

**Northwest Community EMS System – Continuing Education – July 2016 – CE Credit Questions – page 1 of 2**

Name	Date	Employer
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Note: Completion of these questions is worth one (1) CE hour (as this module was primarily psycho-motor simulated cardiac arrest team resuscitation). This may be submitted without penalty until November 30, 2016.

1	What 3 places on the body does rigor mortis first occur?	
2	Complete the quote from Dr. Ortinau, " <i>Of all the medical emergencies where we (EMS) make a difference, for most we just get the ball rolling....</i> "	
3	In cardiac arrest, what 2 things are "known" to improve outcome?	
4	In cardiac arrest, what 2 things have NOT been shown to improve outcome?	
5	List the 5 components of quality CPR.	
6	Are compression rates higher than 120, or less than 100, associated with a decreased likelihood of survival? Why?	
7	Why is releasing completely important?	
8	When using the ResQPOD impedance threshold device what compression rates are associated with best outcome?	
9	Are there improved cardiac arrest outcomes, when using mechanical CPR devices, compared to manual CPR?	
10	In pit crew cardiac arrest resuscitation, what are the 5 roles?	
11	What should be done if only 2 rescuers are on the scene of a cardiac arrest?	
12	Where should defib pads/paddles be placed?	
13	What are 2 acceptable reasons to interrupt chest compressions?	
14	Is it important to minimize pre-shock and post-shock pauses in compressions?	
15	Should patients be moved with CPR in progress?	
16	Why should an OP/NPA be inserted prior to beginning BVM ventilation?	
17	When using the ResQPOD/ITD when is it more important to maintain a tight face-mask seal - during compressions or ventilations?	

18	What are 5 ways capnography is helpful during cardiac arrest resuscitation?	
19	In cardiac arrest resuscitation, which has the higher priority – vascular access & medication administration or advanced airway placement?	
20	While one PM is obtaining vascular access (IO/IV) what can other PM's be doing to assist?	
21	When during cardiac arrest resuscitation can placement of an advanced airway be considered?	
22	What is persistent/refractory VF?	
23	If VF persists after 3-4 defibrillations, what should be done?	
24	What are causes of PEA?	
25	List 5 assessment/interventions appropriate for PEA?	
26	List 4 aspects of post-ROSC care.	
27	What should be done post-ROSC if pt is hypotensive?	
28	Is therapeutic hypothermia still highly recommended by the AHA? Why was it removed from pre-hospital care?	
29	Is re-arrest common? What type of re-arrest is most common? What can be done to promptly detect it?	
30	What should be documented on an ePCR for a cardiac arrest patient?	

July 2016 – Continuing Education

# Cardiac Arrest “Pit-Crew” Team Resuscitation

Diana Neubecker RN BSN PM  
NWC EMSS In-Field Coordinator

Objectives - Related to cardiac arrest resuscitation:

1. Discuss new knowledge.
2. Review selected key elements.
3. Identify & demonstrate treatment priorities.
4. Practice team “pit-crew” approach skills.
5. Improve documentation.



## Crew finds pt non-breathing & pulseless with unmovable jaw, so performs surgical cric



### Rigor Mortis

- At death muscles relax, then stiffen
- Begins in 2-6 hrs, in the **eyelids, neck, and jaw**; then spreads to other muscles, last in fingers & toes
- Onset varies w/ temp, age, physical condition and build
- After 24-hrs muscles relax and flaccidity develops
- Infant/child may not show rigor due to sm. muscle mass



## Cardiac Arrest Resuscitation

“Of all the medical emergencies where we (EMS) make a difference, for most we just get the ball rolling.

In cardiac arrest, we own it. We are the ones that make a difference. Initial care, what we do in the field, determines outcome more than anything else.”



John M. Ortinau, MD, FACEP  
NWC EMSS Medical Director



Do NOT delay something “known” to improve outcome, to do something NOT known to improve outcome

- **“Known” to improve outcome**
  - Quality CPR
  - Defibrillation
- **“Might” improve outcome**
  - Medications, especially if given early
- **NOT shown to improve outcome**
  - Advanced airway
  - Transport w/ CPR in progress



## Quality CPR

1. **Rate** (at least 100, but less than 120)
2. **Depth** (2 - 2¼ ”)
3. **Release completely**
4. **Minimize interruptions**
5. **Do NOT hyperventilate**



## Metronome – USE ONE!

- Monitor
- Smartphone
- Device



# Avoid too Fast Compressions

## Chest Compression Rates and Survival Following Out-of-Hospital Cardiac Arrest\*

Ahmed H. Idris, MD<sup>1</sup>, Danielle Goffin, MD<sup>1</sup>, Paul E. Pepe, MD<sup>1</sup>, Stephen E. Brown, PhD<sup>2</sup>, Steven C. Brinkley, MD<sup>3</sup>, Gabriel N. Calzavara, MD<sup>4</sup>, Paul J. Tomaszewski, MD<sup>5</sup>, Daniel F. Davis, MD<sup>6</sup>, Michaelson E. Chiu, MD<sup>7</sup>, Ronald Gray, BS, MA, MS, NREMT<sup>8</sup>, Peter J. Kudenchuk, MD<sup>9</sup>, Jonathan Larson, EMT<sup>10</sup>, Steve Liu, MD<sup>11</sup>, James I. Montgomerie, PhD<sup>12</sup>, Kelli Shanholtz, BS<sup>13</sup>, George Sopko, MD, MPP<sup>14</sup>, Jon Weil, MD, MS<sup>15</sup>, Graham Nichol, MD<sup>16</sup>, Tom P. Aufderheide, MPP<sup>17</sup> for The Resuscitation Outcomes Consortium Investigators

**“data show that survival peaks with a chest compression rate around 120/min, with rates of 120/min or higher or less than 100/min being associated with a decreased likelihood of survival.”**

**...rates were greater than 120/min in nearly one third of cases.”**

**Abstract:** Objective: To determine the relationship between chest compression rate and survival following out-of-hospital cardiac arrest. Design: Retrospective, observational study. Setting: Out-of-hospital cardiac arrest. Participants: Adults with out-of-hospital cardiac arrest treated by emergency medical services. Measurements and Main Results: Data were analyzed from 10,000 patients with out-of-hospital cardiac arrest. The relationship between chest compression rate and survival was assessed using a cubic spline model. The optimal chest compression rate was 100-120/min. Survival was significantly higher for rates between 100 and 120/min compared to rates less than 100/min or greater than 120/min. Conclusions: Chest compression rates of 100-120/min are associated with the highest survival following out-of-hospital cardiac arrest. Rates less than 100/min or greater than 120/min are associated with decreased survival.

