Get With The Guidelines®-Resuscitation: Physiologic-Directed CPR

March 20, 2019

Presenter:
Robert Michael Sutton MD MSCE FAAP FCCM
Associate Professor of Anesthesia, Critical Care, and Pediatrics
Meet Our Presenter

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Associate Professor of Anesthesia, Critical Care, and Pediatrics
Chair: Resuscitation Committee at The Children’s Hospital of Philadelphia
Medical Lead: Preventing Codes Outside the ICU
Co-Director: Resuscitation Science Research Program
Department of Anesthesiology and Critical Care Medicine
University of Pennsylvania School of Medicine
Personalizing Resuscitation with Physiologic-Directed CPR

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March 16th, 2019
Presenter Disclosure Information

**Current Research Support:**
- NIH NHLBI (PI R01 - clinical)
- NIH NHLBI (PI R01 - clinical)
- NIH NHLBI (Co-I R01 - large animal)
- NIH NICHD (Co-I R21 - large animal)
- Mallinckrodt Pharma (Co-I - large animal)

**Past Research Support:**
- NIH NICHD (PI K23)
- Laerdal Foundation / Laerdal Corporation
- Zoll medical

**Intellectual Conflicts:**
- Chair Elect: AHA GWTG-R Pediatric Research Task Force
- 2015 and 2018 PALS writing group member
- Member: ECC Systems of Care Committee
- Member: ECC Pediatric Emphasis Group
- Member: ECC Science Review Task Force

**Speaking Honoraria:**
- Zoll Medical for Pediatric CPR Quality Talk
Objectives

At the conclusion of this talk, participants will be able to:

Understand the concept, the targets, and the potential practical application of physiologic-directed CPR.
INVASIVE HEMODYNAMIC MONITORING DURING CPR

Recommendation:
• For patients with invasive hemodynamic monitoring in place at the time of arrest, it may be reasonable for rescuers to use blood pressure to guide CPR quality. No specific values can be recommended.

Why?
• Arrests most often occur in ICUs
• Many have arterial lines in place
• Two randomized controlled animal studies demonstrated improved survival with such an approach (Sutton, Resuscitation 2013 and AJRCCM 2014)
• Not studied in humans… yet
Recommendation:

- ETCO$_2$ monitoring may be considered to evaluate the quality of chest compressions, but specific values to guide therapy have not been established in children.

Why?

- No pediatric evidence to support that ETCO$_2$ monitoring during arrest improves outcomes...
- But ETCO$_2$ values during adult CPR correlate with CC depth and ventilation rate.
- Low end tidal associated with death

Sheak, Resuscitation 2015; Sanders, JAMA 1989
A 4 year old presents with fever, bacteremia, and hypotension. The child receives 60cc/kg of isotonic fluid. Blood pressure is now 100/50. Heart rate is normal for age. Extremities are warm. I would start the following vasopressor infusion:

A. Epinephrine  
B. Norepinephrine  
C. Vasopressin  
D. Dobutamine  
E. None
A 4 year old intubated ICU patient has a cardiac arrest. An arterial line is in place at the time of the arrest. Etiology is presumed to be respiratory decompensation. With chest compressions alone, the arterial blood pressure is 100/50. The first vasopressor I would give to treat this cardiac arrest is:

A. Epinephrine
B. Norepinephrine
C. Vasopressin
D. Isoproterenol
E. None
Rescuer-Centric Vs. Patient-Centric

Rescuer

Performance

“Mechanics”
Rate
Depth
Release Velocity
 Interruptions

Patient

Physiology

CoPP
DBP
ETCO₂
NIRS (rSO₂)

Time to Shift the Focus
Near-infrared spectroscopy in vegetables and humans: An observational study.

Kahn RA, Aryanwu A.

Abstract

BACKGROUND: Cerebral near-infrared spectroscopy (NIRS) of tissue oxygen saturation is claimed to be a surrogate marker for global cerebral perfusion. Increasingly, NIRS target-based therapy has been used during cardiac surgery in the hope of decreasing the incidence of adverse neurological outcome.

OBJECTIVES: We report NIRS values for some common vegetables and faculty at a world-class medical institution.

DESIGN: Observational nonblinded study.

