How Sweet It Is!

(Dextrose in Resuscitation?!?)

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Dextrose for OOHCA

Case Study
27 y.o. male unresponsive after a night of partying with friends. Determined to be in arrest on dispatcher assessment (“lips turning blue”).

CCO-CPR instructions given.

CFR arrived on scene first and applied their AED.
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Prehospital Care:
BLS, ALS, and EMS Officer arrived on scene within six minutes.

Treated under PEA / asystole protocol with IV, ETT, vasopressin, epinephrine, atropine → no response.

Narcan administered in light of reported drug abuse.

Patient remained in asystole.

Transport or terminate resuscitative efforts??

Transport (due to age).
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Transported to Bellevue Hospital.

Asystole on arrival.

Initial evaluation included a blood glucose analysis....

..... <35mg/dL.

Patient has been in arrest for 35 minutes.
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ED History
Patient given D50.

ROSC achieved.

Discharged alive.
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Then comes the email...

(paraphrased)

“They gave narcan, but not D50??? Don’t you consider hypoglycemia as a cause for an arrest???”

What to do with this email…
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Email about hypoglycemi a case
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But consider the source...

Call Your Poison Control Center—You Might Save A Life

New York City Poison Control Center
Call 1-800-222-1222
24 hours a day, 7 days a week

POISON Help 1-800-222-1222
212-POISONS (212-764-7667)
212-689-9014 TDD
Visit our website at nyc.gov/health
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ADVANCED EMERGENCY MEDICAL TECHNICIAN (PARAMEDIC) PROTOCOLS

VENTRICULAR FIBRILLATION/PULSELESS VENTRICULAR TACHYCARDIA

1. Continue CPR with minimal interruption.
   NOTE: If arrest is witnessed by EMS, perform CPR until defibrillator is attached.
   In arrests not witnessed by EMS, perform two (2) minutes of CPR prior to defibrillator use.

2. Defibrillate using 100 joules or equivalent biphasic.
   NOTE: If patient has a permanent pacemaker in place, position the paddles or automated defibrillator pads at least one (1) inch away from the pacemaker device.

3. Continue CPR. If after two minutes of additional CPR there is no change in the rhythm, defibrillate a 2nd time as previously stated.

4. Continue CPR. If after two minutes of additional CPR there is no change in the rhythm, defibrillate a 3rd time as previously stated.

5. Perform Endotracheal intubation.

6. If, after every two minute interval of additional CPR, there is no change in the rhythm, defibrillate as previously stated.

7. Begin an IV/IO infusion of Normal saline (0.9% NS) to keep vein open, or a Saline Lock.

8. Administer Vasopressin 40 unit IV/IO/Saline Lock Bolus, single dose.

9. If there is no change in the rhythm, administer Amiodarone 200mg, diluted up to a total of 20mL of D5W, IV/IO/Saline Lock Bolus.

10. If there is no change in the rhythm within 3 – 5 minutes after the administration of vasopressin, administer Epinephrine 1 mg (10 ml of a 1:10,000 solution). IV/IO/Saline Lock Bolus, every 3 – 5 minutes.

11. If there is insufficient improvement in hemodynamic status, contact Medical Control for implementation of one or more of the following MEDICAL CONTROL OPTIONS:

MEDICAL CONTROL OPTIONS:

OPTION A: If ventricular fibrillation or pulseless ventricular tachycardia recurs, a repeat dose of 150 mg Amiodarone diluted up to a total of 10 mL D5W, IV/IO/Saline Lock Bolus may be given.

OPTION B: Administer Sodium Bicarbonate 44-53 mEq IV/IO/Saline Lock Bolus. Repeat doses of Sodium Bicarbonate 44 mEq, IV/IO/Saline Lock bolus, may be given every 10 minutes.