# Principal mechanisms thought to be responsible for producing blood flow during chest compressions:

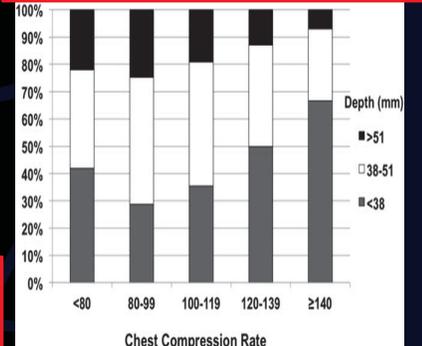
- 1) direct cardiac compression
- 2) ↑ intrathoracic pressure

Forward blood flow depends on venous blood filling the heart & lungs during diastole/release phase of chest compression.

If release phase is too brief, blood available for forward flow during compression may be decreased.

May explain findings that rates faster than 120/min are associated w/ decreased survival, as are rates less than 100/min.

# Compression Rate affects Compression Depth



Chest compression rate versus chest compression depth. The stacked bar graph shows distribution of three categories of chest compression depth (< 38mm indicated in gray, 38-51mm white, > 51mm black) across categories of compression rates (< 80, 80-99, 100-119, 120-139, and ≥ 140 chest compressions/min) (n = 6,399; chi-square test, p < 0.0001).

# RELEASE Completely

- Do **NOT** lean on chest
- Assure chest recoils completely after compressions
- Pressure between compressions creates positive intrathoracic pressure - which decreases heart & coronary artery refilling w/ blood

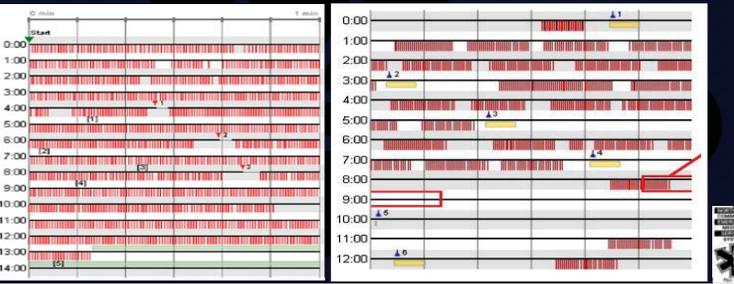


# Real Time CPR Feedback

**Highly Recommended**

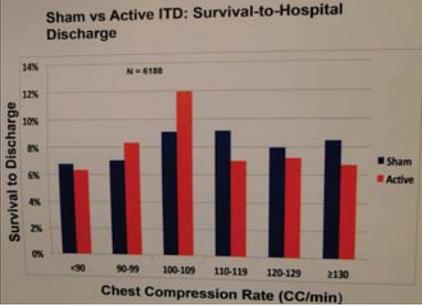
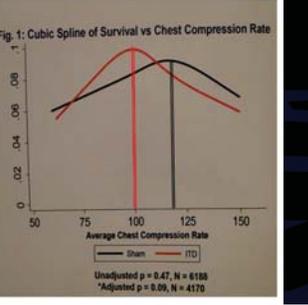
# Chest Compression Fraction (CCF)

- Proportion of each minute interval during which chest compressions are provided
- Associated with survival
- Often expressed as percentage (%)



# ResQPOD Impedance Threshold Device

- Circulation enhancing device
- “Works” during compressions
- Best outcome when compression rate 100-110/min



# What's the story with the RQP ITD?

2015 AHA Guidelines ↓ level of recommendation  
 Study: 8718 pts, 4345 sham, 4373 functioning ITD, did not show a benefit from ITD.  
 No differences in adverse events (pulmonary edema, airway bleeding) between the groups.

AHA Recommendation: Routine use of the ITD as an adjunct during conventional CPR is not recommended. Class of Recommendation indicates that evidence did not demonstrate benefit or harm associated with the ITD when used as an adjunct to conventional CPR.



## Published after 2015 Guidelines written

**Quality of CPR: An important effect modifier in cardiac arrest clinical outcomes and intervention effectiveness trials\***  
 Demetris Yannopoulos<sup>1,2</sup>, Tom P. Aufderheide<sup>3</sup>, Benjamin S. Abella<sup>4</sup>, Sam Durr<sup>5</sup>, Rajah J. Francisco<sup>6</sup>, Jeffrey M. Goodlin<sup>7</sup>, Brian D. Mahoney<sup>8</sup>, Yung M. Nadeau<sup>9</sup>, Henry R. Halperin<sup>10</sup>, Robert O'Connor<sup>11</sup>, Ahmed H. Idrois<sup>12</sup>, Lance B. Becker<sup>13</sup>, Paul E. Pape<sup>14</sup>

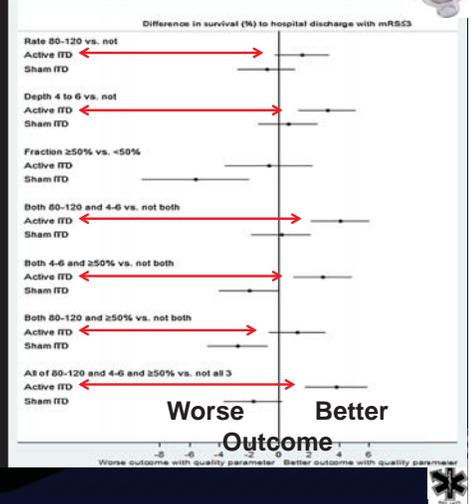
**Abstract:** Objectives: Assessment of the quality of CPR had a significant association with the primary study outcomes in the RQP-PRIMED trial. Design: The entire cohort derived from the RQP-PRIMED trial was included to determine if there was a strong relationship between CPR quality and outcomes. Results: The proportion of patients receiving acceptable quality CPR was 23.5% (n=1012) and 76.5% (n=3306) received unacceptable quality CPR. Acceptable quality CPR was associated with a significantly lower risk of death and a higher rate of survival to hospital discharge with favorable neurological outcome (OR 0.76, 95% CI 0.53, 1.11). Conclusions: The quality of CPR is an important effect modifier in cardiac arrest clinical outcomes and intervention effectiveness trials.