SETTING: Single tertiary care institution and local urban vegetable market.

PARTICIPANTS: Five yams (Dioscorea cayenensis), five courgettes (Cucurbita pepo) and five butternut squashes (Cucurbita moschata) were studied. Five cardiothoracic surgeons and anaesthesiologists were the control group.

INTERVENTIONS: None.

MAIN OUTCOME MEASURES: NIRS value of each species.

RESULTS: Mean NIRS value for the control group was 71% [95% confidence interval (CI) 68 to 74] and was similar to that of the yellow squashes [75% (95% CI 74 to 76)]. These values were significantly greater than the NIRS measurements of both the butternut squash and yam [63% (95% CI 62 to 64) and 64% (95% CI 63 to 65), respectively, P<0.01].

CONCLUSION: Commonly eaten vegetables have NIRS measurements similar to those seen in healthy humans.
Non-Precision Based Medicine

Lots of different physiology

Steroids, chemotherapy?

Septic Shock

Heart Failure

Lots of different physiology

Single Ventricle Physiology

Intracranial Hypertension
Push Hard = Better Pediatric BPs

Blood Pressure (mmHg)

- SBP > 80
- MBP > 50
- DBP > 30

Percent Anterior-Posterior Chest Depth

Sutton, Resuscitation 2012
Simultaneous beat-to-beat assessment of arterial blood pressure and quality of cardiopulmonary resuscitation in out-of-hospital and in-hospital settings

Marko Sainio, Sanna Hoppu, Heini Huhtala, Joar Eilevstjønn, Klaus T. Ollkola, Jyrki Tenhunen

OHCA and IHCA
39 Patients
~42000 Compressions
Substantial heterogeneity

<table>
<thead>
<tr>
<th></th>
<th>Femoral artery recording</th>
<th>Radial artery recording</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male, No. (%)</td>
<td>18 (75)</td>
<td>10 (67)</td>
</tr>
<tr>
<td>Age, mean (SD), y</td>
<td>65.5 (18)</td>
<td>64.1 (17)</td>
</tr>
<tr>
<td>Location of arrest, No.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IHCA</td>
<td>20</td>
<td>15</td>
</tr>
<tr>
<td>OHCA</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Time from CA to EMS or MET arrival, median (IQR), min</td>
<td>2 (0, 3)</td>
<td>1 (0, 2)</td>
</tr>
<tr>
<td>Interval from CA to ABP measure, median (IQR), min</td>
<td>13 (9, 18)</td>
<td>4 (3, 10)</td>
</tr>
</tbody>
</table>
A 2013 Consensus Statement from the AHA recommended which of the following physiologic CPR quality targets*: 

A. Diastolic blood pressure > 30mmHg

B. Coronary Perfusion Pressure > 20mmHg

C. ETCO₂ > 10mmHg

D. NIRs > 50%

*Based on almost NO pediatric data
Monitor the patient’s response to the resuscitation effort

- Coronary perfusion pressure > 20mmHg
- Diastolic pressure > 25mmHg
- End Tidal Carbon Dioxide > 20mmHg
Coronary Perfusion Pressure (CoPP)
Pressure gradient that drives myocardial blood flow

Crile, Experimental Medicine 1906; Sanders, CCM 1984; Michael, CCM 1984; Kern, Resus 1988
What are the right «goals» to target?

"One... presses deep in the heart region with strong movements at a frequency of 120 or more a minute. The effectiveness of the efforts is recognised from the artificially produced carotid pulse and the constriction of the pupils."

Effectiveness Measured by Monitoring Patient Response

Maas, Berlin Klin Wochenschr 1892
CoPP Higher in Patients with ROSC

N=100
Pre-hospital and ED arrests
No ROSC CPP < 15mmHg

Maximal Coronary Perfusion Pressure

Patients with ROSC (%)

- < 15 mmHg (n = 44) 0%
- 15 - 25 mmHg (n = 28) 46%
- > 25 mmHg (n = 16) 79%
Among these markers of CPR quality, which one has been associated with improved survival to hospital discharge with favorable neurological outcome after pediatric cardiac arrest?