OPTION C: Administer Magnesium Sulfate 2 gm, IV/IO/Saline Lock Bolus, diluted in 10 mL of Normal Saline (0.9% NS), over 2 minutes.
Part 7.2: Management of Cardiac Arrest

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Part 10.1: Life-Threatening Electrolyte Abnormalities

Electrolyte abnormalities are commonly associated with cardiovascular emergencies. These abnormalities may cause or contribute to cardiac arrest and may hinder resuscitative efforts. In some cases therapy for life-threatening electrolyte disorders should be initiated before laboratory results become available.

Potassium (K⁺)
The magnitude of the potassium gradient across cell membranes determines excitability of nerve and muscle cells, including the myocardium. Rapid or significant changes in the serum potassium concentration can have life-threatening consequences.

Evaluation of serum potassium must consider the effects of changes in serum pH. When serum pH falls, serum potassium rises because potassium shifts from the cellular to the vascular space. When serum pH rises, serum potassium falls because potassium shifts from the vascular space into the cells. Effects of pH changes on serum potassium should be anticipated during therapy for hyperkalemia or hypokalemia and during any therapy that may cause changes in serum pH (eg, treatment of diabetic ketoacidosis).

Hyperkalemia
Although hyperkalemia is defined as a serum potassium concentration >5 mEq/L, it is moderate (6 to 7 mEq/L) and severe (>7 mEq/L) hyperkalemia that are life-threatening and require immediate therapy. Hyperkalemia is most commonly seen in patients with end-stage renal disease. Other causes are listed in the Table. Many medications can contribute to the development of hyperkalemia. Identification of potential causes of hyperkalemia will contribute to rapid identification and treatment.¹⁻³

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**TABLE: Common Causes of Hyperkalemia**
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Advanced Emergency Medical Technician (Paramedic) Protocols

Ventricular Fibrillation/Pulseless Ventricular Tachycardia

1. Continue CPR with minimal interruption.

   NOTE IN ARRESTS WITNESSED BY EMS, PERFORM CPR UNTIL DEFIBRILLATOR IS ATTACHED.
   IN ARRESTS NOT WITNESSED BY EMS, PERFORM TWO (2) MINUTES OF CPR PRIOR TO DEFIBRILLATOR USE.

2. Defibrillate using 360 joules, or equivalent biphasic.

   NOTE IF PATIENT HAS A PERMANENT PACEMAKER IN PLACE, POSITION THE PADDLES OR AUTOMATED DEFIBRILLATOR PADS AT LEAST ONE (1) INCH AWAY FROM THE PACEMAKER DEVICE.

3. Continue CPR. If after two minutes of additional CPR if there is no change in the rhythm, Defibrillate a 2nd time as previously stated.

4. Continue CPR. If after two minutes of additional CPR if there is no change in the rhythm, Defibrillate a 3rd time as previously stated.

5. Perform Ennobrachial Intubation.

6. If, after every two minute interval of additional CPR, there is no change in the rhythm, Defibrillate as previously stated.

   Begin an IV/IO infusion of Normal Saline (0.9% NS) to keep ven open, or a Saline Lock.

   Administer Vasopressin 40 unit IV/IO/Saline Lock Bolus, single dose.

8. If there is no change in the rhythm, administer Amiodarone 300mg, diluted up to a total of 20mL of D5W/IV/IO/Saline Lock bolus.

9. If there is no change in the rhythm within 3 – 5 minutes after the administration of Vasopressin, administer Epinephrine 1 mg (10 mL of a 1:10,000 solution), IV/IO/Saline Lock bolus, every 3 – 5 minutes.

10. If, after two minutes of additional CPR if there is no change in the rhythm, contact Medical Control for implementation of one or more of the following MEDICAL CONTROL OPTIONS:

   - **OPTION A:** If Ventricular Fibrillation or Pulseless Ventricular Tachycardia recurs, a repeat dose of 150 mg Amiodarone diluted up to a total of 10 mL D5W/IV/IO/Saline Lock Bolus may be given.

   - **OPTION B:** Administer Sodium Bicarbonate 44.88 mEq IV/IO/Saline Lock bolus. Repeat doses of Sodium Bicarbonate 44 mEq, IV/IO/Saline Lock bolus, may be given every 10 minutes.

