

# How Sweet It Is!! (Dextrose in Resuscitation?!?)



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EMS Deputy Medical Director  
New York City Fire Department  
and

Department of Emergency Medicine  
St. Vincent's Hospital - Manhattan



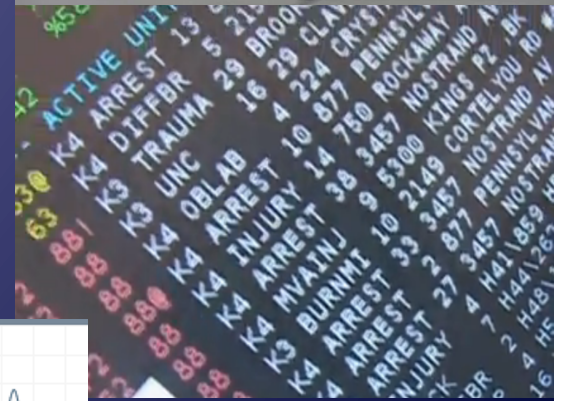
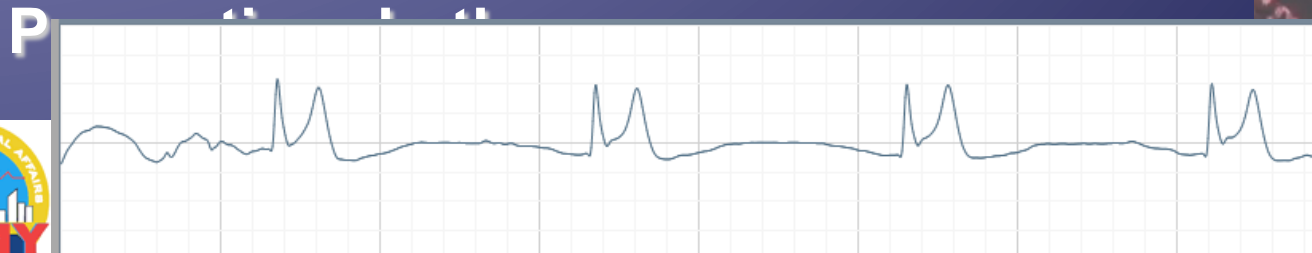
# Dextrose for OOHCA

