Objectives
1. Discuss current 2010 ACLS guidelines
2. Discuss chest compression techniques to optimize return of circulation
3. Describe optimal ventilation strategies during cardiac arrest
4. Discuss post resuscitation strategies to improve outcomes

2010 ILCOR/AHA/Heart & Lung ACLS Guidelines
- Bigger emphasis on compressions
- Early defibrillation
- Post resuscitation algorithm
- Waveform Capnography

Location, Location, Location!!!
Regional Differences: 10 cities

Newsweek
Sept. 23, 2008
Hot Tip: Have Your Cardiac Arrest In Seattle
A new study finds dramatic regional differences in cardiac arrest survival rates. Why some places are better than others when it comes to saving lives.
Designated cardiac arrest centers

- Arizona
  - 24/7 Cardiac Catheterization
  - Therapeutic Hypothermia
  - Center are identified by the AZ DOHS

- NYC
  - January 2009
  - Therapeutic Hypothermia

OPALS Study: Outcomes better at larger teaching hospitals vs. small & rural hospitals with higher volumes

According to the GWTG database, the survival rate from in-hospital cardiac arrest is:

A. 5%
B. 18%
C. 30%
D. 50%

What is the most common type of in-hospital cardiac arrest?

A. PEA & Asystole
B. Vfib & PEA
C. VTach & Vfib
D. Asystole & Vfib

Chances of surviving an In-Hospital Cardiac Arrest?

Defined by ICD-9 code for Cardiac Arrest - Including those admitted through the ED with CA

<table>
<thead>
<tr>
<th>State of Consciousness &amp; Type of Arrhythmia</th>
<th>Survival Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEA &amp; Asystole</td>
<td>5%</td>
</tr>
<tr>
<td>Vfib &amp; PEA</td>
<td>18%</td>
</tr>
<tr>
<td>VTach &amp; Vfib</td>
<td>30%</td>
</tr>
<tr>
<td>Asystole &amp; Vfib</td>
<td>50%</td>
</tr>
</tbody>
</table>

Circulation (2013); Morrison, et al.

In-Hospital Consensus Recommendations

Strategies for Improving Survival After In-Hospital Cardiac Arrest in the United States: 2013 Consensus Recommendations: A Consensus Statement From the American Heart Association

Laurie J. Norris, Robert W. Normand, Todd A. Chisolm, Mark S. Link, L. Kriz’s Needby, Paul W. McMullan, Jr, Tommy Vassan Hock, Calen C. Salemson, Lynn DuBois, Mary Ann Pricbeny and Dana F. Edelson

on behalf of the American Heart Association Emergency Cardiovascular Care Committee, Council on Cardiovascular and Stroke Nursing, Council on Clinical Cardiology, and Council on Peripheral Vascular Disease

May 2013

Cardiac Arrest Survival in Seattle & King County, 2002-2013

0% 10% 20% 30% 40% 50% 60% 70%

Year

**AHA - GWTG**

<table>
<thead>
<tr>
<th>First Documented Pulsatile Rhythm</th>
<th>Pediatric Cardiac Arrest (n=866)</th>
<th>Adult Cardiac Arrest (n=30,902)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asystole</td>
<td>550 (45)</td>
<td>13,824 (35)</td>
<td>0.600</td>
</tr>
<tr>
<td>VF or pulseless VT</td>
<td>120 (14)</td>
<td>8,361 (23)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VF</td>
<td>71 (9)</td>
<td>5,179 (14)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Pulseless VT</td>
<td>40 (9)</td>
<td>2,191 (6)</td>
<td>0.600</td>
</tr>
<tr>
<td>PEA</td>
<td>213 (24)</td>
<td>11,953 (32)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Unknown by documentation</td>
<td>197 (22)</td>
<td>2,054 (6)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Asystole & PEA make up 67% of all adult In-Hospital cardiac arrests

Circulation (2013); Morrison, et al.

