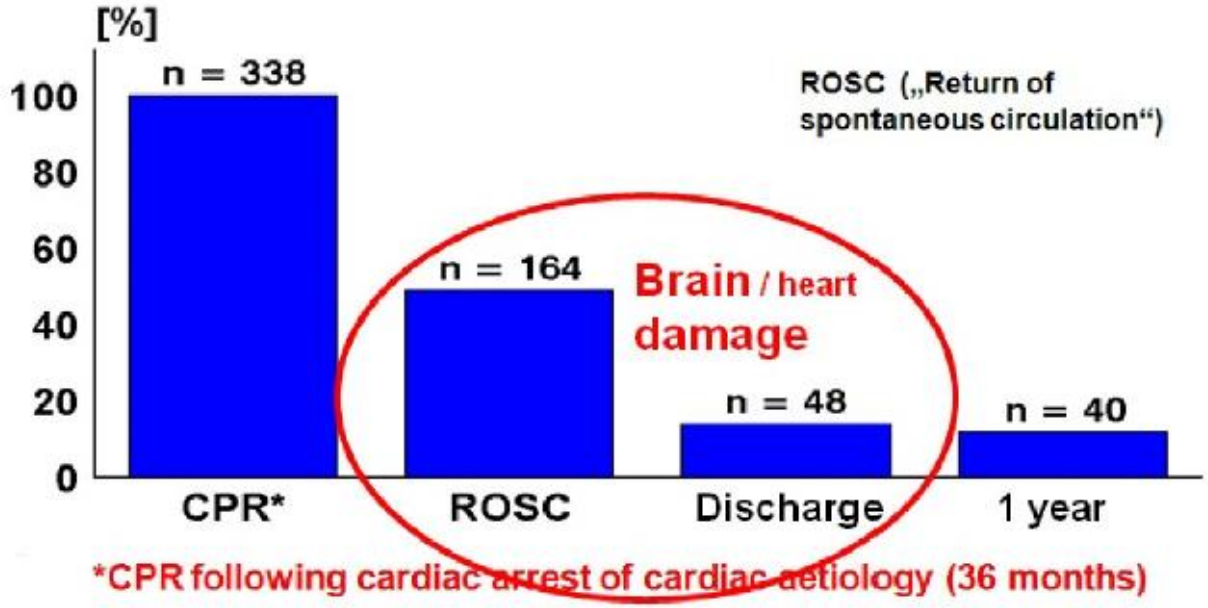
Post "Resuscitation "Syndrome

Hanoch Hod MD, FACC, FESC
ICCU, Leviev Heart Center
Sheba Medical Center
Tel-Aviv University, Israel

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

Harefuah. 2004 Nov;143(11):785-9, 839.

[Out-of-hospital resuscitation in Israel 2000].

[Article in Hebrew]

Canetti M, Feigenberg Z, Caspi A, Leor J, **Hod H**, Green M, Hasin Y, Battler A, Garty M, Mittelman M, Porath A, Grossman E, Behar S.

Heart Institute Meir Hospital, Tel-Aviv.

539 pts'

CPR

203(38%)

Treated by regular ambulance

336(62%)