# 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)

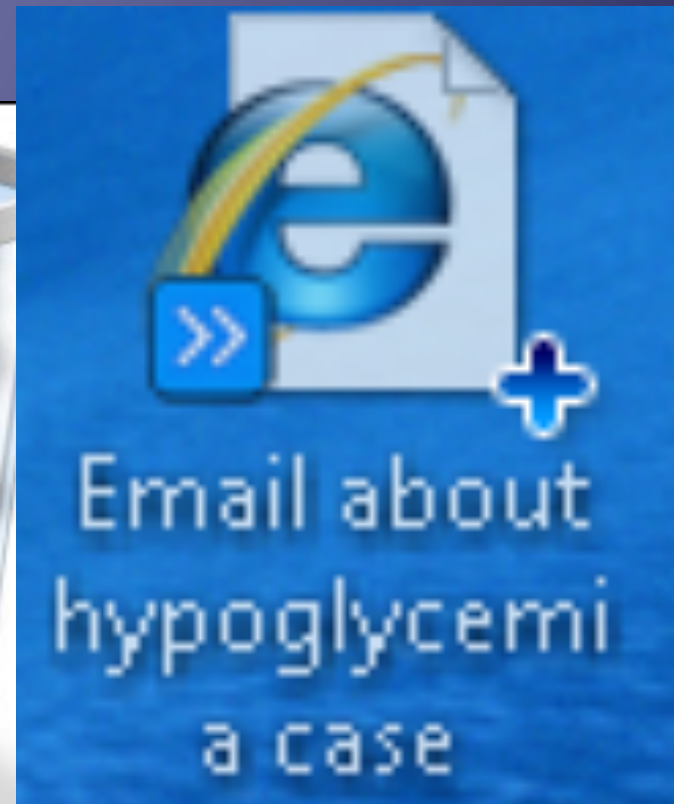
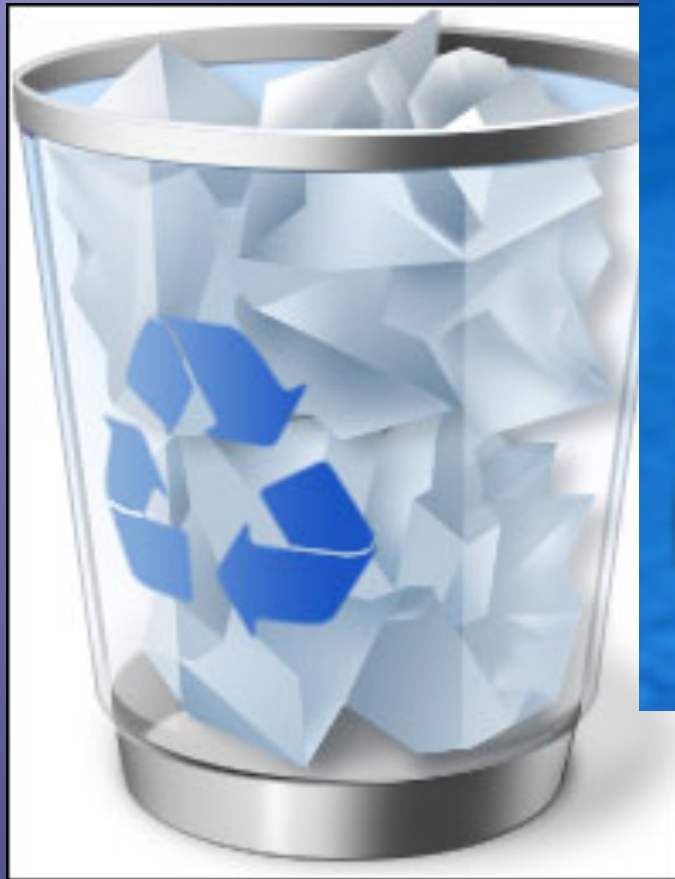
“They gave narcan, but not D50??? Don’t you consider hypoglycemia as a cause for an arrest???”

What to do with this email...





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# Dextrose for OOHCA



But consider the source...



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# Dextrose for OOHCA

THE REGIONAL EMERGENCY MEDICAL ADVISORY COMMITTEE  
NEW YORK CITY



PREHOSPITAL TREATMENT PROTOCOLS

## ADVANCED LIFE SUPPORT (PARAMEDIC) PROTOCOLS

July 2009  
Version 070109a

THE REGIONAL EMERGENCY MEDICAL SERVICES COUNCIL OF NEW YORK CITY

### ADVANCED EMERGENCY MEDICAL TECHNICIAN (PARAMEDIC) PROTOCOLS

503-A

#### 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.

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3. Continue CPR. If after two minutes of additional CPR if there is no change in the rhythm, Defibrillate a 2<sup>nd</sup> time as previously stated.
4. Continue CPR. If after two minutes of additional CPR if there is no change in the rhythm, Defibrillate a 3<sup>rd</sup> 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 300mg, diluted up to a total of 20mL of D<sub>5</sub>W, 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 D<sub>5</sub>W, 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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# Dextrose for OOHCA

## Part 10.1: Life-Threatening Electrolyte Abnormalities

**E**lectrolyte 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.<sup>1-3</sup>

inflammatory agents). Additional treatment is based on the severity of the hyperkalemia and its clinical consequences. The following sequences list the treatments for hyperkalemia in order of priority.