---

**CPR Quality**

- Rate = at LEAST 100/min
- Depth 2 inches
- Allow for full recoil of the chest

Compressions provide 25-33% of normal cardiac output

---

**Is Faster Better?**

Survival favored chest compression rates between 85 to 110 cpm

Circulation Cardiovascular Quality & Outcomes 2013; 6; 148-156

---

**Quality of compressions**

- Rate = at LEAST 100/min
- Depth 2 inches
- Allow for full recoil of the chest

Compressions provide 25-33% of normal cardiac output

---

**Resuscitation Science**

Relationship Between Chest Compression Rates and Outcomes From Cardiac Arrest

Circulation Cardiovascular Quality & Outcomes 2012; 6: 131-138
Compression Fraction

- The amount of time spent providing compressions
- May also be called "compression ratio"
- Goal: At least 80%!

An increased chest compression fraction is independently predictive of better survival in patients who experience a pre-hospital ventricular fibrillation/tachycardia cardiac arrest.

Disco Lives!!!

- 5 Medical students & 10 MDs
- With beat avg. 103/minute
- 5 weeks later repeated

*Use a metronome!!!*

Who provides more effective compressions?

A. Males
B. Females
Who provided more effective CPR?
- 36 RNs (26 females, 20 males)
- 80% effective compressions by males
- Vs. 40% effective compressions by females

What is the best position to provide compressions to a patient in a hospital bed?
A. Standing on the floor
B. Kneeling on the bed
C. Standing on a step stool

Prevent leaning!!!

What are the issues with leaning?

Waveform Capnography
- Used as a marker of perfusion
- Normal is 35 – 40 mmHg
- Goal with compressions is at least 10 mmHg
- Will see increase with ROSC

When to use Waveform Capnography
- When an endotracheal tube is placed
- Gold standard for endotracheal tube placement
- Intra-arrest
  - Quality marker of compressions
  - Information helpful to determine cessation of resuscitation efforts – esp. in asystole
- Post arrest
Meet Howard Snitzer

- 54-years old, collapsed Jan 5, 2011 outside Don’s Foods in Goodhue, MN (pop. 900)
- 2 dozen rescuers took turns providing CPR for 96 minutes
- 6 shocks with first responder AED, 6 more shocks by Mayo Clinic Air Flight Medics
- Transported to Mayo Clinic Cardiac Cath Lab

Why Not Quit?

- Thrombectomy, stent to LAD
- 10 days inpatient
- “The capnography told us not to give up!”
- EtCO₂ averaged 35 (range 32 – 37)

So What’s the Goal During CPR?

- Try to maintain a minimum EtCO₂ of 10 mmHg
- Push
  - HARD (≥ 2” or 5 cm)
  - FAST (at least 100)
- Change rescuer
  - Every 2 minutes

Do we need all this fancy stuff?

- It helps!
- How do you currently evaluate quality of compressions?
- With ineffective compressions CO₂ remains in the tissues
- CO₂ is not making it to the heart to be eliminated by the lungs

Special considerations

- Pulmonary embolus
  - May have a consistently low PetCO₂

- Endotracheal tube dislodgement
  - PetCO₂ reads zero

- Termination of efforts
  - PetCO₂ never above 10 mm Hg

Other adjuncts

- Coronary Perfusion Pressure (CPP)
  - Diastolic pressure
  - Goal > 20 mmHg

- Central venous saturation
  - ScvO₂ – normal 60 – 80%
  - Goal > 30%
  - If < 30%, assess quality of compressions
Rate of ventilations:
- If patient does not have an advanced airway:
  30:2
- If the patient has an advanced airway:
  8 - 10 breaths / min

-2010 AHA Guidelines

Goals of ventilation
- Maintain oxygenation
- Aid in removal of CO₂
- 5 – 6 mL/kg tidal volume
- Breaths 1 second in duration
  ▫ (minimizes risk of gastric inflation)
- EBP lacking:
  ▫ Optimal tidal volume
  ▫ Ventilation rate
  ▫ Inspired O₂ concentration

When an advanced airway is being placed:
- Interrupt compressions for < 10 seconds
  ▫ Enough time to visualize the vocal cords & insert the ETT
- Verify airway placement:
  ▫ Auscultation
  ▫ Chest expansion
  ▫ CO₂ detector
  ▫ Esophageal detection device (Class IIa)

Is too much oxygen a good thing?

