Post-Arrest Hemodynamic Management

What is the best strategy?

David F. Gaieski, MD
Assistant Professor, Department of Emergency Medicine
University of Pennsylvania School of Medicine
Director, Clinical Center for Resuscitation
Hospital of the University of Pennsylvania
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Disclosures

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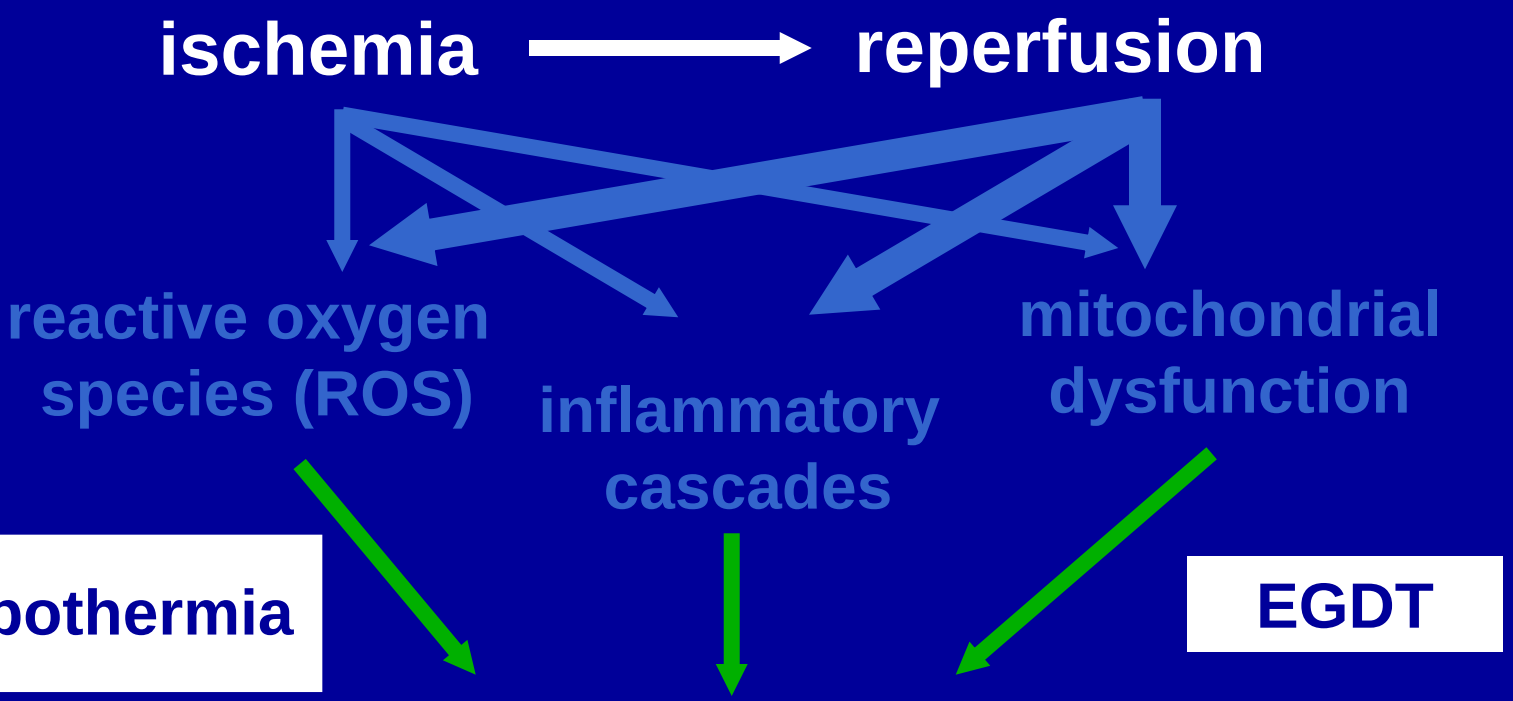
Outline

- Post-Resuscitation Syndrome
- Rapidly changing hemodynamics
- Hemodynamic Optimization Strategies
- Time for a care bundle?
- The Penn Experience
- Conclusions