Treated by MICU

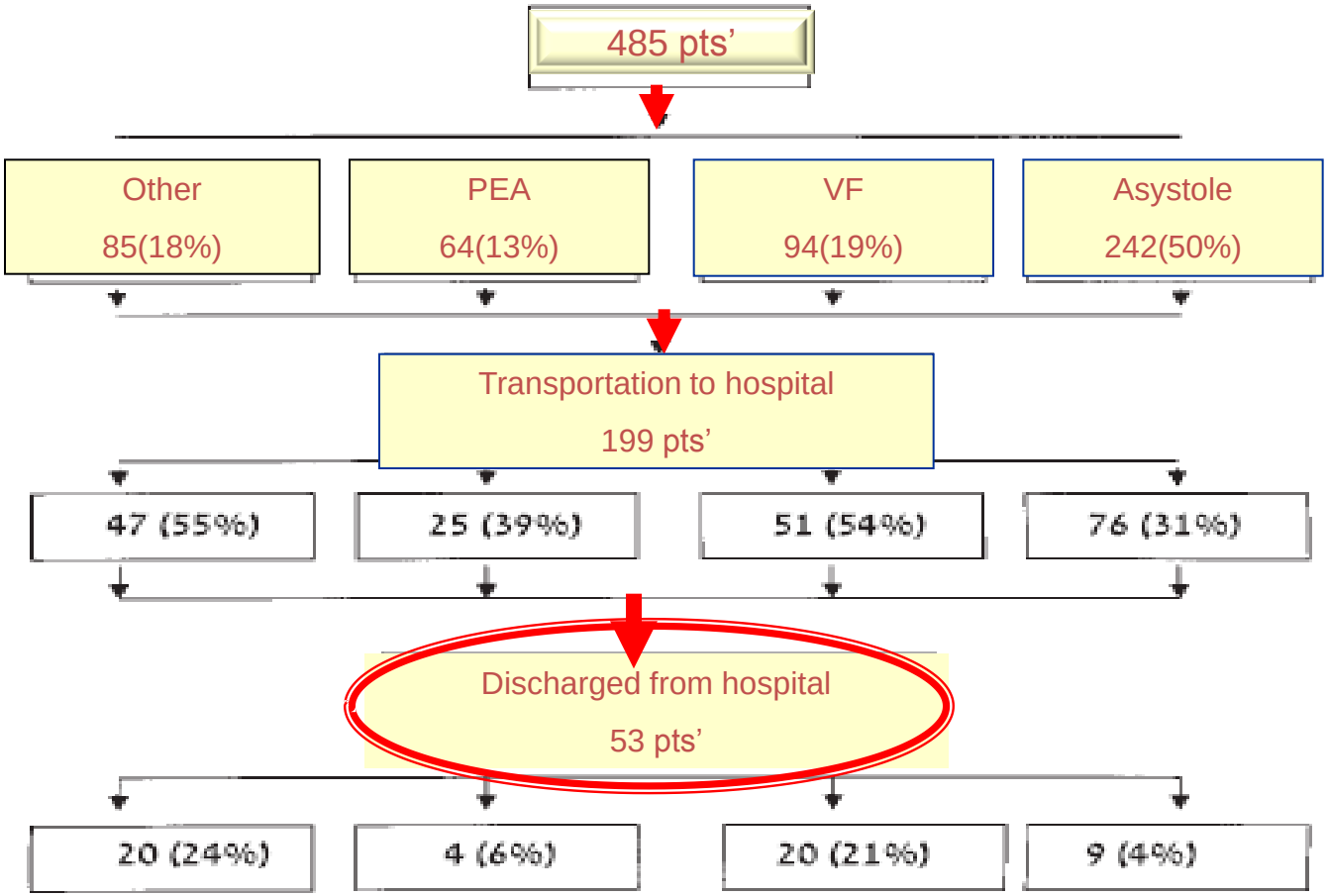
- **149 transferred to MICU**

54pts'

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%)

Resuscitation (1972), **1**, 1

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

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

American Heart Association.
Learn and Live.

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

R.W. Neumar, M.d., PhD, et.al., ILCOR Consensus Statement Post-Cardiac Arrest syndrome; *Circulation* Oct. 2008; 118:2452-2483

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

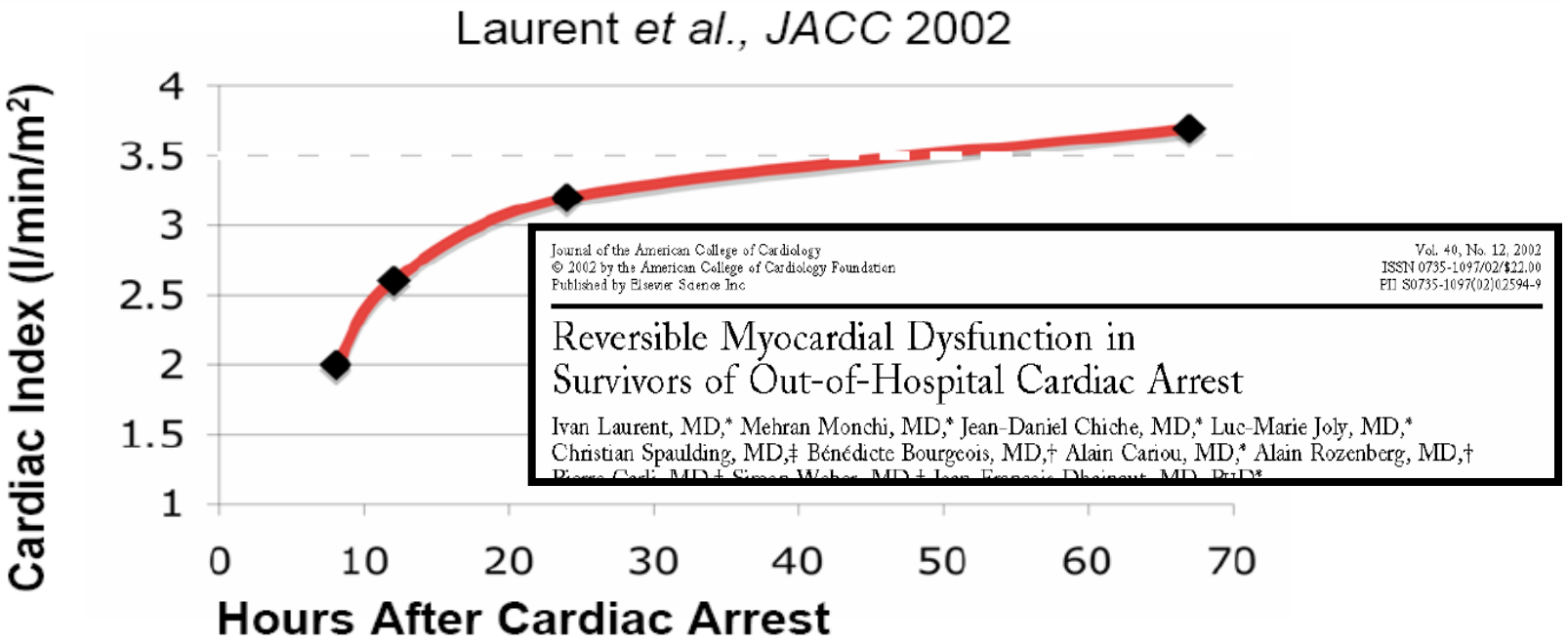
Pathophysiology	Clinical Manifestation	Potential Treatments
• Impaired cerebrovascular autoregulation • Cerebral edema (limited) • Postischemic neurodegeneration	• Coma • Seizures • Myoclonus • Cognitive dysfunction • Persistent vegetative state • Secondary Parkinsonism • Cortical stroke • Spinal stroke • Brain death	• Therapeutic hypothermia • Early hemodynamic optimization • Airway protection and mechanical ventilation • Seizure control