Definitions
Hypoxia: PaO₂ < 60
Hyperoxia: PaO₂ > 300 or P/F Ratio < 300

Kilgannon et al (2010) JAMA
Defibrillation

Biphasic energy
- More effectively depolarizes the heart
- Energy travels bi-directional
- Less energy needed
- Calculated impedance

Pad placement
- Anterior – posterior
- Anterior – lateral
- Skin prep:
  Clean skin
  Remove excess hair
- Special considerations:
  Bariatric
  Large breasts
  Burns
  Pediatrics

Minimize Pre & Post Shock pauses
- Pre-Shock pause < 3 seconds
- Post-Shock pause < 6 seconds

AEDs in non-critical care areas
- 439 patients evaluated in non-monitored areas:
  - 73 with VT/VF: 63 (86%) ROSC; 34 (47%) discharged from hospital
Medications

Which of the following medications has been shown to increase survival to discharge from cardiac arrest?

A. Epinephrine
B. Vasopressin
C. Bicarb
D. Amiodorone
E. None of the above

Emergency medications - V-fib
- **Epinephrine** 1 mg every 3-5 min or
- **Vasopressin** 40 units instead of the 1st or 2nd Epi
- **Amiodorone**
  - 300 mg IV pulseless
  - 150 mg pulse

Epinephrine

Current AHA guideline:
1 mg every 3 – 5 min in any pulseless rhythm

Rationale for use of Epinephrine
- Increased alpha 1, alpha 2 & beta 1 effects
- Increased systemic vascular resistance
- Increased myocardial & cerebral blood flow
- Increased ROSC rates?

Improved myocardial blood flow?

Decreased microcirculatory flow with Epi?

- Reduced sublingual microcirculatory flow with ongoing CPR after Epi administration


Improved cerebral blood flow?

- Animal study evaluating nine 40 kg pigs
- Decreased microvascular perfusion with Epinephrine administration


Cerebral Blood Flow with Epi

Increases in CPP were not accompanied by increases in CBF

This persisted for over 3 min after Epi administration


Japanese EMS introduction of Epi

Short term gain, but long term pain

Increased ROSC rates, however,
Decreased chance of survival at 1 month & decreased neurologic outcomes

All p < 0.001

Japan EMS 2004 – Permitted to start IVs
2006 – Permitted to administer Epi
Hagihara et al (2012) JAMA

Epinephrine in Cardiac Surgery Patients

- Look for causes!
  - Tamponade? Bleeding?
  - Resternotomy
  - If primary V-fib, defibrillation x 3 sequential
  - Do not give Epinephrine unless a senior provider advises to do so!
- What’s the risk?
  - Severe rebound hypertension leading to possible:
    - Aortic rupture
    - Suture line disruption

VSE Study Mentzelopoulos (2013) JAMA

- RCT
- Vasopressin 20 IU + Epi 1mg q 3 min x 5 cycles + 40 mg Steroid – methylprednisolone (1st cycle)
Epinephrine - What’s the evidence?

• Alpha adrenergic effect
  ▫ Increase coronary & cerebral perfusion pressure (animal study - dogs)
  ▫ No evidence linking to increased human survival
  ▫ Optimal dose? Who knows! **NOT high dose!**
    • Possibly < 30 – 45 mcg/kg (< 2 – 3 mg)
  ▫ Optimal interval? Who knows!