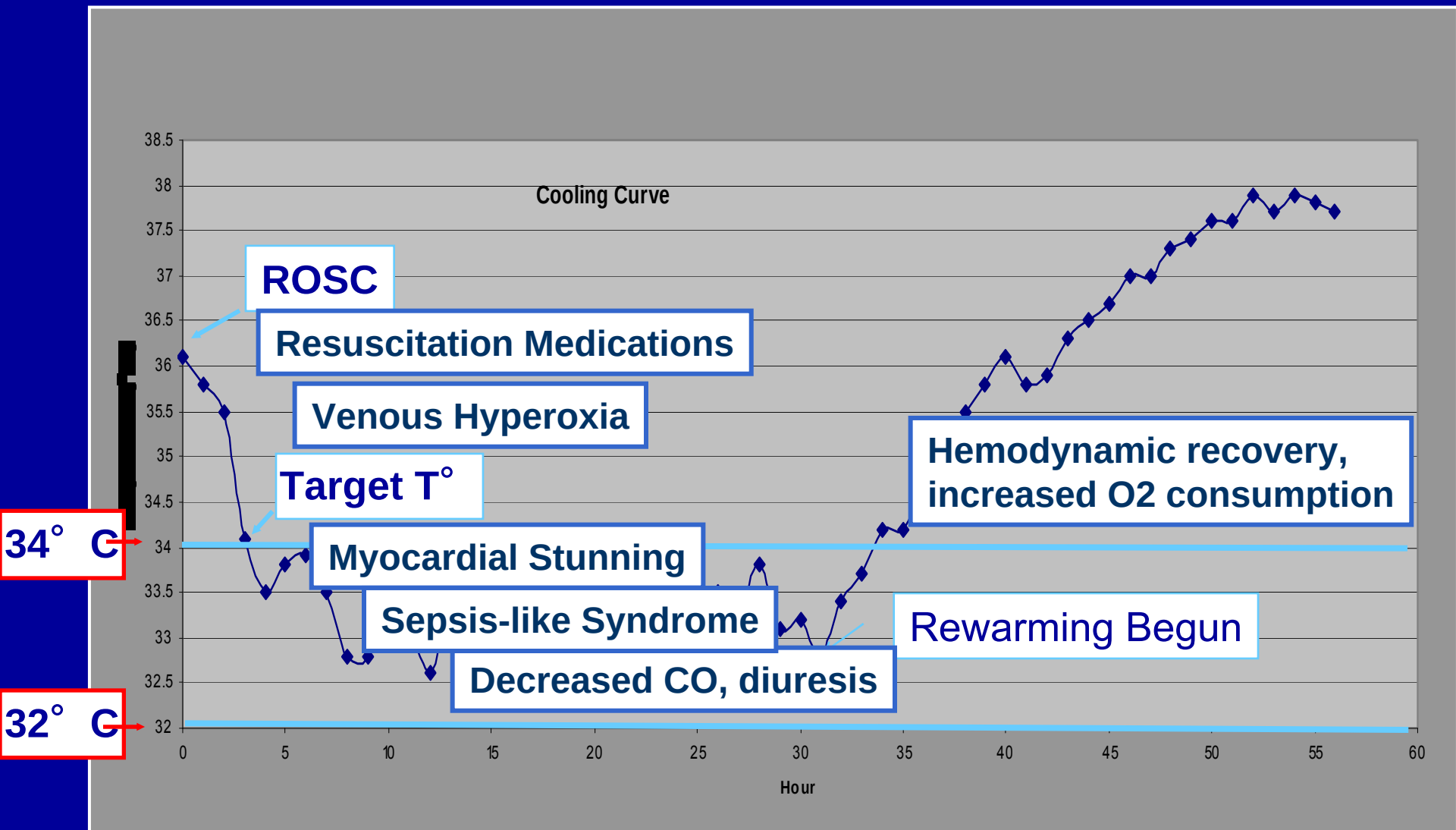
Negovsky—Post-Resuscitation Syndrome

- The second step in resuscitation: the treatment of the 'post-resuscitation disease'
  - Negovsky VA. Resuscitation. 1972; 1: 1-7
- Post-resuscitation disease—a new nosological entity: Its reality and significance
  - Negovsky VA, Gurvitch AM Resuscitation 1995; 30: 23-27

Post-Resuscitation Disease: Mechanisms



Changes with stages post-arrest



The Effect of the Total Cumulative Epinephrine Dose Administered During Human CPR on Hemodynamic, Oxygen Transport, and Utilization Variables in the Postresuscitation Period

Emanuel P. Rivers, Jacobo Wortsman, Mohamed Y. Rady, Heidi C. Blake, Francis T. McGeorge and Nancy M. Buderer

Chest 1994;106;1499-1507

- During era of high dose epinephrine
- Group 1: Cumulative dose < 15 mg
- Group 2: Cumulative dose > 15 mg
- "Inadvertent catecholamine toxicity represents a further complicating factor in the production of postresuscitation disease"

Table 4—*Initial, Mean, and Maximal Hemodynamic, Oxygen Transport, and Utilization Variables During the First 6 h of the Postresuscitation Period Group II is shaded*

	Initial			Mean			Maximal		
Variables	Group 1	Group 2	p Value	Group 1	Group 2	p Value	Group 1	Group 2	p Value
MAP	93 ± 34	118 ± 36	0.02	103 ± 22	96 ± 23	0.47	131 ± 37	130 ± 36	0.83
CI	2.6 ± 1.0	1.7 ± 1.0	0.008	2.9 ± 0.9	2.1 ± 1.1	0.01	3.6 ± 1.1	2.7 ± 1.3	0.01
SVRI	2,578 ± 1,333	6,434 ± 4,488	0.001	3,314 ± 2,709	6,160 ± 5,704	0.18	4,445 ± 3,524	7,494 ± 5,603	0.02
SvO_2	87 ± 8	82 ± 9	0.10	70 ± 13	72 ± 15	0.63	86 ± 8	82 ± 9	0.15
OER	0.12 ± 0.08	0.17 ± 0.09	0.10	0.29 ± 0.13	0.27 ± 0.15	0.60	0.36 ± 0.10	0.40 ± 0.16	0.38
Vo_2	46 ± 30	34 ± 23	0.13	108 ± 27	77 ± 48	0.006	148 ± 37	107 ± 63	0.007
Do_2	406 ± 190	235 ± 152	0.002	408 ± 217	256 ± 194	0.01	541 ± 158	380 ± 211	0.006