For *mild* elevation (5 to 6 mEq/L), remove potassium from the body with

1. Diuretics: furosemide 40 to 80 mg IV
2. Resins: Kayexalate 15 to 30 g in 50 to 100 mL of 20% sorbitol either orally or by retention enema

For *moderate* elevation (6 to 7 mEq/L), shift potassium intracellularly with

1. Glucose plus insulin: mix 25 g (50 mL of D<sub>50</sub>) glucose and 10 U regular insulin and give IV over 15 to 30 minutes
2. Sodium bicarbonate: 50 mEq IV over 5 minutes (sodium bicarbonate alone is less effective than glucose plus insulin or nebulized albuterol, particularly for treatment of patients with renal failure; it is best used in conjunction with these medications<sup>4,5</sup>)
3. Nebulized albuterol: 10 to 20 mg nebulized over 15 minutes

For *severe* elevation ( $>7$  mEq/L with toxic ECG changes), you need to shift potassium into the cells and eliminate potassium from the body. Therapies that shift potassium will act rapidly but they are temporary; if the serum potassium rebounds you may need to repeat those therapies. In order of priority, treatment includes the following:

TABLE. Common Causes of Hyperkalemia



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# 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



# Dextrose for OOHCA

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# Dextrose for OOHCA

## 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



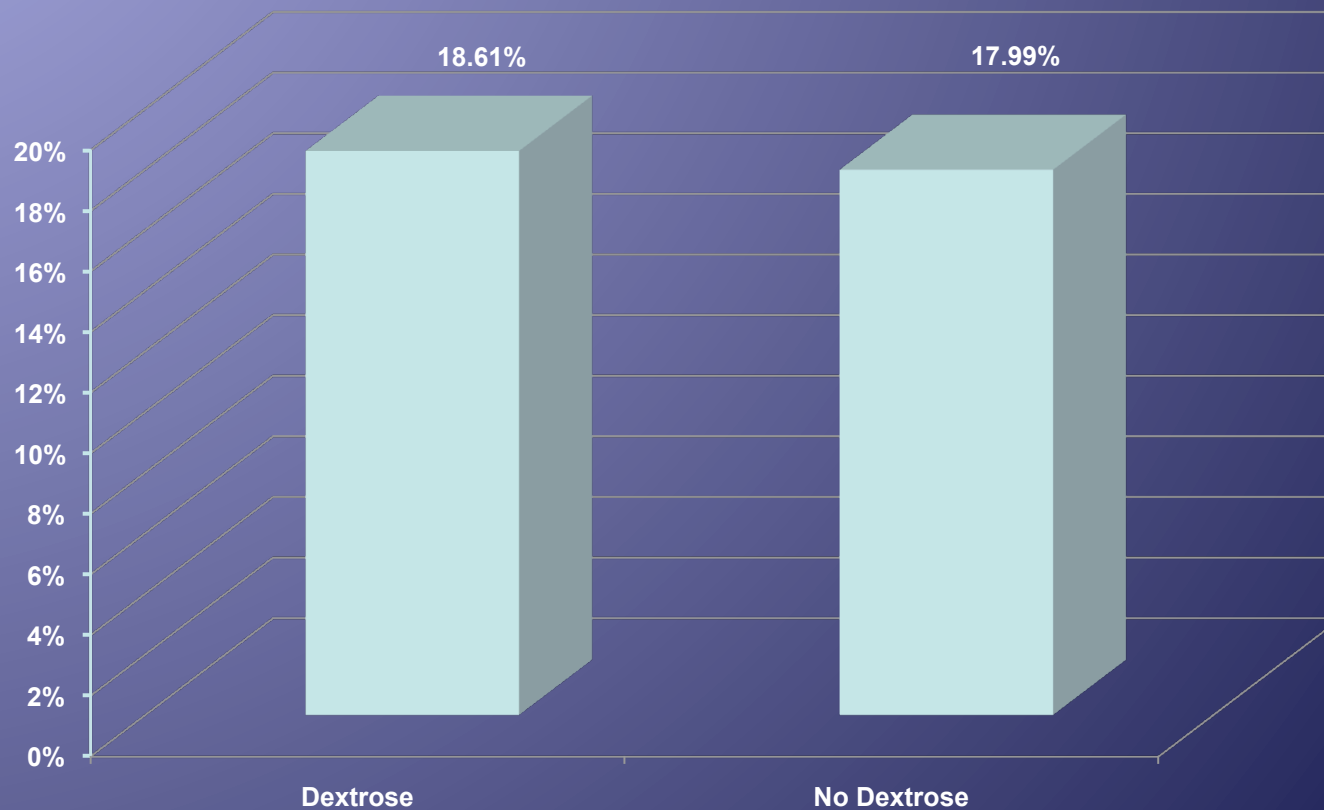


# Dextrose for OOHCA

	No Dextrose	Dextrose	p
All Cases	12,575	3,730	-----



# Dextrose for OOHCA



# 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





# Dextrose for OOHCA

## THE REGIONAL EMERGENCY MEDICAL SERVICES COUNCIL OF NEW YORK CITY, INC.

*Revision/Update of REMAC Prehospital Treatment & Transport Protocols*

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503-B

### PULSELESS ELECTRICAL ACTIVITY (PEA)/ASYSTOLE

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

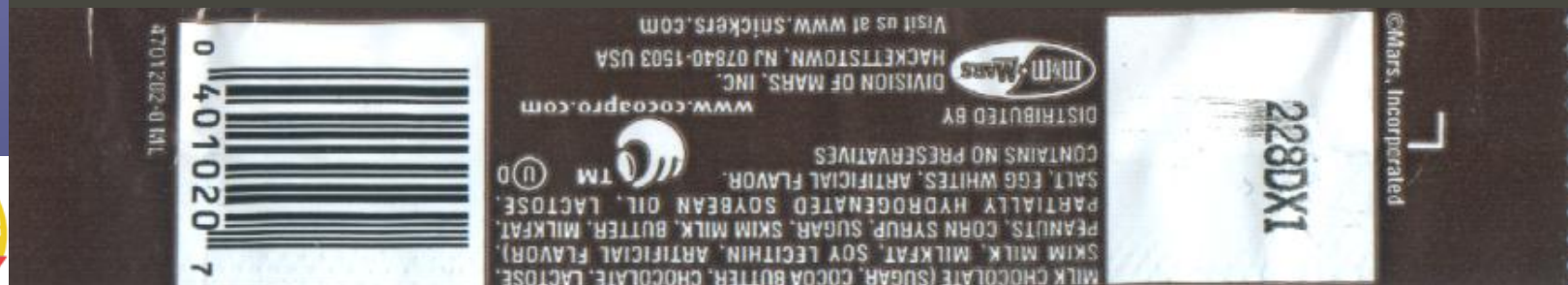
1. Continue CPR with minimal interruption.
