Post “Resuscitation “Syndrome

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ICCU, Leeviev Heart Center
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Outcome After Cardiac Arrest (CA) in Heidelberg, Germany

* CPR following cardiac arrest of cardiac aetiology (36 months)

Böttiger BW et al., Heart 82:674-679, 1999
539 pts’ CPR

- 203 (38%) Treated by regular ambulance
- 336 (62%) Treated by MICU
- 149 transferred to MICU

54 pts’ Dead on scene

485 Treated by MICU
485 pts’

Other
85 (18%)

PEA
64 (13%)

VF
94 (19%)

Asystole
242 (50%)

Transportation to hospital
199 pts’

47 (55%)
25 (39%)
51 (54%)
76 (31%)

Discharged from hospital
53 pts’

20 (24%)
4 (6%)
20 (21%)
9 (4%)
The second step in resuscitation—the treatment of the ‘post-resuscitation disease’

V. A. NEGOVSKY

Laboratory of Experimental Resuscitation, Academy of Medical Sciences of the U.S.S.R., 9, October 25th Street, Moscow, U.S.S.R.

“Extensive experimental studies and clinical findings have clearly proved that after the first step of resuscitation when heart function and respiration have been restored, the second step in resuscitation arises – the more complicated problems of treating the after-effects of general hypoxia”

Negovsky 1972
ILCOR Consensus Statement

Post-Cardiac Arrest Syndrome:

Epidemiology, Pathophysiology, Treatment, and Prognostication

A Consensus Statement From the International Liaison Committee on Resuscitation

Circulation. 2008;118:2452-2483
This syndrome includes all clinical and biological manifestations related to the phenomenon of global ischemia-reperfusion triggered by cardiac arrest and return of spontaneous circulation.

The main component of this syndrome is an early but severe cardiocirculatory dysfunction that may lead to multiple organ failure and death.
4 Key Components:

I. Post-cardiac arrest brain injury
II. Post-cardiac arrest myocardial dysfunction
III. Systemic ischemia/perfusion response
IV. Persistent precipitating pathology

Severity of these disorders is based upon:
- severity of the ischemic insult
- the cause of cardiac arrest
- patient’s prearrest state of health

Post cardiac arrest brain injury is a common cause of morbidity and mortality.

Less than 10% of pts’ admitted to hospital will leave without major neurological impairment.
Post-Cardiac Arrest Brain Injury

**Pathophysiology**
- Impaired cerebrovascular autoregulation
- Cerebral edema (limited)
- Postischemic neurodegeneration

**Clinical Manifestation**
- Coma
- Seizures
- Myoclonus
- Cognitive dysfunction
- Persistent vegetative state
- Secondary Parkinsonism
- Cortical stroke
- Spinal stroke
- Brain death

**Potential Treatments**
- Therapeutic hypothermia
- Early hemodynamic optimization
- Airway protection and mechanical ventilation
- Seizure control
Post–Cardiac Arrest Myocardial Dysfunction

- Global hypokinesis (myocardial stunning)
- ACS
Myocardial Function is Reversibly Depressed for 24 to 72 hours after Cardiac Arrest

Laurent et al., JACC 2002

Cardiac Index (L/min/m²)

Hours After Cardiac Arrest

Post-cardiac arrest myocardial dysfunction is responsive to inotropes and intravenous fluid
Post–Cardiac Arrest Myocardial Dysfunction

**Pathophysiology**
- Global hypokinesis (myocardial stunning)
- ACS

**Clinical Manifestation**
- Reduced cardiac output
- Hypotension
- Dysrhythmias
- Cardiovascular collapse

**Potential Treatments**
- Early revascularization of AMI
- Early hemodynamic optimization
- Intravenous fluid
- Inotropes
- IABP
- LVAD
- ECMO
This is the ultimate systemic representation of shock in response to global ischemia and reperfusion.
Persistent Precipitating Pathology

This is related to any specific disease process that may be the underlying cause for the cardiac arrest
Persistent Precipitating Pathology

**Pathophysiology**
- Cardiovascular disease (AMI/ACS, cardiomyopathy)
- Pulmonary disease (COPD, asthma)
- CNS disease (CVA)
- Thromboembolic disease (PE)
- Toxicological (overdose, poisoning)
- Infection (sepsis, pneumonia)
- Hypovolemia (hemorrhage, dehydration)