Venous Hyperoxia

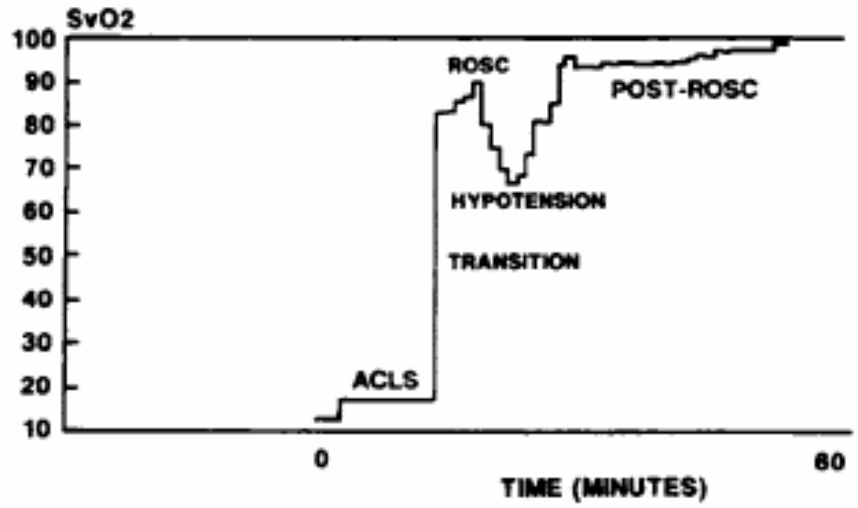


FIGURE 1. The phases of SvO_2 during and after resuscitation from cardiac arrest. This patient received 15 min of ACLS and developed ROSC after the transition phase. After a brief hypotensive episode, the patient responded to vasopressor and fluid therapy to hemodynamic stability. Venous hyperoxia was exhibited during the post-ROSC phase.

Table 1—*Initial Study Variables Obtained Within the First 5 Min After Return of Spontaneous Circulation*

	Survivors (n = 10)		Nonsurvivors (n = 13)		
Variables	Mean	SE	Mean	SE	p-value
PCWP, mm Hg	26.5	5.0	28.8	5.0	0.25
MAP, mm Hg	104	9.5	98	7.0	0.63
CI, L/min·m²	2.6	0.4	1.3	0.4	0.03
SVRI, dynes/s/cm⁵·m²	3,712	577	9,457	1,892	0.01
SvO_2, %	66	4.0	69	3	0.56
OER (CPR), %*	71	12	76	13	0.20
OER (ROSC), %	35	3.6	35	2.7	0.89
Vo_2, ml/min·m²	108	9.1	69	17	0.06
Do_2, ml/min·m²	344	49	218	58	0.12
Epinephrine, mg†	11	4.0	26.4	5.8	0.05
DCA, min	28	5.3	36	5.6	0.28

*CPR, during cardiopulmonary resuscitation or ACLS
†Epinephrine, dose required for ROSC (mg)

Reversible Myocardial Dysfunction

- Median time to hemodynamic instability=6.8 hrs after OHCA
- CI and CVP were low
- It is characterized by a low CI that is reversible in most cases within 24 h, suggesting post-resuscitation myocardial dysfunction

Reversible myocardial dysfunction in survivors of out-of-hospital cardiac arrest. Laurent I et al. JACC, 2002; 40:2110-2116

Dobutamine reversal of myocardial dysfunction

- Prospective, controlled animal trial
- Myocardial dysfunction post-resuscitation
- Reversed by Dobutamine infusion
- Balance O2 consumption versus O2 need
- Paying back the O2 debt
- Full recovery of this postresuscitation myocardial stunning is seen by 48 h

Myocardial dysfunction after resuscitation from cardiac arrest: an example of global myocardial stunning, Kern et al, JACC 1996; 28: 232-240

Successful Cardiopulmonary Resuscitation After Cardiac Arrest as a "Sepsis-Like" Syndrome

Christophe Adrie, MD; Minou Adib-Conquy, PhD; Ivan Laurent, MD; Mehran Monchi, MD; Christophe Vinsonneau, MD; Catherine Fitting, BS; François Fraisse, MD; A. Tuan Dinh-Xuan, MD; Pierre Carli, MD; Christian Spaulding, MD; Jean-François Dhainaut, MD; Jean-Marc Cavaillon, PhD

Background—We investigated the immunoinflammatory profile of patients successfully resuscitated after cardiac arrest, representing a model of whole-body ischemia/reperfusion syndrome.

Methods and Results—Plasma cytokine, endotoxin, and ex vivo cytokine production in whole-blood assays was assessed in 61, 35, and 11 patients, respectively. On admission, high levels of plasma interleukin (IL)-6, IL-8, IL-10, and soluble tumor necrosis factor (TNF) receptor type II could discriminate between survivors and nonsurvivors. Among nonsurvivors, the initial need for a vasopressor agent was associated with higher levels of IL-1 receptor antagonist, IL-10, and IL-6 on day 1. Plasma endotoxin was detected in 46% of the analyzed patients within the 2 first days. Endotoxin-induced TNF and IL-6 productions were dramatically impaired in these patients compared with healthy control subjects, whereas an unaltered production was observed with heat-killed *Staphylococcus aureus*. In contrast, IL-1 receptor antagonist productions were enhanced in these patients compared with healthy control subjects. The productions of T-cell–derived IL-10 and interferon-γ were also impaired in these patients. Finally, using in vitro plasma exchange between healthy control subjects and patients, we demonstrated that the endotoxin-dependent hyporeactivity was an intrinsic property of patients' leukocytes and that an immunosuppressive activity was also present in their plasma.

Conclusions—Altogether, the high levels of circulating cytokines, the presence of endotoxin in plasma, and the dysregulated production of cytokines found in these patients recall the immunological profile found in patients with sepsis. **(*Circulation*. 2002;106:562-568.)**

Key Words: cardiopulmonary resuscitation ■ heart arrest ■ reperfusion ■ inflammation ■ interleukins

TABLE 2. Plasma Cytokine and sTNFRII Concentrations on Hospital Admission in OHCA Patients, in Patients With Sepsis (Positive Control Group), and in Healthy Volunteers (Negative Control Group)

Cytokines and Receptors, pg/mL	OHCA Patients (n=61)	Patients With Sepsis (n=5)	Healthy Volunteers (n=7)
TNF-α	16 (0–30)	16 (0–46)	0 (0–0)*
sTNFRII	5714 (3629–8350)	4000 (7021–12 656)	1458 (1589–3617)‡
IL-1ra	13 972 (1947–40 319)	72 897 (657–94 884)	46 (0–111)‡
IL-6	177 (53–355)	406 (390–4901)*	0 (0–0)‡
IL-8	67 (22–183)	399 (76–529)	0 (0–0)‡
IL-10	122 (41–250)	199 (160–1003)	0 (0–0)‡
RANTES	7035 (3892–20 369)	2021 (583–2184)†	11 957 (9527–12 817)

Data are median (25% to 75% quartile). OHCA patients had a plasma cytokine pattern similar to that observed in patients with sepsis.