2. If a tension pneumothorax is suspected, perform Needle Decompression. (See Appendix O.)
3. Perform Endotracheal Intubation.
4. Begin an IV/IO/ infusion of Normal Saline (0.9% NS) to keep vein open, or a Saline Lock.
5. Administer Vasopressin 40 unit IV/IO/Saline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



# Dextrose for OOHCA



**STILL ASYSTOLE? Inject a SNICKERS™**



**But is there a better answer?**



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DOI: 10.1161/CIRCULATIONAHA.105.166557  
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 72514  
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Done

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# Dextrose for OOHCA

The image is a screenshot of a web browser window. The address bar shows a URL starting with 'http://www.circulationaha.org'. The page title is 'Circulation'. The main content area displays a PDF document titled 'Part 7.2: Management of Cardiac Arrest'. A search overlay from Adobe Reader is visible, indicating that the search has finished and found 27 matches. The overlay includes an 'OK' button. The background shows the journal's masthead and navigation links.

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AHA Statements and Guidelines

## Part 7.2: Management of Cardiac Arrest

Four rhythms produce pulseless cardiac arrest: ventricular fibrillation (VF), rapid ventricular tachycardia (VT), pulseless electrical activity (PEA), and asystole. Survival from these arrest rhythms requires both basic life support (BLS) and advanced cardiovascular life support (ACLS).

The foundation of ACLS care is good BLS care, beginning with prompt high-quality bystander CPR and, for VF/pulseless VT, attempted defibrillation with a defibrillator. For victims of witnessed cardiac arrest, early defibrillation is critical for survival to hospital. ACLS therapies, such as pharmacologic support, are shown to increase rate of survival. This section details the general approach to ACLS and provides an overview of the ACLS Algorithms.