# With "acceptable" CPR, there is a difference

- Rate: 100 ± 20% (80-120)
- Depth: 5 cm ± 20% (4-6 cm)
- Compression fraction ≥50%

45% had "acceptable" CPR

"This analysis supports the notion that the quality of CPR needs to be taken into account during randomized controlled trials of interventions for cardiac arrest. Our analysis of the prospectively collected, well-defined ROC-PRIMED dataset showed statistically significant and clinically important interactions between the quality of CPR provided, the study interventions, and survival to hospital discharge with favorable neurological outcome."



# If "acceptable" quality CPR, when using RQP/ITD, there is improvement in neuro intact survival



Table 5 Rates of ROSC and survival of patients receiving acceptable quality of CPR (rate 80-120 cpm, depth 4-6 cm, fraction ≥50%), and those not receiving acceptable quality CPR (none of rate 80-120 cpm or depth 4-6 cm or fraction ≥50%), stratified by treatment arm.

	Acceptable quality CPR			Unacceptable quality CPR		
	Sham (n=827)	Active (n=848)	p-Value	Sham (n=1061)	Active (n=1012)	p-Value
ROSC	213 (25.8)	233 (27.5)	0.43	244 (23.0)	230 (22.7)	0.88
Survival to hospital discharge	53 (6.4)	81 (9.6)	0.018	75 (7.1)	53 (5.2)	0.082
Discharged alive with mRS ≤3	34 (4.1)	61 (7.2)	0.0064	62 (5.8)	34 (3.4)	0.0071

Values are n (%).

# If "unacceptable" quality CPR, better to not use the RQP/ITD

## "Unacceptable"

- Rate less than 80, or greater than 120
- Depth less than 4 cm, or greater than 6 cm
- Compression fraction less than 50%



5.3. Mechanical CPR A systematic review of randomized trials of mechanical chest compression devices found no advantage to the routine use of mechanical chest compression devices for OHCA (survival to discharge/30 days (average odds ratio (OR) 0.89, 95% CI 0.77, 1.02) and survival with good neurological outcome (average OR 0.76, 95% CI 0.53, 1.11). Gates S, Quinn T, Deakin CD, Blair L, Couper K, Perkins GD. Mechanical chest compression for out of hospital cardiac arrest: systematic review and meta-analysis. Resuscitation 2015;94:91-7.

Per-protocol analysis of the LINC trial observed similar four-hour survival rates between mechanical and manual CPR (23.8% vs. 23.5%, risk difference -0.35%, 95% CI -3.1 to 3.8, p = 0.85). Rubertsson S, Lindgren E, Smekal D, et al. Per-protocol and pre-defined population analysis of the LINC study. Resuscitation 2015;96:92-9.

A prospective evaluation of mechanical CPR in Vienna noted worse neurological outcomes in those receiving mechanical CPR. Zeiner S, Sulzgruber P, Dattler P, et al. Mechanical chest compression does not seem to improve outcome after out of hospital cardiac arrest. A single center observational trial. Resuscitation 2015;96:220-5.

These findings reinforce the ILCOR and ERC recommendations against their routine use."

Resuscitation. 2015 Nov;96:220-5.

# Mechanical chest compression does not seem to improve outcome after out-of hospital cardiac arrest. A single center observational trial.

Zeiner S, Sulzgruber P, Dattler P, Keferböck M, Poppe M, Lobmeyr E, van Tulder R, Zajicek A, Buchinger A, Polz K, Schratzenbacher G, Storz F.

AIM: Recently three large post product placement studies, comparing mechanical chest compression (cc) devices to those who received manual cc, found equivalent outcome results for both groups. Thus the question arises whether those results could be replicated using the devices on a daily routine.

METHODS: We prospectively enrolled 948 patients over a 12 months period. Chi-Square test and Mann-Whitney-U test were used to assess differences between "manual" and "mechanical" cc subgroups. Uni- and multivariate Cox regression hazard analysis were used to assess the influence of cc type on survival.

RESULTS: A mechanical cc device was used in 30.1% (n=283) cases. Patients who received mechanical cc had a significantly worse neurological outcome - measured in cerebral performance category (CPC) - than the manual cc group (56.8% vs. 78.6%, p=0.009). Patients receiving mechanical cc were significantly younger, more were male and were more likely to have bystander CPR and an initially shock-able ECG rhythm. There was no difference in the quality of CPR that might explain the worse outcome in mechanical cc patients.

CONCLUSION: Even with high quality CPR in both, manual and mechanical cc groups, outcome in patients who received mechanical cc was significantly worse. The anticipated benefits of a higher compression ratio and a steadier compression depth of a mechanical cc device remain uncertain. In this study selection for mechanical cc was not standardized, and was non-random. This merits further investigation. Further research on how mechanical cc is chosen and used should be considered.