*$P<0.05$, †$P<0.01$, and ‡$P<0.001$ for patients with sepsis and healthy volunteers vs OHCA patients.

MAP

- MAP = [(2 x diastolic)+systolic] / 3
- Nagao: SBP ≥ 90 mmHg (MAP > 65)
- Bernard: MAP 90 and 100 mm Hg
- Sunde: MAP 65-70 mmHg
- What is the right answer?
- What is this based upon?

Safar: Dog model of VF arrest

- Normothermic VF of 11 minutes
- Defibrillation and controlled reperfusion
- Controlled ventilation for 20 hrs; intensive care to 96 hrs
- Control group (n=8):
  - normothermic (37.5°C)
  - Normotensive
  - Hypocapnic
- Experimental group (n=8):
  - mild hypothermia (34°C) from about 10 minutes to 12 hours
  - cerebral blood flow promotion with induced moderate HTN
  - mild hemodilution
  - Normocapnia

Improved Cerebral Resuscitation From Cardiac Arrest in Dogs With Mild Hypothermia Plus Blood Flow Promotion
Safar P et al, *Stroke.* 1996; 27:105-113

Dog model of VF arrest: Results

- All 16 dogs in the protocol survived
- Control group, n=8:
  - All OPC 3 (severe disability) or 4 (coma)
- Experimental group, n=8:
  - 6/8 (75%)dogs OPC 1 (normal)
  - 1/8 (12.5%) OPC 2 (moderate disability)
  - 1/8 (12.5%) OPC 3 (*P*<.001)

Improved Cerebral Resuscitation From Cardiac Arrest in Dogs With Mild Hypothermia Plus Blood Flow Promotion.
Safar P et al, *Stroke.* 1996; 27:105-113

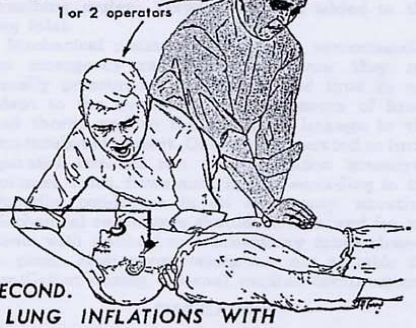
Rapidly Changing Hemodynamics

ECHO		EF
1 Hr	The LV is underfilled with hyperdynamic function	80%
11 Hr	The LV is normal in size with severe global systolic dysfunction	15%
29 Hr	The LV is normal in size with normal systolic function	65%

How should these dynamic changes be managed clinically?

HEART–LUNG RESUSCITATION

I FIRST AID: OXYGENATE THE BRAIN IMMEDIATELY



IF UNCONSCIOUS

Airway – *TILT HEAD BACK*

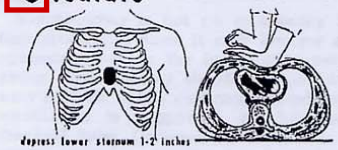
IF NOT BREATHING

Breathe – *INFLATE LUNGS 3-5 TIMES, MAINTAIN HEAD TILT*

MOUTH-TO-MOUTH, MOUTH-TO-NOSE, mouth-to-adjunct, bag-mask

- FEEL PULSE
- IF PRESENT – CONTINUE LUNG INFLATIONS
- IF ABSENT – [illegible]

Circulate – *COMPRESS HEART ONCE A SECOND. ALTERNATE 2-3 LUNG INFLATIONS WITH 15 STERNAL COMPRESSIONS UNTIL SPONTANEOUS PULSE RETURNS.*



for physicians only

II START SPONTANEOUS CIRCULATION

Drugs – *EPINEPHRINE:* 1.0 mg (1.0 CC OF 1:1000) I.V. OR 0.5 mg INTRACARDIAC. REPEAT LARGER DOSE IF NECESSARY

SODIUM BICARBONATE: APPROXIMATELY 3.75 G/50 CC (1/2 DOSE IN CHILDREN) I.V.

III SUPPORT RECOVERY (physician-specialist)

Gauge *EVALUATE AND TREAT CAUSE OF ARREST*

Hypothermia *START WITHIN 30 MINUTES IF NO SIGN OF CNS RECOVERY*

Intensive Care *SUPPORT VENTILATION:* TRACHEOTOMY, PROLONGED CONTROLLED VENTILATION, GASTRIC TUBE AS NECESSARY

SUPPORT CIRCULATION
CONTROL CONVULSIONS
MONITOR

card or for a poster which may be obtained from the Pennsylvania Heart Association or the Pennsylvania Department of Health, Harrisburg.