A. Chest compression rate 100-120 per minute

B. DBP ≥ 25mmHg in infants, ≥ 30mmHg in older children

C. ETCO₂ during CPR ≥ 20mmHg

D. Depth of compression > 50 mm

DBP indicates diastolic blood pressure
DBP Higher in Kids Who Survive

164 Children with an ICU arrest (25/30 mmHg)
Survival more than doubled when DBP cutoffs achieved

Berg, Circulation 2017
Hemodynamic-Directed Training Tools

Collaboration with Biomedical Engineers

Monitoring and Titrating to a Clinical Endpoint makes sense to Clinical Care Providers

TABLE 3. Multivariable Model Adjusted for Clustering on Subject

<table>
<thead>
<tr>
<th></th>
<th>OR</th>
<th>95% CI</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acquisition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posttraining</td>
<td>5.2</td>
<td>1.3-21.2</td>
<td>0.02</td>
</tr>
<tr>
<td>Retention</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12 h</td>
<td>4.4</td>
<td>1.3-14.9</td>
<td>0.018</td>
</tr>
<tr>
<td>3 mo</td>
<td>4.1</td>
<td>1.2-13.9</td>
<td>0.023</td>
</tr>
</tbody>
</table>
In-Hospital CPR ≈ Out-of-Hospital CPR

~200,000 IHCA annually in USA

Majority in ICUs

~95% among children

>60% among adults

Invasive monitoring

Highly trained teams

JUST LOOK UP!

Get with the Guidelines - Resuscitation

Berg et. al., CCM 2013

Merchant, Crit Care Med 2011; Girotra, NEJM 2012
Pressure ≠ Flow
Expired CO$_2$ (ETCO$_2$) Represents Flow

Blood flows out to the body

Gas exchange in the tissues: CO$_2$ is picked up

Better CCs
Move more blood

Higher ETCO$_2$
Translational Evidence:
End-Tidal CO₂ is Proportional to Cardiac Output

Weil, Crit Care Med 1985


Slide adapted from Neumar SCCM 2018
ETCO$_2$ in Adult IHCA / OHCA

N=34 patents

Average end tidal CO2

Resuscitated: 15 ± 4
Nonresuscitated: 7 ± 5

Sanders, JAMA 1989
Quantitative relationship between end-tidal carbon dioxide and CPR quality during both in-hospital and out-of-hospital cardiac arrest

Kelsey R. Sheak, Douglas J. Wiebe, Marion Leary, Saeed Babaeizadeh, Trevor C. Yuen, Dana Zive, Pamela C. Owens, Dana P. Edelson, Mohamud R. Daya, Ahamed H. Idris, Benjamin S. Abella

583 cardiac arrest events (both OHCA and IHCA) ~30,000 chest compressions analyzed

Table 4
Assessment of overall case resuscitation characteristics by clinical outcomes.

<table>
<thead>
<tr>
<th></th>
<th>ROSC</th>
<th>No ROSC</th>
<th>p-Value</th>
<th>Survival to discharge</th>
<th>No survival to discharge</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate (cpm)</td>
<td>109.0 ± 8.4</td>
<td>110.0 ± 8.7</td>
<td>NS</td>
<td>430.0 ± 12.1</td>
<td>440.0 ± 8.3</td>
<td>NS</td>
</tr>
<tr>
<td>Depth (mm)</td>
<td>44.5 ± 8.0</td>
<td>41.8 ± 8.5</td>
<td>NS</td>
<td>372.0 ± 7.7</td>
<td>340.0 ± 8.3</td>
<td>NS</td>
</tr>
<tr>
<td>ETCO₂ (mmHg)</td>
<td>34.5 ± 4.5</td>
<td>31.1 ± 3.7</td>
<td>&lt;0.001</td>
<td>323.2 ± 12.1</td>
<td>311.1 ± 15.2</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Rescuer-Centric Targets Not Different
Patient-Centric Physiology Different

All values are means ± standard deviation. cpm, compressions per minute; NS, not significant.
No Signal During Pediatric CPR

Multicenter study of 43 intubated children (HLHS excluded)
Primary evaluation: AHA target of 20mmHg

