Post Cardiac Arrest Management

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MEDICINE

Disclosure: No Industry or commercial support
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5R01HL071568 (co-PI: mechanisms of neuro recovery after lab CPR)
and R01 NS074425 (IDEF - co-I: multicenter ICH study)
Chair – "Evidence-based guideline: Reducing brain injury following cardiopulmonary resuscitation"
American Academy of Neurology – *under peer review*

Member – AHA-ILCOR 2015 CPR Guideline Review Panel
Writing Panel – Post Arrest Chapter of 2015 CPR Guidelines
Science Subcommittee member, ECC AHA (CPR Guidelines)
Science Taskforce of the Get-with-the-Guidelines Resuscitation of AHA

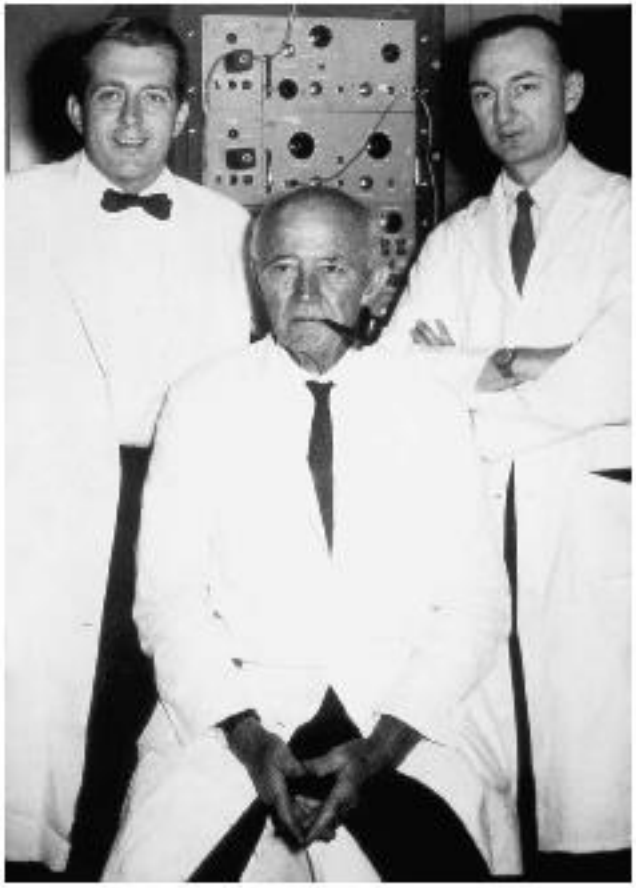
Immediate Past President /
Chair, Global Partners – Neurocritical Care Society

Objectives:

- Review Post Cardiac Arrest Syndrome: brain injury
- Impact of temperature on brain injury
- Clinical studies of temperature management after CPR
- Existing guidelines and upcoming considerations

Kouwenhoven WB, Jude J, Knickerbocker G.
Closed chest cardiac massage.
JAMA 1960;173:1064–7.

Fig. 2.



Johns Hopkins Hospital 1950-1960

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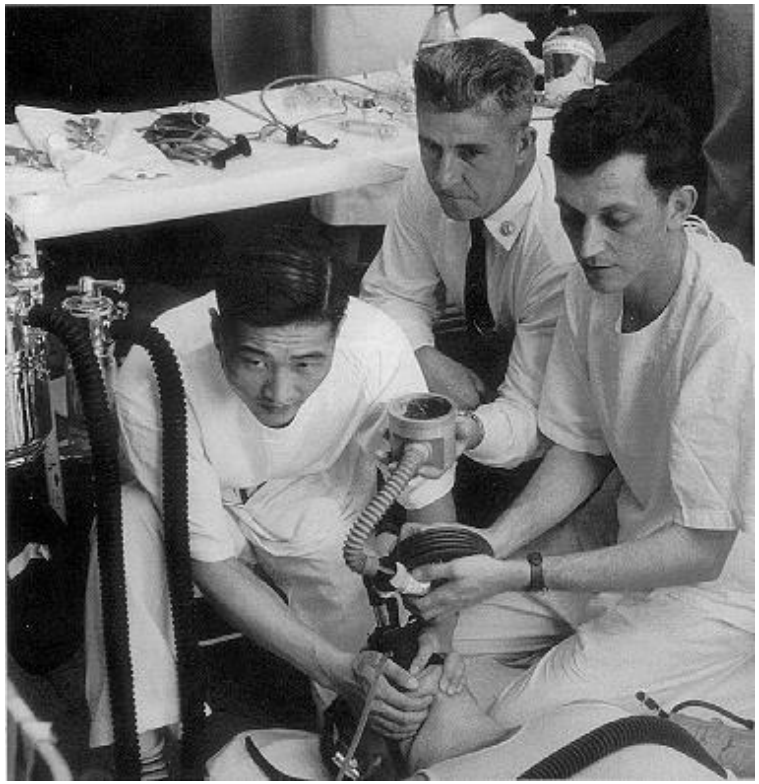
SAFAR P, McMAHON. Mouth-to-airway emergency artificial respiration. M.J Am Med Assoc. 1958 Mar 22;166(12):1459-60.



Dr. C. Park, Anesthesia Resident, Baltimore City Hospital; Capt. Martin McMahon, Chief, Baltimore Fire Department Ambulance Service and Dr. Peter Safar, Chief, Department of Anesthesia, Baltimore City Hospital, performing one of the earliest resuscitation studies using CPR.

ABCs of resusciation

Baltimore City Hospitals 1950s-1960
(now Johns Hopkins Bayview Medical Center)

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Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION
Circulation. 2008 Dec 2;118(23):2452-83.

ILCOR Consensus Statement

Resuscitation. 2008 Dec;79(3):350-79.
Int Emerg Nurs 2009 Oct;17(4):203-25

Post–Cardiac Arrest Syndrome

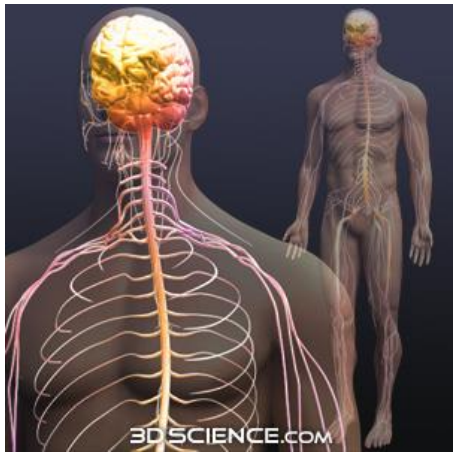
Epidemiology, Pathophysiology, Treatment, and Prognostication

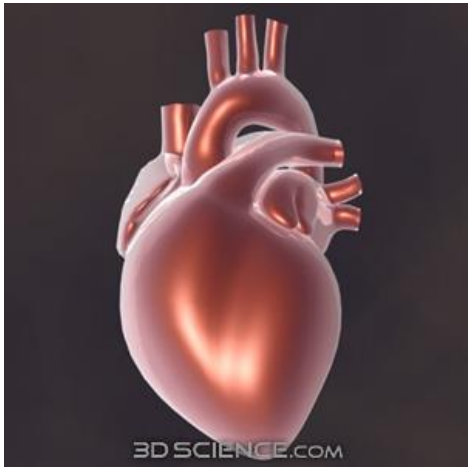
A Consensus Statement From the International Liaison Committee on Resuscitation (American Heart Association, Australian and New Zealand Council on Resuscitation, European Resuscitation Council, Heart and Stroke Foundation of Canada, InterAmerican Heart Foundation, Resuscitation Council of Asia, and the Resuscitation Council of Southern Africa); the American Heart Association Emergency Cardiovascular Care Committee; the Council on Cardiovascular Surgery and Anesthesia; the Council on Cardiopulmonary, Perioperative, and Critical Care; the Council on Clinical Cardiology; and the Stroke Council

Endorsed by the American College of Emergency Physicians, Society for Academic Emergency Medicine, Society of Critical Care Medicine, and Neurocritical Care Society

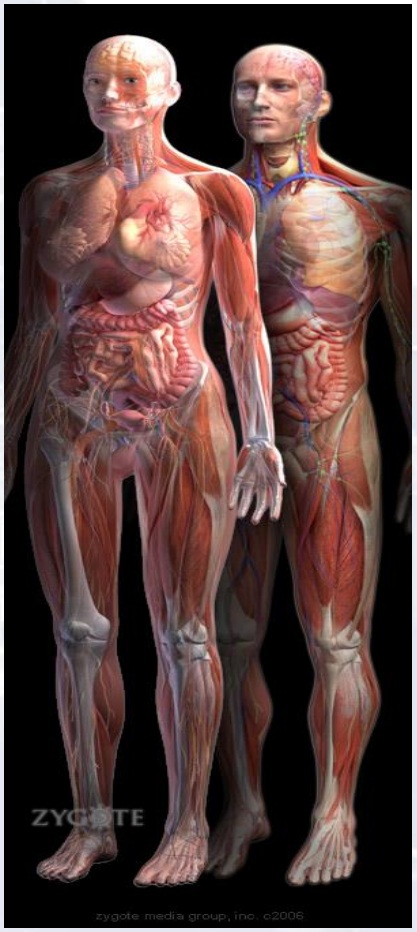
Robert W. Neumar, MD, PhD, Co-Chair; Jerry P. Nolan, FRCA, FCEM, Co-Chair; Christophe Adrie, MD, PhD; Mayuki Aibiki, MD, PhD; Robert A. Berg, MD, FAHA; Bernd W. Böttiger, MD, DEAA; Clifton Callaway, MD, PhD; Robert S.B. Clark, MD; Romergryko G. Geocadin, MD; Edward C. Jauch, MD, MS; Karl B. Kern, MD; Ivan Laurent, MD; W.T. Longstreth, Jr, MD, MPH; Raina M. Merchant, MD; Peter Morley, MBBS, FRACP, FANZCA, FJFICM; Laurie J. Morrison, MD, MSc; Vinay Nadkarni, MD, FAHA; Mary Ann Peberdy, MD, FAHA; Emanuel P. Rivers, MD, MPH; Antonio Rodriguez-Nunez, MD, PhD; Frank W. Sellke, MD; Christian Spaulding, MD; Kjetil Sunde, MD, PhD; Terry Vanden Hoek, MD

Post–Cardiac Arrest Syndrome

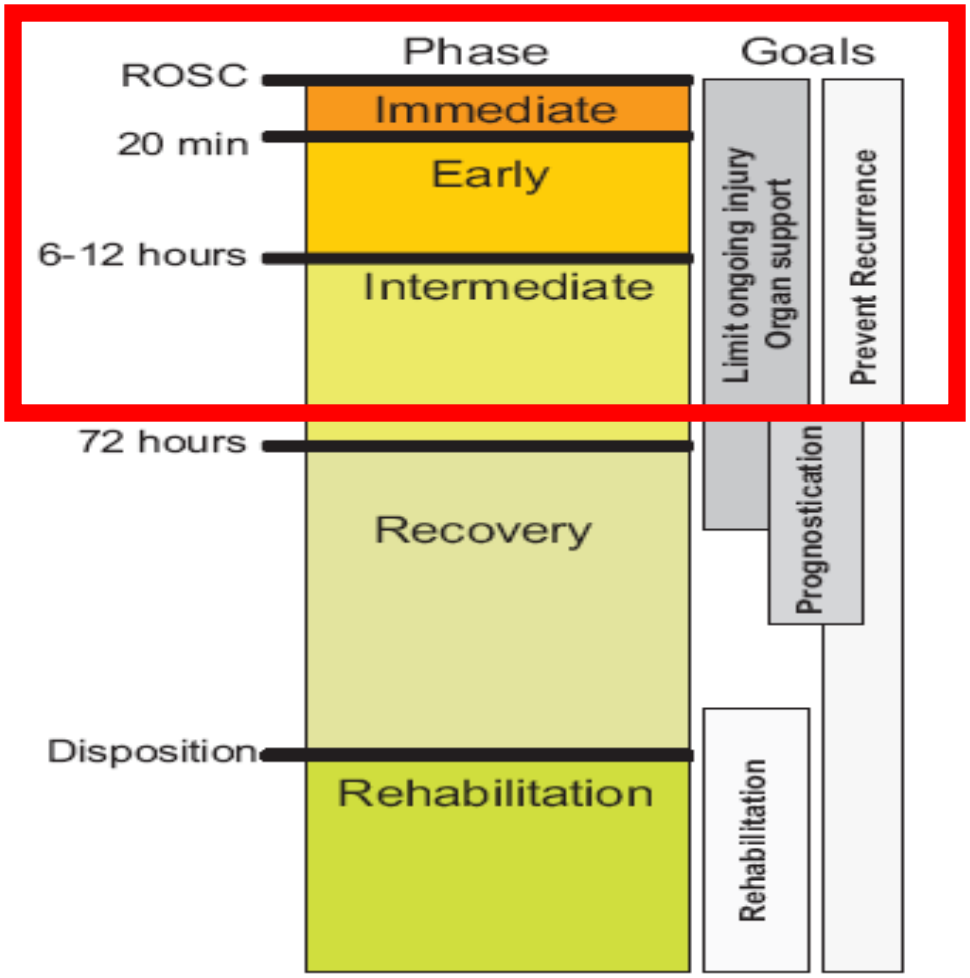




- Post– cardiac arrest brain injury
  - Systemic ischemia-reperfusion response
  - Persistent precipitating pathology
- Post–cardiac arrest myocardial dysfunction