# "Pit-Crew"



# Seconds make a Difference

# "Pit-Crew" Roles - in order

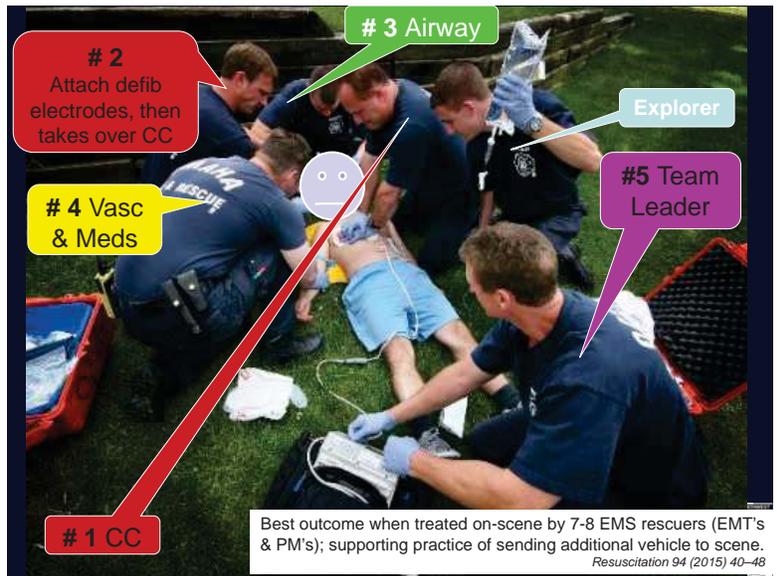
**# 1 - Compressor**  
 ✓ responsive & pulse  
 Begin Compressions

**# 3 - Airway**  
 Insert OP/NPA  
 Attach BVM to RQP, capno, O2  
 Tight face-mask seal w/ 2 hands

**# 2 - Monitor**  
 Attach combo-pads to pt & monitor  
 Take over compressions

**# 5 - Team Leader**  
 Code Commander  
 Coach crew & document

**# 4 - Meds**  
 Obtain IV/IO access  
 Administer meds



Best outcome when treated on-scene by 7-8 EMS rescuers (EMT's & PM's); supporting practice of sending additional vehicle to scene. Resuscitation 94 (2015) 40-48

Resuscitation 94 (2015) 40-48

Contents lists available at ScienceDirect

**Resuscitation**

Journal homepage: www.elsevier.com/locate/resuscitation

European Resuscitation Council

**Does number of EMS personnel on scene affect outcome?**

Study of 16,122 cases, **7-8 EMS personnel on-scene** was associated w/ highest survival compared with fewer personnel on-scene.

EMS personnel on-scene	No. of patients (%)	Unadjusted survival to discharge No. (%)	Unadjusted Odds Ratio for Survival (95%CI)	Adjusted Odds Ratio for Survival (95%CI)**	Adjusted Odds Ratio for Survival (95%CI) Pooled (I <sup>2</sup> = unadjusted OR)
0	30 (1.0)	3 (10.0)	0.46 (0.16, 1.29)	0.67 (0.16, 2.81)	
1-2	352 (11.7)	47 (13.4)	0.71 (0.53, 0.93)	0.88 (0.61, 1.25)	
3-4	373 (12.3)	62 (16.5)	0.82 (0.58, 1.16)	0.84 (0.60, 1.18)	
5-6	1325 (44.2)	247 (18.6)	1.04	1.04	
7-8	707 (23.6)	184 (26.0)	1.43 (1.14, 1.74)	<b>1.48 (1.11, 1.99)</b>	
>8	216 (7.2)	55 (25.5)	1.21 (0.86, 1.71)	1.36 (0.93, 1.99)	
Total	3095 (100)				

Fig. 3. Survival to hospital discharge by category of number of EMS personnel on-scene within 15 min of 9-1-1 call and prior to patient death or transport, among shockable initial rhythm. Shockable rhythms represent 24.8% of the study population and 68.7% of the survivors. \*Adjusted for age (years), sex, median income of census tract where cardiac arrest occurred, whether the arrest occurred in a public setting, whether the arrest was witnessed by bystanders, whether resuscitation was attempted by bystanders, and time (minutes) from 9-1-1 call except to first EMS vehicle arrival, and first arriving EMS agency. EMS agency level variables included for each cardiac arrest: first responding EMS agency wave; population density arrival; arrival EMS time per Advanced Life Support (ALS) capable vehicle; paid ALS full-time equivalents (FTE) per 100k service population; paid BLS FTE per 100k service population.

## What if only 2 responders?

**# 1**  
 ✓ responsive & pulse  
 Begin Compressions  
 After ECG ✓/defib, move to airway

**# 2**  
 Call for help  
 Attach combo-pads to pt & monitor  
 then  
 Take over compressions

Insert OP/NPA  
 Attach BVM RQP, capno, O2  
 Tight face-mask seal w/ 2 hands

**Do 2 person BLS CPR until help arrives**

## Defib Pad/Paddle Placement

Location

- Just under Clavicle Right of Sternum
- Upper chest, to R of sternum, under clavicle
- Apex of heart, L of nipple, mid-axillary line

Apply firm pressure when using paddles

No advantage anterior-posterior position for defib

Anterior view: Pad on upper chest (right of sternum), pad on mid-axillary line (left of nipple).

Lateral view: Pad on upper chest (right of sternum), pad on mid-axillary line (left of nipple).

~V6 position (L) armpit  
 Mid-axillary line  
 Horizontal to nipple

## Defibrillation Pad Placement

**Goal Placement**

**Pad Placement**

Labels in diagrams: HEART, R lung, L lung, R arm, L arm, Spine.

## Chest Compressions (CC)

Acceptable reasons to interrupt compressions

1. ✓ **ECG every 2 minutes** (goal less than 5 sec)  
Should it be shocked? Is it organized?

Change compressor at the same time  
If reliever NOT in place/ready to take over – speak up!