**Clinical Manifestation**
- Central nervous system
  - Encephalopathy
  - Stroke-like episodes
  - Seizures and dementia
  - Psychosis and depression
  - Ataxia
  - Migraine
- Cardiac
  - Hypertrophic cardiomyopathy
  - Dilated cardiomyopathy
  - Heart block
  - Pre-excitation syndrome
- Renal
  - Renal tubular defects
  - Toni-Fanconi-Debre syndrome
- Muscle
  - Myopathy
- Gastrointestinal
  - Dysphagia
  - Pseudo-obstruction
  - Constipation
  - Hepatic failure
- Eye
  - External ophthalmoplegia
  - Ptosis
  - Cataract
  - Pigmentary retinopathy
  - Optic atrophy
- Hearing
  - Bilateral sensorineural deafness
- Skin
  - Palmo plantar keratoderma
- Endocrine
  - Diabetes mellitus
  - Hypoparathyroidism
  - Hypothyroidism
  - Gonadal failure
- Peripheral nervous system
  - Axonal sensorimotor neuropathy
Persistent Precipitating Pathology

Pathophysiology

Clinical Manifestation

• Specific to cause but complicated by concomitant PCAS

Potential Treatments

• Disease-specific interventions guided by patient condition and concomitant PCAS
Treatment must focus on reversing the pathophysiological manifestation of the post cardiac arrest syndrome
Monitoring Options

1. General intensive care monitoring
   - Arterial catheter
   - Oxygen saturation by pulse oximetry
   - Continuous ECG
   - CVP
   - ScvO₂
   - Temperature (bladder, esophagus)
   - Urine output
   - Arterial blood gases
   - Serum lactate
   - Blood glucose, electrolytes, CBC, and general blood sampling
   - Chest radiograph

2. More advanced hemodynamic monitoring
   - Echocardiography
   - Cardiac output monitoring (either noninvasive or PA catheter)

3. Cerebral monitoring
   - EEG (on indication/continuously): early seizure detection and treatment
   - CT/MRI
Early Hemodynamic Optimization

• Early Goal-Directed Therapy
  – CVP: 8 to 12 mm Hg,
  – MAP: 65 to 90 mm Hg,
  – ScvO₂ >70%,
  – Hematocrit >30% or hemoglobin >8 g/dL,
  – lactate <2mmol/L,
  – urine output >0.5 mL · kg⁻¹ · h⁻¹,
  – oxygen delivery index >600 mL · min⁻¹ · m⁻²
Significance of arterial hypotension after resuscitation from cardiac arrest*

Stephen Trzeciak, MD, MPH; Alan E. Jones, MD; J. Hope Kilgannon, MD; Barry Milcarek, PhD; Krystal Hunter, MBA; Nathan I. Shapiro, MD, MPH; Steven M. Hollenberg, MD; R. Phillip Dellinger, MD; Joseph E. Parrillo, MD

Hypotension = SBP < 90 mmHg within 1 hour of ICU arrival

Objective: Expert guidelines advocate hemodynamic optimization after return of spontaneous circulation (ROSC) from cardiac arrest despite a lack of empirical data on prevalence of post-ROSC hemodynamic abnormalities and their relationship with outcome. Our objective was to determine whether post-ROSC arterial hypotension predicts outcome among postcardiac arrest patients who survive to intensive care unit admission.

Design: Cohort study utilizing the Project IMPACT database (intensive care unit admissions from 120 U.S. hospitals) from 2001–2005.

Setting: One hundred twenty intensive care units.

Patients: Inclusion criteria were: 1) age ≥ 18 yrs; 2) nontrauma; and 3) received cardiopulmonary resuscitation before intensive care unit arrival.

Interventions: None.

Measurements and Main Results: Subjects were divided into two groups: 1) Hypotension Present—one or more documented systolic blood pressure < 90 mm Hg within 1 hr of intensive care unit arrival; or 2) Hypotension Absent—all systolic blood pressure ≥ 90 mm Hg. The primary outcome was in-hospital mortality. The secondary outcome was functional status at hospital discharge among survivors. A total of 8736 subjects met the inclusion criteria. Overall mortality was 50%. Post-ROSC hypotension was present in 47% and was associated with significantly higher rates of mortality (65% vs. 37%) and diminished discharge functional status among survivors (49% vs. 38%), p < .001 for both. On multivariable analysis, post-ROSC hypotension had an odds ratio for death of 2.7 (95% confidence interval, 2.5–3.0).

