MY PATIENT WAS RESUSCITATED, NOW WHAT?
Continuing Nursing and Allied Health Education Provider:

Pfiedler ENTERPRISES
Developed for:

HEALTHTRUST EDUCATION

Funding provided by:

PHYSIOCONTROL
Nicole Kupchik MN, RN, CCNS, CCRN, PCCN-CMC

- Clinical Nurse Specialist
- Former Code Blue Committee Co-Chair
- Currently consultant
- Staff Nurse

National resuscitation presentations:
- American Heart Association (AHA)
- Emergency Cardiovascular Care Updates (ECCU)
- Society of Critical Care Medicine (SCCM)
- National Teaching Institute (NTI)
- Emergency Nurses Association (ENA)
MY PATIENT WAS RESUSCITATED, NOW WHAT?

HealthTrust Resuscitation Webinar Series
4-Part Resuscitation Webinar Series

September 28th – What’s New with the ACLS & BLS Guidelines?

December 20th – High Quality CPR & Why It Matters!

February 1st – Capnography: It’s about more than ventilation!

March 1st – My Patient was Resuscitated, Now What?
Objectives

- Discuss the 2015 AHA Guideline Updates for Post Cardiac Arrest Care
- Discuss oxygenation & hemodynamic targets
- Discuss the literature supporting Targeted Temperature Management post Cardiac Arrest
Trends in Resuscitation

- Pre-hospital phase
- Early defibrillation
- High Quality CPR
- Post arrest: Supportive care
Return of spontaneous circulation (ROSC)

Optimize ventilation and oxygenation
- Maintain oxygen saturation ≥94%
- Consider advanced airway and waveform capnography
  Do not hyperventilate

Treat hypotension (SBP <90 mm Hg)
- IV/IO bolus
- Vasopressor infusion
- Consider treatable causes

Coronary reperfusion

12-Lead ECG: STEMI
OR high suspicion of AMI

Follow commands?
- Yes
- No

Initiate targeted temperature management

Advanced critical care

Doses/Details

Ventilation/oxygenation:
Avoid excessive ventilation. Start at 10 breaths/min and titrate to target PETCO₂ of 35-40 mm Hg. When feasible, titrate FiO₂ to minimum necessary to achieve SpO₂ ≥94%.

IV bolus:
Approximately 1-2 L normal saline or lactated Ringer’s

Epinephrine IV infusion:
0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)

Dopamine IV infusion:
5-10 mcg/kg per minute

Norepinephrine IV infusion:
0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)

Reversible Causes
- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo-/hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary
Overall ROSC Goals

- Does the patient need to go to the cath lab?
  - Assess the 12 Lead ECG
- Hemodynamic goals
  - Avoid hypotension
  - Monitor Capnography post arrest!
- Avoid post arrest Hypoxemia or Hyperoxemia
- Targeted Temperature Management
Hemodynamic goals?

- **SBP < 90 mmHg** associated with worse outcomes
  - Trzeciak et al (2009), Crit Care Med
  - Bray et al (2014) Resuscitation

- **MAP > 100 mmHg** during 2 hrs after ROSC associated with better neurologic recovery

- Study with “bundle” of care including MAP > 80 mmHg associated with higher survival & neuro outcomes
Avoid & immediately correct SBP < 90 mmHg, MAP < 65 mmHg
- Class IIb, LOE C-LD

Identify optimal MAP for the patient
Oxygenation

Hypoxia:
\[ \text{PaO}_2 < 60 \]
\[ \text{P/F ratio} < 300 \]

Hyperoxia:
\[ \text{PaO}_2 > 300 \] or

2015 ACLS Update:
- 100% FiO\(_2\) until ROSC
- Avoid hypoxia or hyperoxia

Kilgannon et al (2010) JAMA

Graph showing survival proportion over days with norms and hyperoxia, with p < 0.001 for everyone lives and everyone dies.
To avoid hypoxia in adults with ROSC after CA, it is reasonable to use the highest available concentration of oxygen until the $O_2$ sat can be measured.

- Maintain $O_2$ sat $\geq 94$
  - Class IIa, LOE C-LD
What about the brain?