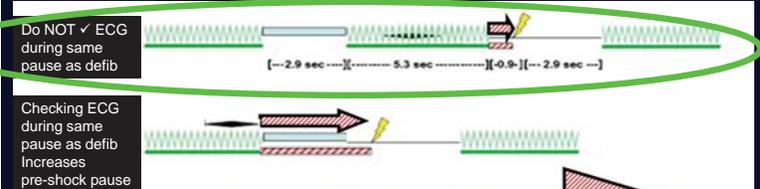
2. **Defibrillate** (goal less than 5 sec)



Goal: Minimize peri-shock pauses

Minimize time from last compression to shock, and from shock to next compression

Do NOT ✓ ECG during same pause as defib



Checking ECG during same pause as defib  
Increases pre-shock pause

### Pre-Shock Pause Comparison

Adapted from Perkins, Dawes, Grant, Thickety, Manick and simulation studies: The impact of manual defibrillation technique on in-hospital cardiac arrest resuscitation. Resuscitation, Vol 71, No 1, Aug 2007, 109-114

## Should pts be moved w/ CPR in progress?

NO (unless very unique situation), interrupts & decreases CPR quality



## OPA/NPA & BVM Ventilation

Insert OP/NPA before beginning BVM ventilation to minimize gastric distention, vomiting & aspiration



Use 2-hand method before adv. airway to maintain tight face-mask seal esp. w/ RQP/ITD during compressions

## Ventilation

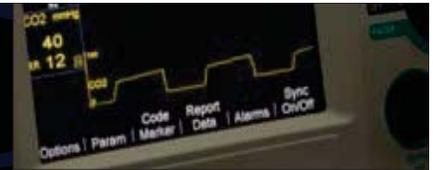
- **Hyperventilation is Lethal**
  - Watch both RATE and VOLUME
- Do **NOT** squeeze bag right before:
  - ECG ✓ (can cause artifact)
  - Defibrillation (↓ effectiveness)



## Capnography

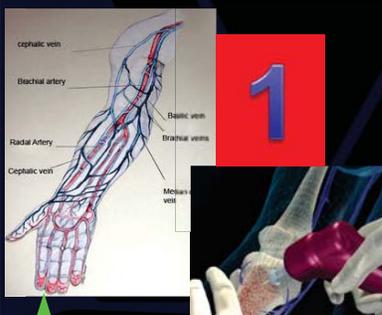
✓ every 2 minutes to:

1. Confirm airway patency & ventilation
2. Prevent hyperventilation (shows ventilation rate)
3. Monitor compression quality
4. Predict ROSC (before pulse detected)
5. Identify when ROSC unlikely



# ★ NEW – Priority Emphasis ★

## Complete vascular access & 1<sup>st</sup> round meds before preparing for adv. airway placement




# Vascular Access - ASAP

## Dedicate additional personnel, if available to help

1. ID vascular access site (IV or IO)
2. Prep site w/ CHG/IPA
3. Prep 10mL NS flush, if IO
4. Prime connecting tubing, if IO
5. Prep IVF & tubing
6. Insert & secure IO (or IV)
7. Prep epinephrine
8. Prep amiodarone
9. Place IVF in pressure infuser, if IO

# Medications

- 1<sup>st</sup> med all pulseless = vasopressor  
Prepare/administer epinephrine ASAP
- Prepare meds in advance, so ready when time to give
- Give based on last ECG  
do NOT delay until next ECG ✓
- Follow w/ 20-50 mL IVF bolus  
If extremity IV: elevate x 20 sec



## ORIGINAL CONTRIBUTIONS

### PROBABILITY OF RETURN OF SPONTANEOUS CIRCULATION AS A FUNCTION OF TIMING OF VASOPRESSOR ADMINISTRATION IN OUT-OF-HOSPITAL CARDIAC ARREST

Michael W. Habibe, MBA, NREMT-P, Christopher Johnson, BS, EMT-P, Jamie Blackwelder, BS, EMT-P, Kevin Collopy, BA, NREMT-P, Sara Houston, BS, NREMT-P, Melissa Martin, MHS, EMT-P, Delbert Wilkes, EMT-P, Jonina Wiser, BS, EMT-P

**OBJECTIVE:** Introduction. Vasopressors (epinephrine and vasopressin) are associated with return of spontaneous circulation (ROSC). Recent retrospective studies reported a greater likelihood of ROSC when vasopressors were administered within the first 10 minutes of arrest. However, it is unclear that the relationship between ROSC and the timing of vasopressor administration is a binary function (i.e., <math>0-10</math> minutes). More likely this relationship is a function of time measured as a continuum, with diminishing effectiveness even within the first 10 minutes of arrest, and potentially, some lingering benefit beyond 10 minutes. However, this relationship remains unexplored. **Objective:** To develop a model describing the likelihood of ROSC as a function of the call receipt to vasopressor interval (CRVI).

**DESIGN:** measured as a continuum. **Methods:** We conducted a retrospective study of cardiac arrest using the North Carolina Prehospital Care Reporting System (PREHRS). Inclusionary criteria were all adult patients suffering a witnessed, non-traumatic arrest during January-June 2012. Chi-square and t-tests were used to analyze the relationship between ROSC and CRVI, patient age, race, and gender; endotracheal intubation (ETI), advanced external defibrillation (AED) use; presenting cardiac rhythm; and bystander cardiopulmonary resuscitation (CBR). A multivariate logistic regression model calculated the odds ratio (OR) of ROSC as a function of CRVI while controlling for potential confounding variables. **Results:** Of the 1,122 patients meeting inclusion criteria, 542 (48.3%) experienced ROSC. ROSC was less likely with increasing CRVI (OR = 0.96,  $p < 0.01$ ). Compared to patients with shockable rhythm, patients with asystole (OR = 0.42,  $p < 0.01$ ) and pulseless electrical activity (OR = 0.52,  $p < 0.01$ ) were less likely to achieve ROSC. Older (OR = 0.04,  $p < 0.02$ ) and patients receiving bystander CBR (OR = 0.42,  $p < 0.01$ ) were less likely to attain ROSC, although emergency medical services response time was significantly longer among patients receiving bystander CBR. Race, sex, ETI, and AED were not predictors of ROSC. **Conclusions:** We found that time to vasopressor administration is significantly associated with ROSC, and the odds of ROSC declines by 4% for every 1-minute delay between call receipt and vasopressor administration. These results support the notion of time-dependent function of vasopressor effectiveness as one the entire range of administration delay rather than just the first 10 minutes. Larger, prospective studies are needed to determine the relationship between the timing of vasopressor administration and long-term outcomes. **Keywords:** epinephrine, cardiac arrest, emergency medical services, paramedic, resuscitation, CTR

PREHOSPITAL EMERGENCY CARE 2013;19:457-463

**INTRODUCTION**  
Sudden cardiac death accounts for more than half of all coronary heart disease deaths in the United States.