Conclusions: Half of postcardiac arrest patients who survive to intensive care unit admission die in the hospital. Post-ROSC hypotension is common, is a predictor of in-hospital death, and is associated with diminished functional status among survivors. These associations indicate that arterial hypotension after ROSC may represent a potentially treatable target to improve outcomes from cardiac arrest. (Crit Care Med 2009; 37:2895–2903)

Key words: heart arrest; cardiopulmonary resuscitation; resuscitation; shock; hemodynamics
Ventilation with 100% oxygen for the first hour after ROSC resulted in worse neurological outcome than immediate adjustment of FIO2 to produce an arterial oxygen saturation of 94%-96%.
Association Between Arterial Hyperoxia Following Resuscitation From Cardiac Arrest and In-Hospital Mortality

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Investigators

Context Laboratory investigations suggest that exposure to hyperoxia after resuscitation from cardiac arrest may worsen anoxic brain injury; however, clinical data are lacking.

Objective To test the hypothesis that postresuscitation hyperoxia is associated with increased mortality.

Design, Setting, and Patients Multicenter cohort study using the Project IMPACT

First ABG in ICU (within 24 hours of ICU arrival)

Hyperoxia = \( \text{PaO}_2 \geq 300 \text{ mmHg} \)

Hypoxia = \( \text{PaO}_2 \leq 60 \text{ mmHg} \)

Main Outcome Measure In-hospital mortality.

Results Of 6326 patients, 1156 had hyperoxia (18%), 3999 had hypoxia (63%) and 1171 had normoxia (19%). The hyperoxia group had significantly higher in-hospital mortality (732/1156 [63%; 95% confidence interval {CI}, 60%-66%]) compared with the normoxia group (532/1171 [45%; 95% CI, 43%-48%]; proportion difference, 18% [95% CI, 14%-22%]) and the hypoxia group (2297/3999 [57%; 95% CI, 56%-59%]; proportion difference, 6% [95% CI, 3%-9%]). In a model controlling for potential confounders (eg, age, preadmission functional status, comorbid conditions, vital signs, and other physiological indices), hyperoxia exposure had an odds ratio for death of 1.8 (95% CI, 1.5-2.2).

Conclusion Among patients admitted to the ICU following resuscitation from cardiac arrest, arterial hyperoxia was independently associated with increased in-hospital mortality compared with either hypoxia or normoxia.

JAMA. 2010;303(21):2165-2171
Ventilation

**Association Between Postresuscitation Partial Pressure of Arterial Carbon Dioxide and Neurological Outcome in Patients With Post–Cardiac Arrest Syndrome**

Brian W. Roberts, MD; J. Hope Kilgannon, MD; Michael E. Chansky, MD; Neil Mittal, MD; Jonathan Wooden, MD; Stephen Trzeciak, MD, MPH

Prospective registry of 193 patients

- **Hypocapnia**: Paco2 ≤ 30mmHg
- **Hypercapnia**: Paco2 ≥ 50mmHg

**Conclusions**—Hypocapnia and hypercapnia were common after cardiac arrest and were independently associated with poor neurological outcome. These data suggest that Paco2 derangements could be potentially harmful for patients after resuscitation from cardiac arrest. *(Circulation. 2013;127:2107-2113.)*
During hypothermia PaCO2 should be corrected to patient temperature. When core temperature is 33°C the patient’s actual PaCO2 may be 6 to 7 mmHg lower.

\[
\text{PaCO2} = 40 - 45 \text{mmHg}
\]
\[
\text{ETCO2} = 35 - 40 \text{mmHg}
\]
Ventilation

Surviving Sepsis Campaign Recommends:

- Assist control mode – volume ventilation
- Reduce tidal volume to 6 mL/kg lean body weight
- Keep inspiratory plateau pressure (Pplat) ≤ 30 cm H2O
  - Reduce TV as low as 4 mL/kg predicted body weight to limit Pplat
- Maintain SaO2/SpO2 88–95%
- Anticipated PEEP settings at various FiO2 requirements
  - FiO2 \(0.3\) 0.4 0.4 0.5 0.5 0.6 0.7 0.7 0.7 0.8 0.9 0.9 0.9 1.0
  - PEEP \(5\) 5 8 8 10 10 10 10 12 14 14 14 16 18 20–24
- Predicted Body Weight Calculation
  - Male – 50 + 2.3 (height (inches) – 60) or 50 + 0.91 (height (cm) – 152.4)
  - Female – 45.5 + 2.3 (height (inches) – 60) or 45.5 + 0.91 (height (cm) – 152.4)