Peter Safar:

Journal of the Iowa Medical Society, November, 1964

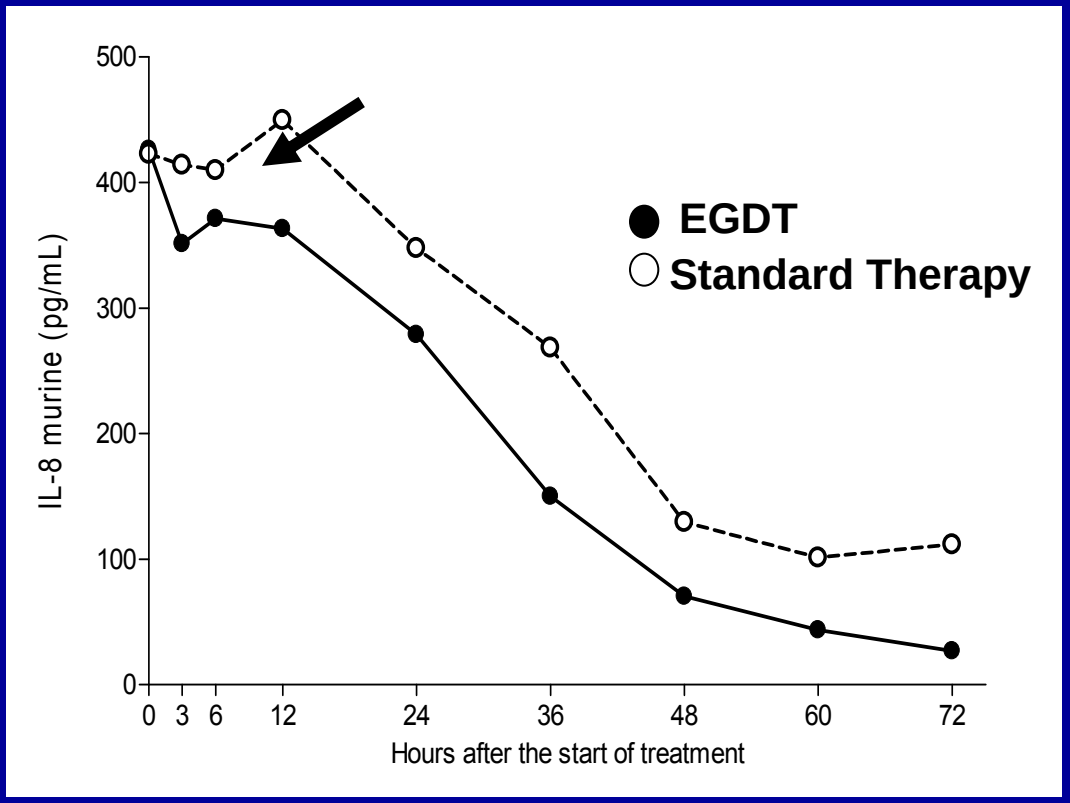
ABCs

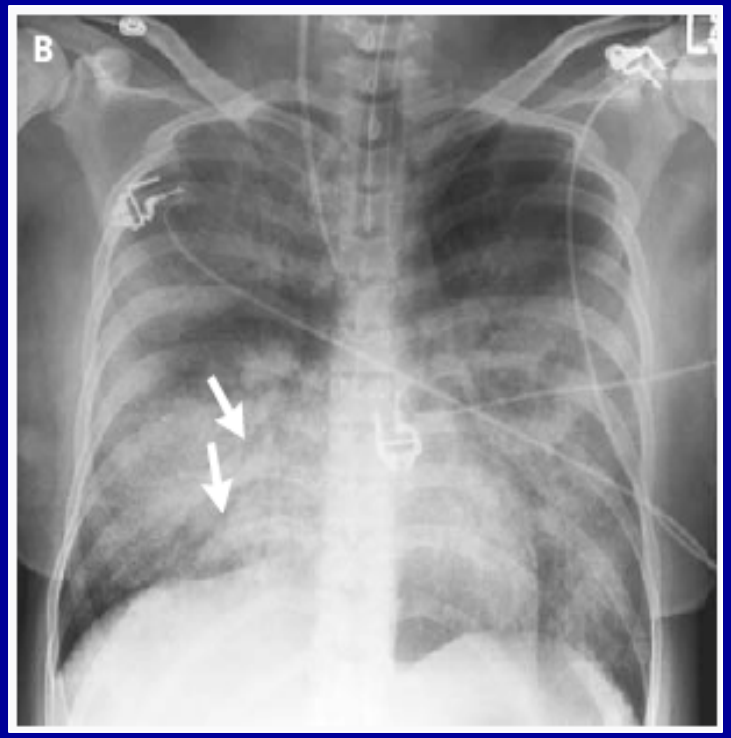
Historically, evidence for and against Hemodynamic Optimization Strategies

- Shoemaker:
  - Pre-operative hemodynamic optimization of high-risk cardiac surgery patients
  - RESULTS:
    - Normal MAP, CVP, UOP: Mortality 33%
    - Supranormal DO_2, VO_2, CI: Mortality 4%
- Gattinoni:
  - ICU enrollment
  - Normal CI vs. Supranormal CI vs. SvO_2 Optimization
  - RESULTS:
    - No difference in mortality at ICU discharge and at 6 months
- Rivers:
  - ED enrollment of severe sepsis, septic shock patients
  - Algorithmic care optimizing CVP, MAP, ScvO2
  - RESULTS:
    - 16% absolute mortality reduction

The influence of early hemodynamic optimization on biomarker patterns of severe sepsis and septic shock*