## The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812      MAY 5, 2016      VOL 374, NO 19

### Amiodarone, Lidocaine, or Placebo in Out-of-Hospital Cardiac Arrest

F.J. Klotzsch, S.P. Brown, M. Daya, T. Rea, G. Nichol, L.J. Morrison, B. Lemus, C. Vallancourt, L. Winters, C.W. Callaway, J. Christenson, D. Egan, J.P. Ornato, M.L. Waisfeld, G.G. Sivil, A.H. Ikin, T.P. Aufderheide, J.V. Dunford, M.R. Callaly, G.M. Wilk, A.M. Brienzo, P. Devroeghe-Nickens, P.C. Gray, R. Gray, N. Seale, R. Straight, and P. Doran, for the Resuscitation Outcomes Consortium Investigators\*

**ABSTRACT**  
**BACKGROUND:** Antiarhythmic drugs are used commonly in non-hospital cardiac arrest for shock-refractory ventricular fibrillation or pulseless ventricular tachycardia, but without proven survival benefit.

**DESIGN:** In this randomized, double-blind trial, we compared parenteral amiodarone, lidocaine, and saline placebo, along with standard care, in adults who had nontraumatic out-of-hospital cardiac arrest, shock-refractory ventricular fibrillation or pulseless ventricular tachycardia after a first one shock, and tracheal intubation. Randomized enrolled patients at 30 North American sites. The primary outcome was survival to hospital discharge; the secondary outcome was favorable neurologic function at discharge. The preplanned primary analysis population included all randomly assigned participants who met eligibility criteria and received any dose of a trial drug and whose initial cardiovascular rhythm of ventricular fibrillation or pulseless ventricular tachycardia was refractory to shock.

**RESULTS:** In the per-protocol population, 3020 patients were randomly assigned to amiodarone (9%), lidocaine (9%), or placebo (82%). Of these, 24.6%, 23.7%, and 21.0%, respectively, survived to hospital discharge. The difference in survival rate for amiodarone versus placebo was 3.2 percentage points (95% confidence interval [CI], -0.4 to 7.0;  $P=0.08$ ), for lidocaine versus placebo, 2.4 percentage points (95% CI, -0.9 to 5.3;  $P=0.15$ ), and for amiodarone versus lidocaine, 0.7 percentage points (95% CI, -1.2 to 4.3;  $P=0.79$ ). Neurologic outcome at discharge was similar in the three groups. There was heterogeneity of treatment effect with respect to whether the arrest was witnessed (0.81), active drugs were associated with a survival rate that was significantly higher than the rate with placebo among patients with bystander-witnessed arrest but not among those with unwitnessed arrest. More amiodarone recipients required temporary cardiac pacing than did recipients of lidocaine or placebo.

**CONCLUSIONS:** Overall, neither amiodarone nor lidocaine resulted in a significantly higher rate of survival or favorable neurologic outcome than the rate with placebo among patients with shock-refractory cardiac arrest due to initial shock-refractory ventricular fibrillation or pulseless ventricular tachycardia. (Funded by the National Heart, Lung, and Blood Institute and others; ClinicalTrials.gov number, NCT01401547.)

**MECHANISMS OUTCOMES**  
After randomization, placebo recipients were more likely to require an additional dose of blinded trial drug than recipients of amiodarone or lidocaine, and they received a greater number of subsequent shocks and other rhythm-control medications (Table 2). More lidocaine recipients than placebo recipients had sustained return of spontaneous circulation on hospital arrival (Table 3). Patients were more likely to survive to hospital admission after receipt of placebo. Fewer recipients of amiodarone or lidocaine than of placebo required CPR during hospitalization (Table 5) in the Supplementary Appendix. The

Rates of survival with favorable neurologic status (the secondary outcome) were similar in the amiodarone group (182 patients [18.9%]), lidocaine group (172 [17.8%]), and placebo group (175 [16.6%]). The risk difference for the secondary outcome for amiodarone versus placebo was 2.2 percentage points (95% CI, -1.1 to 5.6;  $P=0.19$ ); for lidocaine versus placebo, 0.9 percentage points (95% CI, -2.4 to 4.2;  $P=0.59$ ); and for amiodarone versus lidocaine, 1.1 percentage points (95% CI, -2.1 to 4.6;  $P=0.44$ ).

0.5 to 0.9;  $P=0.03$ ), but did not differ significantly between amiodarone and lidocaine (-0.1 percentage points [95% CI, -0.5 to 0.4;  $P=0.07$ ]). The survival rate was also higher among amiodarone recipients than placebo recipients with EMS-witnessed arrest, a risk difference of 21.9 percentage points (95% CI, 5.8 to 38.0;  $P<0.001$ ). Conversely, among 839 patients in whom out-of-hospital cardiac arrest was unwitnessed, survival did not differ significantly between trial groups. No other significant interaction with treatment was found in other prespecified subgroups.

## Scenario – Prep Meds Before Need

Time	EtCO2	ECG	DF	Med Administration	What med person doing?
0004	29	VF	120j		Prepare Epi, give ASAP, then prepare amiodarone
0006	33	AS		Epi 1mg	Prep next epi
0008	42	VF	150j	Amio 300mg	Give amio, then prep next amio
0010	37	IVR		Epi 1mg	Give epi, then prep next epi
0012	29	AS			
0014	41	VF	200j	Epi 1mg & Amio 150mg	Give epi & amiodarone Then prep next epi
0016	39	IVR			
0018	45	IVR		Epi 1mg	Give epi & then prep next epi
0020	44	VF	200j		
0022	38	AS		Epi 1mg	Give epi & then prep next epi
0024	40	AS			
0026	41	VF	200j	Epi 1mg	Give epi & the prep next epi
0028	64	AS			
0030	56	ST			Get dopamine out while BP ✓

# Medication Double Cross-Check

Beyond the Rights.....