Post Cardiac Arrest Syndrome Therapeutic Strategies



Goal Directed Therapy
Early Hemodynamic Optimization

Oxygenation

Ventilation

Circulatory Support

Management of ACS

Therapeutic hypothermia

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Cardiac Arrest and Death

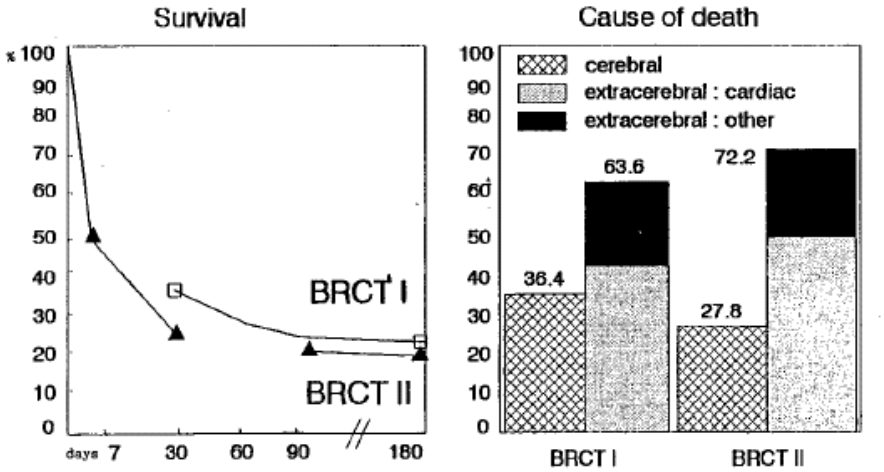
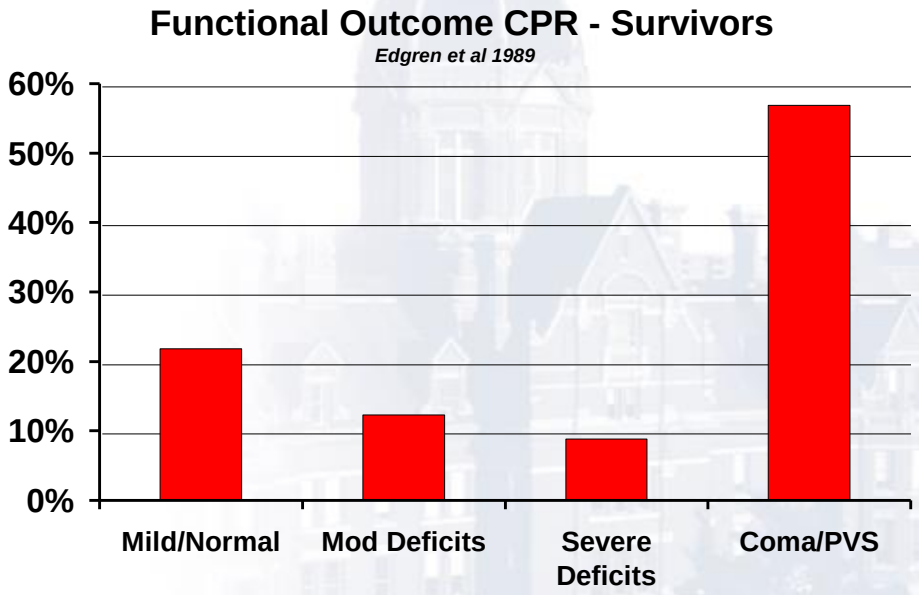


Figure 47.2. Mortality/survival and cause of death in comatose survivors of cardiac arrest (Brain Resuscitation Clinical Trial I [BRCT I], $n = 262$; Brain Resuscitation Clinical Trial II [BRCT II], $n = 516$).



Brain Injury after Global Ischemia

Brain Selective Vulnerability

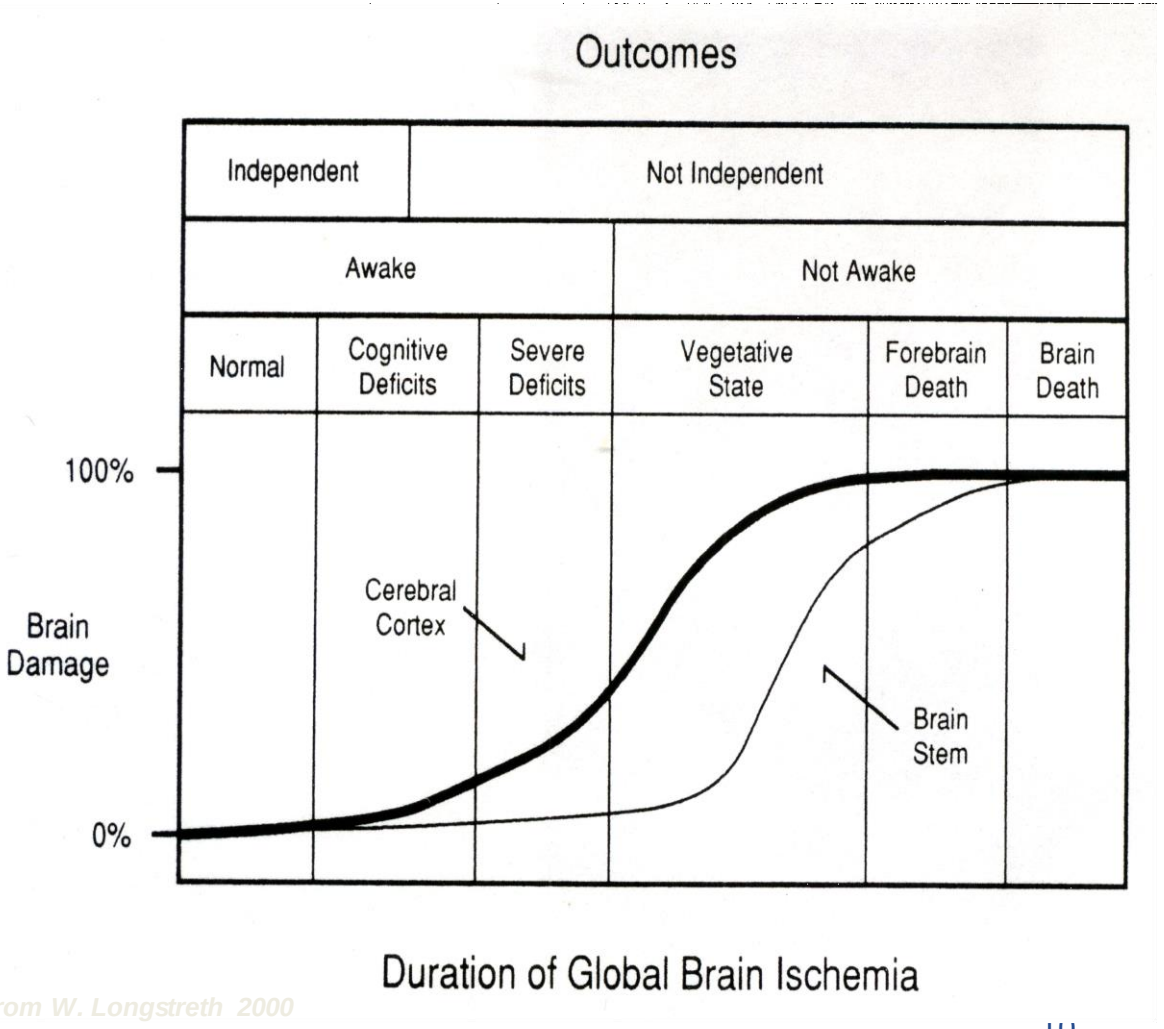
- CA-1 hippocampus
- Neocortex cell layers

Brain Relative Vulnerability

- Thalamus
- Cerebellum (Purkinjie cells
- Putamen and Caudate

Relative Tolerance to Ischemia

- Brainstem



From W. Longstreth 2000

Global Ischemia

Acute Presentation

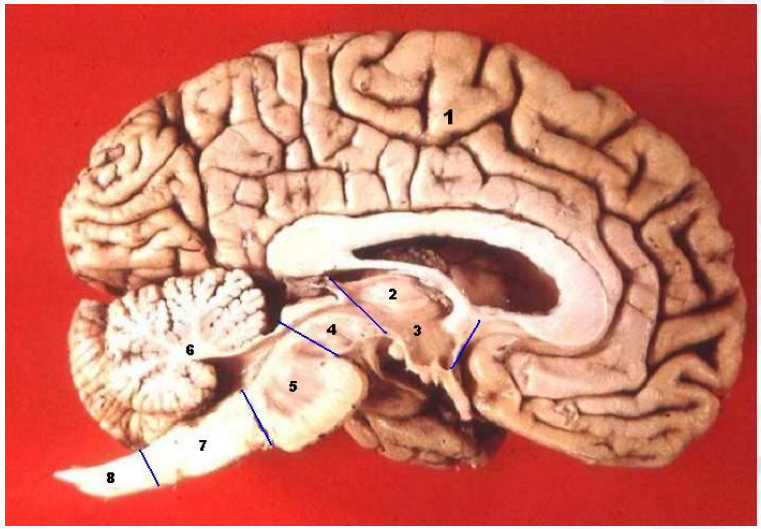
Brain Death

Coma/PVS

Stuporous/ Delirious

Cognitive Deficit

Seizures (cortex only)



Problem: Multi-systems

Cortex

Subcortex

Thalamus

Upper Brainstem

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Graveyard of Clinical Trials:
Neuroprotection in Global Cerebral Ischemia

Thiopental LD - no benefit
BRCT1 (*NEJM*, 1986) n=262

Glucocorticoid - associated with complications
BRCT1 (*JAMA*, 1989) n=262

Nimodipine – no benefit
Roine, et al (JAMA, 1990) n=748

Lidoflazine - no benefit
BRCT 2 (*NEJM*, 1991) n=520

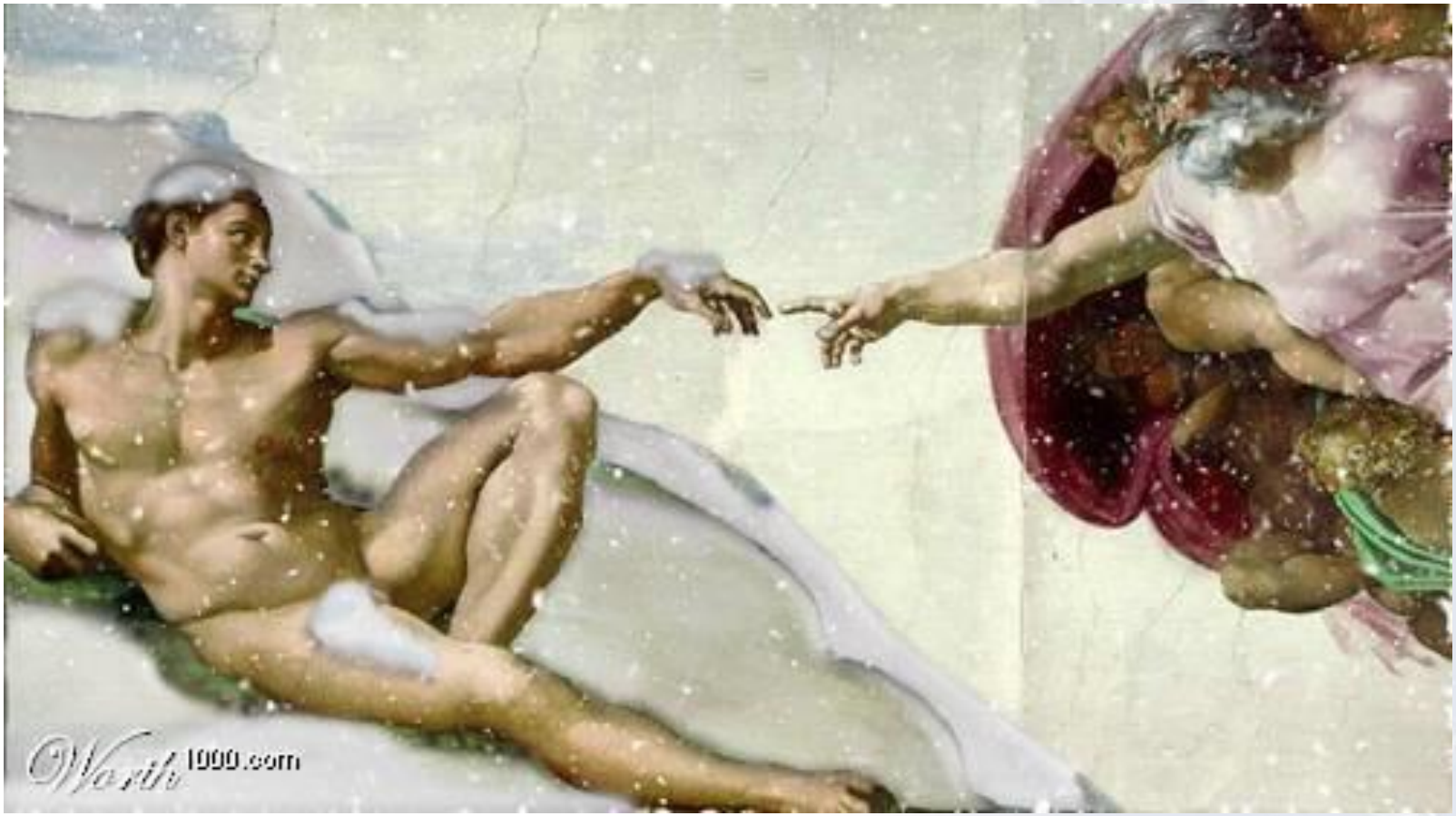
Non-Glucose IV Fluid – no benefit
Longstreth, et al (Neurology 1993) n=748

Magnesium/Diazepam – no benefit
Longstreth, et al (Neurology 2002) n=300

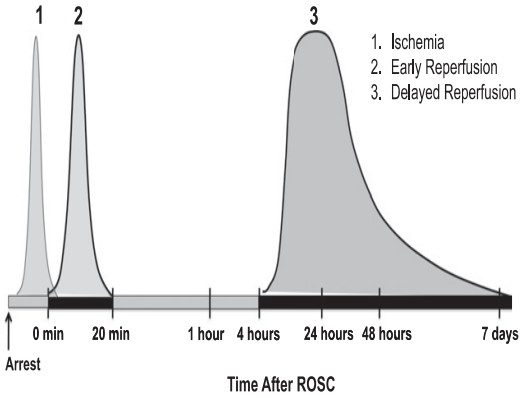
Vasopressin - no neuro benefit
Vasopressin–OHA V-Fib Arrest (Lancet, 2001) n=40
Canadian Vasopressin - Epi Study (Lancet, 2001) n=200

Except for IV-TPA no drug improves outcome in brain ischemia!!