Post–Cardiac Arrest Myocardial Dysfunction

Pathophysiology	Clinical Manifestation	Potential Treatments

- Global hypokinesis (myocardial stunning)
- ACS

Myocardial Function is Reversibly Depressed for 24 to 72 hours after Cardiac Arrest



Post-cardiac arrest myocardial dysfunction is responsive to inotropes and intravenous fluid

Post–Cardiac Arrest Myocardial Dysfunction

Pathophysiology	Clinical Manifestation	Potential Treatments
• Global hypokinesis (myocardial stunning) • ACS	• Reduced cardiac output • Hypotension • Dysrhythmias • Cardiovascular collapse	• Early revascularization of AMI • Early hemodynamic optimization • Intravenous fluid • Inotropes • IABP • LVAD • ECMO

Systemic Ischemia/Reperfusion Response

Pathophysiology	Clinical	Potential
oxygen delivery and utilization • Impaired resistance to infection		• Antibiotics for documented infection

This is the ultimate systemic representation of shock in response to global ischemia and reperfusion

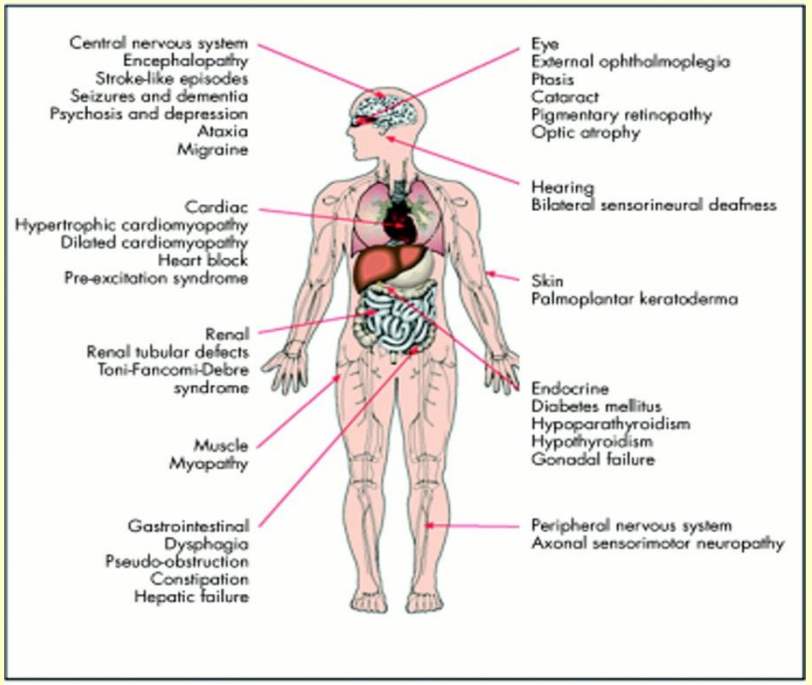
Persistent Precipitating Pathology

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

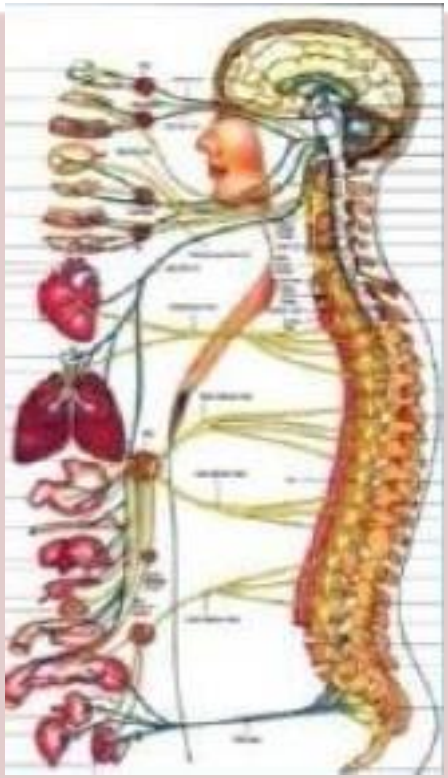
Persistent Precipitating Pathology

Pathophysiology	Clinical Manifestation	Potential Treatments

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



Persistent Precipitating Pathology

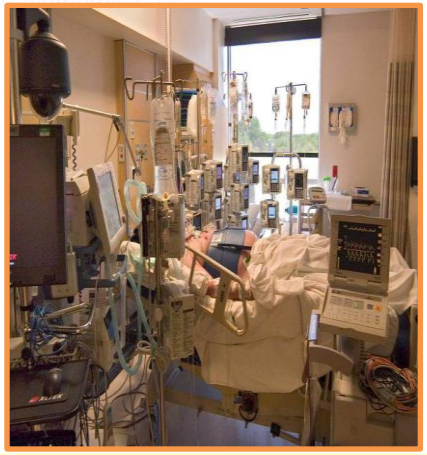
Pathophysiology	Clinical Manifestation	Potential Treatments
	• Specific to cause but complicated by concomitant PCAS	• Disease-specific interventions guided by patient condition and concomitant PCAS

General Therapeutic Strategies

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
   - $ScvO_2$
   - 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,
  - $ScvO_2$ >70%,
  - Hematocrit >30% or hemoglobin >8 g/dL,
  - lactate <2mmol/L,
  - urine output >0.5 $mL \cdot kg^{-1} \cdot h^{-1}$,
  - oxygen delivery index >600 $mL \cdot min^{-1} \cdot m^{-2}$

Significance of arterial hypotension after resuscitation from cardiac arrest*

CPP=(MAP-ICP)

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

Oxygenation

Ventilation with100% 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%