**TV**, tidal volume; **SaO2**, arterial oxygen saturation; **SpO2**, pulse oximetry oxyhemoglobin saturation; **PEEP**, positive end-expiratory pressure


Higher TV in post cardiac arrest
PEEP<10cmH2O ↑ ICP
Glucose Control

- Tight control blood glucose (80 to 110 mg/dL) with insulin decreases hospital mortality rates in critically ill adults.
Intensive versus Conventional Glucose Control in Critically Ill Patients

The NICE-SUGAR Study Investigators

**Intensive:** 81-108 mg/dl
**Conventional:** ≤180 mg/dl

**BACKGROUND**
The optimal target range for blood glucose in critically ill patients remains unclear.

**METHODS**
Within 24 hours after admission to an intensive care unit (ICU), adults who were expected to require treatment in the ICU on 3 or more consecutive days were ran-

**Strict Glycemic Control Worsens Outcomes**

![Graph showing the impact of glycemic control on survival]

*P* = 0.03

**NICE-SUGAR, NEJM 2010**
Other Issues:

1. Pyrexia
2. Hyperglycemia
3. Seizures
4. Adrenal dysfunction
5. Renal Failure
6. Infection

When serial temperatures were monitored in 151 patients for 48 hours after out-of-hospital cardiac arrest... the risk of unfavorable outcome increased for every degree Celsius that the peak temperature exceeded 37°C...

Other Issues:

1. Pyrexia
2. Hyperglycemia
3. Seizures
4. Adrenal dysfunction
5. Renal Failure
6. Infection

Hyperglycemia is common in post-cardiac arrest patient and is associated with a poor neurological outcome after out-of-hospital cardiac arrest.

**Other Issues:**

1. Pyrexia  
2. Hyperglycemia  
3. Seizures  
4. Adrenal dysfunction  
5. Renal Failure  
6. Infection

---

...Seizures in the post–cardiac arrest period are associated with **worse prognosis** and are likely to be caused by, as well as **exacerbate, post–cardiac arrest brain injury**...

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Continuous EEG in therapeutic hypothermia after cardiac arrest

Prognostic and clinical value

Amy Z. Crepeau, MD, Alejandro A. Rabinstein, MD, Jennifer E. Fugate, DO, Jay Mandrekar, PhD, Eelco F. Wijdicks, MD, Roger D. White, MD and Jeffrey W. Britton, MD

Published online before print January 2, 2013, doi: 10.1212/WNL.0b013e31827f089d Neurology January 22, 2013 vol. 80 no. 4 339-344

Results: Fifty-four patients were included; 51 remained on cEEG through NT. Nineteen died. EEG severity grading during both TH and NT statistically correlated with outcome (grade 1 = good, grade 3 = poor). Other EEG features correlating with poor outcome included seizures, nonreactive background, and epileptiform discharges. Changes in EEG grade during monitoring did not statistically correlate with outcome. Five patients had seizures; all occurred in patients with grade 3 EEG backgrounds and all had a poor outcome.

Conclusion: Grades 1 and 3 on our EEG severity grading scale during TH and NT correlated with outcome. Treating seizures did not improve outcome in our cohort.
<table>
<thead>
<tr>
<th>Other Issues:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Pyrexia</td>
</tr>
<tr>
<td>2. Hyperglycemia</td>
</tr>
<tr>
<td>3. Seizures</td>
</tr>
<tr>
<td>4. Adrenal dysfunction</td>
</tr>
<tr>
<td>5. Renal Failure</td>
</tr>
<tr>
<td>6. Infection</td>
</tr>
</tbody>
</table>

Steroids? Controversial
Other Issues:

1. Pyrexia
2. Hyperglycemia
3. Seizures
4. Adrenal dysfunction
5. Renal Failure
6. Infection

Indication for use of RRT same with that of critically ill patients.
Other Issues:

1. Pyrexia
2. Hyperglycemia
3. Seizures
4. Adrenal dysfunction
5. Renal Failure
6. Infection

Ventilation associated aspiration pneumonia
Management of ACS

- Patients resuscitated from cardiac arrest who have STEMI should undergo immediate angiography, with subsequent PCI if indicated.
- It is appropriate to consider immediate coronary angiography in all post cardiac arrest patients in whom ACS is suspected.
Immediate Percutaneous Coronary Intervention Is Associated With Better Survival After Out-of-Hospital Cardiac Arrest

Insights From the PROCAT (Parisian Region Out of Hospital Cardiac Arrest) Registry

Florence Dumas, MD; Alain Cariou, MD; Stéphane Manzo-Silberman, MD; David Grimaldi, MD; Benoît Vivien, MD; Julien Rosencher, MD; Jean-Philippe Empana, MD; Pierre Carli, MD; Jean-Paul Mira, MD; Xavier Jouven, MD; Christian Spaulding, MD

Background—Acute coronary occlusion is the leading cause of cardiac arrest. Because of limited data, the indications and timing of coronary angiography and angioplasty in patients with out-of-hospital cardiac arrest are controversial. Using data from the Parisian Region Out of hospital Cardiac ArresT prospective registry, we performed an analysis to assess the effect of an invasive strategy on hospital survival.