• High dose epi?
  • 0.1 – 0.2 mg/kg (3 mg, 5 mg doses)
  • No difference in survival or neurologic outcomes

Amiodorone vs. Placebo
(after 3 successive shocks in OHCA)

- **Survival to admission (%)**
  - All Patients: 86%
  - VF: 86%
  - Asystole or PEA: 36%
  - ROSC: 34%
  - No ROSC: 34%

N = 504
Kudenchuk et al (1999) NEJM

Amiodorone vs. Lidocaine

- **Survival to hospital admission**
  - Early = < 24 min of dispatch call
  - Amiodorone superior to Lidocaine regardless of time administered
  - Note: Survival to hospital admission

Dorian et al (2002) NEJM

V-fib

**Amiodorone**
- 2 RCTs (OHCA) increased survival to hospital admission (vs. Lidocaine or placebo)
- Lacking evidence that it makes a difference in survival to discharge
  - Dorian et al (2002) NEJM

- For REFRACTORY V-fib, use **Amiodorone**
- 300 mg, may re-bolus with 150 mg

ALP Trial

- **Amiodorone vs. Lidocaine vs. Placebo**
  - Out of hospital v-fib arrest
  - Goal is drug administration < 10 minutes after arrival on scene

- Resuscitation Outcome Consortium (ROC) study group
- Multi-city EMS trial
- Still enrolling patients
- Goal: 3,000 patients

The evidence behind vasopressors?

- Vasopressors
- To date no placebo-controlled trials have shown that administration of any vasopressor agent at any stage during management of VF, pulseless VT, PEA, or asystole increases the rate of neurologically intact survival to hospital discharge.
- There is evidence, however, that the use of vasopressor agents is associated with an increased rate of ROSC.

Circulation 2010 AHA Guidelines

Amiodorone vs. Placebo
N = 504
Kudenchuk et al (1999) NEJM
Intraosseous access

How can we improve in-hospital cardiac arrest outcomes?

- Prevent the arrest!!!
- Compressions 100 – 120 per minute
- 2 inch depth
- Minimize interruptions
  - Chest Compression Fraction >80%
  - Avoid unnecessary pulse checks
  - Allow full recoil
- Defibrillate early
- Use feedback devices
  - EtCO2
- Avoid excessive ventilation
- Designated teams
- Practice!!!

Quality Improvement

Circulation 2013, Meaney et al

Response teams

- Dedicated team with defined, clear roles
- Practice, practice, practice!
- Team Debriefing

Clinical Investigations

Residents feel unprepared and unsupervised as leaders of cardiac arrest teams in teaching hospitals: A survey of internal medicine residents®

Chris W. Hayes, MD, MSc; Augustine Hsue, MD; Michael E. DeBias, MD; Vicki A. LeBlanc, PhD; Randy S. Wax, MD, MSc

Publication: Critical Care Medicine, 2007; vol. 35, no. 7.
The Code Team & Defined Roles

Data:
- 86,748 in-patient cardiac arrests
  - 58,593 day/evening arrests
  - 28,155 night arrests
- Cardiac arrest events at night were less likely to be monitored
- **Day/evening:**
  - 33.5% asystole - 36.9% PEA - 22.9% VF
- **Night**
  - 39.6% asystole – 34.6% PEA – 19.8% VF

Feedback Devices

Survival From In-Hospital Cardiac Arrest During Nights and Weekends

Real Time Feedback Devices
The Mattress Issue:
- Mattress compression = 35 – 40% of total compression depth
- Accelerometer feedback devices fail to account for mattress compression
- Use of a backboard fails to compensate for mattress compression

AHA Consensus Recommendation
2013 Consensus Recommendation:
"...resuscitation data from the defibrillator or any other device or source documentation that captures data at the scene should be used for feedback to the team"
*Circulation, 2013*
Intra-Arrest Data Report

1 Second
Vfib – No Shock, but stopped to assess?

Issues here?