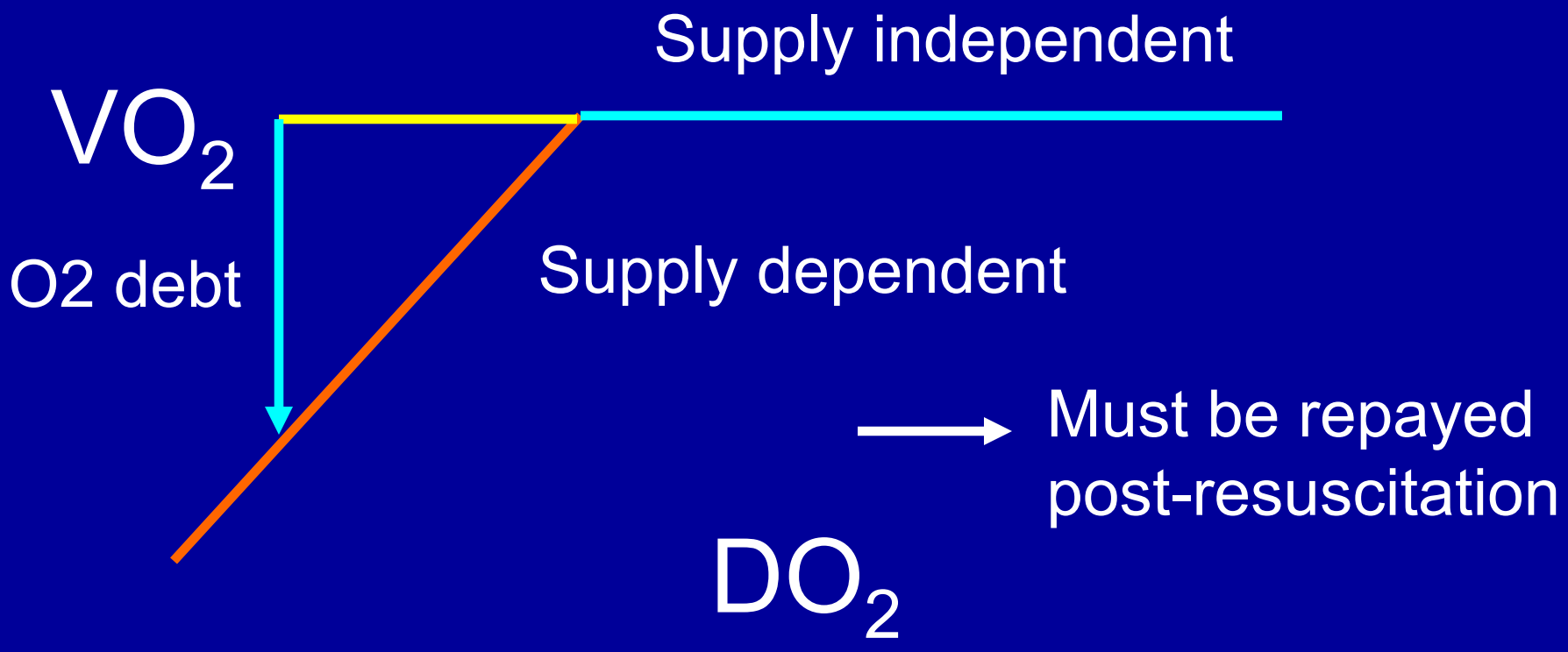
Emanuel P. Rivers, MD, MPH; James A. Kruse, MD; Gordon Jacobsen, MS; Kant Shah, MD; Manisha Loomba, MD; Ronny Otero, MD; Ed W. Childs, MD





Crit Care Med, 2007

Delivery vs. Consumption



AHA Post-Arrest Recommendations

- 2005 AHA guidelines recommend hemodynamic optimization of patients post-arrest:
  - Invasive monitoring
  - Titrate volume infusion to CVP
  - Titrate vasoactive, inotropic, and vasodilator drugs as needed to support blood pressure, cardiac index, and systemic perfusion

AHA PostResuscitation Support
Circulation, 2005

Are people applying these concepts to post-arrest patients?

- Is anyone doing this?
- Literature survey looking for studies using a goal-directed hemodynamic optimization strategy post-arrest
- Inclusion criteria:
  - a clearly defined intervention consisting of a structured cardiovascular resuscitation protocol
  - a control group that received standard of care therapy
- NO studies found where the AHA recommendations were being followed

Jones, Trceziak, Shapiro: Resuscitation, 2007

Post resuscitation care
Time for a care bundle?

- Editorial examining whether it is time for a care bundle for systematic delivery of post-resuscitation care
- Proposed bundle elements include:
  - Therapeutic hypothermia
  - Early percutaneous coronary intervention
  - Hemodynamic optimization
  - Other adjuncts to intensive care
- Question—Do bundles improve care?
- For example, surviving sepsis campaign's bundles:
  - Do we believe the evidence for each component?
  - Are bundles revised with changing evidence?
  - Is the evidence objectively analyzed?

Nolan J, Soar J, Resuscitation. 2008; 76: 161 – 162

Nagao et al - Hemodynamic Parameters

- This study was not included because there is no control group
- However, there is a clearly defined hemodynamic resuscitation strategy:
  - SBP ≥ 90 mmHg
  - CI ≥ 2.2L/min/m^2
  - Systemic O_2 Delivery ≥ 520mL/min/m^2
  - Oxygen Extraction Ratio between 20-30
  - Hemoglobin ≥ 12 g/dL

Nagao et al, CardioCerebral Resuscitation
JACC 2000; 36: 776-83

Bernard's Landmark Trial

- What hemodynamic strategy was pursued?
- "The mean arterial blood pressure was maintained between 90 and 100 mm Hg by infusion of epinephrine or nitroglycerin, as indicated."
- "After the admission of the patient to the intensive care unit, a pulmonary-artery catheter was inserted, and hemodynamic data were obtained 1 to 3, 6, 12, 18, and 24 hours after arrival at the hospital."