Oximetry-Guided Reoxygenation Improves Neurological Outcome After Experimental Cardiac Arrest

Irina S. Balan, PhD; Gary Fiskum, PhD; Julie Hazelton, MS; Cynthia Cotto-Cumba, MD; Robert E. Rosenthal, MD

Stroke 2006

Hyperoxia: *100% FiO_2 for first hour after ROSC*

Oximetry: *FiO_2 titrated to pulse-ox 94-96% first one hour ROSC*

Association Between Arterial Hyperoxia Following Resuscitation From Cardiac Arrest and In-Hospital Mortality

J. Hope Kilgannon, MD
Alan E. Jones, MD
Nathan I. Shapiro, MD, MPH
Mark G. Angelos, MD
Barry Milcarek, PhD
Krystal Hunter, MBA
Joseph E. Parrillo, MD
Stephen Trzeciak, MD, MPH
for the Emergency Medicine Shock Research Network (EMShockNet) 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 = $PaO_2 \geq 300$ mmHg

Hypoxia = $PaO_2 \leq 60$ 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 www.jama.com

SUDDEN CARDIAC ARREST IS THE most common lethal consequence of cardiovascular disease. Even if return of spontaneous circulation (ROSC) from cardiac arrest is achieved, approximately 60% of patients will not survive to hospital discharge.[1,2] The high mortality is attributed to the postcardiac arrest syndrome, which involves global ischemia-reperfusion injury, myocardial stunning, and anoxic brain injury.[3] The recent success of therapeutic hypother-

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 $Paco_2$ derangements could be potentially harmful for patients after resuscitation from cardiac arrest. (*Circulation*. 2013;127:2107-2113.)

Ventilation

During hypothermia PaCO2 should be corrected to patient temperature. When core temperature is 33© the patient' actual PaCO2 may be 6 to 7 mmHg lower

PaCO2=40-45mmHg

ETCO2=35-40mmHg

Ventilation

Surviving Sepsis Campaign Recommends:

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

- Assist control mode – volume ventilation
- ~~Reduce tidal volume to 6 mL/kg lean body weight~~
- Keep inspiratory plateau pressure (Pplat) ≤ 30 cm H_2O
  - Reduce TV as low as 4 mL/kg predicted body weight to limit Pplat
- Maintain SaO_2/SpO_2 88–95%
- Anticipated PEEP settings at various FIO_2 requirements

FiO_2	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	12	14	14	14	16	18	20–24