The New England Journal of Medicine

VOLUME 346  FEBRUARY 21, 2002  NUMBER 8

MILD THERAPEUTIC HYPOTHERMIA TO IMPROVE THE NEUROLOGIC OUTCOME AFTER CARDIAC ARREST

The Hypothermia after Cardiac Arrest Study Group*

INDUCED HYPOTHERMIA AFTER OUT-OF-HOSPITAL CARDIAC ARREST

TREATMENT OF COMATOSE SURVIVORS OF OUT-OF-HOSPITAL CARDIAC ARREST WITH INDUCED HYPOTHERMIA

Median initiation of cooling = 105 min.
Median time to goal temp = 8 hours
Active surface cooling protocol to induce mild therapeutic hypothermia after out-of-hospital cardiac arrest: A retrospective before-and-after comparison in a single hospital*

Creighton W. Don, MD, PhD; W. T. Longstreth Jr, MD; Charles Maynard, PhD; Michele Olsufka, RN; Graham Nichol, MD; Todd Ray, RN; Nicole Kupchik, RN; Steven Deem, MD; Michael K. Copass, MD; Leonard A. Cobb, MD; Francis Kim, MD

491 patients from January 1, 2001 – December 31, 2004

Critical Care Medicine, 2009
Why cool?
To minimize reperfusion injury!

- Depleted stores of $O_2$ & glucose
- Intracellular calcium influx
- Formation of $O_2$ free radicals
- Release of glutamate
- Intracellular acidosis
- Disruption in blood brain barrier
- Mitochondrial injury
- Apoptosis

Polderman, KH Crit Care Med (2009); 37:S186-202
CT scan (ED)
30 y.o. s/p asystolic arrest
Hypoxic-Ischemic Brain Injury 36 hours later

Note: Loss of distinction between gray & white matter in the cerebral hemispheres
Who should be cooled?

- Out-of-Hospital
- Ventricular Fibrillation
- Ventricular Tachycardia

What about:
- Asystole?
- PEA?
- In-Hospital arrests?
- Drowning?
- Electrocution?
- Asphyxiation?

Common side effects of mild hypothermia (32 - 34°C) include(s):

A. Bradycardia
B. Diuresis
C. Decreased cardiac output
D. Hypokalemia
E. Decreased medication clearance
F. Hyperglycemia
G. All of the above
Shivering Management

- Should have a protocol for it!
- Skin counter-warming
- Magnesium
- Buspirone
- Acetaminophen (consider IV dosing)

- Propofol
- Dexmedetomidine (Precedex)
- Neuromuscular blockade + sedative

<table>
<thead>
<tr>
<th>Step</th>
<th>Intervention</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Baseline</td>
<td>Acetaminophen 650–1000 mg Q 4–6 h</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Buspirone 30 mg Q 8 h</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Magnesium sulfate 0.5–1 mg/h IV Goal (3–4 mg/dl)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Skin counterwarming 43°C/MAX Temp</td>
</tr>
<tr>
<td>1</td>
<td>Mild sedation</td>
<td>Dexmedetomidine 0.2–1.5 mcg/kg/h</td>
</tr>
<tr>
<td></td>
<td></td>
<td>or Fentanyl starting dose 25 mcg/h</td>
</tr>
<tr>
<td></td>
<td></td>
<td>or Meperidine 50–100 mg IM or IV</td>
</tr>
<tr>
<td>2</td>
<td>Moderate sedation</td>
<td>Dexmedetomidine and Opioid Doses as above</td>
</tr>
<tr>
<td>3</td>
<td>Deep sedation</td>
<td>Propofol 50–75 mcg/kg/min</td>
</tr>
<tr>
<td>4</td>
<td>Neuromuscular blockade</td>
<td>Vecuronium 0.1 mg/kg/min</td>
</tr>
</tbody>
</table>

Choi et al (2011) Neuro Crit Care

Columbia University shivering protocol
Should we be infusing iced saline post ROSC to “kick start” cooling?

