOUS

- If IV access cannot be quickly obtained, next preferred type of access
- Fluids/drugs enter venous sinusoids of the medullary cavity, drain into central venous channel
  - Blood through bone marrow is rapid
- Can give virtually any drug and fluids via this route
  - Currently no resuscitation drugs for which IO is considered CI
- Time for drug to reach heart 10 secs - 5 minutes
- Most of the data was conducted during normal perfusion states
- Biggest risk = extravasation

Pharmacother 2007;41:1679-86.
Pharmacother 2006;26:1703-1729
Circulation 2010;122:S729-S767
ENDOTRACHEAL TUBE

- Consider only when IV and IO access is not possible
- Peak cardiac concentrations may be lower with ETT administration as compared to IV or IO
- Time for drug to reach heart ~1-2 minutes
- Drugs should be diluted in 5-10ml fluid to allow adequate delivery
- Dilution with SWFI may improve absorption of epinephrine compared to normal saline
- Animal data suggest only β-agonist effects of epinephrine (due to ↓concentrations) → vasodilation

*Pharmacother 2006;26:1703-1729
Circulation 2010;122:S729-S767*
ACLS – Alternate Administration Sites

ENDOTRACHEAL TUBE

N- naloxone
A- atropine
V- vasopressin/Valium (diazepam)
E- epinephrine
L- lidocaine
ACLS – Drug Administration Pearls

- Appropriate CPR may only provide 25-30% of normal cardiac output → shunting of blood
  - Distribution of drugs to appropriate site may be suboptimal
  - Normal saline 10-20ml after drug facilitates drug distribution
  - CPR should not be interrupted to give drugs

- Time of pharmacologic effect depends on distance from heart (peripheral vs central) → central preferred
  - If peripheral is used: 20 ml flush; raise arm

*Pharmacother* 2006;26:1703-1729
*Circulation* 2010;122:S729-S767
ACLS – Drug Administration Pearls

- After administration of each drug, CPR should be continued to facilitate drug distribution in order to optimize a response
  - Don’t give drug and them shock
- Avoid unnecessary treatment delays
  - 5ml of air poses no threat
- Hyperosmolar therapies
  - Infusion of dextrose, sodium bicarbonate, calcium chloride into a peripheral line should be administered into free flowing fluid to prevent loss of access
- Compatibility Issues
  - Calcium + Sodium bicarbonate = precipitate (Ca Carbonate)
  - Epinephrine + Sodium bicarbonate = incompatible

Pharmacother 2006;26:1703-1729
Circulation 2010;122:S729-S767
Symptomatic Bradycardia

Treatment may involve

- Reversing potential causes (ie. hypoxemia)
- Internal or external pacemakers
- Pharmacotherapy
  - Blocking parasympathetic activity → atropine
  - Stimulating $\beta_1$ activity → catecholamines

- 2010 ACLS guidelines: atropine is the first line for symptomatic bradycardia

Pharmacother 2006;26:1703-1729
Circulation 2010;122:S729-S767
Symptomatic Bradycardia

1. Assess appropriateness for clinical condition. Heart rate typically <50/min if bradycardia.

2. Identify and treat underlying cause
   - Maintain patent airway; assist breathing as necessary
   - Oxygen (if hypoxic)
   - Cardiac monitor to identify rhythm; monitor blood pressure and oximetry
   - IV access
   - 12-Lead ECG if available; don’t delay therapy

3. Persistent bradycardia causing:
   - Hypotension?
   - Acutely altered mental status?
   - Signs of shock?
   - Ischemic chest discomfort?
   - Acute heart failure?

4. Monitor and observe

5. Yes
   - Atropine
     - If atropine ineffective:
       - Transcutaneous pacing
       - Dopamine infusion
       - Epinephrine infusion

6. Consider:
   - Expert consultation
   - Transvenous pacing

Doses/Details
- **Atropine IV Dose:**
  - First dose: 0.5 mg bolus
  - Repeat every 3-5 minutes
  - Maximum: 3 mg
- **Dopamine IV Infusion:**
  - 2-10 mcg/kg per minute
- **Epinephrine IV Infusion:**
  - 2-10 mcg per minute

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Symptomatic Bradycardia – Role of Drug Therapy

Atropine – why?