<table>
<thead>
<tr>
<th>Relative Risk (95% CI)</th>
<th>P-value</th>
<th>Relative Risk (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean ETCO2 (mmHg) over the first ten minutes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ETCO2 over the first ten minutes &gt; 20 mmHg</td>
<td>1.32 (0.89, 1.95)</td>
<td>0.162</td>
<td>0.92 (0.65, 1.29)</td>
</tr>
<tr>
<td>Mean ETCO2 over the first ten minutes &gt; 25 mmHg</td>
<td>1.02 (0.74, 1.43)</td>
<td>0.899</td>
<td>0.86 (0.56, 1.32)</td>
</tr>
<tr>
<td>Mean ETCO2 over the first ten minutes &gt; 30 mmHg</td>
<td>1.05 (0.64, 1.74)</td>
<td>0.848</td>
<td>0.86 (0.24, 3.06)</td>
</tr>
<tr>
<td>Mean ETCO2 categories</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 20 mmHg</td>
<td>Reference</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20 - &lt; 25 mmHg</td>
<td>1.56 (1.01, 2.42)</td>
<td>0.267</td>
<td>1.03 (0.36, 2.93)</td>
</tr>
<tr>
<td>25 - &lt; 30 mmHg</td>
<td>1.17 (0.72, 1.92)</td>
<td>0.688</td>
<td>0.88 (0.29, 2.63)</td>
</tr>
<tr>
<td>&gt; 30 mmHg</td>
<td>1.26 (0.73, 2.21)</td>
<td>0.052</td>
<td>0.84 (0.23, 3.00)</td>
</tr>
<tr>
<td>All ETCO2 &lt; 10 mmHg</td>
<td>0.71 (0.25, 2.00)</td>
<td>0.520</td>
<td>0.60 (0.11, 3.39)</td>
</tr>
<tr>
<td>All ETCO2 &lt; 15 mmHg</td>
<td>0.88 (0.55, 1.41)</td>
<td>0.593</td>
<td>1.17 (0.45, 3.02)</td>
</tr>
<tr>
<td>All ETCO2 &lt; 20 mmHg</td>
<td>1.03 (0.70, 1.50)</td>
<td>0.886</td>
<td>1.28 (0.55, 3.00)</td>
</tr>
</tbody>
</table>
A Sudden Increase in Partial Pressure End-Tidal Carbon Dioxide at the Moment of Return of Spontaneous Circulation

Pokorná 2010

Out-of-hospital cardiac arrest
Adults (n=108)
2 Groups
• 59 with single episode of ROSC
• 49 with no signs of ROSC

ROSC = pulse

ETCO₂ ~10mmHg higher after ROSC (27 vs. 37)

End-tidal carbon dioxide monitoring during CPR may be unreliable in the following clinical circumstances:

A. Leak around invasive airways
B. Vasopressor administration
C. Sodium bicarbonate administration
D. All of the above

Cerebral Oxygen Saturation: NIRS

Non-invasive
Subtle differences
- emitters, detectors
- lowest detectable values
- ambient light sensitivity

Can measure CPR quality when pulsatile flow is not present

Genbrugge, Journal of Emergency Medicine, 2016
Cerebral Oximetry During Cardiac Arrest: A Multicenter Study of Neurologic Outcomes and Survival*

5 sites (US and UK)
N=183
ED patients with OHCA (n=100)
AUCs were similar (~0.7)
Poor correlation
ETCO₂ more sensitive
rSO₂ more specific
A new method to detect cerebral blood flow waveform in synchrony with chest compression by near-infrared spectroscopy during CPR

Yasuaki Koyama, MD *, Takafumi Wada, MD, Brandon D. Lohman, MD, Yuka Takamatsu, MD, Junichi Matsumoto, MD, Shigeki Fujitani, MD, Yasuhiko Taira, MD
Pulse Oximetry: A Non-Invasive, Novel Marker for the Quality of Chest Compressions in Porcine Models of Cardiac Arrest

Jun Xu*, Chen Li†, Liangliang Zheng‡, Fei Han†, Yan Li†, Joseph Walline§, Yangyang Fu†, Dongqi Yao†, Xiaocui Zhang†, Hui Zhang†, Huadong Zhu†, Shubin Guo†, Zhong Wang§, Xuezhong Yu†