* Predicted Body Weight Calculation

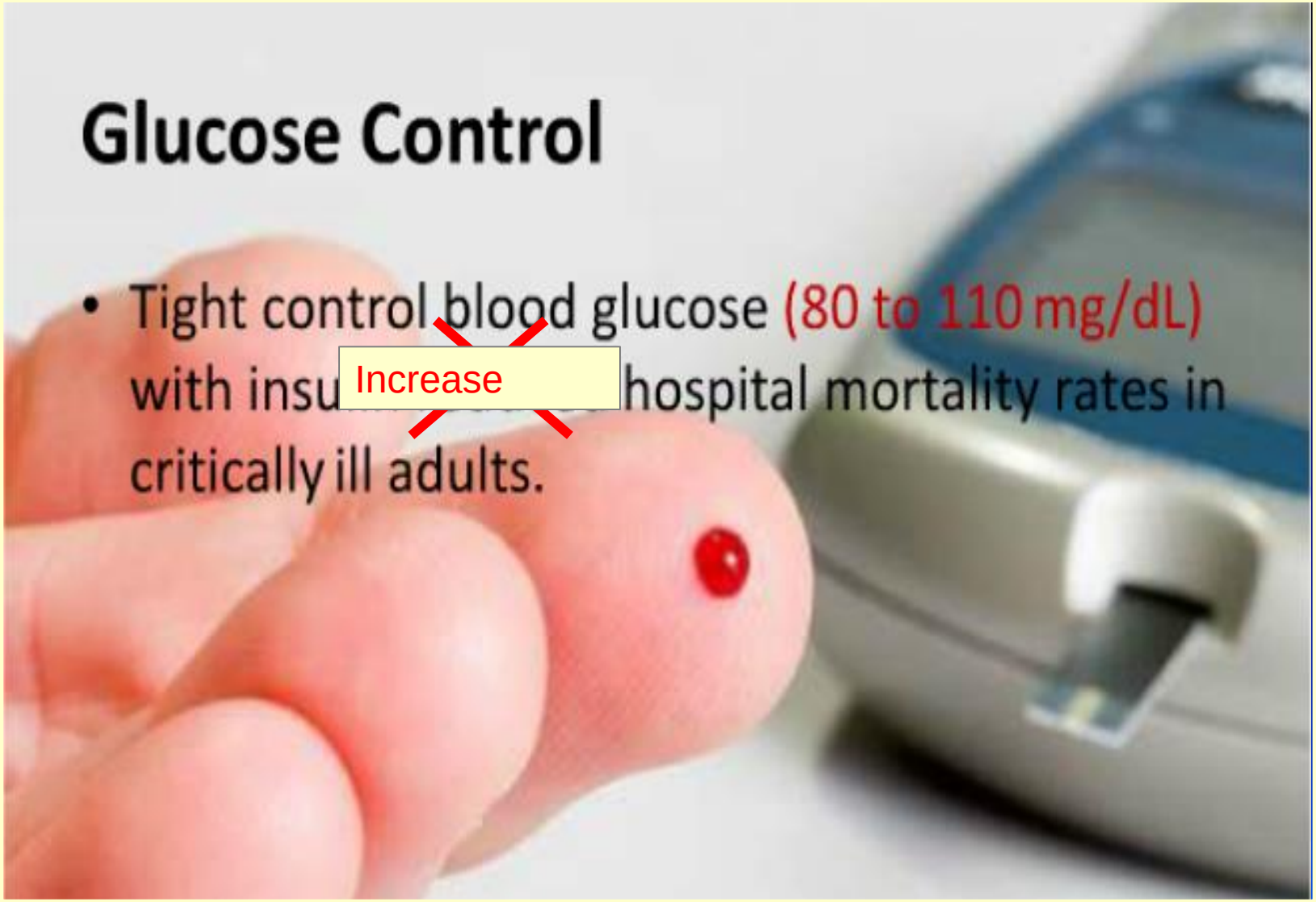
- Male – 50 + 2.3 (height (inches) – 60) or 50 + 0.91 (height (cm) – 152.4)
- Female – 45.5 + 2.3 (height (inches) – 60) or 45.5 + 0.91 (height (cm) – 152.4)

TV, tidal volume; *SaO_2*, arterial oxygen saturation; *SpO_2*, pulse oximetry oxyhemoglobin saturation; *PEEP*, positive end-expiratory pressure

ICOR Consensus Statement: Post cardiac arrest syndrome. Circulation. 2008;118:2452-2483.

Videtta W, Villarejo F, Cohen M et al. Effect of positive and expiratory pressure on intracranial pressure and cerebral perfusion. ActaNeurochirSuppl. 2003;81:93-97.

Surviving Sepsis Campaign


Glucose Control
• Tight control blood glucose (80 to 110 mg/dL)
with insu
Increase
hospital mortality rates in
critically ill adults.