Methods and Results—Between January 2003 and December 2008, 714 patients with out-of-hospital cardiac arrest were referred to a tertiary center in Paris, France. In 435 patients with no obvious extracardiac cause of arrest, an immediate coronary angiogram was performed at admission followed, if indicated, by coronary angioplasty. At least 1 significant coronary artery lesion was found in 304 (70%) patients, in 128 (96%) of 134 patients with ST-segment elevation on the ECG performed after the return of spontaneous circulation, and in 176 (58%) of 301 patients without ST-segment elevation. The hospital survival rate was 40%. Multivariable analysis showed successful coronary angioplasty to be an independent predictive factor of survival, regardless of the postresuscitation ECG pattern (odds ratio, 2.06; 95% CI, 1.16 to 3.66).

Conclusions—Successful immediate coronary angioplasty is associated with improved hospital survival in patients with or without ST-segment elevation. Therefore, our findings support the use of immediate coronary angiography in patients with out-of-hospital cardiac arrest with no obvious noncardiac cause of arrest regardless of the ECG pattern. (Circ Cardiovasc Interv. 2010;3:200-207.)
Successful PCI Associated With Improved Outcome With or Without STEMI

Survival rate (%)

ST segment elevation

Other ECG patterns

Successful PCI
No or failed PCI

p<0.001
ns

p<0.001
ns
Immediate angiography and PCI when indicated should be performed in resuscitated out-of-hospital cardiac arrest patients whose initial ECG shows STEMI.34-49 (Level of Evidence: B)
Patients who cannot follow basic commands generally need treatment with mild therapeutic hypothermia.
2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction: Executive Summary

A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines

Therapeutic hypothermia should be started as soon as possible in comatose patients with STEMI and out-of-hospital cardiac arrest caused by VF or pulseless VT (Level of Evidence :B)
Is Hypothermia After Cardiac Arrest Effective in Both Shockable and Nonshockable Patients?
Insights From a Large Registry

Florence Dumas, MD; David Grimaldi, MD; Benjamin Zuber, MD; Jérôme Fichet, MD; Julien Charpentier, MD; Frédéric Pène, MD, PhD; Benoît Vivien, MD, PhD; Olivier Varenne, MD; Pierre Carli, MD, PhD; Xavier Jouven, MD, PhD; Jean-Philippe Empana, MD, PhD; Alain Cariou, MD, PhD

Background—Although the level of evidence of improvement is significant in cardiac arrest patients resuscitated from a shockable rhythm (ventricular fibrillation or pulseless ventricular tachycardia [VF/Vf]), the use of therapeutic mild hypothermia (TMH) is more controversial in nonshockable patients (pulseless electric activity or asystole [PEA/asystole]). We therefore assessed the prognostic value of hypothermia for neurological outcome at hospital discharge according to first-recorded cardiac rhythm in a large cohort.

Methods and Results—Between January 2000 and December 2009, data from 1145 consecutive out-of-hospital cardiac arrest patients in whom a successful resuscitation had been achieved were prospectively collected. The association of TMH with a good neurological outcome at hospital discharge (cerebral performance categories level 1 or 2) was quantified by logistic regression analysis. TMH was induced in 457/708 patients (65%) in VF/Vf and in 261/437 patients (60%) in PEA/asystole. Overall, 342/1145 patients (30%) reached a favorable outcome (cerebral performance categories level 1 or 2) at hospital discharge, respectively 274/708 (39%) in VF/Vf and 68/437 (16%) in PEA/asystole (P<0.001). After adjustment, in VF/Vf patients, TMH was associated with increased odds of good neurological outcome (adjusted odds ratio, 1.90; 95% confidence interval, 1.18 to 3.06) whereas in PEA/asystole patients, TMH was not significantly associated with good neurological outcome (adjusted odds ratio, 0.71; 95% confidence interval, 0.37 to 1.36).