And then there was …THERAPEUTIC HYPOTHERMIA



Injury Mechanism TH Protective Mechanism



Box 1
Mechanisms of anoxic-ischemic brain injury

Immediate

1. Cellular energy depletion, with anaerobic metabolism
2. Collapse of transmembrane sodium and potassium gradients
3. Failure of synaptic transmission, axonal conduction, and action potential firing
4. Intracellular acidosis
5. Hypercalcemia
6. Glutamate release, with neuronal hyperexcitability
7. Activation of intracellular enzymatic systems (protein kinase C and B, calcium/calmodulin-dependent protein kinase II, mitogen-activated protein kinases, phospholipase A2, C and D).
8. Mitochondrial dysfunction
9. Reperfusion, with generation of reactive oxygen species and lipid peroxidation
10. Elevated production of nitric oxide and peroxynitrite
11. Blood-brain barrier dysfunction
12. Loss of cerebral autoregulation

Delayed

1. Release of proinflammatory mediators (eg, tumor necrosis factor-α and interleukin-1)
2. Inflammatory cells recruitment
3. Complement activation
4. Caspase activation with apoptosis
5. Coagulation activation

Data from Refs.[27,133–135]

Box 2
Protective mechanism of therapeutic hypothermia

Early

1. Decrease of cerebral metabolism
2. Decrease in mitochondrial injury and dysfunction
3. Improve ion pump function, decrease intracellular influx of calcium
4. Improve cell membrane leakage, decrease intracellular acidosis
5. Decrease production of reactive oxygen species
6. Decrease formation of cytotoxic edema

Late

1. Decrease of local production of endothelin and thromboxane A2, increase generation of prostaglandins
2. Improve tolerance for ischemia
3. Decrease neuroinflammation
4. Decrease apoptosis
5. Decrease cerebral thermo-pooling
6. Decrease vascular permeability
7. Activation of protective genes
8. Suppression of cortical spreading depression
9. Suppression of seizure activity
10. Decrease coagulation activation and formation of microthrombi

Data from Refs.[71,80]

Da Silva & Frontera
Cardiol Clin 31 (2013) 637–655

Cardiac Rhythms/Place of Arrest:

Markers of Severity-Not effectiveness of TX

Place of Arrest:

OOHCA – Generally healthier

IHCA – Sicker pts in hospital

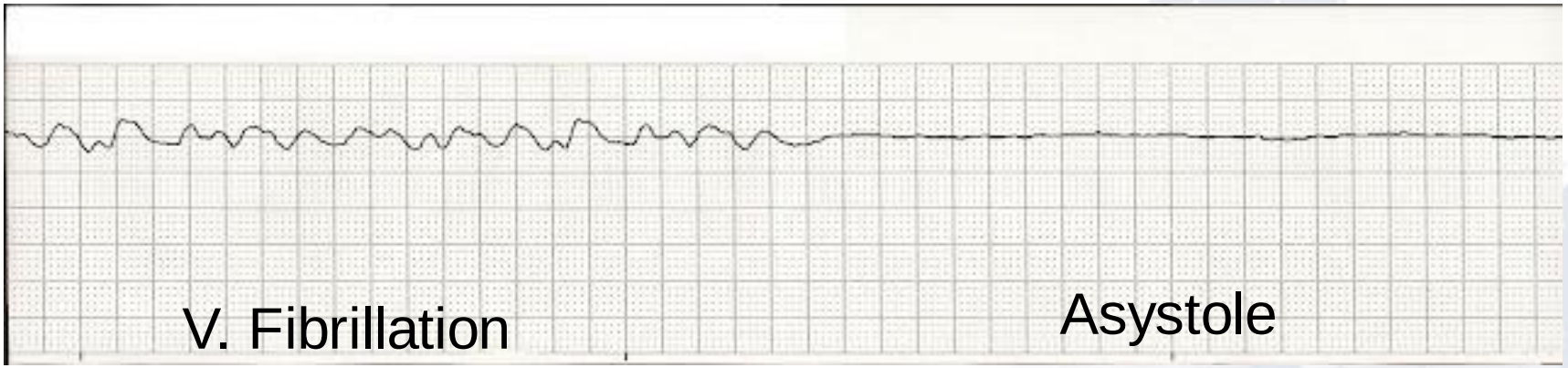
Cardiac Rhythms:

Shockable (pulseless VT/VF)

Non-Shockable Rhythms (PEA/Asystole)

Outcomes:
Cardiac Rhythms & Cardiac Arrest

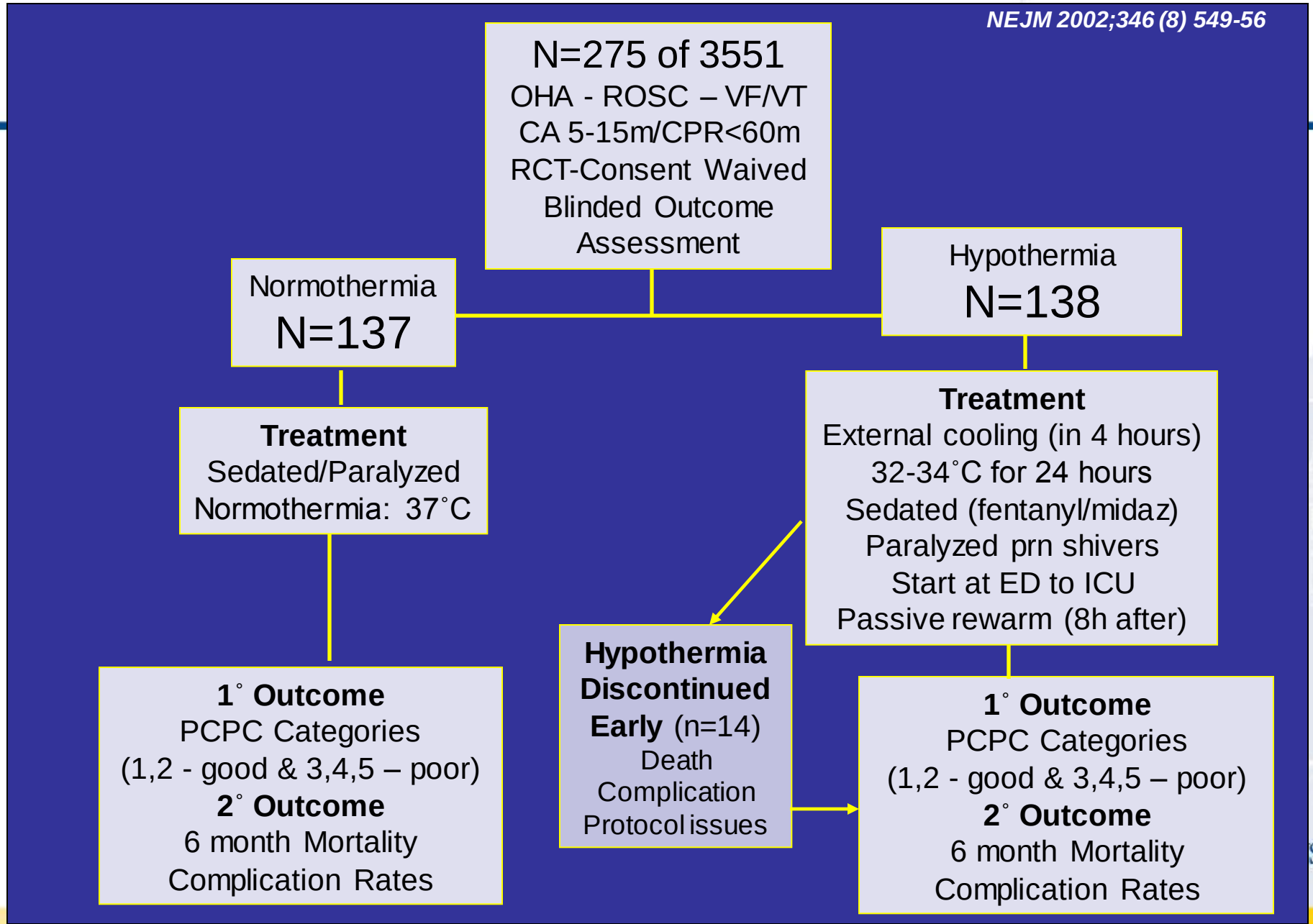
Ultimately all malignant arrythmias will deteriorate to asystole

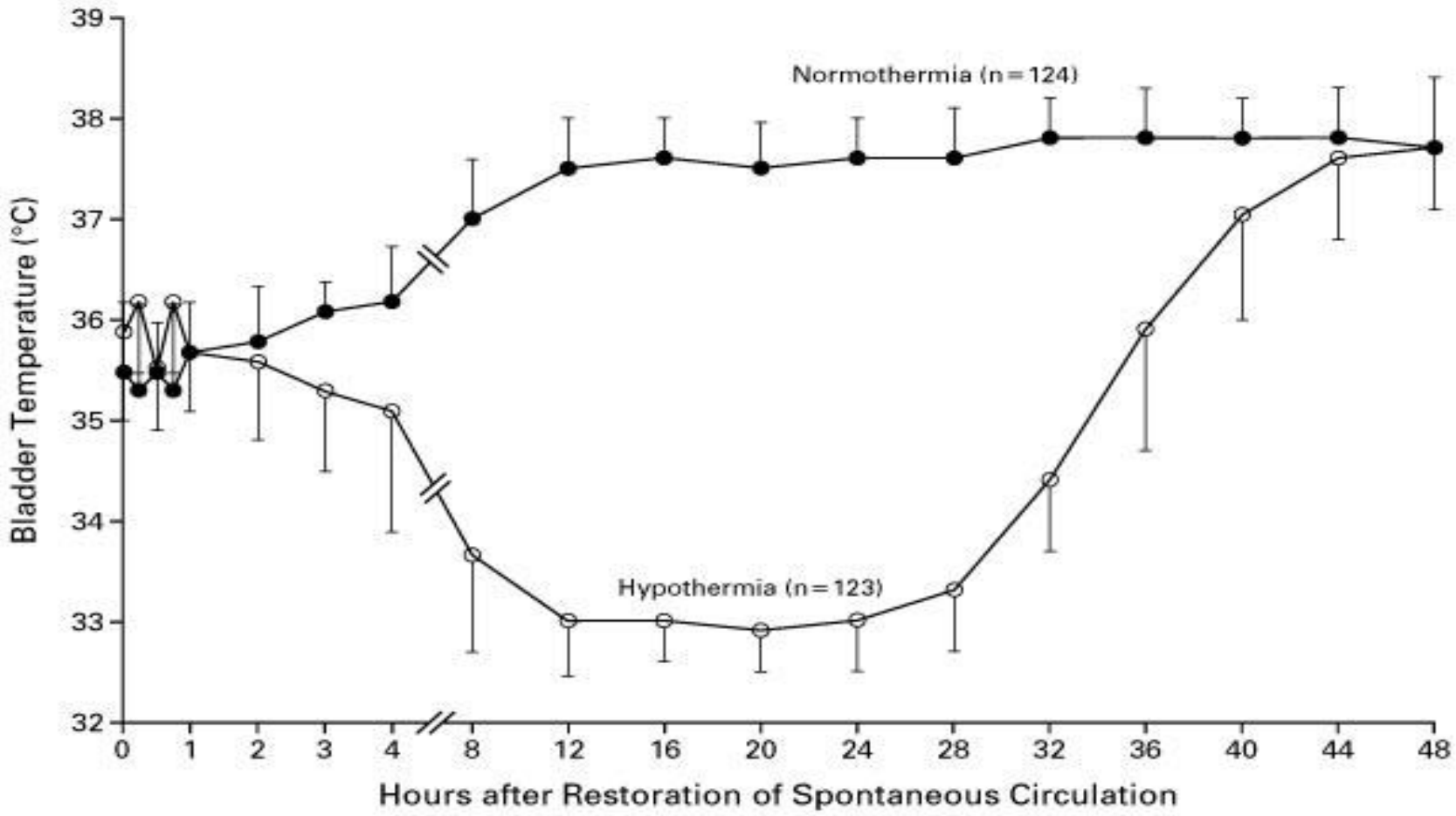


Better outcome	Poor Outcome
Shorter Arrest Duration	Longer Arrest Duration
Less co-morbidity	More co-morbidity

Which rhythm results in more brain injury?

HACA Study Group





The Hypothermia after Cardiac Arrest Study Group. N Engl J Med 2002;346:549-556.