17 second pause

Minimal pre/post shock pause

LUCAS mechanical chest compressions

Post Cardiac Arrest: Targeted Temperature Management

Trends in Resuscitation

Pre-hospital phase
Early defibrillation
Quality CPR
Post arrest: Supportive care
ILCOR/ACLS Advisory Statement

October 2002, the Advanced Life Support (ALS)

Task Force recommended:

- “Unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest should be cooled to 32-34°C for 12 to 24 hours when the initial rhythm was ventricular fibrillation”
  - IIa recommendation

- “Such cooling may also be beneficial for other rhythms or in-hospital arrest”
  - IIb recommendation

Included in AHA Guidelines since 2005
Updated into post-arrest algorithm in 2010

A new therapy?

THE USE OF HYPOTHERMIA AFTER CARDIAC ARREST

Donald W. Benson, M.D.

Benson DW et al. Anesthesia and Analgesia 1959; 38: 423-428

1992 - Hypothermia after Cardiac Arrest in Dogs

1992 - Hypothermia after Cardiac Arrest in Dogs

A New Option?

The New England Journal of Medicine

MILD THERAPEUTIC HYPOTERMIA TO IMPROVE THE NEUROLOGIC OUTCOME AFTER CARDIAC ARREST

The Hypothermia After Cardiac Arrest Study Group

TREATMENT OF CONSCIOUS SURVIVORS OF OUT-OF-HOSPITAL CARDIAC ARREST WITH INDUCED HYPOTERMIA


HACA

NEJM 2002; 346:549-556

Cooling after Cardiac Arrest

<table>
<thead>
<tr>
<th>n</th>
<th>Randomization</th>
<th>Temp.</th>
<th>Rhythm</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bernard et al.</td>
<td>77</td>
<td>43 Hypothermia 34 Control</td>
<td>37° x 12 hrs</td>
<td>V-fib</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HACA</td>
<td>275</td>
<td>137 Hypothermia 138 Control</td>
<td>32-34° x 24 hrs</td>
<td>V-fib/ V-tach</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

RR 1.4 (95% CI 1.08–1.81)
<table>
<thead>
<tr>
<th>Condition</th>
<th>Number (Original)</th>
<th>Number (Outcome)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NS</td>
<td>2/138 (0.2)</td>
<td>2/119 (0.2)</td>
</tr>
<tr>
<td>Numb</td>
<td>4/138 (2.9)</td>
<td>8/138 (6.9)</td>
</tr>
<tr>
<td>Nausea</td>
<td>8/138 (6.4)</td>
<td>17/138 (12.6)</td>
</tr>
<tr>
<td>Numb</td>
<td>2/138 (1.5)</td>
<td>2/138 (1.5)</td>
</tr>
<tr>
<td>Numb</td>
<td>14/138 (10.1)</td>
<td>13/138 (9.9)</td>
</tr>
<tr>
<td>Hypotension</td>
<td>2/138 (1.5)</td>
<td>2/138 (1.5)</td>
</tr>
<tr>
<td>Numb</td>
<td>4/138 (3.2)</td>
<td>6/138 (4.5)</td>
</tr>
<tr>
<td>Pyelonephritis</td>
<td>1/138 (0.7)</td>
<td>9/138 (6.7)</td>
</tr>
<tr>
<td>Lactate</td>
<td>3/138 (2.2)</td>
<td>2/138 (1.5)</td>
</tr>
<tr>
<td>Lactate</td>
<td>4/138 (3.3)</td>
<td>49/150 (32.7)</td>
</tr>
<tr>
<td>Numb</td>
<td>9/138 (6.6)</td>
<td>6/138 (4.5)</td>
</tr>
</tbody>
</table>

Note: Some of the comparisons between the two groups were performed with a two-tailed *t*-test, with *p* values shown in parentheses. *p* values < 0.05 were considered significant differences.

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.

Critical Care Medicine, 2009

CT scan (ED)
30 y.o. s/p asystolic arrest

Used with permission of Dr. W. Longstreth, Harborview Medical Center

Why cool?
To minimize reperfusion injury!

