What’s New in Managing Cardiac Arrest

South Miami Heart Center Comprehensive Cardiovascular Symposium
J. Francisco Rodriguez-Moran MD, FACCP, FAASM
Director of Critical Care Services
South Miami Hospital

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Cardiac Arrest Epidemiology

- 350000 people/year suffer a cardiac arrest and receive attempted resuscitation in the US and Canada.
- Half of those occur in the hospital.
- There are 50 to 55/100000 persons/year EMS-treated cardiac arrest in the US and Canada.
- There is an incidence of 3 to 6/1000 admissions of in-hospital cardiac arrest.
- 25% of all cardiac arrest present with pulseless ventricular arrhythmias.
- The vast majority of victims are adults.

Major Changes in the 2010 Guidelines

- Change from “A-B-C” to “C-A-B”.
- “Look, Listen and Feel” has been eliminated.
- Encouraged Hands-Only CPR for the untrained lay rescuer.
- Compression-ventilation ratio of 30:2 for the trained rescuer.
- Minimize interruptions in effective chest compressions.
• No stacked defibrillations.
• No transcutaneous pacing for asystolic arrest.
• No more atropine for PEA or asystolic arrest.
• Monitor patient’s physiological response with advanced techniques (CPP, Arterial diastolic pressure, Capnography, ScVO2).
• Monitor CPR performance.
• Post-cardiac arrest care.

CPR Quality: Improving Cardiac Resuscitation Outcomes Both Inside and Outside the Hospital
A Consensus Statement From the American Heart Association
on behalf of the CPR Quality Summit Investigators, the American Heart Association Emergency Cardiovascular Care Committee, and the Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation.
Circulation. 2013: published online before print June 25, 2013, 10.1161/CIR.0b013e31829d8654

Why now? Between Guidelines?

High Quality CPR is the Primary Component Influencing Survival
Chest Compression Fraction

Recommendations
1. High-quality CPR should be recognized as the foundation on which all other resuscitative efforts are built. Target CPR performance metrics include:
   a. CCF greater than 80%
   b. Compression rate of 100 to 120/min
   c. Compression depth of at least 50 mm in adults with no residual leaning
      i. (At least one third the anterior-posterior dimension of the chest in children)
   d. Avoid excessive ventilation by achieving minimal chest rise
      i. With BVM during CPR deliver 2 synchronized breaths with every 30 compressions.
      ii. With protected (e.g. intubated) airway by delivering 1 breath, asynchronous with compressions, every 7 to 8 seconds.
      iii. During rescue breathing (with a pulse & no compressions) at a rate of less than 12 breaths per minute.

Recommendations
2. At every cardiac arrest attended by professional rescuers
   a. Use at least 1 modality of monitoring the team’s CPR performance
   b. Depending on available resources, use at least 1 modality of monitoring the patient’s physiological response to resuscitative efforts
   c. Continually adjust resuscitative efforts based on the patient’s physiological response

How are those Components Measured?
1. Patient’s response to resuscitative efforts
2. Rescuers performance
Patient Physiological Response to Resuscitative Efforts

- CPP should be the primary endpoint when arterial and central venous catheters are in place.
- Should be kept > 20 mmHg.
- Arterial diastolic pressure > 25 mmHg if arterial catheter only.
- Scvo2 > 30%
- ETCO2 > 20 mmHg

Recommendations

4. Systems of care (EMS system, hospital, and other professional rescuer programs) should
   a. Determine a coordinated code team response with specific role responsibilities to ensure that high-quality CPR is delivered during the entire event.
   b. Capture CPR performance data in every cardiac arrest and use an ongoing CPR CQI program to optimize future resuscitative efforts.
   c. Implement strategies for continuous improvement in CPR quality and incorporate education, maintenance of competency, and review of arrest characteristics that include available CPR quality metrics.

Therapeutic Hypothermia for Comatose Survivor of Cardiac Arrest

* Image of a Newsweek cover with the headline: "This Man Was Dead. He Isn’t Anymore."
Sudden Cardiac Arrest (SCA): The Facts

- Only 40,000 patients survive to hospital admission.
- About one third of this patients survive to hospital discharge.
- 80% of patients remain comatose for over 1 hour after resuscitation.
- Fewer than 50% of admitted patients have good neurological recovery.

NEJM 2010; 363: 1256-64

Double Trouble

Phase I: Ischemia
- Lost of oxygen, glucose and ATP stores.
- Loss of transmembrane electrochemical gradients.
- Failure of synaptic transmission, axonal conduction and action-potential firing.
- Release of excitatory neurotransmitters (glutamate).
- Accumulation of intracellular calcium.
- Cell death by neuronal necrosis and apoptosis.

Phase II: Reperfusion
- Initial transient hyperemia due to vasomotor paralysis.
- Delayed global and multifocal hypoperfusion.
- Production of reactive oxygen species (lipid peroxidation).
- Endothelial activation.
- Leukocyte infiltration.
- Release of inflammatory products (cytokines, interleukins).
- Brain edema.
Brain Injury After Cardiac Arrest

Mild Resuscitative Hypothermia: “Neuroprotection”

- TH reduces brain metabolism (oxygen and ATP consumption).
- Inhibits the release of glutamate and dopamine.
- Induces brain derived neurotrophic factor.
- Reduces calcium overload.
- Attenuates oxidative stress and reduces lipid peroxidation.
- Inhibits cell death (apoptosis) by inducing Bcl-2 and suppressing BAX.
- TH Attenuates brain edema and blood-brain barrier damage.