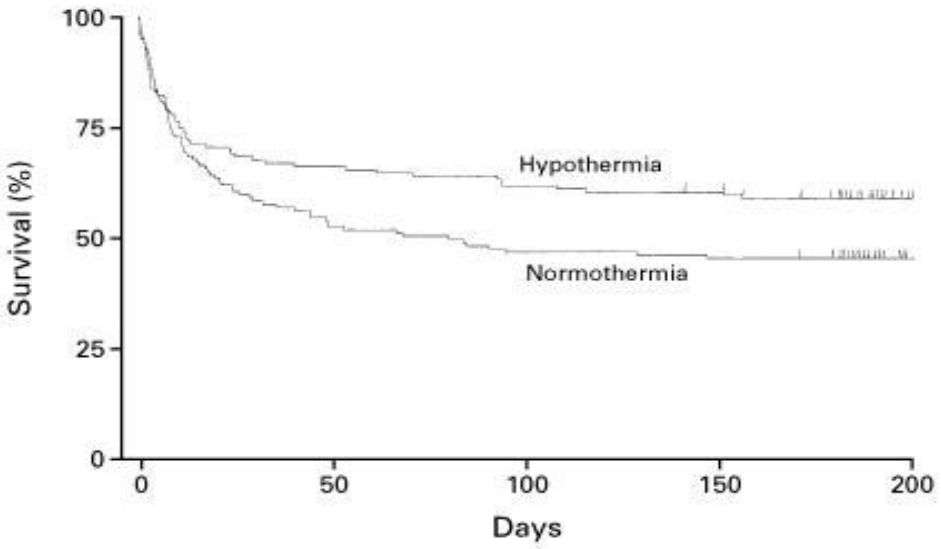


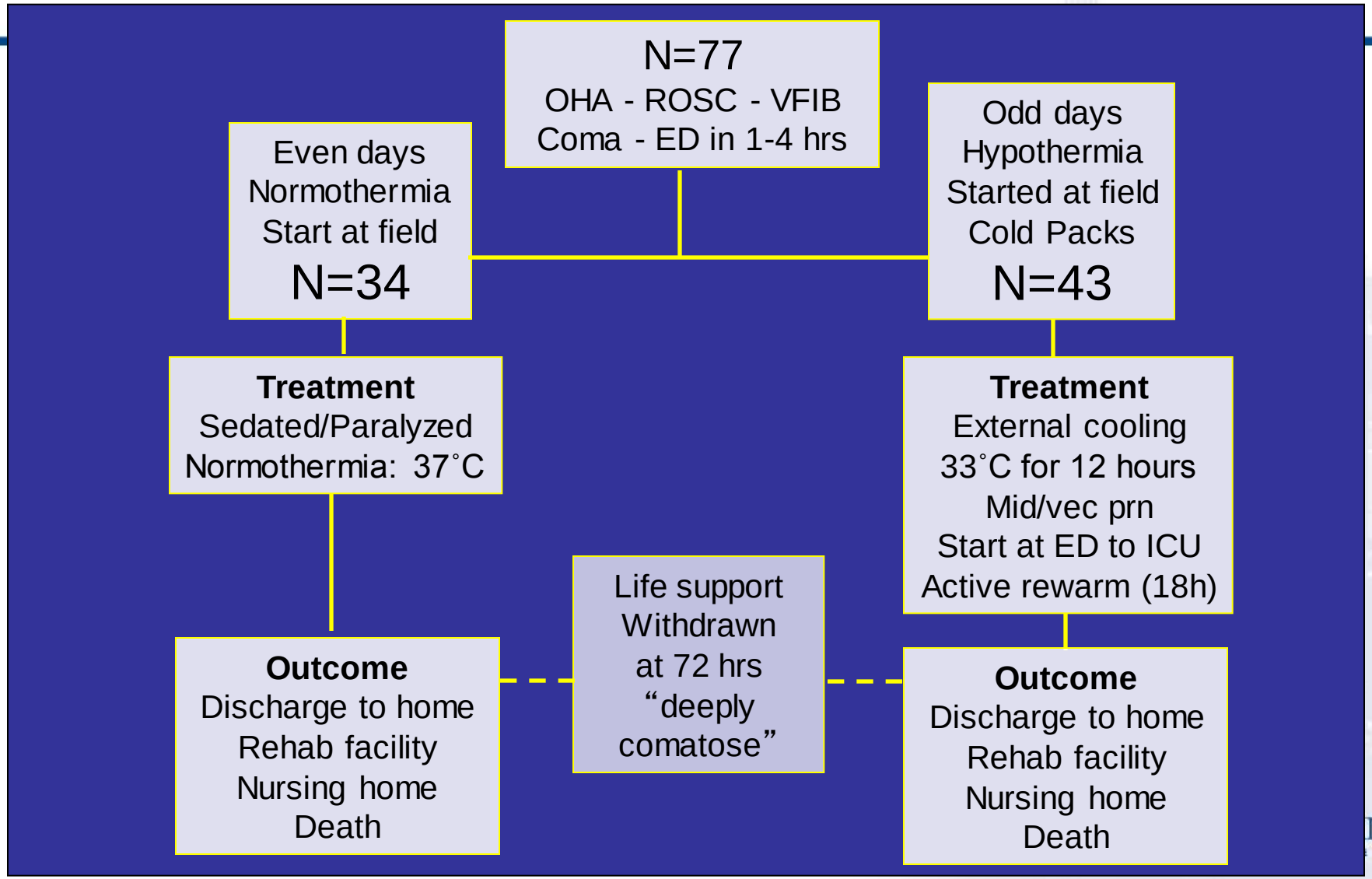
Table 2. Neurologic Outcome and Mortality at Six Months.

Outcome	Normothermia	Hypothermia	Risk Ratio (95% CI)*	P Value†
	no./total no. (%)			
Favorable neurologic outcome‡	54/ 137 (39)	75/ 136 (55)	1.40 (1.08–1.81)	0.009
Death	76/ 138 (55)	56/ 137 (41)	0.74 (0.58–0.95)	0.02

The Hypothermia after Cardiac Arrest Study Group. N Engl J Med 2002;346:549-556.

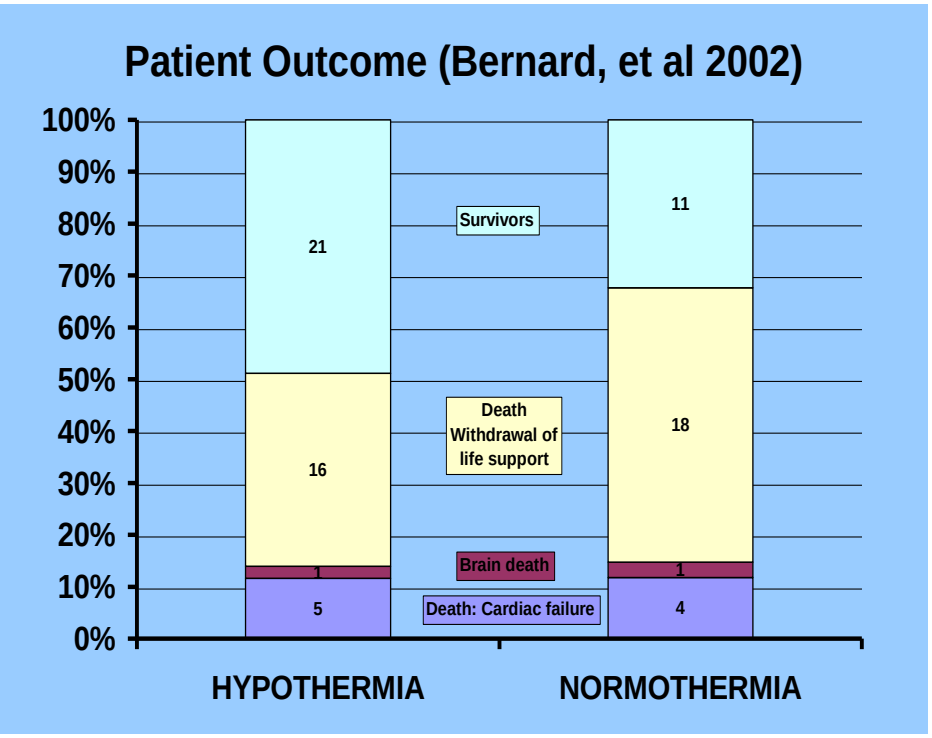
Induced Hypothermia

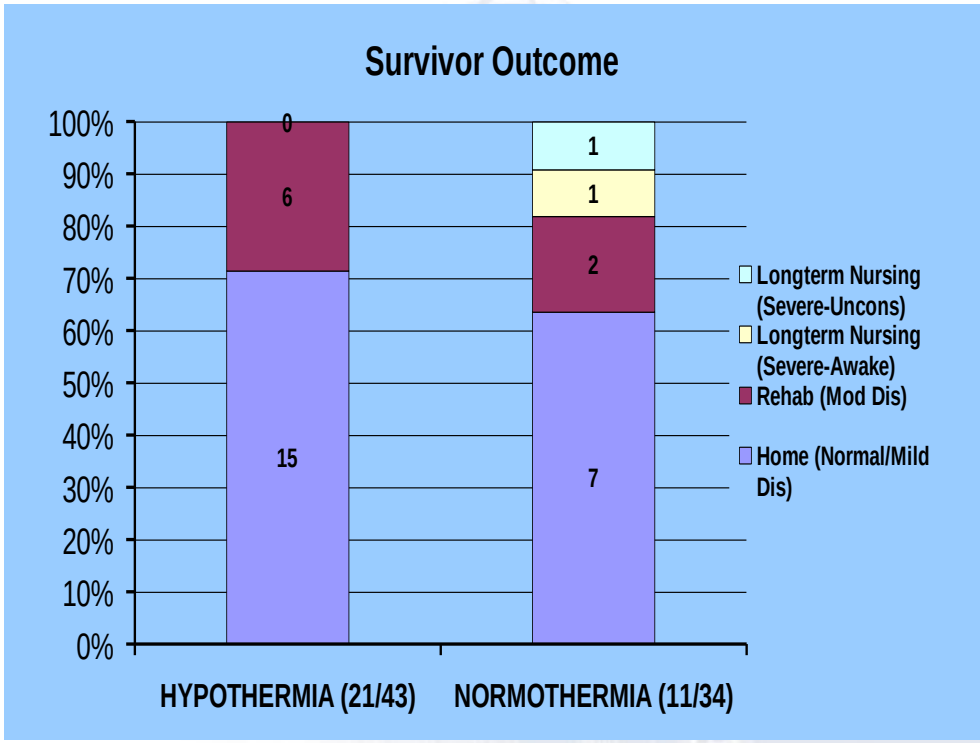
***Bernard, et al NEJM** 2002;346 (8) 557-63*



Induced Hypothermia

***Bernard, et al NEJM** 2002;346 (8) 557-63*





Good Outcome Rates
Hypothermia: 49% Normothermia: 26%
(95%CI: 13-43, p=0.046)

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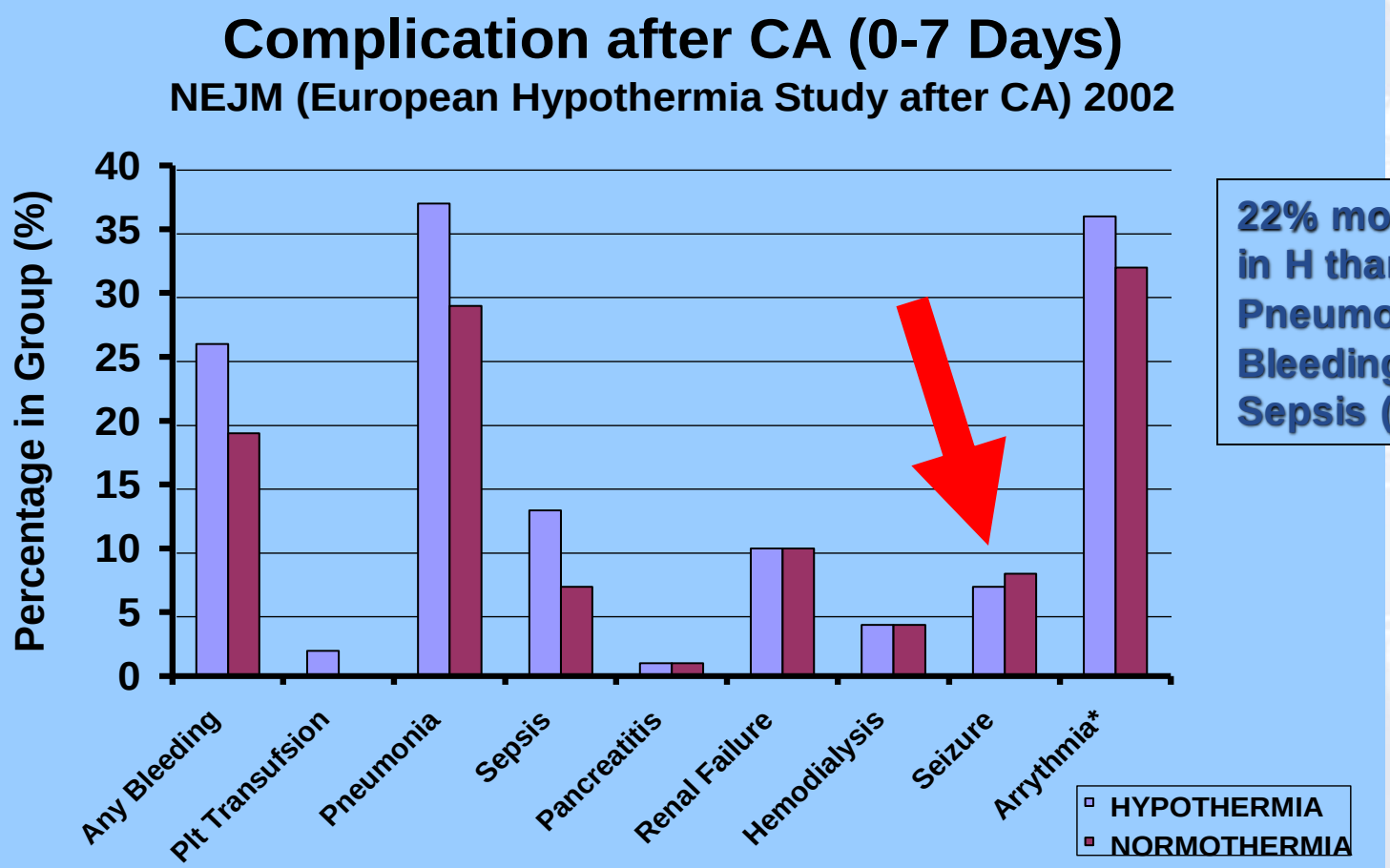
Therapeutic Hypothermia Complications after CA (0-7 days)



22% more complications in H than N (NS)
Pneumonia (NNH=12)
Bleeding (NNH=14)
Sepsis (NNH=16)

Part 9: Post Cardiac Arrest Care: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

Mary Ann Peberdy, Clifton W. Callaway, Robert W. Neumar, Romergryko G. Geocadin, Janice L. Zimmerman, Michael Donnino, Andrea Gabrielli, Scott M. Silvers, Arno L. Zaritsky, Raina Merchant, Terry L. Vanden Hoek and Steven L. Kronick

Circulation 2010;122;S768-S786

Chapter 9 S768

A comprehensive, structured, multidisciplinary system of care should be implemented in a consistent manner for the treatment of post–cardiac arrest patients **(Class I, LOE B)**. Programs should include as part of structured interventions therapeutic hypothermia; optimization of hemodynamics and gas exchange; immediate coronary reperfusion when indicated for restoration of coronary blood flow with percutaneous coronary intervention (PCI); glycemic control; and neurological diagnosis, management, and prognostication.