- Inhibits cholinergic responses that diminish HR and SVR
Symptomatic Bradycardia—Atropine

- Recommended for symptomatic bradycardia
- MOA: blocks acetylcholine at parasympathetic sites
  - ↑ HR through increasing SA and AV nodal conduction velocity
- Dose 0.5 mg every 3-5 minutes (maximum 3 mg)
  - Higher doses may be necessary in organophosphate, carbamate or nerve agent poisoning is suspected
- Doses < 0.5mg and slow administration (>1 min) should be avoided, associated with paroxysmal parasympathetic response, further slowing of HR and exacerbation of bradycardia
- Precautions: patients with ACS, heart transplant
- Can be given via ETT if IV/IO access unavailable
  - 2x the dose
Tachyarrhythmias

Most important questions to ask prior to giving drugs:

1. Is the patient hemodynamically stable?
   1. SBP <90, severe AMS, severe chest pain
   2. If not → forget the drugs → synchronized cardioversion

2. Is the QRS narrow or wide (>0.12sec)?
   1. In general, narrow QRS signifies atrial origin, wide QRS signifies ventricular origin

3. Is the rhythm regular or irregular?
   1. Will make a difference in what you give the patient

Pharmacother 2006;26:1703-1729
Tachyarrhythmias

In general:

- If the QRS is narrow (<0.12secs) regardless of regular or irregular:
  - Can give adenosine
  - Could consider metoprolol if not CI and Afib with RVR

- If the QRS is wide (>0.12sec) and regular:
  - Can consider adenosine → be careful with very high rates

- If the QRS is wide (>0.12sec) and irregular:
  - DO NOT give adenosine, metoprolol, diltiazem
  - Stabilize and get to hospital asap

Pharmacother 2006;26:1703-1729
Tachyarrhythmias

**Adult Tachycardia (With Pulse)**

1. Assess appropriateness for clinical condition. Heart rate typically ≥150/min if tachyarrhythmia.

2. Identify and treat underlying cause
   - Maintain patent airway; assist breathing as necessary
   - Oxygen (if hypoxemic)
   - Cardiac monitor to identify rhythm; monitor blood pressure and oximetry

3. Persistent tachyarrhythmia causing:
   - Hypotension?
   - Acutely altered mental status?
   - Signs of shock?
   - Ischemic chest discomfort?
   - Acute heart failure?

4. Synchronized cardioversion
   - Consider sedation
   - If regular narrow complex, consider adenosine

5. Wide QRS? ≥0.12 second

6. IV access and 12-lead ECG if available
   - Consider adenosine only if regular and monomorphic
   - Consider antiarrhythmic infusion
   - Consider expert consultation

**Doses/Details**

**Synchronized Cardioversion**
Initial recommended doses:
- Narrow regular: 50-100 J
- Narrow irregular: 120-200 J biphasic or 200 J monophasic
- Wide regular: 100 J
- Wide irregular: defibrillation dose (NOT synchronized)

**Adenosine IV Dose:**
First dose: 6 mg rapid IV push; follow with NS flush.
Second dose: 12 mg if required.

**Antiarrhythmic Infusions for Stable Wide-QRS Tachycardia**

**Procainamide IV Dose:**
20-50 mg/min until arrhythmia suppressed, hypotension ensues, QRS duration increases >50%, or maximum dose 17 mg/kg given. Maintenance infusion: 1-4 mg/min. Avoid if prolonged QT or CHF.

**Amiodarone IV Dose:**
First dose: 150 mg over 10 minutes. Repeat as needed if VT recurs. Follow by maintenance infusion of 1 mg/min for first 8 hours.

**Sotalol IV Dose:**
100 mg (1.5 mg/kg) over 5 minutes. Avoid if prolonged QT.

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Figure 4. Tachycardia Algorithm.

Circulation
2010;122:
S729-S767
Tachyarrythmias – Role of Drug Therapy

Adenosine– why?

- Resets the heart
- Can aid in arrhythmia identification
  - Will usually restore PSVT to sinus rhythm
  - Will not convert atrial fibrillation/flutter but transiently slows the ventricular rate to uncover the underlying rhythm
Tachyarrhythmias – Adenosine

- Drug of choice for hemodynamically narrow complex tachycardia (ie. PSVT)
- Can be used safely in some (regular/monomorphic) wide-complex rhythms
- If used for unstable, irregular, or polymorphic VT → may precipitate VF
- MOA: inhibits adenylate cyclase → ↓ Ca current in AV node
  - temporarily inhibits conduction through the SA/AV node
  - Restoring normal sinus rhythm
- If PSVT → adenosine will terminate
- If VT → adenosine will have no effect
Tachyarrythmias – Adenosine

- Dose 6 mg rapid IVP → 12 mg IVP → 12 mg IVP
  - Consider 3 → 6 → 6 if patient has central line or takes carbamazepine/dipyridamole
  - Effect may be diminished in those taking theophylline/caffeine
  - If 30 mg doesn’t work, giving more likely won’t work

- Onset = 15-60 secs

- $t_{1/2} = \sim 10$ secs

- AE: chest pain, flushing, dyspnea, bronchospasm sinus pause (transient)
  - Prolonged asystole may require atropine

*Pharmacother* 2006;26:1703-1729
*Circulation* 2010;122:S729-S767
Tachyarrythmias – Adenosine
Tachyarrhythmias – Adenosine
Tachyarrythmias – Adenosine
Beta Blockers – why?