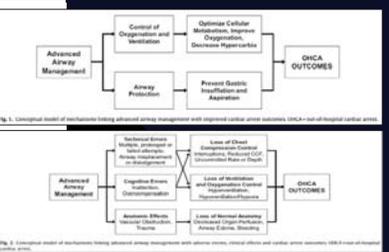
Check meds w/ another PM prior to giving

# Advanced Airways (ETI, KLTSO)

- No evidence to support early placement
- Preoxygenate for at least 3 min prior
- Consider after epi & amiodarone (if VF) given
- Insert sooner - if unable to BVM
- Avoid interrupting compressions



# Resuscitation



Commentary and concepts  
Mechanisms linking advanced airway management and cardiac arrest outcomes  
Justin L. Benoit<sup>1</sup>, David K. Prince<sup>2</sup>, Henry E. Wang<sup>3</sup>

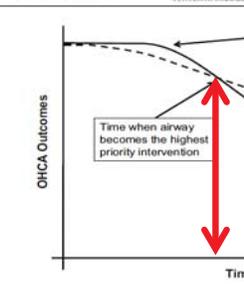
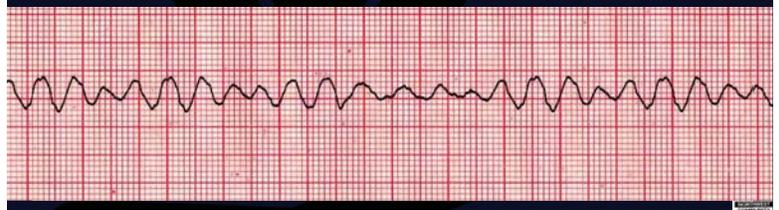


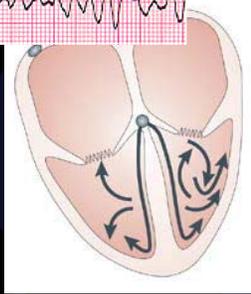
Fig. 3. Conceptual model of the time-dependent benefit of advanced airway management relative to other cardiac arrest interventions. Depicts a hypothetical survival curve for patients with and without an advanced airway intervention as a function of elapsed time. OHCA=out-of-hospital cardiac arrest.

# Ventricular Fibrillation (VF)

- "Recurrent" VF
  - Other rhythms between episodes of VF
- "Refractory/Persistent" VF
  - VF despite multiple defibrillation attempts



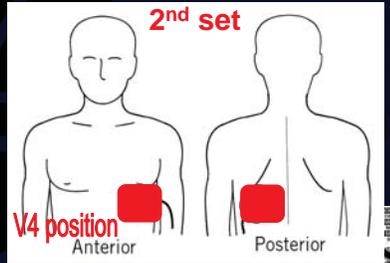
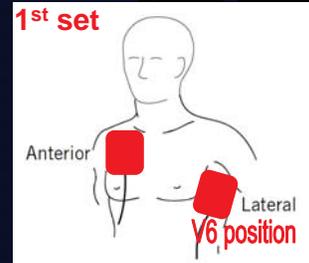
# Persistent/Refractory VF



- Defib goal - stop electrical activity, to allow normal pacemakers to function
- VF has different vectors
- While anterior-lateral placement works for most VF....
- If it does not, changing pad placement has been shown to be effective

# Persistent/Refractory VF

- After ~3<sup>rd</sup> or 4<sup>th</sup> defibrillation
  - Leaving original (anterior-lateral) electrodes in place
    - Apply fresh/new set defib pads in Anterior-Posterior position
    - Minimize compression interruption placing posterior pad
    - Switch cable from 1<sup>st</sup> set to 2<sup>nd</sup> set electrodes
  - Defib using - new pads- in AP position



# Pulseless Electrical Activity

- PEA is not a rhythm; any rhythm can be PEA
- PEA can be fast or slow; wide or narrow
- "Pseudo" vs true PEA (cannot [yet] determine in field)
- 55% had mechanical activity ("pseudo PEA") and a much higher rate survival to discharge. Flato UA, Palva EF, Carballo MT, Buehler AM, Marco R, Timmerman A. Echocardiography for prognostication during the resuscitation of intensive care unit patients with non-shockable rhythm cardiac arrest. Resuscitation 2015;92:1-6.



<ul style="list-style-type: none"> <li>&gt; HypoVOLEMIA</li> <li>&gt; HypOXIA</li> <li>&gt; HypoGLYCEMIA</li> <li>&gt; Hydrogen ion</li> <li>&gt; Hypo/hyperKALEMIA</li> <li>&gt; HypoTHERMIA</li> <li>&gt; Tension pneumo</li> <li>&gt; Toxins</li> <li>&gt; Tamponade</li> <li>&gt; Thrombosis</li> <li>&gt; Trauma</li> </ul>	<ul style="list-style-type: none"> <li>• <b>IV</b> <ul style="list-style-type: none"> <li>• begin rapid IVF bolus</li> <li>• using pressure infuser</li> </ul> </li> <li>• <b>Glucose</b> ✓                     <ul style="list-style-type: none"> <li>• ? hypoglycemia</li> </ul> </li> <li>• <b>Lungs</b> ✓                     <ul style="list-style-type: none"> <li>• ? tension pneumo</li> </ul> </li> <li>• <b>Oxygen</b> ✓                     <ul style="list-style-type: none"> <li>• ? airway, O2 supply</li> </ul> </li> <li>• <b>PMH &amp; meds</b> ✓                     <ul style="list-style-type: none"> <li>• ? renal failure, toxins</li> </ul> </li> </ul>
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# Post ROSC Care