A. Yes, it works!!!

B. No, the evidence doesn’t support it

C. Only if you have it available
Pre-Hospital Iced Saline

1359 Cardiac Arrest

583 VFIB Arrest
292 Intervention, 291 control

776 without VFIB
396 Intervention, 380 control

- 2 L - 4°C Iced Saline
- Nearly all VFIB cases were admitted to the hospital and received cooling (despite randomization group)
- Decreased temperature by 1.2°C
- Decreased time to goal temperature by ~ 1 hour

Kim et al JAMA (2013)
# Table 1. Baseline Characteristics of Randomized Eligible Patients (n=1359)\textsuperscript{a}

<table>
<thead>
<tr>
<th></th>
<th>With Ventricular Fibrillation</th>
<th>Without Ventricular Fibrillation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intervention (n = 292)</td>
<td>Control (n = 291)</td>
</tr>
<tr>
<td>Age, y</td>
<td>62.1 (14.2)</td>
<td>62.1 (15.6)</td>
</tr>
<tr>
<td>Men, No. (%)</td>
<td>227 (78)</td>
<td>217 (75)</td>
</tr>
<tr>
<td>Witnessed cardiac arrest, No. (%)</td>
<td>208 (71)</td>
<td>215 (74)</td>
</tr>
<tr>
<td>CPR before EMS arrival, No. (%)</td>
<td>199 (68)</td>
<td>186 (64)</td>
</tr>
<tr>
<td>Time from call to randomization, min</td>
<td>(n = 288)</td>
<td>32.9 (10.6)</td>
</tr>
<tr>
<td>Time from call to first responder arrival, min</td>
<td>(n = 290)</td>
<td>5.3 (2.0)</td>
</tr>
<tr>
<td>Sustained ROSC, No. (%)</td>
<td>273 (94)</td>
<td>274 (94)</td>
</tr>
<tr>
<td>Time from call to sustained ROSC, min</td>
<td>(n = 142)</td>
<td>25 (14)</td>
</tr>
<tr>
<td>Time to first shock, min\textsuperscript{b}</td>
<td>(n = 175)</td>
<td>9.4 (3.3)</td>
</tr>
<tr>
<td>Heart rate at randomization, beats/min</td>
<td>(n = 284)</td>
<td>109 (28)</td>
</tr>
<tr>
<td>Systolic blood pressure at randomization, mm Hg</td>
<td>(n = 271)</td>
<td>140 (37)</td>
</tr>
</tbody>
</table>
# Table 2: Status at Time of Discharge

<table>
<thead>
<tr>
<th></th>
<th>With Ventricular Fibrillation (n = 583)</th>
<th>Without Ventricular Fibrillation (n = 776)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Intervention (n = 292)</td>
<td>Control (n = 291)</td>
<td></td>
</tr>
<tr>
<td>No. (%) [95% CI]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dead</td>
<td>109 (37.3) [32.0-43.0]</td>
<td>104 (35.7) [30.5-41.4]</td>
<td>.69</td>
</tr>
<tr>
<td>Alive</td>
<td>183 (62.7) [57.0-68.0]</td>
<td>187 (64.3) [58.6-69.5]</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>.30</td>
</tr>
<tr>
<td></td>
<td>Intervention (n = 396)</td>
<td>Control (n = 380)</td>
<td></td>
</tr>
<tr>
<td>No. (%) [95% CI]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vital status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dead</td>
<td>320 (80.8) [76.6-84.4]</td>
<td>318 (83.7) [79.6-87.1]</td>
<td></td>
</tr>
<tr>
<td>Alive</td>
<td>76 (19.2) [15.6-23.4]</td>
<td>62 (16.3) [12.9-20.4]</td>
<td></td>
</tr>
<tr>
<td>Neurological status at discharge</td>
<td></td>
<td></td>
<td>.59</td>
</tr>
</tbody>
</table>
Figure 2. The Proportion of Comatose Patients Achieving Either Death Without Awakening or Awakening as a Function of Days After Cardiac Arrest for Enrolled Patients

A With ventricular fibrillation

B Without ventricular fibrillation
Results

- Pre-Hospital cooling (via iced saline) made no difference in mortality or neurologic outcomes.

- Increased diuretic use & higher incidence of pulmonary edema on initial chest x-ray with pre-hospital iced-saline.

- Re-arrest 26% (treatment group) vs. 21% ($p = 0.008$).
Post-Arrest Optimal Temperature?

33°C vs. 36°C
Which temperature goal is preferred?