Conclusions—In this large cohort of cardiac arrest patients, hypothermia was independently associated with an improved outcome at hospital discharge in patients presenting with VF/Vf. By contrast, TMH was not associated with good outcome in nonshockable patients. Further investigations are needed to clarify this lack of efficiency in PEA/asystole. (Circulation. 2011;123:877-886.)
Usefulness of Mild Therapeutic Hypothermia for Hospitalized Comatose Patients Having Out-of-Hospital Cardiac Arrest

Avishag Laish-Farkash, MD, PhD<sup>a,c</sup>, Shlomi Matetzky, MD<sup>a,c</sup>, Dan Oieru, MD<sup>a</sup>, Amir Sandach, PhD<sup>d</sup>, Niza Levi, SRN<sup>a</sup>, Jacob Or, MD<sup>b,c</sup>, Johnatan Rieck, MD<sup>b</sup>, Alon Barsheshet, MD<sup>a,c</sup>, and Hanooh Hod, MD<sup>a,c</sup>,<sup>•</sup> (Am J Cardiol 2011;108:173–178)

Overall outcomes and complications

<table>
<thead>
<tr>
<th>Variable</th>
<th>VF</th>
<th>p Value</th>
<th>Asystole</th>
<th>p Value vs VF</th>
<th>PEA</th>
<th>p Value vs VF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes (n = 86)</td>
<td>No (n = 24)</td>
<td>p Value</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>26 (30%)</td>
<td>15 (63%)</td>
<td>0.004</td>
<td>11 (61%)</td>
<td>0.01</td>
<td>4 (67%)</td>
</tr>
<tr>
<td>CPC 1 or 2</td>
<td>57 (66%)</td>
<td>2 (8%)</td>
<td>&lt;0.0001</td>
<td>2 (11%)</td>
<td>&lt;0.0001</td>
<td>0</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>35 (62%)</td>
<td>13 (24%)</td>
<td>0.51</td>
<td>11 (61%)</td>
<td>0.97</td>
<td>2 (33%)</td>
</tr>
<tr>
<td>Sepsis</td>
<td>13 (15%)</td>
<td>8 (33%)</td>
<td>0.05</td>
<td>6 (33%)</td>
<td>0.07</td>
<td>2 (33%)</td>
</tr>
<tr>
<td>Any bleeding</td>
<td>11 (13%)</td>
<td>6 (25%)</td>
<td>0.14</td>
<td>5 (28%)</td>
<td>0.11</td>
<td>1 (17%)</td>
</tr>
<tr>
<td>Seizures (clinical/electroencephalography)</td>
<td>21 (24%)</td>
<td>17 (71%)</td>
<td>&lt;0.0001</td>
<td>12 (67%)</td>
<td>0.0005</td>
<td>5 (83%)</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>13 (15%)</td>
<td>0</td>
<td>0.04</td>
<td>0</td>
<td>0.08</td>
<td>0</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>16 (19%)</td>
<td>2 (8%)</td>
<td>0.23</td>
<td>1 (5.5%)</td>
<td>0.18</td>
<td>1 (17%)</td>
</tr>
<tr>
<td>Pulmonary congestion</td>
<td>14 (16%)</td>
<td>8 (33%)</td>
<td>0.07</td>
<td>5 (28%)</td>
<td>0.25</td>
<td>3 (50%)</td>
</tr>
<tr>
<td>Need for catecholamines during MTH</td>
<td>15 (17%)</td>
<td>5 (21%)</td>
<td>0.70</td>
<td>3 (17%)</td>
<td>0.94</td>
<td>2 (33%)</td>
</tr>
</tbody>
</table>

Therefore, although the use of MTH in VF is recommended, its use in non-VF rhythms is debatable and needs further investigation.
Return of spontaneous circulation (ROSC)

Ventilatory and oxygenation optimization
- Maintain O2 saturation between 94 et 96%
- Capnography and airway securization
- Do not hyperventilate

Cardio-circulatory management
- Fluid status assessment
- Curable cause (physical examination, ECG)?

Consider hypothermia

Adapted response to verbal command?

Consider coronary angiogram

Evidence of extra-cardiac cause?

ICU management
- Prevention of complications (hypothermia side effects, infections...)
- Organ failure assistance
- Neurological evaluation

Mongardon et al. Annals of Intensive Care 2011, 1:45
Long term management

- Cardiac and neurological rehabilitation
- Nutrition
- Family counseling
- AICD?