



## Episode 199

### Chapter 8 – Adult Resuscitation

**NOTE: CONTENT CONTAINED IN THIS DOCUMENT IS TAKEN FROM ROSEN'S EMERGENCY MEDICINE 9th Ed.**

*Italicized text is quoted directly from Rosen's.*

#### Key Concepts:

1. *CPR quality is critical to successful resuscitation from cardiac arrest. Important benchmarks of quality CPR include compression rate between 100 and 120 compressions/min, compression depth of 5 to 6 cm, chest compression fraction of 80% or more, full chest recoil, and ventilation rate of 10 breaths/min.*
2. *Restoration of adequate cardiac function is the defining factor of ROSC. Restoration of good neurologic function is the defining factor of successful resuscitation.*
3. *Resuscitation of a cardiac arrest victim does not end with ROSC. Rapid diagnosis and proper management of the pathologic conditions that precipitated and resulted from the arrest, as well as goal-directed post-cardiac arrest care, can improve outcome.*
4. *Immediate PCI is indicated in patients with demonstrated ST segment elevation MI following ROSC without regard to neurological status.*
5. *Hypothermic targeted temperature management (32°–36° C for 24 hours) is the first and only post-ROSC intervention that has been shown to improve survival and functional outcome of comatose cardiac arrest survivors.*

#### Core Questions:

1. Describe your history and physical exam in the patient being actively resuscitated.
2. Discuss the process of deterioration to cardiac arrest with respiratory failure and cardiac obstruction.
3. List 6 aspects of optimal CPR.
4. What medications have been shown to improve outcomes in cardiac arrest?
5. List 8 differential diagnoses for PEA arrest (See Table 8.4)
6. What is electromechanical dissociation (EMD) and how does it differ from pseudo electromechanical dissociation (pseudo EMD)?
7. What is echo-guided life support (EGLS) and how is it used?
8. What are your targets during CPR for the following metrics? (See Table 8.3)
  - a. Carotid or femoral pulse



- b. CPP
- c. Arterial relaxation (diastolic) pressure
- d. PETCO<sub>2</sub>
- e. SCVO<sub>2</sub>

### **Wisecracks:**

1. What is cough CPR and when should it be used?
2. What is the only antidysrhythmic shown to improve rates of VF conversion to a perfusing rhythm?
3. What is the minimum coronary perfusion pressure (CPP) is needed to achieve return of spontaneous circulation (ROSC)?
4. What is the triad of cardiac arrest?

### **Rosen's in Perspective**

Alright guys, we are back at it. As practitioners of emergency medicine, we are expected to be masters of the resuscitation game. As such, we must go about taking it upon ourselves to ensure our life-saving interventions are exactly that... life saving. Our podcast today will go about giving you information that will better inform your resuscitative efforts and will better help you to recall physiologic endpoints to guide further management of your sickest patients.

It is important to note here that we will not be reviewing ACLS algorithms in our podcast today. We ask that you take it upon yourself to review those to compliment your learning. Additionally, we want to stress the importance of using this podcast in conjunction with textbook readings and the flashcard set available on CanadiEM to enhance your learning. It is like Owen and I always say - spaced repetition is key.

### **Core Questions:**

**[1] Describe your history and physical exam in the patient being actively resuscitated.**

History:

- Exact time of arrest
- Witnessed vs unwitnessed arrest
- Bystander CPR
- CPR duration – crucial
- ECG rhythm



- Prehospital interventions
- past medical/psychiatric history
- drug ingestion or environmental exposures? do we need to give an antidote?
- Allergies

## Physical Exam

### Head to toe:

- Primary Survey ABCDE ...
- airway secretions and ease of ventilation abdominal exam
- Consideration for rectal exam (r/o massive GI bleed)

TABLE 8.2 Physical Examination Findings<sup>a</sup>

PHYSICAL EXAMINATION	ABNORMALITIES	POTENTIAL CAUSES
General	Pallor	Hemorrhage
	Cold	Hypothermia
Airway	Secretions, vomitus, or blood	Aspiration
		Airway obstruction
	Resistance to positive-pressure ventilation	Tension pneumothorax Airway obstruction
		Bronchospasm
Neck	Jugular venous distention	Tension pneumothorax
		Cardiac tamponade
		Pulmonary embolus



	Tracheal deviation	Tension pneumothorax
Chest	Median sternotomy scar	Underlying cardiac disease
Lungs	Unilateral breath sounds	Tension pneumothorax
		Right mainstem intubation
		Aspiration
	Distant or no breath sounds or no chest expansion	Esophageal intubation Airway obstruction
		Severe bronchospasm
	Wheezing	Aspiration
		Bronchospasm
		Pulmonary edema
	Rales	Aspiration
		Pulmonary edema
		Pneumonia
Heart	Diminished heart tones	Hypovolemia
		Cardiac tamponade
		Tension pneumothorax
		Pulmonary embolus