   - **OPTION C:** Administer Magnesium Sulfate 2 gm IV/IO/Saline Lock Bolus, diluted in 10 mL of Normal Saline (0.9% NS), over 2 minutes.

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Prehospital Treatment Protocols

Advanced Life Support (Paramedic) Protocols

July 2009
Version 070100a
Dextrose for OOHCA

**New York-isms**

“Syncopized” – Make-believe verb meant to describe the act of experiencing syncope / a syncopal episode

“The Bus” – The ambulance

“The Ultimate AMS” – Cardiac arrest
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NYC Cardiac Arrest Database (2007 – present)
  - 20,319 OOHCA Cases
  - excluded 725 traumatic arrests
  - excluded 3,319; no ALS meds given
  - 16,305 nontraumatic arrests who received ALS medications
  - perhaps there will be a few D50 administrations
  - OLMC contacts
  - people practicing “old” NYC EMS
## Dextrose for OOHCA

<table>
<thead>
<tr>
<th></th>
<th>No Dextrose</th>
<th>Dextrose</th>
<th>p</th>
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</thead>
<tbody>
<tr>
<td>All Cases</td>
<td>12,575</td>
<td>3,730</td>
<td>-----</td>
</tr>
<tr>
<td>ROSC</td>
<td>25.7%</td>
<td>28.8%</td>
<td>0.0002</td>
</tr>
<tr>
<td>Sustained ROSC</td>
<td>19.5%</td>
<td>24.6%</td>
<td>&lt;0.0001</td>
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<tr>
<td>VF ROSC</td>
<td>34.7%</td>
<td>34.5%</td>
<td>1</td>
</tr>
<tr>
<td>Sustained VF ROSC</td>
<td>25.4%</td>
<td>24.2%</td>
<td>0.7496</td>
</tr>
<tr>
<td>Non-VF ROSC</td>
<td>22.2%</td>
<td>26.9%</td>
<td>&lt;0.0001</td>
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<tr>
<td>Sustained Non-VF ROSC</td>
<td>17.0%</td>
<td>19.6%</td>
<td>0.0132</td>
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<tr>
<td>Non-Diabetics</td>
<td>19.2%</td>
<td>27.6%</td>
<td>0.0002</td>
</tr>
<tr>
<td>Sustained Non-Diabetics ROSC</td>
<td>11.5%</td>
<td>19.0%</td>
<td>0.0006</td>
</tr>
</tbody>
</table>
Dextrose for OOHCA

- Dextrose: 18.61%
- No Dextrose: 17.99%
Dextrose for OOHCA

Makes physiologic sense

- myocardium and CNS are glucose-dependent organs
- myocardium reverts to FFA metabolism during ischemia
- glucose resistance increases during ischemia
- epinephrine-induced glycogolysis / gluconeogenesis may not be sufficient
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Revision/Update of REMAC Prehospital Treatment & Transport Protocols

503-B

PULSELESS ELECTRICAL ACTIVITY (PEA)/ASYSTOLE

NOTE: CONSIDER THE POSSIBILITY OF CONDITIONS MASQUERADING AS PEA/ASYSTOLE WHICH REQUIRE IMMEDIATE TREATMENT.

1. Continue CPR with minimal interruption.
2. If a tension pneumothorax is suspected, perform Needle Decompression. (See Appendix O.)
3. Perform Endotracheal Intubation.
4. Begin an IV/IO/ infusion of Normal Saline (0.9% NS) to keep vein open, or a Saline Lock.
5. Administer Vasopressin 40 unit IV/IO/Saline Lock Bolus, single dose.
6. Administer Dextrose 25 gm (50 ml of a 50% solution), IV/Saline Lock bolus.
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STILL ASYSTOLE? Inject a SNICKERS™
But is there a better answer?
Dextrose for OOHCA
Newest Drug for OOHCA

PEPE-NEPHRINE

Injection, USP
1:10,000
1 mg (0.1 mg/mL)

PROTECT FROM LIGHT
Thank you.