1. Monitor closely
2. BP support HIGH priority; maintain heart & brain perfusion
  - ✓ & support BP/MAP
  - If hypotensive:
    - Administer IVF until Dopamine ready
    - Begin DOPAMINE, titrate to response
    - Start additional IV, if needed
3. Do NOT hyperventilate - even if ↑ ETCO<sub>2</sub>
4. Acquire 12L



regulate DOPAMINE drip rate here

turn OFF

connect mini-drip tubing here

Prime this line/space w/ dopamine

## American Heart Association GUIDELINES CPR ECC 2015

### Therapeutic Hypothermia

- Not harmful; Still highly recommended by AHA
- Removed from prehospital SOP's because no evidence of benefit when given prior to hospital arrival
- Was distracting from other priorities in prehospital care (BP support, 12L ECG)

2015 Recommendations — We recommend that comatose (ie, lack of meaningful response to verbal commands) adult patients with ROSC after cardiac arrest have TTM (Class I, LOE B-R for VF/pVT OHCA; Class I, LOE C-EO for non-VF/pVT (ie, "nonshockable") and in-hospital cardiac arrest)...Of note, there are essentially no patients for whom temperature control somewhere in the range between 32o C and 36o C is contraindicated.

Hypothermia in the Prehospital Setting - When cooling maneuvers were initiated in the prehospital setting, neither survival nor neurologic recovery differed for any of these trials alone or when combined in a meta-analysis... Current evidence indicates that there is no direct patient benefit from these interventions and that the intravenous fluid administration in the prehospital setting may have some potential harm, albeit with no increase in overall mortality.

## Rearrest

- Re-arrest occurs in ~38%, most often in first 10 minutes
- Most common type: PEA (so ECG rhythm may not change)
- Risk w/ re-arrest: Not detected quickly, not treated aggressively
- Detected quickly & treated aggressively; does not worsen outcome!
- Keep finger on pulse; watch O2 sat pleth on monitor to detect

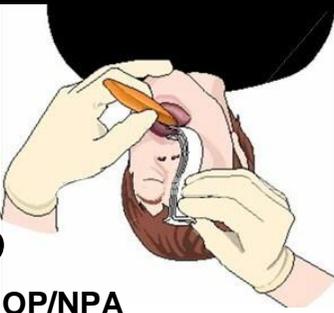
## Documentation – Key Points

- Document ECG & EtCO<sub>2</sub> - every 2 minutes
  - Not BP & pulse; not checked every 2 min
  - Pulse is a palpable pulse (not HR on ECG)
- CPR started when started and stopped due to ROSC or TOR (termination of resuscitation)
  - Not every 2 minutes with rhythm check
- O2 sat should not be documented during CPR
  - Number is meaningless without a pulse



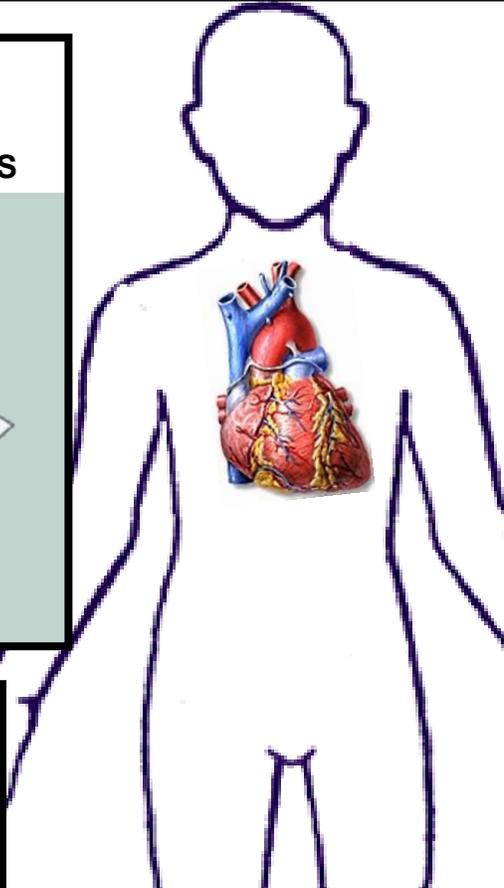
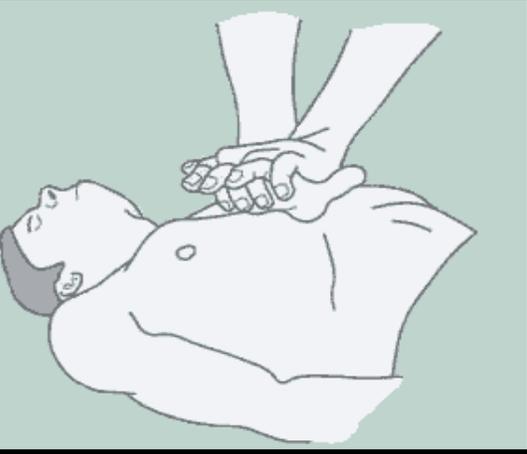
# # 3

Insert OP/NPA  
 Attach RQP/ITD & capnography to BVM w/ O2  
 Maintain tight 2-hand face-mask seal during compressions & ventilations  
 Compressor squeezes bag, after compression sets, until advanced airway placed



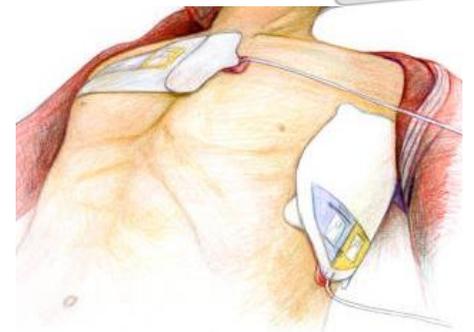
# # 1

begin CHEST COMPRESSIONS



# # 2

TURN ON MONITOR & ATTACH ELECTRODES/DEFIB PADS  
 (Will relieve compressor)



# # 4

Establish IV/IO  
 Administer MEDICATIONS



# # 5

TEAM LEADER  
 Code Commander



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 COMMUNITY  
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 SERVICES  
 SYSTEM**  
 EST 1972