Multidisciplinary Critical Care at its best!

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Part 9: Post Cardiac Arrest Care: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

Mary Ann Peberdy, Clifton W. Callaway, Robert W. Neumar, Romergryko G. Geocadin, Janice L. Zimmerman, Michael Donnino, Andrea Gabrielli, Scott M. Silvers, Arno L. Zaritsky, Raina Merchant, Terry L. Vanden Hoek and Steven L. Kronick

Circulation 2010;122;S768-S786

Chapter 9 page S772

In summary, we recommend that comatose (ie, lack of meaningful response to verbal commands) adult patients with ROSC after out-of-hospital VF cardiac arrest should be cooled to 32° C to 34° C (89.6° F to 93.2° F) for 12 to 24 hours **(Class I, LOE B)**.

Induced hypothermia also may be considered for comatose adult patients with ROSC after in-hospital cardiac arrest of any initial rhythm or after out-of-hospital cardiac arrest with an initial rhythm of pulseless electric activity or asystole **(Class IIb, LOE B)**.

Non-shockable Rhythms: Mixed signal but not harmful

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

American Heart Association. Learn and Live.

Circulation 2011, 123:877-886:

Is Hypothermia After Cardiac Arrest Effective in Both Shockable and Nonshockable Patients? : Insights From a Large Registry

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

Clinical paper

Resuscitation 83 (2012) 202–207

Therapeutic hypothermia is associated with improved neurologic outcome and survival in cardiac arrest survivors of non-shockable rhythms☆

Justin B. Lundbye[a,b,*], Mridula Rai[a,b], Bhavadharini Ramu[a,b], Alireza Hosseini-Khalili[a], Dadong Li[a], Hanna B. Slim[a], Sanjeev P. Bhavnani[a,b], Sanjeev U. Nair[a], Jeffrey Kluger[a,b]

Resuscitation 82 (2011) 1162–1167

Clinical paper

Mild therapeutic hypothermia is associated with favourable outcome in patients after cardiac arrest with non-shockable rhythms☆

Christoph Testori, Fritz Sterz*, Wilhelm Behringer, Moritz Haugk, Thomas Uray, Andrea Zeiner, Andreas Janata, Jasmin Arrich, Michael Holzer, Heidrun Losert

Many more papers….

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The NEW ENGLAND JOURNAL of MEDICINE

Original Article

Targeted Temperature Management at 33°C versus 36°C after Cardiac Arrest

Niklas Nielsen, M.D., Ph.D., et al - TTM Trial Investigators

N Engl J Med Volume 369(23):2197-2206 December 5, 2013

Study objective: To compare two target temperatures, both intended to prevent fever.

International RCT: 950 unconscious adults after out-of hospital cardiac arrest of presumed cardiac cause to targeted temperature management at either 33°C or 36°C.

The primary outcome: all-cause mortality to end of the trial.

Secondary outcomes: composite of poor neurologic function (CPC and mRS) or death at 180 days

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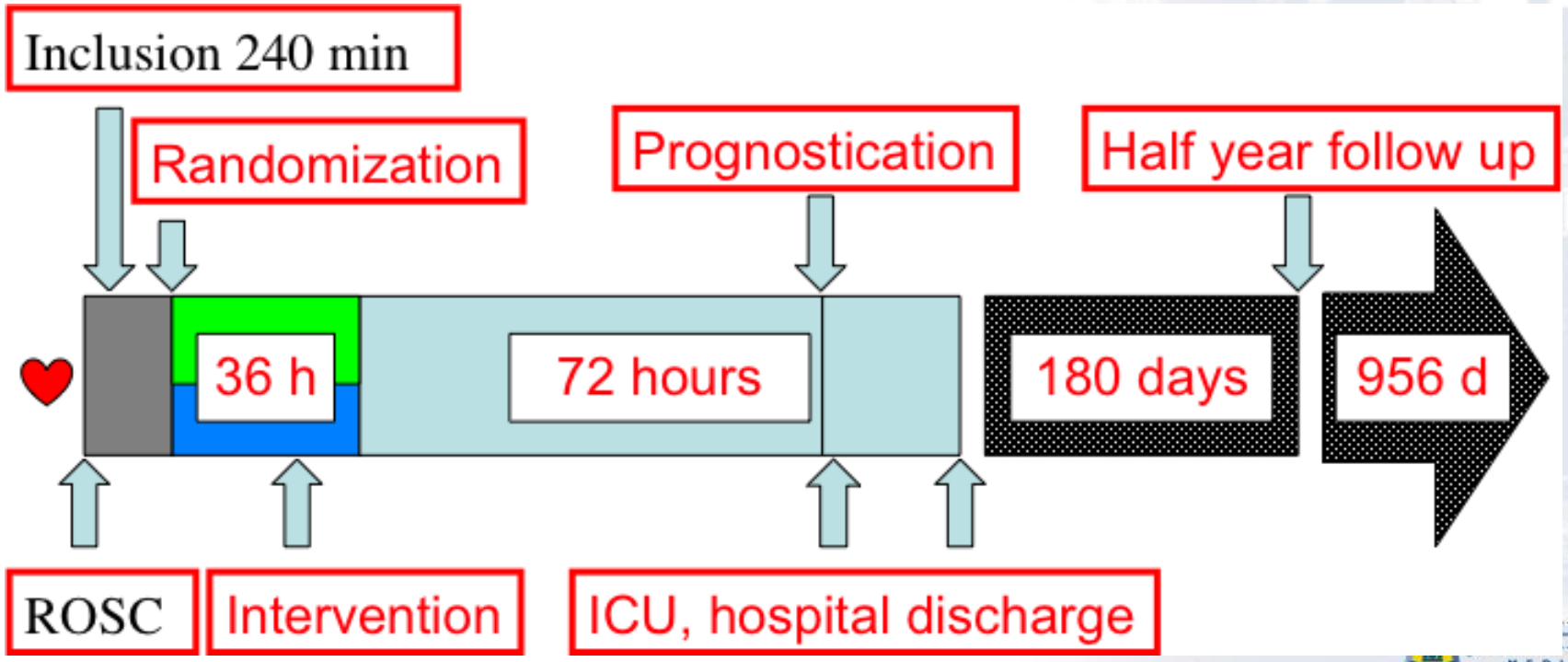
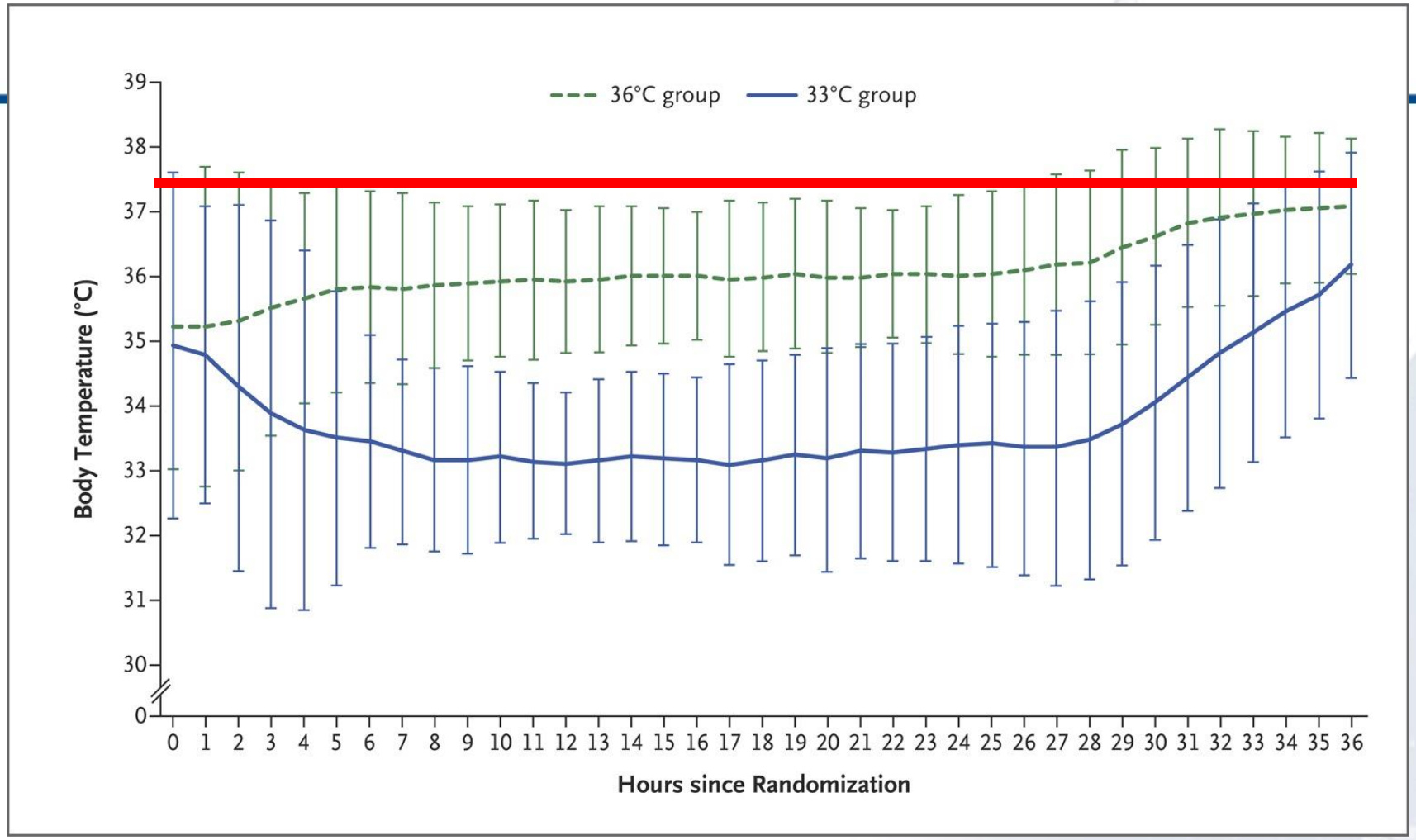
Design and timeline *(modified from N Nielsen)*

Inclusion Criteria

- Age >= 18 years
- Out-of-hospital cardiac arrest of presumed cardiac cause
- Unconsciousness (Glasgow Coma Score <8) after sustained return of spontaneous circulation (ROSC) (20 minutes of circulation)

Cooling device with feedback control; surface and endovascular mixed



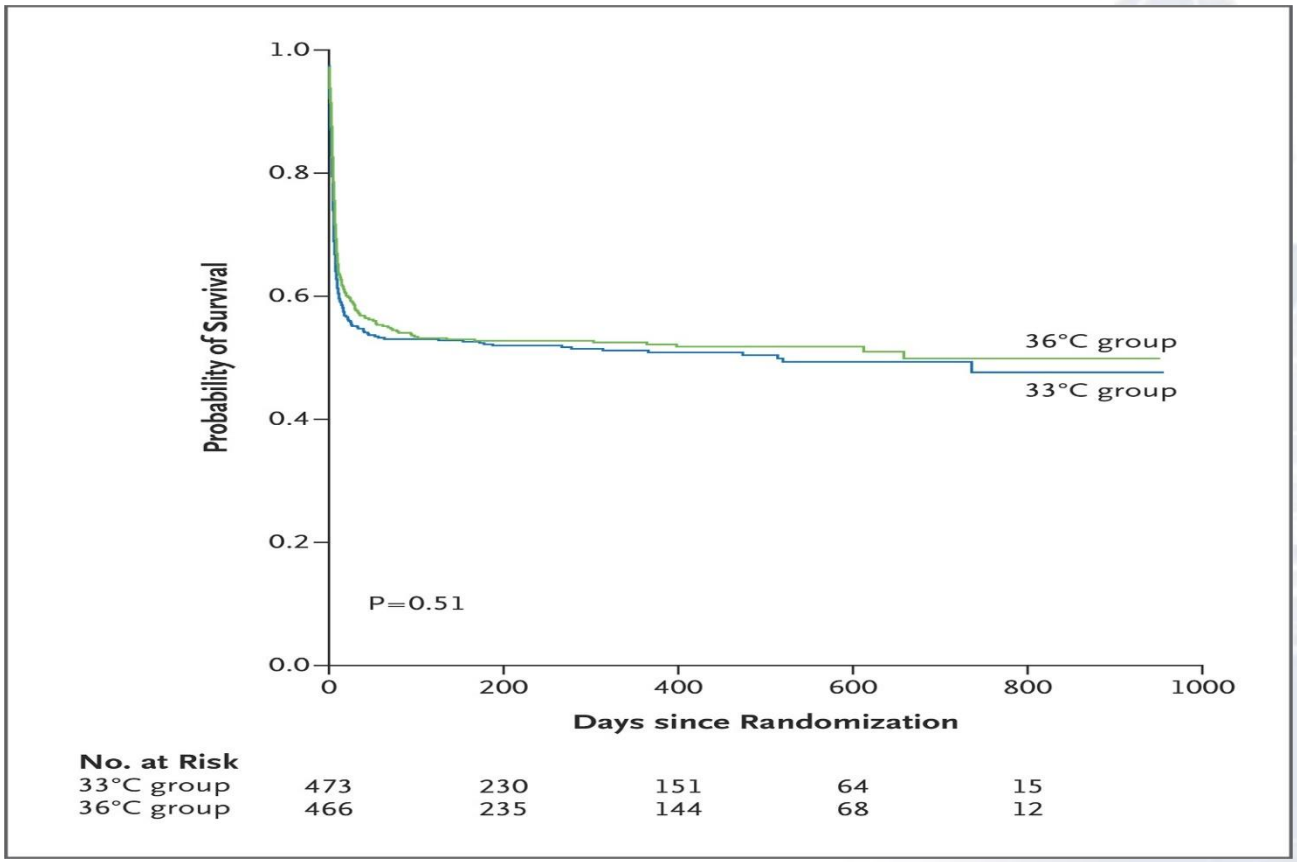


Nielsen N et al. N Engl J Med 2013;369:2197-2206

The NEW ENGLAND JOURNAL of MEDICINE

The NEW ENGLAND JOURNAL of MEDICINE

Nielsen N et al. N Engl J Med 2013;369:2197-2206



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The NEW ENGLAND JOURNAL of MEDICINE

Nielsen N et al. N Engl J Med 2013;369:2197-2206

Table 2. Outcomes.