Treatment of comatose survivors…
Bernard SA et al. NEJM. 2002; 346: 557-563

Bernard: PAC Hemodynamics

TABLE 2. PHYSIOLOGICAL AND HEMODYNAMIC VALUES.*

VARIABLE	TREATMENT GROUP	ADMISSION TO ED	ADMISSION TO ICU	6 HR	12 HR	18 HR	24 HR
Number of patients	Hypothermia	43	39	39	39	39	38
	Normothermia	34	33	32	32	32	31
Temperature (°C)	Hypothermia	35.0±1.18	33.3±0.98†	32.7±1.19†	33.1±0.89†	36.0±1.24†	37.4±0.85†
	Normothermia	35.5±0.90	36.0±0.76†	37.1±0.75	37.4±0.58†	37.3±0.56†	37.3±0.59†
	P value‡	0.02	<0.001	<0.001	<0.001	<0.001	0.60
Mean arterial blood pressure (mm Hg)	Hypothermia	90.4±18.89	108.7±20.89†	97.0±14.92	89.5±13.16	88.8±9.17	89.1±12.9
	Normothermia	87.2±21.46	94.4±18.80	92.2±13.00	90.8±14.16	91.3±12.96	92.1±11.76
	P value‡	0.51	0.02	0.16	0.82§	0.38	0.24
Pulse (per minute)	Hypothermia	97±22.5	82±21.6§	72±17.1§	70±17.6	80±18.2§	89±17.9†
	Normothermia	105±30.4	100±17.0	100±21.9	94±17.9	97±16.8	99±15.5
	P value‡	0.18	0.001	<0.001	<0.001	<0.001	0.02
Cardiac index (liters/min/m² of body-surface area)¶	Hypothermia		2.0 (1.2–4.4)	2.1 (0.9–4.2)	2.4 (0.8–4.9)	2.9 (1.5–7.3)§	3.4 (1.6–6.8)§
	Normothermia		2.6 (1.4–5.5)	2.7 (1.4–6.1)	3.2 (1.2–6.1)	3.3 (1.5–5.8)	3.0 (1.8–5.7)
	P value‡		0.01	0.16	0.10	0.12	0.54
Systemic vascular resistance (dyn·sec·cm⁻⁵)¶	Hypothermia		2213 (599–4645)	1808 (836–4531)	1564 (439–4280)	1198 (402–2833)§	987 (551–2500)§
	Normothermia		1356 (481–2545)	1278.5 (346–2841)	1056 (340–3163)	964 (479–2204)†	1072 (591–1998)
	P value‡		0.02	<0.001	0.002	0.23	0.50

Treatment of comatose survivors…
Bernard SA et al. NEJM. 2002; 346: 557-563

Studies that employ some of a hemodynamic optimization protocol

- Since the publication of Bernard and HACA studies several implementation studies have incorporated components of hemodynamic optimization:
  - Oddo et al, CCM, 2006
    - MAP: 90-100 mm Hg
  - Hovdenes et al, Acta Anaesth Scand, 2007
    - Early PCI
    - IABP
    - PA Catheter
  - Sunde et al, Resuscitation, 2007

Hovdenes et al

Table 4

Cardiac index and systemic vascular resistance index (SVRI) during the first 32 h in the intensive care unit (ICU).

	Cardiac index (l/min/m^2)	SVRI (dyn s/cm^5/m^2)	Central venous oxygen saturation (%)
At 4 h in ICU*	2.1 (1.1–3.8) ($n = 22$)	1990 (1260–4200)	69 (56–85)
At 8 h in ICU*	2.2 (1.6–4.2) ($n = 23$)	2100 (800–3300)	76 (57–85)
At 12 h in ICU*	2.3 (1.0–3.6) ($n = 25$)	1785 (1163–4975)	75.5 (63–87)
At 16 h in ICU*	2.6 (1.1–4.3) ($n = 27$)	1650 (645–3520)	77 (61–83)
At 20 h in ICU*	2.6 (1.8–3.9) ($n = 27$)	1540 (980–3110)	77 (65–86)
At 24 h in ICU*	2.8 (1.7–5.5) ($n = 28$)	1500 (580–2810)	75 (63–85)
At 28 h in ICU*	3.15 (2.0–5.4) ($n = 26$)	1400 (860–2660)	79.5 (65–88)
At 32 h in ICU*	3.3 (2.2–5.7) ($n = 24$)	1250 (740–1860)	74 (55–85)

*Median values (minimum–maximum).