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 MARCH 26, 2009 VOL. 360 NO. 13

Intensive versus Conventional Glucose Control in Critically Ill Patients

The NICE-SUGAR Study Investigators*

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

BACKGROUND

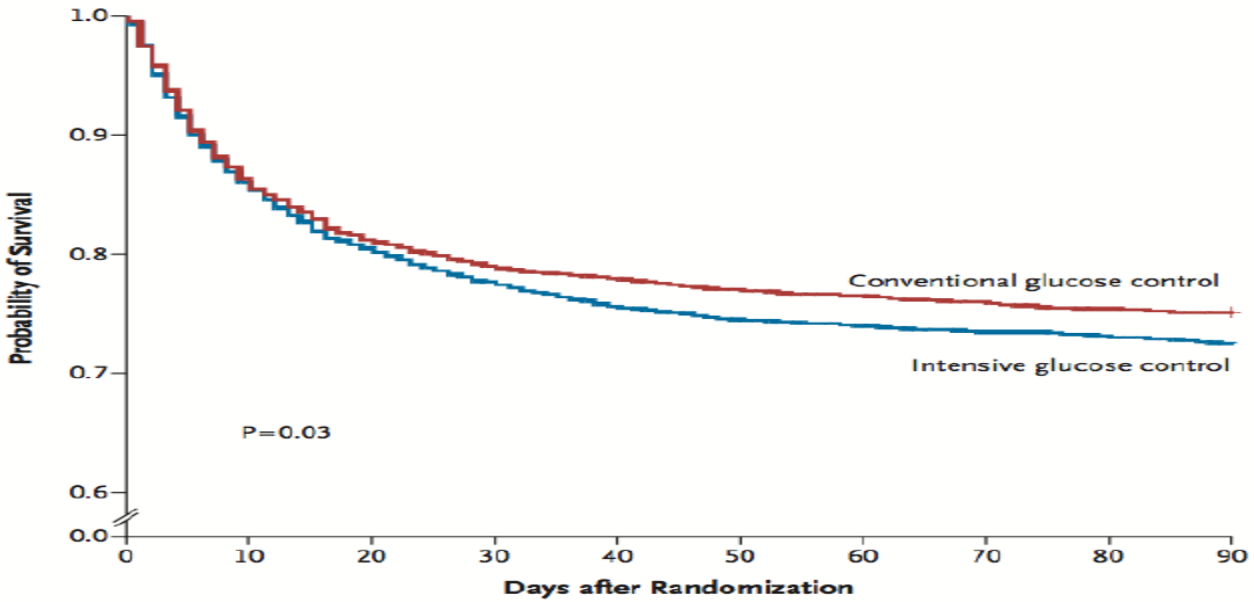
The optimal target range for blood glucose in critically ill patients remains unclear.

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-

The NICE-SUGAR study is a collaboration of the Australian and New Zealand Intensive Care Society Clinical Trials Group, the George Institute for International Health (University of Sydney), the Canadian Critical Care Trials Group, and the Vancouver Coastal Health Research

Strict Glycemic Control Worsens Outcomes



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…

Zeiner A. et al, . Hyperthermia after cardiac arrest is associated with unfavorable neurologic outcome. Arch intern. Med. 200; 161: 2007-2012

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.

Langhelle A, et al. In-hospital factors associated with improved outcome after out-of-hospital cardiac arrest: a comparison between four regions in Norway. *Resuscitation*. 2003; 56: 247–263

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…

Krumholz A, Stern BJ, Weiss HD. Outcome from coma after cardiopulmonary resuscitation: relation to seizures and myoclonus. *Neurology*. 1988; 38: 401–405.

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.

FOOTNOTES

Other Issues:

1. Pyrexia
2. Hyperglycemia
3. Seizures
4. **Adrenal dysfunction** → Steroids? Controversial
5. **Renal Failure**
6. **Infection**

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 appropiate 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