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

* The hazard ratio is shown for the primary outcome, and risk ratios are shown for the secondary outcomes. CI denotes confidence interval.

† The neurologic follow-up was specified in the protocol to be performed at 180 days ±2 weeks, but the time to follow-up was in some cases several weeks longer for logistic reasons. The Cerebral Performance Category (CPC) scale ranges from 1 to 5, with 1 representing good cerebral performance or minor disability, 2 moderate cerebral disability (function is sufficient for independent activities of daily life), 3 severe cerebral disability, 4 coma or vegetative state, and 5 brain death. Scores on the modified Rankin scale range from 0 to 6, with 0 representing no symptoms, 1 no clinically significant disability despite some symptoms, 2 slight disability (patient is able to look after own affairs without assistance), 3 moderate disability (patient requires some help but is able to walk unassisted), 4 moderately severe disability (patient is unable to attend to own bodily needs), 5 severe disability (patient is bedridden), and 6 death.

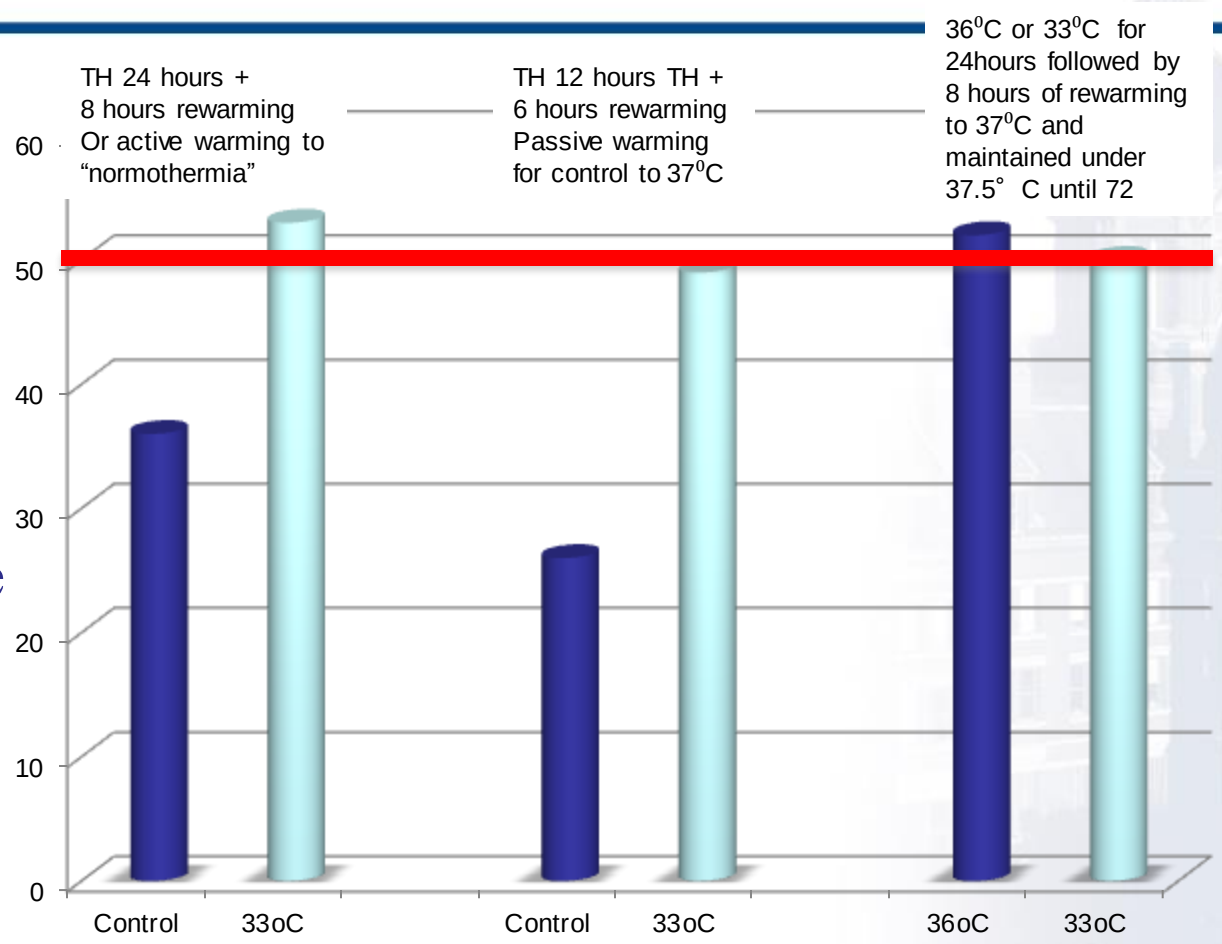
Table 3. Neurologic Scores.*

Variable	33°C Group	36°C Group
CPC at follow-up†		
Total no. of patients	469	464
Category — no. (%)		
1	195 (42)	183 (39)
2	23 (5)	39 (8)
3	17 (4)	20 (4)
4	6 (1)	2 (0.5)
5	228 (49)	220 (47)
P value for trend	0.85	
Best, or lowest numerical, CPC during trial		
Total no. of patients	472	466
Category — no. (%)		
1	209 (44)	205 (44)
2	25 (5)	41 (9)
3	37 (8)	37 (8)
4	201 (43)	183 (39)
5	NA	NA
P value for trend	0.89	
Modified Rankin scale score at follow-up†		
Total no. of patients	469	464
Score — no. (%)		
0	88 (19)	89 (19)
1	69 (15)	83 (18)
2	50 (11)	34 (7)
3	17 (4)	19 (4)
4	8 (2)	11 (2)
5	9 (2)	8 (2)
6	228 (49)	220 (47)
P value for trend	0.67	

* P values for trend were calculated with the use of the Cochran–Armitage test. NA denotes not applicable.

† The neurologic follow-up was specified in the protocol to be at 180±14 days, but the time to follow-up was in some cases several weeks longer for logistic reasons.

Good Outcomes Comparison: 3 Trials



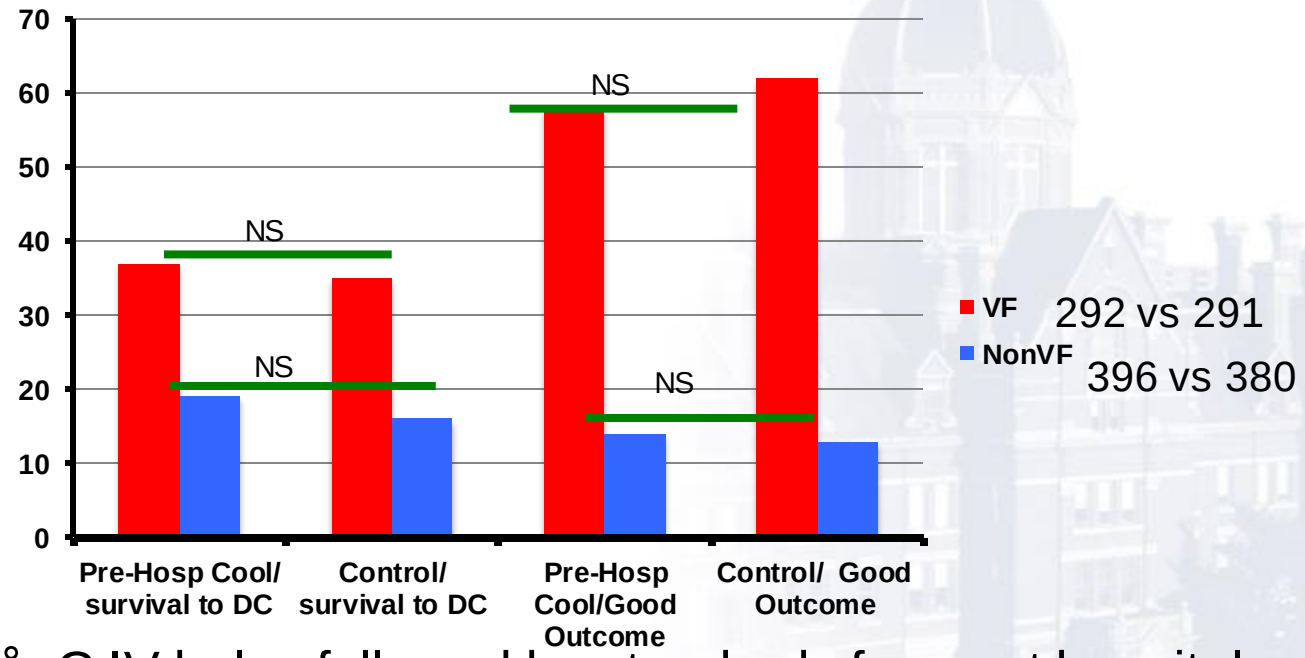
Original Investigation

Effect of Prehospital Induction of Mild Hypothermia on Survival and Neurological Status Among Adults With Cardiac Arrest

A Randomized Clinical Trial

Francis Kim, MD; Graham Nichol, MD, MPH; Charles Maynard, PhD; Al Hallstrom, PhD; Peter J. Kudenchuk, MD; Thomas Rea, MD, MPH; Michael K. Copass, MD; David Carlbom, MD; Steven Deem, MD; W. T. Longstreth Jr, MD; Michele Olsufka, RN; Leonard A. Cobb, MD

JAMA January 1, 2014 Volume 311, Number 1



2 liters of 4° C IV bolus followed by standard of care at hospital

**re-arrest in the treatment group (26% vs 21%, p=0.008)

lower O2 saturation (PaO2 189 vs 218, p<0.001),

pulmonary edema on CXR (41% vs 30%, p<0.001)

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Targeted temperature management following cardiac arrest : An update December 2013

- "…Pending formal Consensus on the optimal temperature, we suggest that clinicians provide postresuscitation care based on the current treatment recommendations (ILCOR/AHA). We accept that some clinicians may make a local decision to use a target temperature of 36° C pending this further guidance."

Part 8: Post–Cardiac Arrest Care

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2015 Recommendations—Updated

We recommend that comatose (ie, lack of meaningful response to verbal commands) adult patients with ROSC after cardiac arrest have TTM (Class I, LOE B-R for VF/pVT OHCA; Class I, LOE C-EO for non-VF/pVT (ie, “nonshockable”) and in-hospital cardiac arrest).

We recommend selecting and maintaining a constant temperature between 32ºC and 36ºC during TTM (Class I, LOE B-R).

Nuances of temperature range 32 to 36 degree Celsius

"essentially no patients for whom temperature control somewhere in the range between 32*C and 36*C is contraindicated"

Specific features of the patient may favor selection of one temperature over another for TTM. Higher temperatures (~36*C) might be preferred in patients for whom lower temperatures convey some risk (eg, bleeding),

Lower temperatures (~32*C) might be preferred when patients have clinical features that are worsened at higher temperatures (eg, seizures, cerebral edema).

caveat – lower temps (32*C) may be selected for worse neurologic injuries and result in the impression that it is less effective in practice

Hypothermia in the Prehospital Setting

2015 Recommendation—New

We recommend **against** the routine prehospital cooling of patients after ROSC with rapid infusion of cold intravenous fluids (Class III: No Benefit, LOE A).

Whether different methods or devices for temperature control outside of the hospital are beneficial is unknown.

Avoidance of Hyperthermia

2015 Recommendation—New

It may be reasonable to actively prevent fever in comatose patients after TTM (Class IIb, LOE C-LD).

Fever in the post–cardiac arrest patient who is not treated with TTM is associated with poor outcome.

After rewarming to normothermia from TTM, fever occurs in a significant proportion of patients. Occurrence of hyperthermia during the first few days after cardiac arrest was associated with worse outcome in some studies

Summary for TTM
I hope for now - 2014

OOHCA
VF/nonVF
Pre-hospital Cooling
No Benefit (?harmful)
Kim/Castren/Bernard

OOHCA
Pulseless VT/VF
TTM @ in-hospital
Beneficial outcome/qol
HACA/Bernard/Neilsen
Class 1

OOHCA/ PEA/Asystole
Neilsen subgroup
Class 1(?)