A. Hypothermia ranging from 32 - 34° C
B. 36° C
C. Normothermia
D. 32 - 36 ° C
E. Controlling temperature hasn’t been shown to be beneficial
Characteristics

- ~ 80% VFIB
- Received BLS within 1 min

Nielsen et al (2013) NEJM
**Results (at 180 days):**

- **RCT 950 patients** – Temp 33°C vs. 36°C
- **36 Hospitals** – 10 countries
- **Catheter 24%**, surface cooling 76%

### Table 2. Outcomes.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>33°C Group</th>
<th>36°C Group</th>
<th>Hazard Ratio or Risk Ratio (95% CI)*</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>no./total no. (%)</td>
<td>no./total no. (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Primary outcome: deaths at end of trial</strong></td>
<td>235/473 (50)</td>
<td>225/466 (48)</td>
<td>1.06 (0.89–1.28)</td>
<td>0.51</td>
</tr>
<tr>
<td><strong>Secondary outcomes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurologic function at follow-up†</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPC of 3–5</td>
<td>251/469 (54)</td>
<td>242/464 (52)</td>
<td>1.02 (0.88–1.16)</td>
<td>0.78</td>
</tr>
<tr>
<td>Modified Rankin scale score of 4–6</td>
<td>245/469 (52)</td>
<td>239/464 (52)</td>
<td>1.01 (0.89–1.14)</td>
<td>0.87</td>
</tr>
<tr>
<td>Deaths at 180 days</td>
<td>226/473 (48)</td>
<td>220/466 (47)</td>
<td>1.01 (0.87–1.15)</td>
<td>0.92</td>
</tr>
</tbody>
</table>

Nielsen et al (2013) NEJM
## 2015 Targeted Temperature Management Levels of Evidence – ILCOR/AHA

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Class</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Recommend against routine pre-hospital cooling of patients with ROSC with rapid infusion of cold IV fluids – No Harm</td>
<td>III</td>
<td>A</td>
</tr>
<tr>
<td>Comatose adult patients with ROSC after CA should have Targeted Temperature Management. For Vfib/pVT OHCA: For non Vfib/pVT (PEA &amp; Asystole) &amp; IHCA:</td>
<td>I</td>
<td>B-R</td>
</tr>
<tr>
<td>Maintain temperature 32 - 36° C</td>
<td>I</td>
<td>CB-R</td>
</tr>
<tr>
<td>TTM for a minimum of 24 hours after achieving ROSC</td>
<td>Ila</td>
<td>C-EO</td>
</tr>
<tr>
<td>It may be reasonable to actively prevent fever in comatose patients after TTM</td>
<td>Iib</td>
<td>C-LD</td>
</tr>
</tbody>
</table>

## Physiologic parameters 32 – 34°C vs. 36°C

<table>
<thead>
<tr>
<th>Physiologic parameter</th>
<th>32 - 34°C</th>
<th>36°C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bradycardia</td>
<td>Yes, but not harmful</td>
<td>Not as much</td>
</tr>
<tr>
<td>Shivering</td>
<td>Yes, threshold less once at goal temperature</td>
<td>Yes!!!</td>
</tr>
<tr>
<td>Electrolyte shifts</td>
<td>Yes, especially potassium &amp; magnesium</td>
<td>Not as much</td>
</tr>
<tr>
<td>Drug clearance</td>
<td>Prolonged</td>
<td>Not as much</td>
</tr>
<tr>
<td>Cold induced diuresis</td>
<td>Yes</td>
<td>Not as much</td>
</tr>
</tbody>
</table>
Clinical assessment:

- Does mild hypothermia (32 - 34°C) reduce mortality & improve neurologic outcomes post cardiac arrest?
  - YES!!!

- Does 36°C have the same benefit?
  - YES!!!

- Does “normothermia” have the same benefit?
  - We don’t know!!!

- Is fever bad post-cardiac arrest?
  - Very Likely!!!!
Re-warming

- Important to re-warm slowly
  - Vasodilation, hypotension if too quick
- Minimum 8 - 12 hours
  - ~0.15 - 0.25°C per hour
- If re-warm too quickly, can possibly negate benefits
  - Poor neuro outcomes in TBI/ Stroke*
- Rebound hyperthermia

Re-warming
In conclusion,

- Resuscitation involves a system of care, all being inter-dependent on improving outcomes.
- Oxygen should be normalized.
- Hemodynamic goals should be clearly identified & individualized for the patient.
- Temperature should be managed to 32 - 36°C for 24 hours in patients resuscitated cardiac arrest.
Following the Q & A session, the webinar will adjourn, and you will be directed to the Pfiedler Enterprises website to complete a course evaluation and will receive a printable certificate.
Thank you for attending this continuing education presentation.