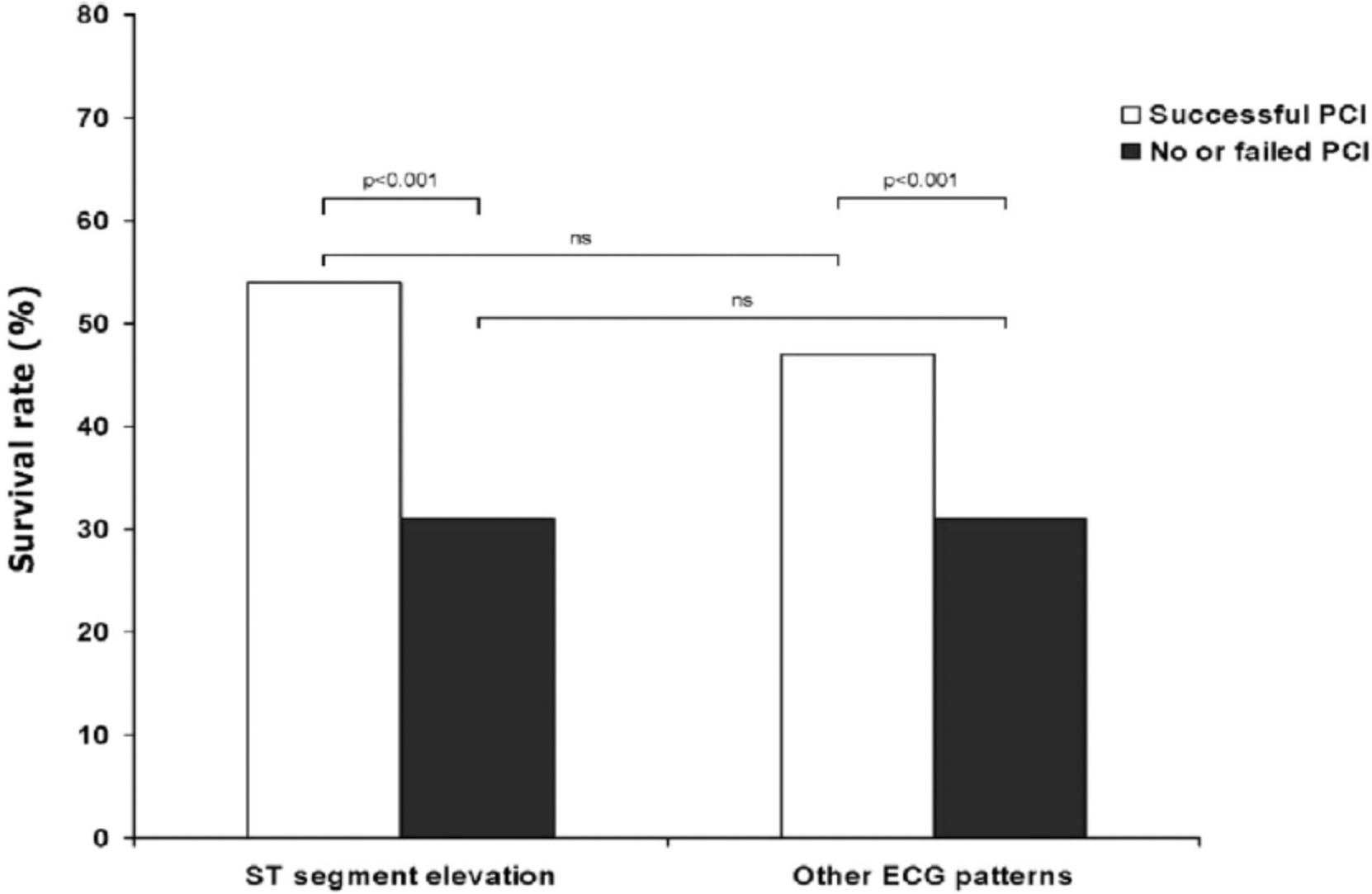
Florence Dumas, MD; Alain Cariou, MD; Stéphane Manzo-Silberman, MD; David Grimaldi, MD; Benoît Vivien, MD; Julien Rosencher, MD; Jean-Philippe Empana, MD; Pierre Carli, MD; Jean-Paul Mira, MD; Xavier Jouven, MD; Christian Spaulding, MD

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



ACCF/AHA Guideline

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

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*)

VOLUME 38, NUMBER 6 — NOVEMBER-DECEMBER, 1959

THE USE OF HYPOTHERMIA

Patients who cannot follow basic commands generally need treatment with mild therapeutic hypothermia

DONALD W. BENSON, M.D.
G. RAINEY WILLIAMS, JR., M.D.
FRANK C. SPENCER, M.D.
ADOLPH J. YATES, M.D.
Baltimore, Maryland*

Benson DW et al. *Anesthesia and Analgesia*. 1959 38(6):423–428

ACCF/AHA Guideline

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/VT]), 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/VT 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/VT and 68/437 (16%) in PEA/asystole ($P<0.001$). After adjustment, in VF/VT 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/VT. 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