Abdomen	Distended and dull	Ruptured abdominal aortic aneurysm or ruptured ectopic pregnancy
	Distended, tympanitic	Esophageal intubation
	Gastric insufflation	
Rectal	Blood, melena	Gastrointestinal hemorrhage
Extremities	Asymmetrical pulses	Aortic dissection
	Arteriovenous shunt or fistula	Hyperkalemia
Skin	Needle tracks or abscesses	Intravenous drug abuse
	Burns	Smoke inhalation
		Electrocution

**[2] Discuss the process of deterioration to cardiac arrest with respiratory failure, cardiac obstruction, and hypovolemia.**

**Respiratory Failure:**

Primary respiratory failure leads to hypertension and tachycardia initially, followed by hypotension and bradycardia, progressing to PEA, VF, or asystole.

**Cardiac Obstruction**

Circulatory obstruction (eg, tension pneumothorax, pericardial tamponade) and hypovolemia lead to tachycardia and hypotension initially, progressing through bradycardia and then to PEA, but may deteriorate to VF or asystole as well.



### [3] List 6 aspects of optimal CPR.

1. Place two hands over lower half of sternum
2. Compression rate (100–120 compressions/min)
3. Compression depth (5–6 cm)
4. Chest compression fraction  $\geq 80\%$  (CPR performed 80 out of every 100 seconds) or limit interruptions to less than 10 seconds
5. Full chest recoil
6. Proper ventilation rate (10 breaths ventilations/min)

**Throwing back to original episode: P's of high quality CPR: placement, pace, pressure, pauses, and puffs (don't over ventilate)**

### [4] What medications have been shown to improve outcomes in cardiac arrest?

#### From the book:

*“Pharmacologic therapy during CPR improves the proportion of patients who achieve ROSC.<sup>17</sup> However, there is yet to be a randomized prospective placebo-controlled clinical trial that has been adequately powered to determine if pharmacologic therapy during CPR improves long-term survival or neurologic outcome.”*

That trial has since been done - PARAMEDIC-2 showed an increased likelihood of ROSC in patients who received epi compared to those that didn't. However, there was no increase in 30 day survival or survival to hospital discharge with favourable neurologic outcome. (Rebel EM Summary [here](#)).

Also in Rosen's

*“For refractory VF or pVT, antidysrhythmics can be administered up to their maximum loading dose. Amiodarone (300 mg IV) is the only antidysrhythmic agent that has been shown to improve the rate of VF conversion to a perfusing rhythm.”*

However, according to the [2018 AHA Focused Update on ACLS](#), both amiodarone and lidocaine can be considered to treat shock-refractory VF or VT.

### [5] List 8 differential diagnoses for PEA arrest (See Table 8.4)

Alright, so let's set the scene. You are working a nice weekend afternoon shift - things have been fairly quiet thus far. As soon as you go to take a sip of your coffee, EMS rolls in the door



with a patient in PEA. They have gone through the ACLS algorithm twice already, and the patient remains in that rhythm. WHAT ARE YOU GOING TO DO?

Don't sweat it, we got you. When considering the differential diagnosis for PEA arrest, it is important to have a concise manner to conceptualize and address the potential causes of said arrhythmia. Luckily, Rosen's presents a concise table that will allow you to work through the major causes of PEA arrest. Of note, we have adapted the list to include both hypo- and hyperkalemia, and both PE and MI to mirror the causes noted in the ACLS algorithm.

*The following table has been adapted from Table 8.4 in Rosen's Emergency Medicine, 9th Edition.*

CAUSE	DIAGNOSIS	PALLIATIVE THERAPY	DEFINITIVE THERAPY
<b>Hypovolemia</b>	Response to volume infusion	Volume infusion	Hemostasis if hemorrhage
<b>Hypoxia</b>	Response to oxygenation	Oxygenation, assisted ventilation	Treat underlying cause
<b>Hypothermia</b>	Rectal temperature		Warm peritoneal or thoracic lavage, venoarterial ECMO
<b>Hyperkalemia</b>	History of renal failure or elevated serum potassium level	Calcium chloride, insulin and glucose, sodium bicarbonate	Hemodialysis
<b>Hypokalemia</b>	Serum Potassium, U waves/prolonged QTc on ECG, history of poor intake or losses		Potassium supplementation
<b>Acidosis</b>	Arterial blood gas	Hyperventilation, sodium bicarbonate	Treat underlying cause
<b>Cardiac Tamponade</b>	Echocardiogram; jugular venous	Pericardiocentesis	Thoracotomy and pericardiotomy



	distention		
<b>Toxicity</b>	History of drug ingestion	Drug-specific	Drug-specific
<b>Tension Pneumothorax</b>	Asymmetric breath sounds, tracheal deviation	Needle thoracostomy	Tube thoracostomy
<b>Thrombus Formation - PE</b>	Risk factors or evidence of deep venous thrombosis	Venoarterial ECMO	Lytic therapy, pulmonary embolectomy
<b>Thrombus Formation - MI</b>	STEMI, Heart Block, or VF arrest	DAPT +/- lytic depending on availability of cath lab	Angiography and revascularization