(Kochanek et al., 2009)

**Effect of therapeutic hypothermia during the early phase after initial brain injury**

<table>
<thead>
<tr>
<th>Timing</th>
<th>Molecular Events</th>
<th>Effect of Hypothermia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early (0-40 min)</td>
<td>Decreased cerebral flow causes consumption of ATP, Lactate also decreased.</td>
<td><em>TH reduces O2 consumption, lowering metabolic and oxygen demand. ATP stores may be preserved.</em></td>
</tr>
<tr>
<td>Hypothermia (b)</td>
<td>This environment leads to increased glucose and other excitatory amino acids.</td>
<td><em>Inhibition of glutamate release</em></td>
</tr>
<tr>
<td></td>
<td>Glutamate union lowers intracellular Ca^2+ through activation of NMDA and AMPA receptors.</td>
<td><em>Suppression of the synthesis of excitatory neurotransmitters</em></td>
</tr>
<tr>
<td></td>
<td>Ca^2+ triggers in over release from intracellular stores.</td>
<td><em>Reduction of intracellular Ca^2+</em></td>
</tr>
<tr>
<td></td>
<td>Glutamate union increases intracellular anoxia, free radicals and excitotoxicity, which have pro-inflammatory effects.</td>
<td><em>Suppression of excitotoxicity</em></td>
</tr>
<tr>
<td>Late (up to 24 h)</td>
<td>Breakdown of the blood-brain barrier (BBD), with resultant brain edema and hemorrhagic transformation.</td>
<td><em>Diminution of breakdown of BBB, with less cerebral edema and possible reduction of hemorrhagic transformation.</em></td>
</tr>
</tbody>
</table>

(Kochanek et al., 2009)

<table>
<thead>
<tr>
<th>Hypothermia</th>
<th>Normothermia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Favorable</td>
<td>Hypothermia: 75/136 (55%)</td>
</tr>
<tr>
<td>Death</td>
<td>Hypothermia: 56/137 (41%)</td>
</tr>
</tbody>
</table>

HACA Study Group NEJM 2002;346:549-556
Primary percutaneous coronary intervention and mild induced hypothermia in comatose survivors of ventricular fibrillation with ST-elevation acute myocardial infarction

Rihard Knafej, Peter Radel, Tom Ploj, Marko Noc

- **Treated with TH**
  - 32 patients presented with STEMI and VF CA
  - Underwent primary PCI
  - 18 of the 32 pt (55%) had good outcomes based on cerebral performance categories (CPC 1 or 2)

- **Normothermia group**
  - 40 patients presented with STEMI and VF CA
  - Underwent primary PCI
  - Only 6 of the 40 pt (16%) had good outcomes based on cerebral performance categories (CPC 1 or 2)

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Therapeutic hypothermia after cardiac arrest
a successful translation from bench to bedside

- Hypothermia
- Normothermia

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


**Table 5. Outcome of Patients at Discharge from the Hospital:**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Hypothermia (N=40)</th>
<th>Normothermia (N=50)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal or minimal disability</td>
<td>15</td>
<td>7</td>
</tr>
<tr>
<td>Moderate disability (discharged to a rehabilitation facility)</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Severe disability, awake but completely dependent</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Severe disability, unconscious (discharged to a long-term nursing facility)</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Death</td>
<td>22</td>
<td>31</td>
</tr>
</tbody>
</table>

Bernard, SA, NEJM 2002
• TH is now a Class I intervention for out-of-hospital VF arrest
• Control body temperature to optimize survival and neurological recovery
• Concurrent PCI and hypothermia are safe, with good outcomes reported for some comatose patients who undergo PCI.

Traditional methods of cooling

Issues with ice and water blankets:
• Poor control during cooling and warming
• Not cardiac cath. lab friendly
• Difficulty to access the patient
• Labor intensive for nursing staff
• Imprecise: either unable to reach target or overcool to dangerously low

Ice-cold infusions: Out of hospital

Research
Mild hypothermia during advanced life support: a preliminary study in out-of-hospital cardiac arrest
Cédric Briesi, Jean-Jacques Parenti, William Marie, Xavier Arrêt, Cédric Baudoin, Damien Du Chuyren, Maxime Massot and Pierre Chauffournon

Key messages
• Therapeutic infusion of 2 L of normal saline at 4°C over 30 minutes during resuscitation following cardiac arrest safely and effectively lowers body temperature and can be used as part of ALS after OHCA.
• Inducing mild hypothermia in the field by EMS personnel is reproducible and cost-effective.
Surface Cooling

- Allows for rapid cooling
- Automatic precise patient temperature control: 32 C to 38 C utilizing esophageal probe
- Circulating water temperature automatically controls between 4 C to 42 C.
- Water temperature rises to 25 C to 30 C during maintenance
- Precise controlled gradual re-warming (0.05 C to 0.5 C/hour)

(Inzer, 2006)

Intravascular Temperature Management

- Nurses have a 63% chance of overcooling their patients when using surface cooling methods.
- 14% of patients never reached target temperatures with external cooling methods.
- Rapid cooling rates
- Precise controlled re-warming rates
- More control
- More power

(http://www.alsius.com/index.html)

Re-Warming Phase

- Rapid re-warming may undo any positive effects from hypothermia.
- Rapid re-warming can lead to sudden vasodilatation and can increase intracranial pressures.
- While there is no concrete data on the rate of re-warming, many institutions re-warm at a rate of 0.25 – 0.5°C per hour.
- Controlled, gradual re-warming to 36°C over 6-8 hours helps prevent overshoot.
- When temperature >36°C sedation, analgesia and paralysis should be discontinued as feasible.

(Povlishock, 2009)

Thank You

Any Questions?
References