- Depleted stores of O₂ & glucose
- Intracellular calcium influx
- Formation of O₂ free radicals
- Release of glutamate
- Intracellular acidosis
- Disruption in blood brain barrier
- Mitochondrial injury
- Apoptosis

Polderman, KH Crit Care Med (2009); 37:S186-202
Who should be cooled?

- Ventricular Fibrillation
- Ventricular Tachycardia

What about:
- Asystole?
- PEA?
- Drowning?
- Electrocution?
- Asphyxiation?

American Heart Association, 2010

Can we safely cool cardiogenic shock/PCI?

- Previous trials excluded patients with shock symptoms, few with PCI
- Outcomes of 50 patients
  - 23/50 received IABP, 36/50 PCI
  - 41 (82%) survived until 6 mos.
  - 34 (68%) CPC 1 or 2
  - 7 (14%) CPC 3


When should cooling be initiated?

- Pre-Hospital?
  - Safe, relatively easy with cold saline
  - Does it improve outcomes???
  - Emergency Department? *YES!!*
  - Cardiac Catheterization Lab? *YES!!*
  - Critical Care Unit?

  - How late is too late?

Pre-Hospital Cooling?

- Iced saline via paramedics

Pre-Hospital Iced Saline

- RCT 1359 patients with cardiac arrest
- 2 L - 4 °C Iced Saline
- No difference
- Higher incidence of pulmonary edema
- Re-arrest 26% vs. 21% (p = 0.008)

Kim et al (2013) JAMA
Goal temperature

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

<table>
<thead>
<tr>
<th></th>
<th>RCT 9°C</th>
<th>RCT 35°C</th>
<th>RR (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality</td>
<td>50%</td>
<td>44%</td>
<td>1.09 (0.99-1.19)</td>
<td>0.09</td>
</tr>
<tr>
<td>Neurological Function (worse)</td>
<td>50%</td>
<td>54%</td>
<td>0.71 (0.55-1.04)</td>
<td>0.07</td>
</tr>
<tr>
<td>Serious Adverse Events</td>
<td>57%</td>
<td>50%</td>
<td>1.12 (1.03-1.20)</td>
<td>0.009</td>
</tr>
</tbody>
</table>

Nielsen et al (2013) NEJM

Dosing Therapy?

- Duration of post-arrest TTM
- Depth of post-arrest TTM for select patients
- Optimal injury measurement post-arrest
- Pharmacologic adjuncts to TTM
- Early vs. late post-arrest cardiac cath

How to Cool?

Iced Saline Nasopharyngeal

Surface
- Ice packs (not rec.)
- External water blankets
- Forced cool air blankets
- Hydrogel pads

Endovascular Catheters

**Need continuous temperature feedback loop to prevent overcooling!!

Overcooling with ice packs & cooling blankets

20/32 (63%) reached temp <32°C
9/32 (28%) reached temp <33°C
4/32 (13%) reached temp <30°C

Overcooling: 6/20 (30%) survived to hospital discharge
Without overcooling: 7/12 (58%) survived to hospital discharge
(No statistical significance)


Challenging patients to cool:

- Obese patients
  (Adipose insulates 3x’s as well as muscle)
- Young patients
  (react to changes in body temperature)

Easy cooling:
Older patients
- Lower BMI
- Lower rate of metabolism
- Less effective vascular response

Temperature Source

- Pulmonary Artery (PA) catheter
  - Not practical
- Esophageal**
- Bladder
- Rectal

Neuro-Protective Qualities:

- Cerebral Metabolic Rate
- Intracranial pressure
- Inflammation & cytokine release
- Cerebral edema

Ning, XH  Journal of Appl Physiol (2002); 92:2200-2207

ICU Management & Physiologic Effects of Cooling

Common side effects of mild hypothermia include:

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

Danzl & Pozos, NEJM (1994); 331:1756-1760

Systemic Effects of Cooling

- ↓ Heart rate (May see tachycardia with induction)
- ↓ Cardiac output (by up to 25%)
- ↑ BP & SVR (d/t vasoconstriction)
- ↑ Urine output – cold induced diuresis
- ↑ Lactate – prolonged clearance
- Prolonged clotting times
- ↓ Intestinal motility

Danzl & Pozos, NEJM (1994); 331:1756-1760

Arrhythmias/ECG

- Negative chronotropic effects of myocardial pacemaker cells
- Prolonged PR, QT intervals
- J wave
- More susceptible to atrial fibrillation (<32° C)
- Ventricular arrhythmias at lower temps (Below 28-30° C)

Danzl & Pozos, NEJM (1994); 331:1756-1760

Electrolyte Imbalance

- Decreased: Due to intracellular shifts
  - Hypokalemia
  - Hypomagnesemia (has neuro-protective qualities)
  - Hypophosphatemia
  - Hypocalcemia
- Assess electrolytes upon initiation, goal, then at regular intervals (every 4-6 hrs.)
- Hyperglycemia: ↓ insulin secretion

Danzl & Pozos, NEJM (1994); 331:1756-1760
Glucose Control
- 234 subjects post cardiac arrest
- Assessed glucose levels 12 hours after ROSC with 6 month survival
- Conclusion: Tight control may not be needed -Losert et. al. (2007); Resuscitation
- 90 subjects post v-fib arrest/therapeutic hypothermia
- Strict glycemic control group, moderate group
- Conclusion: No survival benefit from strict glycemic control -Okanen et. al. (2007); Intensive Care Medicine

Ventilation Management
- Shift to the left
- CO₂ production slows
- Target normal CO₂
- Hyperventilation leads to cerebral ischemia
- Standard Tidal Volume
- Quickly wean FiO₂ – hyperoxia damages post-ischemic neurons
- Overall decreased O₂ consumption -Danzl & Pozos, NEJM (1994); 331:1756-1760

Oxygen Saturation Monitoring
- Digit sensor often unreliable d/t peripheral vasoconstriction
- Study compared forehead sensors with digit sensors

Shivering:
- Increases O₂ consumption by 40-100%
- Neuromuscular blockade
- Ensure adequate sedation
- Train Of Four monitoring- unreliable <35º C (slowing of neuromuscular junction)

Other strategies:
- Propofol/Sedatives
- Opiates
- Meperidine (avoid)

Columbia Shiver Scale

Score | Term | Description
--- | --- | ---
0 | NO | Absence of shivering on palpation of neck or periorbital muscles
1 | MILD | Localized to the neck and/or thorax. May be present only on palpation
2 | MODERATE | Involvement of the upper extremities +/- neck or periorbital muscles
3 | SEVERE | Generalized, whole body involvement

Re-warming
- Important to re-warm slowly
- Vasodilation, hypotension if too quick
- Minimum 8-12 hours
- If re-warm too quickly, can possibly negate benefits Poor neuro outcomes in TBI/Stroke*
- Rebound hyperthermia

Prognostication

- More research needs to be completed
- EEG monitoring
- Reports of delayed wake-up
- Wait at least 72 hours before withdrawal of support
- Many prognostic indicators not tested with cooling
- AHA recommendations, 2010

Future research:

- Duration of cooling
  - 12 hrs, 24 hrs, longer?
- Pre-hospital cooling
- Intranasal cooling
- Cooling intra-arrest
- Ideal cooling methods
- Medication clearance
- Biggest benefit? Asystole? PEA?
- Prognostication
- Ultrafiltration

Need more info?


- Society of Critical Care Medicine
  www.sccm.org

- http://www.med.upenn.edu/resuscitation/Hypothermia

In conclusion:

- Provide good quality compressions
- Minimize interruptions in compression
- Defibrillate early!
- Avoid over-ventilation
- Post-resuscitation care
- Practice!
- Identify roles and establish clear expectations
- Control the temperature post arrest