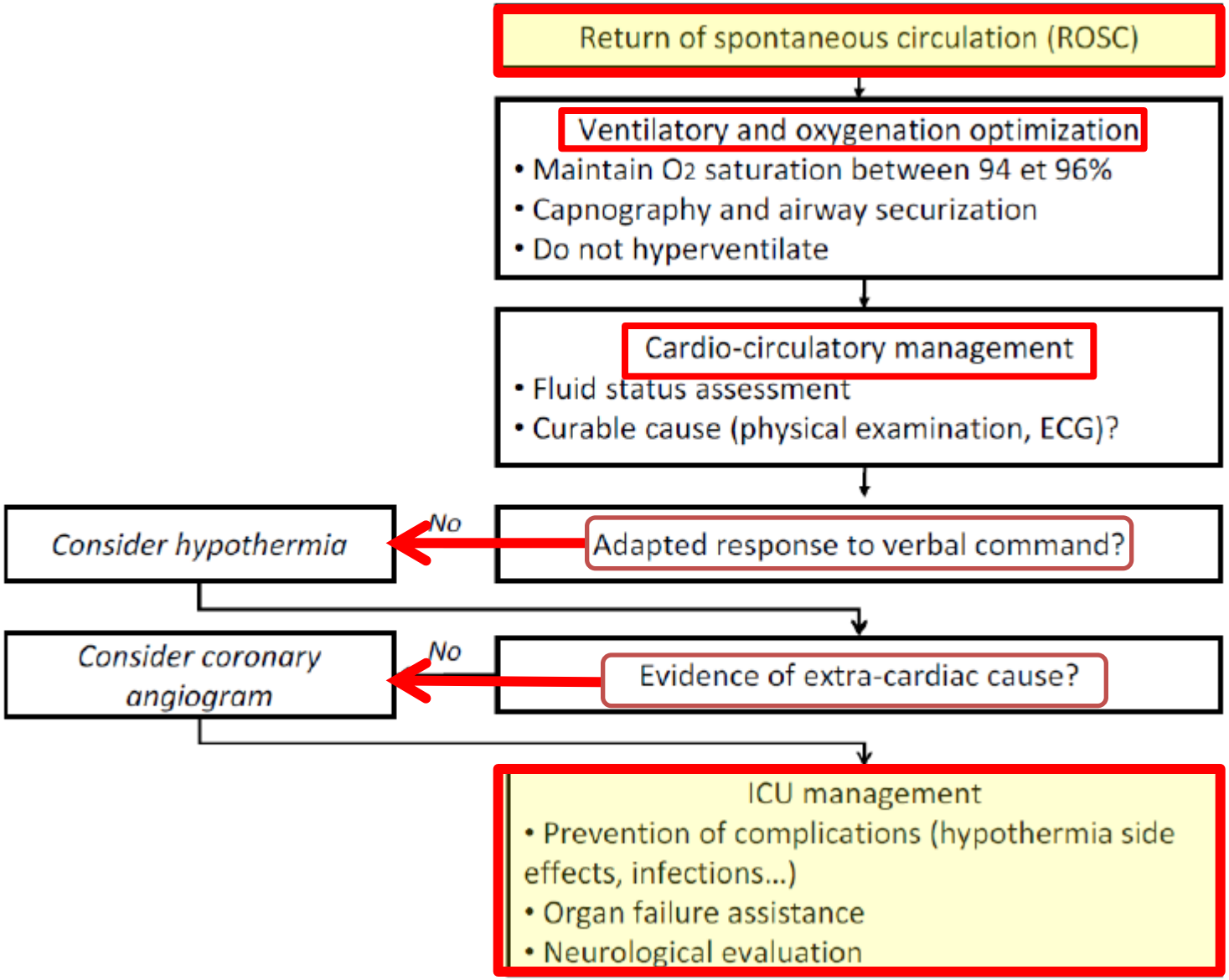
Avishag Laish-Farkash, MD, PhD[a,c], Shlomi Matetzky, MD[a,c], Dan Oieru, MD[a], Amir Sandach, PhD[d], Niza Levi, SRN[a], Jacob Or, MD[b,c], Johonatan Rieck, MD[b], Alon Barsheshet, MD[a,c], and Hanoch Hod, MD[a,c,*]

(Am J Cardiol 2011;108:173–178)

Overall outcomes and complications

Variable	VF		p Value	Asystole (n = 18)	p Value vs VF	PEA (n = 6)	p Value vs VF
	Yes (n = 86)	No (n = 24)					
Death	26 (30%)	15 (63%)	0.004	11 (61%)	0.01	4 (67%)	0.07
CPC 1 or 2	57 (66%)	2 (8%)	<0.0001	2 (11%)	<0.0001	0	0.001
Pneumonia	53 (62%)	13 (54%)	0.51	11 (61%)	0.97	2 (33%)	0.17
Sepsis	13 (15%)	8 (33%)	0.05	6 (33%)	0.07	2 (33%)	0.25
Any bleeding	11 (13%)	6 (25%)	0.14	5 (28%)	0.11	1 (17%)	0.79
Seizures (clinical/electroencephalography)	21 (24%)	17 (71%)	<0.0001	12 (67%)	0.0005	5 (83%)	0.002
Cardiogenic shock	13 (15%)	0	0.04	0	0.08	0	0.31
Arrhythmia	16 (19%)	2 (8%)	0.23	1 (5.5%)	0.18	1 (17%)	0.91
Pulmonary congestion	14 (16%)	8 (33%)	0.07	5 (28%)	0.25	3 (50%)	0.04
Need for catecholamines during MTH	15 (17%)	5 (21%)	0.70	3 (17%)	0.94	2 (33%)	0.33

Therefore, although the use of MTH in VF is recommended, its use in non-VF rhythms is debatable and needs further investigation.



Mongardon *et al. Annals of Intensive Care* 2011, **1**:45

Long term management

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


Thank You