IHCA PEA/Asystole
TTM @ in-hospital
May be beneficial
Multiple studies
Class 2A-2B

IHA-CA (No RCT)
Multiple studies
Class 2B

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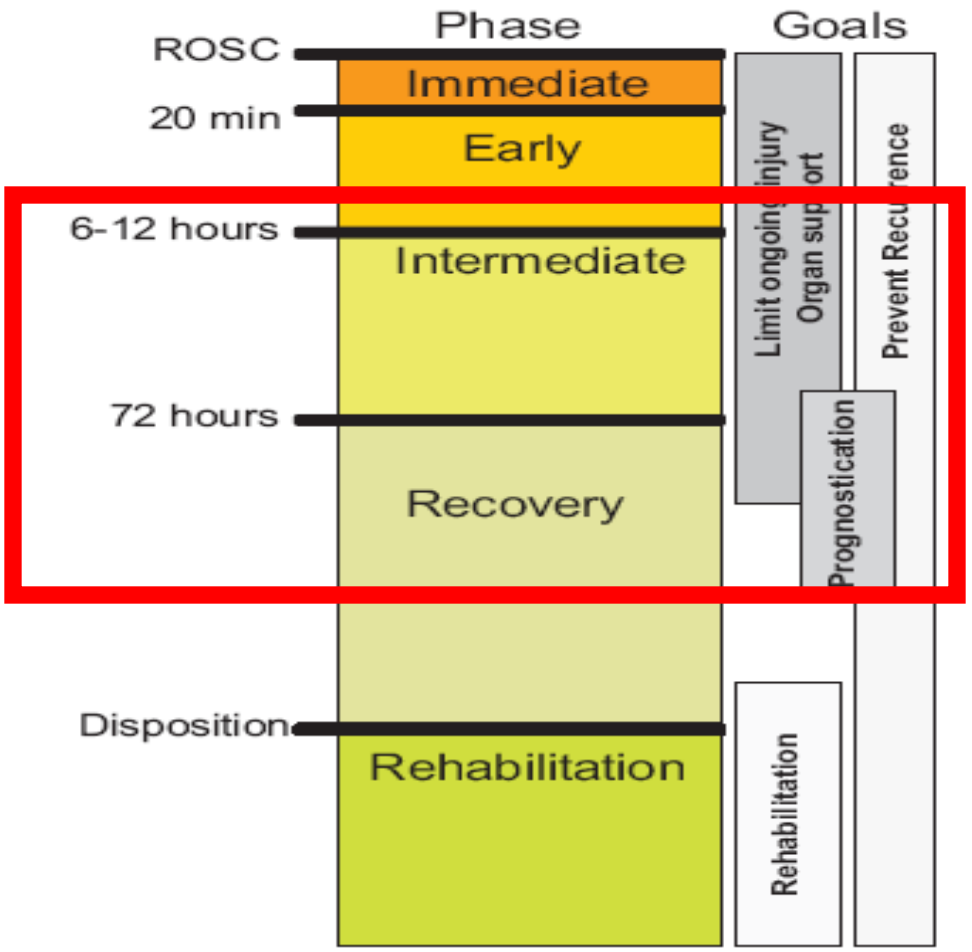
Post Cardiac Arrest Syndrome Therapeutic Strategies



Therapeutic hypothermia

Sedation and Neuromuscular Blockade

Seizure control

Glucose Control

Neuroprotection

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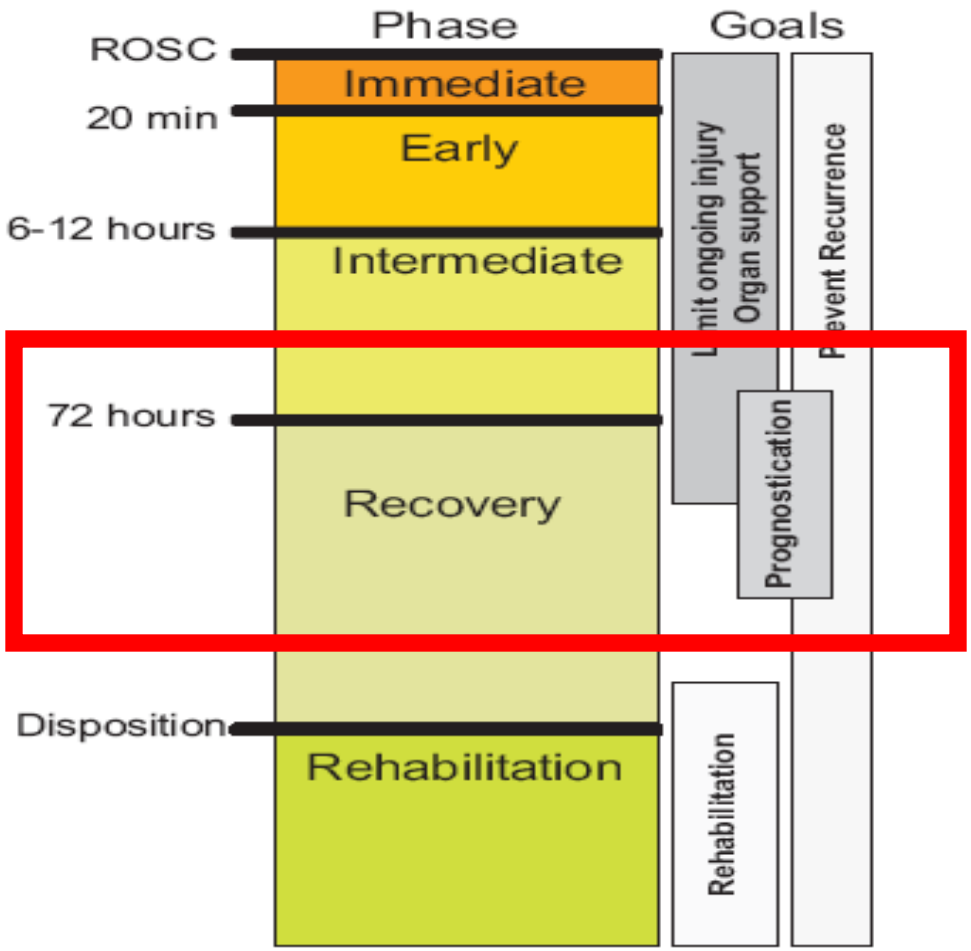
Post Cardiac Arrest Syndrome Therapeutic Strategies



Prognostication

Continue Care

Withdrawal of life-supporting therapies

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2015 Recommendation—New

Avoiding and immediately correcting hypotension (systolic blood pressure less than 90 mm Hg, MAP less than 65 mm Hg) during postresuscitation care may be reasonable (Class IIb, LOE C-LD).

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2015 Recommendations—Updated

Coronary angiography should be performed emergently (rather than later in the hospital stay or not at all) for OHCA patients with suspected cardiac etiology of arrest and ST elevation on ECG (Class I, LOE B-NR).

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2015 Recommendation—Updated

The benefit of any specific target range of glucose management is uncertain in adults with ROSC after cardiac arrest (Class IIb, LOE B-R).

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2015 Recommendation—Updated

Maintaining the PaCO2 within a normal physiological range, taking into account any temperature correction, may be reasonable (Class IIb, LOE B-NR).

To avoid hypoxia in adults with ROSC after cardiac arrest, it is reasonable to use the highest available oxygen concentration until the arterial oxyhemoglobin saturation or the partial pressure of arterial oxygen can be measured (Class IIa, LOE C-EO).

When resources are available to titrate the FIO2 and to monitor oxyhemoglobin saturation, it is reasonable to decrease the FIO2 when oxyhemoglobin saturation is 100%, provided the oxyhemoglobin saturation can be maintained at 94% or greater (Class IIa, LOE C-LD).

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2015 Recommendations—Updated

An EEG for the diagnosis of seizure should be promptly performed and interpreted, and then should be monitored frequently or continuously in comatose patients after ROSC (Class I, LOE C-LD).

The same anticonvulsant regimens for the treatment of status epilepticus caused by other etiologies may be considered after cardiac arrest (Class IIb, LOE C-LD).

AMERICAN ACADEMY OF NEUROLOGY

Special Article

Practice Parameter: Prediction of outcome in comatose survivors after cardiopulmonary resuscitation (an evidence-based review)

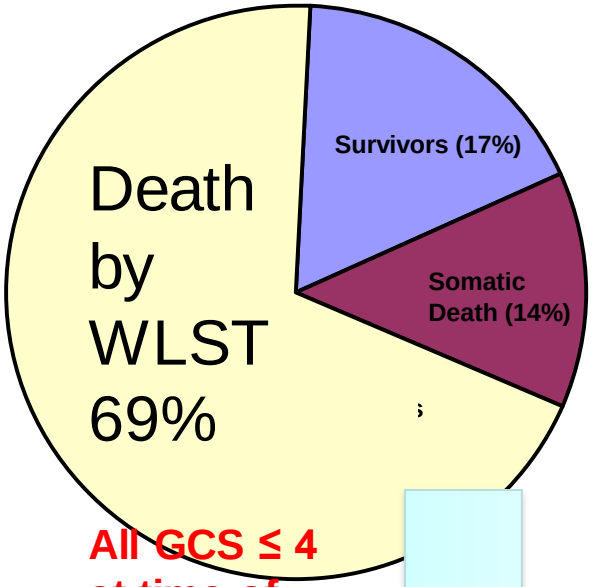
Report of the Quality Standards Subcommittee of the American Academy of Neurology

E.F.M. Wijdicks, MD; A. Hijdra, MD; G.B. Young, MD; C.L. Bassetti, MD; and S. Wiebe, MD

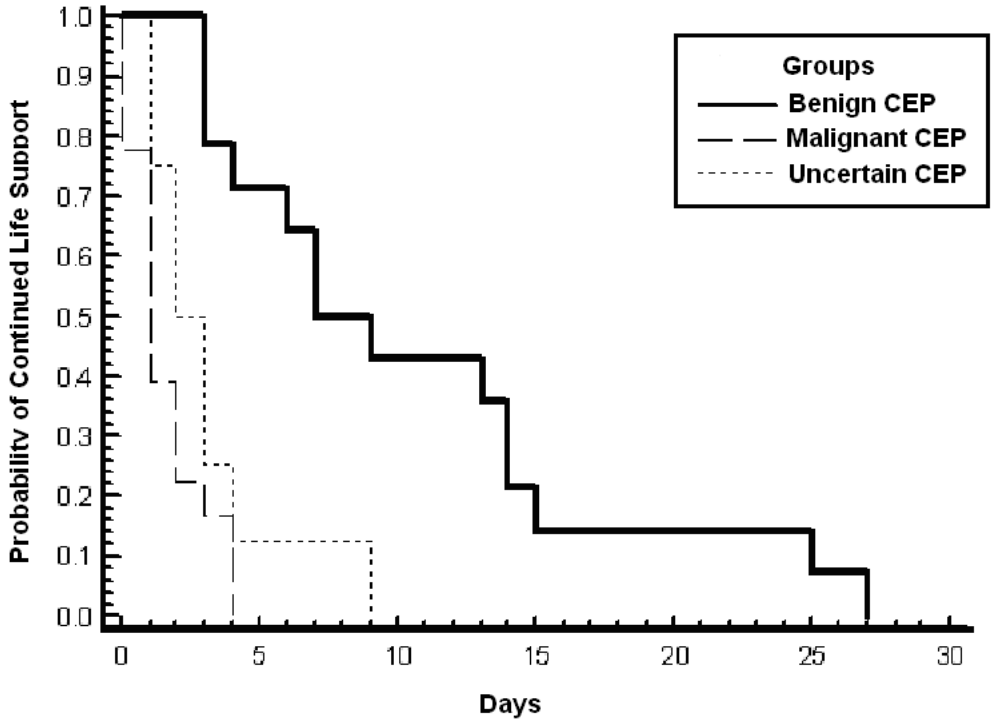
Neurology 2006;67;203-210

All studies focused on prognostication of poor outcome
Patients NOT treated with hypothermia

Does neuro prognostication have an impact on patient care?

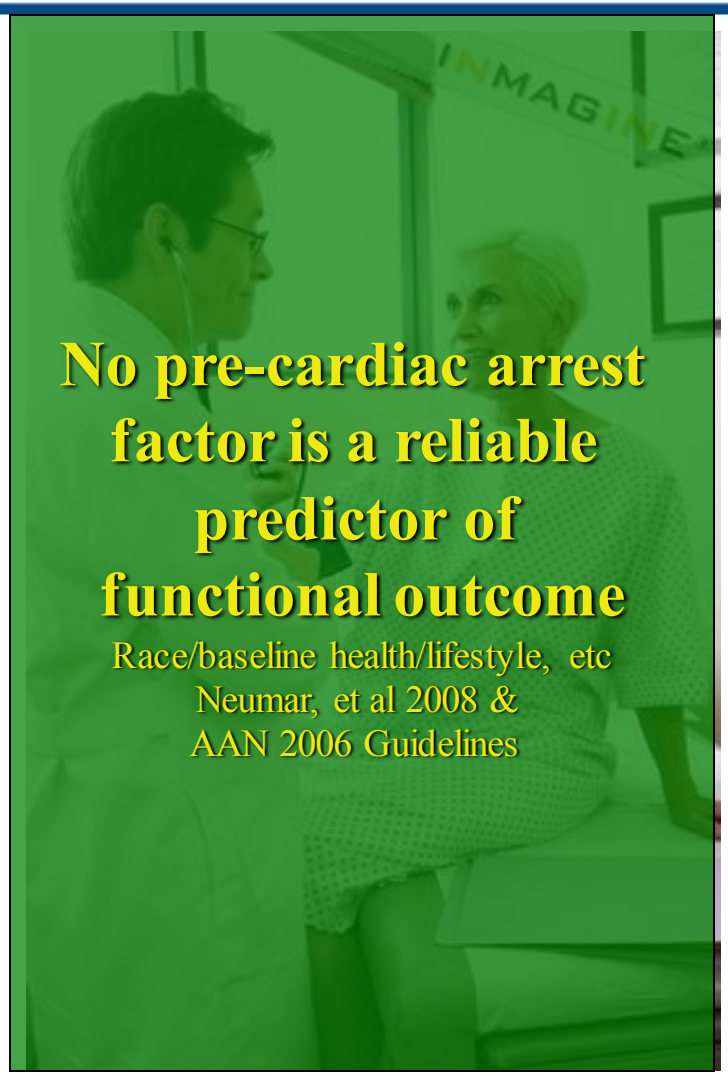


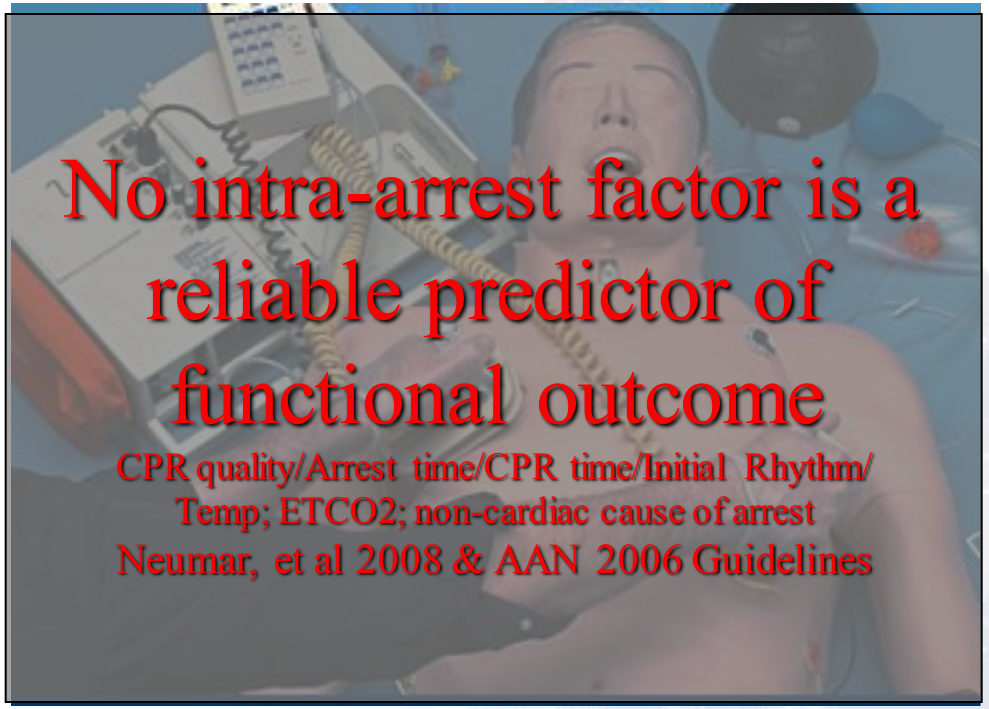
All GCS ≤ 4 at time of decision To withdraw life sustaining therapy