PLOS ONE | DOI: 10.1371/journal.pone.0139707 October 20, 2015

No Pulse = No Pulse Ox
Poor BP = Low Amplitude
Good BP = Better Amplitude

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Low Quality (3cm)</th>
<th>High Quality (5cm)</th>
<th>t/U</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (bpm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3min</td>
<td>104±6</td>
<td>104±5</td>
<td>0.251</td>
<td>0.803</td>
</tr>
<tr>
<td>6min</td>
<td>103±3</td>
<td>103±3</td>
<td>0.245</td>
<td>0.808</td>
</tr>
<tr>
<td>9min</td>
<td>103±2</td>
<td>103±2</td>
<td>0.532</td>
<td>0.593</td>
</tr>
<tr>
<td>Pao2/CO2 (mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3min</td>
<td>12±4</td>
<td>19±4</td>
<td>-4.830</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>6min</td>
<td>13±4</td>
<td>19±4</td>
<td>-3.948</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>9min</td>
<td>12±4</td>
<td>18±4</td>
<td>-4.690</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>3min</td>
<td>14±10</td>
<td>25.0±17.5</td>
<td>-2.146</td>
<td>0.040</td>
</tr>
<tr>
<td>3min</td>
<td>14±10</td>
<td>21±5</td>
<td>-2.301</td>
<td>0.028</td>
</tr>
<tr>
<td>CPP (mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3min</td>
<td>15±10</td>
<td>21±5</td>
<td>-2.221</td>
<td>0.034</td>
</tr>
<tr>
<td>3min</td>
<td>14±3</td>
<td>22±6</td>
<td>-1.823</td>
<td>0.036</td>
</tr>
<tr>
<td>Amp (PVA)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3min</td>
<td>70±82</td>
<td>189±129</td>
<td>-3.317</td>
<td>0.003</td>
</tr>
<tr>
<td>6min</td>
<td>71±63</td>
<td>194±132</td>
<td>-3.379</td>
<td>0.003</td>
</tr>
<tr>
<td>9min</td>
<td>79±81</td>
<td>188±119</td>
<td>-3.081</td>
<td>0.005</td>
</tr>
<tr>
<td>AUC (PVPG)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3min</td>
<td>221±852</td>
<td>319±556</td>
<td>-3.872</td>
<td>0.001</td>
</tr>
<tr>
<td>6min</td>
<td>221±781</td>
<td>319±517</td>
<td>-4.231</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>9min</td>
<td>202±665</td>
<td>307±422</td>
<td>-5.001</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
Can we alter our resuscitation technique to improve outcomes?

A translational perspective
Both Groups: VF Induced and manual CPR started

Standard Care: Depth = 1/3 anterior-posterior chest diameter
HD-CPR: Depth titrated to SBP 90/100 mmHg

$\text{t}_{7\text{ min}}$ ETT Clamp

$\text{t}_{7\text{ min}}$ Both Groups: VF Induced and manual CPR started
Standard Care: Depth = 1/3 anterior-posterior chest diameter
HD-CPR: Depth titrated to SBP 90/100 mmHg

$\text{t}_{9\text{ min}}$ HD-CPR: First vasopressor given if CoPP < 20 mmHg
Dosing order: Epinephrine $\rightarrow$ Epinephrine $\rightarrow$ Vasopressin (0.4U/kg) if CoPP < 20
Dosing Interval: 1 min. after Epinephrine; 2 min. after Vasopressin

$\text{t}_{9\text{ min}}$ Standard Care: Epinephrine (0.02mg/kg)

$\text{t}_{13\text{ min}}$ Standard Care: Epinephrine

$\text{t}_{17\text{ min}}$ First Defibrillation Attempt

$\text{t}_{27\text{ min}}$ Experiment end if no ROSC

**Asphyxial Period**

**CPR Period**

VENTRICULAR FIBRILLATION

ETT Clamped

CPR (Rate 100 min$^{-1}$, ventilations 6-10 min$^{-1}$, 100% FiO$_2$) and Vasopressors
# Asphyxia-Associated Cardiac Arrest

![Graph showing coronary perfusion pressure during CPR](image)