- Rate control
Tachyarrhythmias – Metoprolol

- May consider in PSVT/Afib/Aflutter after adenosine
- Not many reasons to do this prior to arriving at ED
- Class II antiarrhythmic
- MOA: competitively blocks $\beta_1$ receptors
  - ↓ HR through ↓ conduction/automaticity and ↑ refractoriness
- Dose 2.5-5 mg IV over 2 minutes (max total dose = 15 mg)
- If atrial fibrillation/flutter is due to WPW, use of metoprolol may favor the alternative pathway and lead to ventricular arrhythmias
- CI: 2\textsuperscript{nd}/3\textsuperscript{rd} degree heart block; severe lung disease; hypotension; decompensated heart failure
- Should NOT be used in irregular/polymorphic VT

*Pharmacother* 2006;26:1703-1729
Tachyarrhythmias – Role of Drug Therapy

Antiarrhythmics – why?

- Potential to normalize abnormally depolarizing/conducting myocardial cells
Tachyarrhythmias – Amiodarone

- Initial antiarrhythmic in hemodynamically stable wide-complex tachycardia (VT); be careful Afib/flutter
- Class III antiarrhythmic
- MOA: Na, K, Ca channel antagonist aka “King of Dirty”
  - Prolongs action potential and refractory period in myocardial tissue
  - Decreases AV conduction and sinus node function
- Dose 150 mg IV diluted in 100ml D5W administered over 10 mins (in hemodynamically stable patient), q10mins PRN
- Undiluted amiodarone can cause:
  - Bradycardia
  - Hypotension (polysorbate 80) – rate related
  - Phlebitis
- Recommendation: to dilute and give slowly  

Pharmacother 2006;26:1703-1729
Acute Coronary Syndrome (ACS)

Remember…

M- morphine
O- oxygen
N- nitroglycerin
A- aspirin

But not in this order…
Aspirin—why?

- Diminishes platelet aggregation
- Four randomized trials have each demonstrated that, compared with placebo, aspirin reduces the risk of death or MI by more than 50% for patients presenting with UA/NSTEMI → MORTALITY DATA!!!
ACS – Aspirin

- Aside from ABC’s, the most important drug you can give a patient with ACS
- MOA: blocks the synthesis of thromboxane A2 by irreversibly inhibiting cyclooxygenase 1
  - ↓ platelet aggregation
- Dose 324 mg (4x 81mg chew) asap
- CI: documented aspirin allergy (eg, asthma or anaphylaxis), active bleeding, or platelet disorder

Mayo Clin Proc 2009;84:917-938
ACS – Role of Drug Therapy

Nitroglycerin – why?

- Vasodilator that reduces myocardial oxygen demand by decreasing ventricular preload via venodilation
- Enhances myocardial oxygen delivery by dilating large coronary arteries and improving collateral flow to ischemic areas
- Intended primarily to relieve ischemic pain for patients with STEMI
- NO MORTALITY DATA!!!

Mayo Clin Proc 2009;84:917-938
Drug of choice for hemodynamically stable STEMI/NSTEMI patients to relieve pain associated with ACS

MOA: relaxation of smooth muscle, with a more prominent effect on the veins; dilates coronary arteries
- ↓ cardiac oxygen demand via ↓ preload
- Improves collateral flow to ischemic regions

Dose 0.3 mg SL every 5 minutes x 3 doses

CI: hypotension (SBP <90) or the use of sildenafil/vardenafil w/n 24h or tadalafil w/n 48h; HR <50; right ventricular infarction

*Mayo Clin Proc* 2009;84:917-938
Morphine – why?

- A potent analgesic and anxiolytic
- Hemodynamic effects may be beneficial
- NO MORTALITY DATA!!!
ACS – Morphine

- Morphine is recommended when ischemia-related symptoms are unrelieved after 3 doses of nitroglycerin or when such symptoms recur during treatment.
- MOA: opiate agonist.
- Dose 1-5mg IV q5-30 mins PRN with monitoring of BP, RR.
- AE: hypotension, respiratory depression.

Mayo Clin Proc 2009;84:917-938
The Cardiac Drug Box

Christina Candeloro, PharmD, BCPS
Clinical Pharmacy Specialist, Emergency Medicine, VCUHS
Assistant Clinical Professor, VCU School of Pharmacy
Richmond, VA