Geocadin, et al 2006

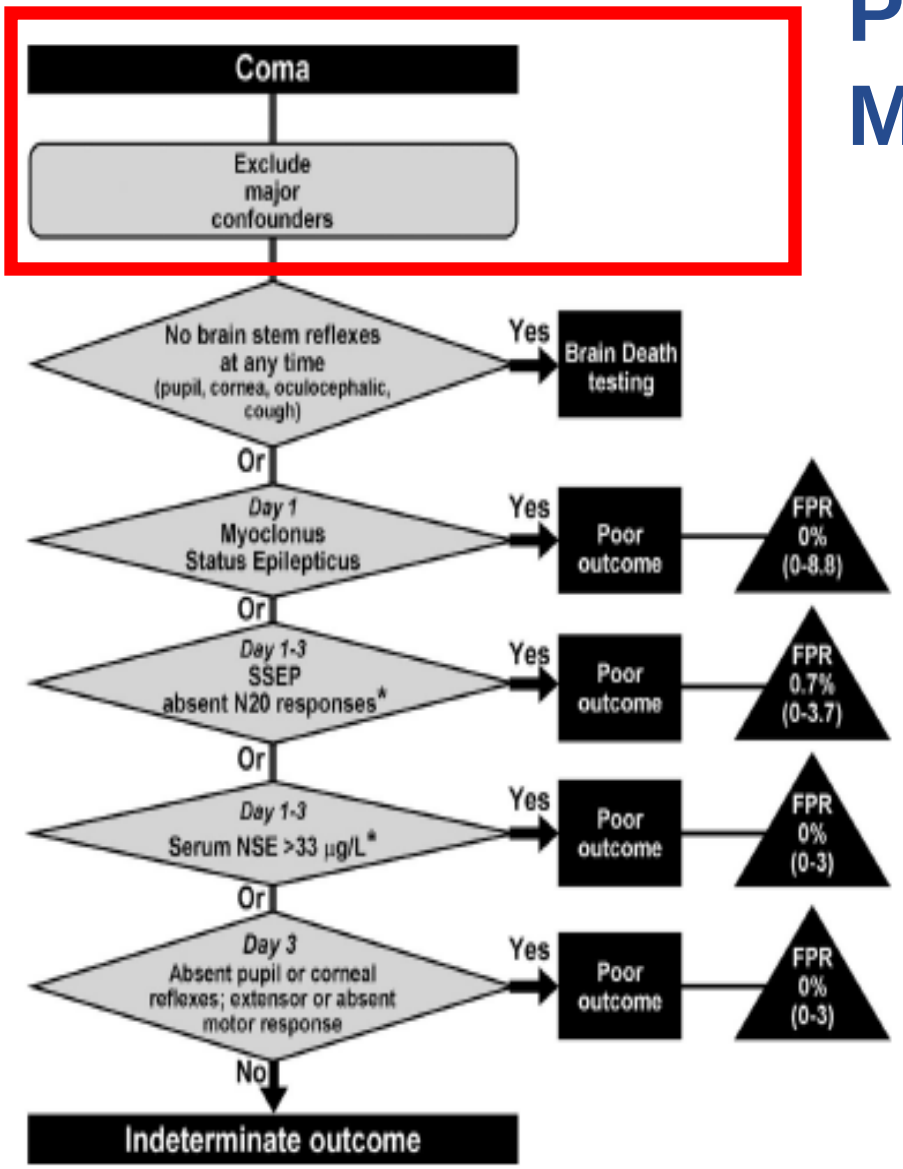
Can we predict the outcome?





Only in post arrest: Neuro exam
But not earlier than 3 days,
With TH ~5-7 days

From Wijdicks, et al 2006



Post-Arrest Predictors Major Confounders

- Hemodynamic instability, severe metabolic derangement and drugs may mask neurologic evaluation – error in prognosis
- Hypothermia patients – have delayed clearance of sedative and paralytics
- Neurologic recovery may be delayed by hypothermia

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2015 Recommendations—New and Updated

In comatose patients who are not treated with TTM, the absence of pupillary reflex to light at 72 hours or more after cardiac arrest is a reasonable exam finding with which to pre- dict poor neurologic outcome (FPR, 0%; 95% CI, 0%–8%; Class IIa, LOE B-NR).

In comatose patients who are treated with TTM, the absence of pupillary reflex to light at 72 hours or more after cardiac arrest is useful to predict poor neurologic outcome (FPR, 1%; 95% CI, 0%–3%; Class I, LOE B-NR).

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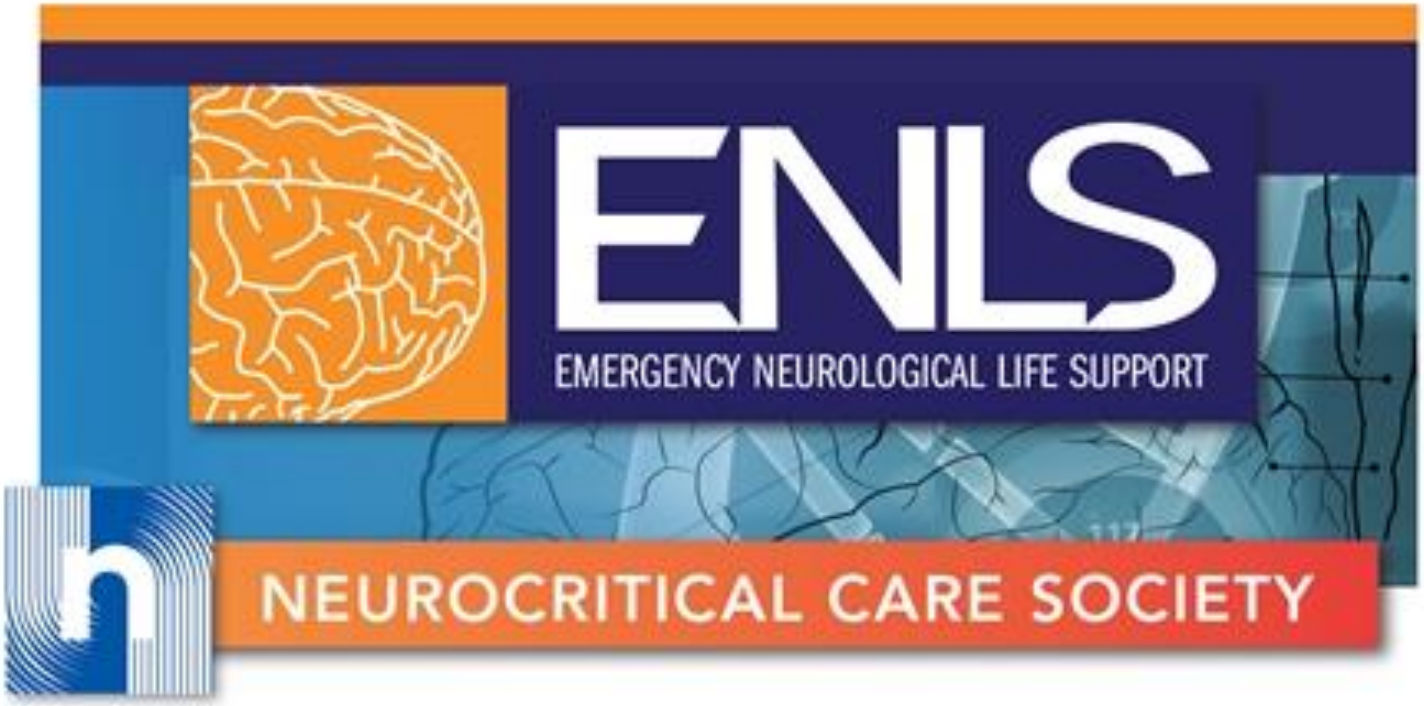
2015 Recommendations—Updated

In comatose post–cardiac arrest patients who are treated with TTM, it may be reasonable to consider persistent absence of EEG reactivity to external stimuli at 72 hours after cardiac arrest, and persistent burst suppression on EEG after rewarming, to predict a poor outcome (FPR, 0%; 95% CI, 0%–3%; Class IIb, LOE B-NR).

Intractable and persistent (more than 72 hours) status epilepticus in the absence of EEG reactivity to external stimuli may be reasonable to predict poor outcome (Class IIb, LOE B-NR).



EMERGENCY NEUROLOGICAL LIFE SUPPORT (ENLS):

WHAT TO DO IN THE CRITICAL FIRST HOURS OF A NEUROLOGICAL EMERGENCY

http://www.neurocriticalcare.org/

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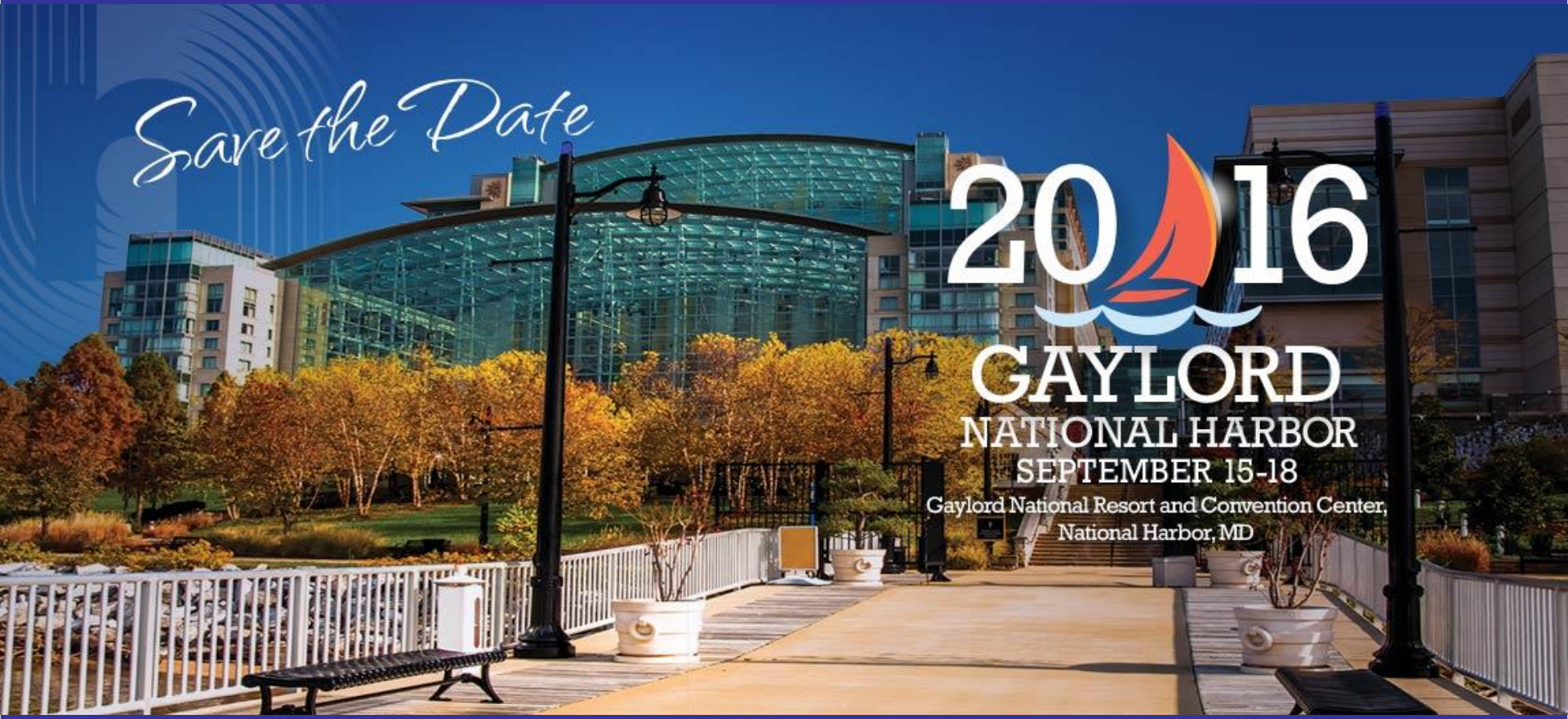

Contact: info@neurocriticalcare.org