## First 10 Minutes of CPR

<table>
<thead>
<tr>
<th>Coronary Perfusion Pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP-Care</td>
</tr>
<tr>
<td>Guideline care</td>
</tr>
</tbody>
</table>

## Table: Survival and Neurological Outcome

<table>
<thead>
<tr>
<th>Survival</th>
<th>Depth 51 (n=10)</th>
<th>CPP-20 (n=10)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>45 Minute ICU Survival</td>
<td>1 (10)</td>
<td>9 (90)</td>
<td>p = 0.001</td>
</tr>
<tr>
<td>24 Hour Survival</td>
<td>0 (0)</td>
<td>8 (80)</td>
<td>p = 0.001</td>
</tr>
<tr>
<td>Good Neurological Outcome</td>
<td>0 (0)</td>
<td>7 (70)</td>
<td>p = 0.003</td>
</tr>
</tbody>
</table>

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Sutton, Resuscitation 2013; Friess, CCM 2013; Sutton, AJRCCM 2014; Naim, CCM 2016; Morgan, AJRCCM 2017; Morgan, Resuscitation 2017
What’s next?

Pediatric asphyxia model
Adult asphyxia model
Adult VF model

But what about a progressive shock state?
LPS model
The first 3 animals with HD-CPR died
Pulmonary HTN key physiologic roadblock
Shock Associated Cardiac Arrest

Coronary Perfusion Pressure (mmHg)

First Ten Minutes of Cardiopulmonary Resuscitation

Higher CPPs with iNO
Better Survival: 100% (10/10) vs. 27% (3/11); p=0.001

Morgan, AJRCCM 2017
Bolus vs. Bolus + Continuous Epinephrine During CPR

N=24 animals
Laser Doppler CBF

Johansson Resuscitation 2003

Epi bolus: 20 ug/kg
Epi Infusion: 10 ug/kg/min

P=0.009
End Tidal CO₂ Guided Cardiopulmonary Resuscitation

Feedback optimized

VS

ETCO₂ Guided

Hamrick, JAHA 2014; PCCM 2017
Experimental Timeline

-20 minutes: Begin 20 minutes of asphyxia (ETT clamped).

-6 minutes: Induce fibrillation (asphyxia continues).

0 minutes: Begin 10 min BLS (chest compressions and ventilation).

10 minutes: Begin 10 min ALS (BLS plus epinephrine every 4 min and defibrillation every 2 min).

20 minutes: If no ROSC then non-survivor. If ROSC during ALS then additional 20 min observation for continuance of ROSC without intervention.
End Tidal CO₂ Guided CPR: Neonatal Asphyxial Arrest

**Mean Arterial Pressure**

- **Variables:** MAP mmHg
- **Graphs:**
  - STD vs. ETCO₂
  - X-axis: Minutes: BLS → ALS → ALS

**Cerebral Perfusion Pressure**

- **Variables:** CPP mmHg
- **Graphs:**
  - STD vs. ETCO₂
  - X-axis: Minutes: BLS → ALS → ALS

**Outcome Results**

<table>
<thead>
<tr>
<th>Variables</th>
<th>End-Tidal CO₂ Group</th>
<th>Standard Group</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Successful ROSC, n (%)</td>
<td>7/14 (50)</td>
<td>2/14 (14)</td>
<td>0.04</td>
</tr>
</tbody>
</table>

Hamrick, J AHA 2014; PCCM 2017
So if you had to pick...
Pilot Study to Compare the Use of End-Tidal Carbon Dioxide–Guided and Diastolic Blood Pressure–Guided Chest Compression Delivery in a Swine Model of Neonatal Asphyxial Cardiac Arrest

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Method specific physiologic changes
A quantitative comparison of physiologic indicators of cardiopulmonary resuscitation quality: Diastolic blood pressure versus end-tidal carbon dioxide

DBP performed better than ETCO₂
True across all models
asphyxia
VF
Optimal DBP target: ~34mmHg

N = 60

P = 0.025
The technique is not broadly implemented...