### Access for Medical Professionals

During cardiac arrest, BLS is of primary importance, and ACLS is of secondary importance. Few drugs are supported by strong evidence, and attempting defibrillation without ACLS is dangerous. Intravenous (IV) access, central venous access (CVA), and advanced airway.

### Central Versus Peripheral Access

Central line access is the preferred route for ACLS attempts. If IV access is not available, IO access should be used. In adults, a large peripheral vein (e.g., antecubital) should be used. In children, a large peripheral vein (e.g., femoral) should be used. In infants, a large peripheral vein (e.g., umbilical) should be used. In all cases, IO access should be used if IV access is not available. IO access is not recommended for long-term use. IO access is not recommended for the administration of blood products. IO access is not recommended for the administration of contrast media. IO access is not recommended for the administration of chemotherapy. IO access is not recommended for the administration of radioactive contrast media. IO access is not recommended for the administration of radioactive contrast media.

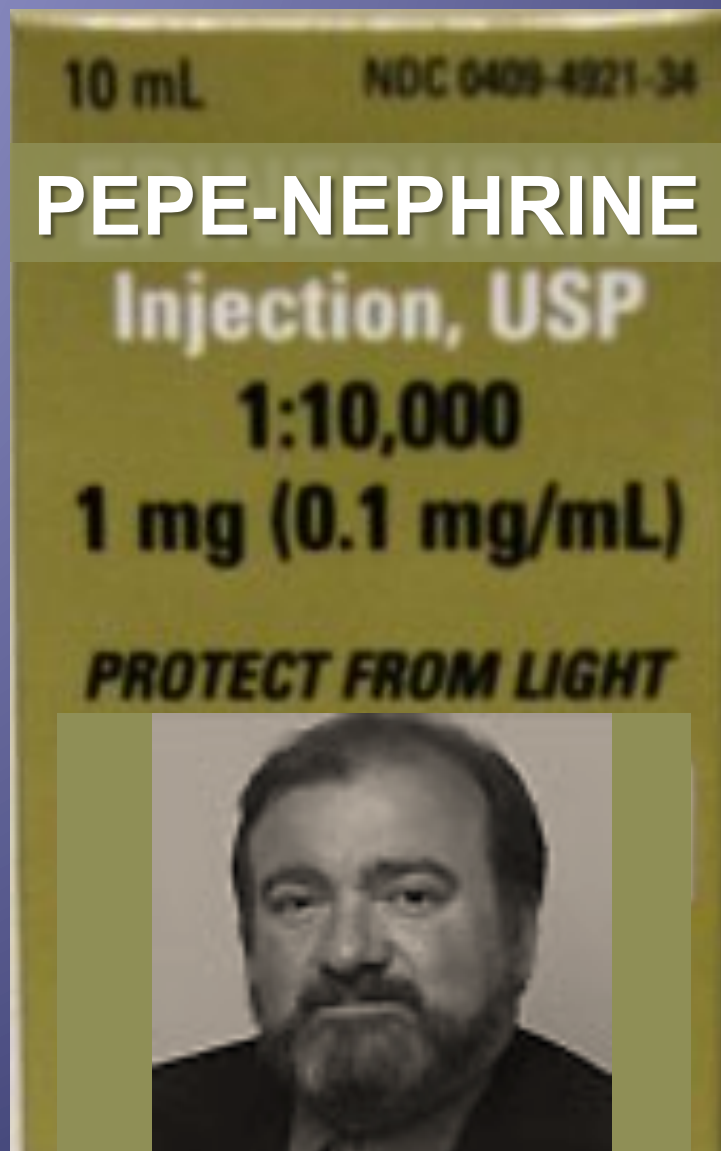
### Arrest Rhythms

The management of pulseless arrest is highlighted in the ACLS Pulseless Arrest Algorithm (Figure). Box numbers in the text refer to the numbered boxes in the algorithm.

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# Newest Drug for OOHCA





Thank you.