**[6] What is electromechanical dissociation (EMD) and how does it differ from pseudo electromechanical dissociation (pseudo EMD)?**

**True EMD**

- Cause: abnormal automaticity and conduction. Associated with global myocardial depression and acidosis 2ndary to ischemia or hypoxia
- Rhythm: bradycardia with wide QRS complex
- This typically occurs after defib following prolonged VF.
- Associated with: hyperkalemia, hypothermia, drug OD

**Pseudo EMD**

- The step before true EMD...
- Has all the same causes as true EMD. Additional causes of pseudo EMD include papillary mm rupture and myocardial wall rupture (ventricle contracting but forward flow severely reduced), hypovolemia, tension PTX, tamponade, massive PE.
- Pseudo EMD of extracardiac origin is typically **narrow and tachycardic**, progressing to bradycardia and wide QRS

**[7] What is echo-guided life support (EGLS) and how is it used?**

**5 clinical questions to be applied to patient in shock/extremis**

At each stage - consider associated diagnoses and treatments





- 1. Is there PTX**
  - a. US: lung slide, lung point
  - b. Rx: needle decompression/finger thoracostomy, tube thoracostomy
- 2. Is tamponade present?**
  - a. US: pericardial effusion, fixed dilated IVC, RA and RV diastolic collapse
  - b. Rx: pericardiocentesis, thoracotomy, pericardotomy
- 3. Is patient hypovolemic?**
  - a. US: no or few B lines, hyperdynamic LV, collapsible IVC
  - b. Rx: volume infusion
- 4. Is LV dysfunction main cause of shock**
  - a. US: Hypokinetic LV, B lines, fixed and dilated IVC
  - b. Rx: pressors and inotropes
- 5. Is there RV strain?**
  - a. US: RV dilation, D sign, paradoxical septal wall movement, fixed and dilated IVC
  - b. Rx: consider thrombolysis, DVT search, further diagnostic workup for PE/MI, consider pulmonary hypertension

<https://www.echoguidedlifesupport.com>

**[8] What are your targets during CPR for the following metrics? (See Table 8.3)**

- f. Carotid or femoral pulse**
- g. CPP**
- h. Arterial relaxation (diastolic) pressure**
- i. PETCO<sub>2</sub>**
- j. SCVO<sub>2</sub>**

Alright, guys. Rapid fire here. Knowing this list will better arm you for your next critical care pimp session and will give you physiologic parameters to steward the resuscitation process. We have adapted Table 8.3 in Rosen's to give you targets to reach as opposed to a list of values to avoid - enjoy.



The following table has been adapted from Table 8.3 in Rosen's Emergency Medicine, 9th Edition.

<b>Monitoring Technique</b>	<b>Indicator</b>
<b>Carotid or femoral pulse</b>	Present
<b>CPP</b>	>15 mmHg
<b>Arterial relaxation (diastolic) pressure</b>	> 20-25 mmHg
<b>PETCO<sub>2</sub></b>	>10 mmHg
<b>ScvO<sub>2</sub></b>	> 40%

### **Wisecracks:**

#### **[1] What is cough CPR and when should it be used?**

Cough CPR may sound like something you do to resuscitate your best friend that has just choked on that stray Dorito after seeing that hilarious meme you posted, but it is actually a resuscitation strategy that you can use to save a life.

As per Rosen's, cough CPR is best used in the patient who enters into ventricular fibrillation or pulseless ventricular tachycardia while on cardiac monitors. The patient should be constructed to cough "vigorously" until a defibrillator is available. There is some evidence to suggest that this strategy can keep a patient conscious for up to a minute or longer.

#### **[2] What is the only antidysrhythmic shown to improve rates of ventricular fibrillation conversion to a perfusing rhythm?**

For all you guys thinking that the answer to this question was epinephrine, you are wrong. In cases of refractory ventricular fibrillation, amiodarone dosed at 300 mg IV is the ONLY antidysrhythmic to improve rates of conversion to a perfusing rhythm.

#### **[3] What is the minimum coronary perfusion pressure (CPP) is needed to achieve return of spontaneous circulation (ROSC)?**



Some spaced repetition here, CRACKCast listeners. Remember, you want to target coronary perfusion pressure to 15 mmHg or more during resuscitation. This is the recommended value given the results of human and animal studies that identified a minimum CPP of at least 15 mm Hg to achieve return of spontaneous circulation.

#### **[4] What is the triad of cardiopulmonary arrest?**

We are easing you out of the podcast with this one. The triad of cardiopulmonary is as follows: unconsciousness, apnea, and pulselessness.

If there is any suspicion that the patient is in cardiac arrest, CPR should be started immediately. Also, be sure to use a large artery to assess for pulselessness, as distal pulses become faint and undetectable at lower mean arterial pressures.