- 4% used ETCO$_2$
- 30% used diastolic pressure
- Event survival increased more than 20% when physiology was used
Evaluation of out-of-hospital cardiac arrest using transesophageal echocardiography in the emergency department

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9 / 17 cases with AMC over LV outflow tract
TEE used to reposition to appropriate AMC

\textbf{MELAX**}

LV, LA and RV chambers
LV Outflow Tract
Aortic root
Mitral and Aortic Valves

Determination of area of maximal compression (AMC) during CPR
Disclaimer

The interpretation of the literature / opinions rendered by me do not necessarily reflect the official policy or position of the American Heart Association or its volunteers, the Children’s Hospital of Philadelphia, the University of Pennsylvania, or anyone else for that matter.

Please direct complaints to Dr. Robert A. Berg, Division Chief, bergra@chop.edu
Minute 1
Ensure High Quality Chest Compressions
Evaluate Rhythm:
• If VF/pVT, prompt defibrillation
• If PEA, consider early echocardiogram (Box A)
Draw arterial blood gas (q6 min)
Consider physiologic monitor placement
Prepare for vasopressor, fluid, and inhaled nitric oxide administration

Minute 2-6
Assessment of Hemodynamic Goals
Consider reassessment every 1 minute

CVP during CC pauses
< 10
• Consider fluid bolus
• Consider ITD
> 20
Consider POCUS

Diastolic BP During CC
< Age specific target
• Epinephrine up to every 1 minute
• Consider epinephrine infusion (minute 5)
• Consider vasopressor rescue

Systolic BP During CC
< Age specific target
• Increase CC Depth / Rate
• Consider epinephrine
• Optimize CC Impulse with POCUS

Minute 6
Consider activation of E-CPR Protocol
Consider Ventilation Adjustment (Box B)

Details
High Quality CPR Mechanics
• Depth: > 1/3 AP Chest Depth
• Rate: 100-120/min
• Ensure full chest recoil
• Minimize interruptions

Diastolic BP Targets
≥ 25mmHg in infants
≥ 30mmHg in children > 1 year

Systolic BP Targets
≥ 60mmHg in infants
≥ 80mmHg in children > 1 year

Epinephrine
Bolus: 0.01 mg/kg
Infusion: 0.001 mg/kg/min

Vasopressin Rescue / Replacement
Consider 0.4 units/kg/dose:
• after 2nd epinephrine dose
• high probability of PHTN

Box A: POCUS
PEA:
• Treat pneumothoraces / effusions
• Sepsis / PHTN: Consider iNO 80ppm
• If high probability of PE, consider tPA

Box B: Ventilation
Respiratory etiology and BP at goal:
• Consider ventilation rate increase
Adult Goal-Directed CPR Algorithm
Univ. of Michigan Emergency Department

1. **Standard ACLS** (*quality CPR, rhythm check, shock VF/VT, Epi 1mg IV/IO q5min*)

2. **Place Advanced Airway & Attach Waveform Capnography**
   - **ENTER PATHWAY**
     - If ETCO2 <20mmHg
   - **Optimize Chest Compressions**
   - **Switch to LUCAS-2**
   - **Add Res-Q Pod**
   - **Switch to CARDIO-PUMP**
   - **EXIT PATHWAY**
     - If ETCO2 >20mmHg

   A. May switch to LUCAS-2 regardless of ETCO2
   B. REMOVE Res-Q Pod after ROSC or ECPR started

3. **Place Right Femoral Arterial + Venous 5Fr Catheters & transduce arterial BP**
   - **ENTER PATHWAY**
     - If DBP <35mmHg
   - **Start Epinephrine Infusion:** 1 mcg/kg/min, continue until ROC
   - **Epinephrine 1mg q5min**, until DBP>35 +/- **Vasopressin 40u, x1 only**
   - **EXIT PATHWAY**
     - If DBP >35mmHg

   C. May titrate Epi pushes to achieve DBP>35
   D. May substitute Vasopressin for one Epi dose
Conclusions

• Physiologic-directed CPR is not a new concept
• Decades of experimental evidence
• Targeting underlying pathophysicsiology is key
  – More than just BP or end tidal or regional saturation
• Human trials are needed to obtain the evidence necessary to support widespread implementation
It’s why we do what we do!

If not us, who?
THANK YOU
Personalizing Resuscitation with Physiologic-Directed CPR

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