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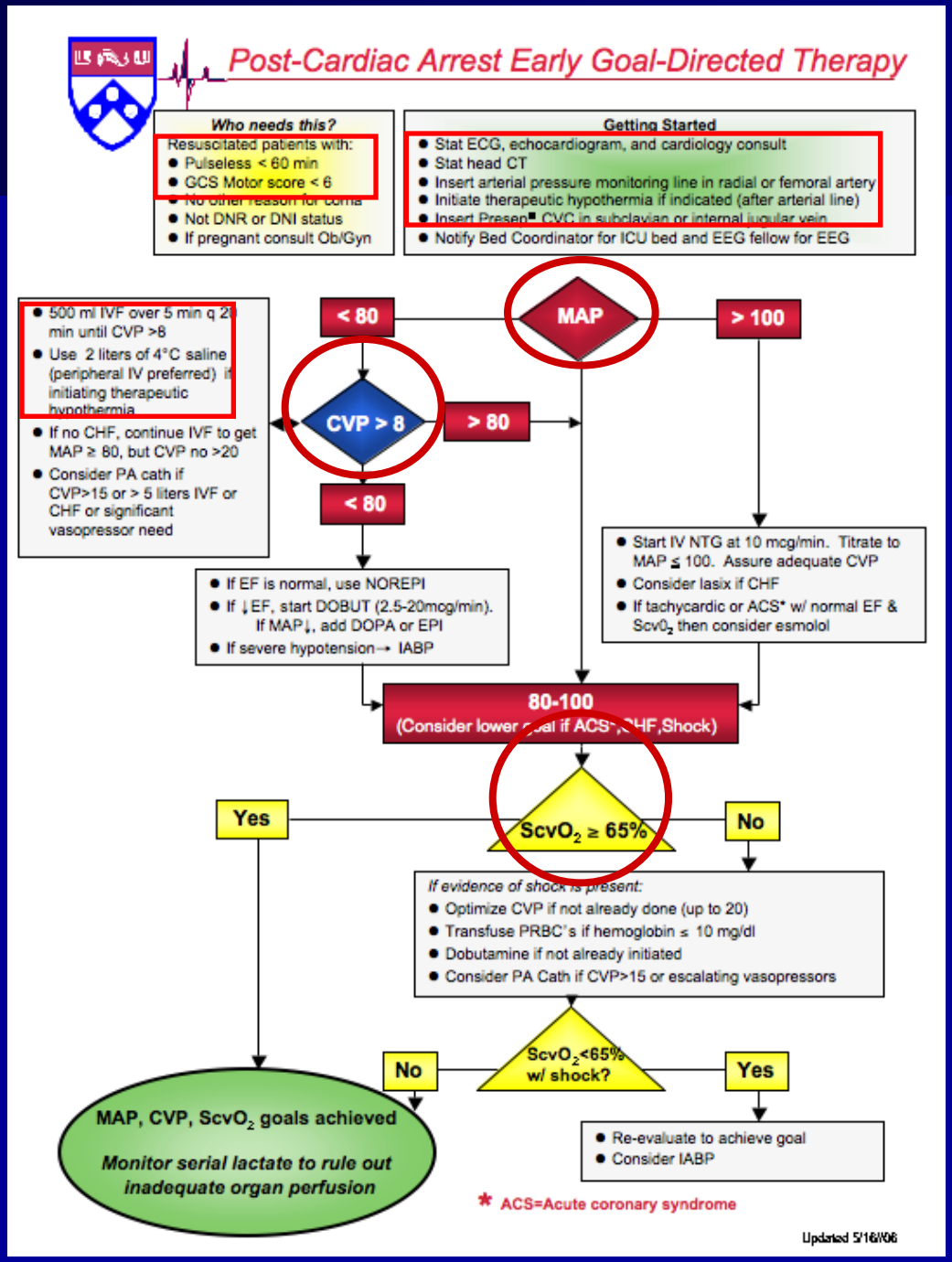
How do we apply this knowledge and a hemodynamic optimization strategy to patients who have ROSC after OHCA?

Aspects of Post-Resuscitation Care Bundle

- STEMI→Early PCI
- Therapeutic Hypothermia
- Early Hemodynamic Optimization
- Hyperglycemia→Glucose Management Protocol
- ALI/ARDS→Low Stretch Procotol
- Antibiotic, GI, DVT Prophylaxis
- Assessment for Relative Adrenal Insufficiency or HPA Dysfunction


Post-Cardiac Arrest Early Goal-Directed Therapy
Who needs this?
Resuscitated patients with:
Pulseless < 60 min
GCS Motor score < 6
Not DNR or DNI status
If pregnant consult Ob/Gyn
Getting Started
Stat ECG, echocardiogram, and cardiology consult
Stat head CT
Insert arterial pressure monitoring line in radial or femoral artery
Initiate therapeutic hypothermia if indicated (after arterial line)
Notify Bed Coordinator for ICU bed and EEG fellow for EEG
MAP
< 80
> 100
CVP > 8
> 80
< 80
500 ml IVF over 5 min q 2
min until CVP >8
Use 2 liters of 4°C saline
(peripheral IV preferred)
initiating therapeutic
hypothermia
If no CHF, continue IVF to get MAP ≥ 80, but CVP no >20
Consider PA cath if CVP>15 or > 5 liters IVF or CHF or significant vasopressor need
If EF is normal, use NOREPI
If ↓EF, start DOBUT (2.5-20mcg/min). If MAP↓, add DOPA or EPI
If severe hypotension→ IABP
Start IV NTG at 10 mcg/min. Titrate to MAP ≤ 100. Assure adequate CVP
Consider lasix if CHF
If tachycardic or ACS* w/ normal EF & ScvO2 then consider esmolol
80-100
ScvO2 ≥ 65%
Yes
No
If evidence of shock is present:
Optimize CVP if not already done (up to 20)
Transfuse PRBC's if hemoglobin ≤ 10 mg/dl
Dobutamine if not already initiated
Consider PA Cath if CVP>15 or escalating vasopressors
ScvO2<65% w/ shock?
No
Yes
MAP, CVP, ScvO2 goals achieved
Monitor serial lactate to rule out inadequate organ perfusion
Re-evaluate to achieve goal
Consider IABP
* ACS=Acute coronary syndrome
Updated 5/16/06

Early goal-directed hemodynamic optimization combined with therapeutic hypothermia in comatose survivors of out-of-hospital cardiac arrest

- Feasibility study
- Hypothesis

  "We can implement early goal-directed hemodynamic optimization while inducing TH w/o negatively impacting on time to target T°"
- Analyzed first 18 patients since start of TH
- Versus 18 historic controls from 2001-2005
- Examined differences in
  - Volume resuscitation
  - Vasoactive drug use
  - Mortality
  - Good neurologic outcomes

Gaieski et al. Resuscitation, 2009; 80: 418-424

GDR Endpoints Over Time

Resuscitation End-Point (hr)	0	1	2	3	4	5	6
CVP ≥ 8 ≤ 20 mmHg	77.8	Intravenous Fluid Boluses					81.3
MAP 80-100 mmHg	50.0	Vasoactive Medications					83.3
ScvO2 > 65	83.3	Inotropic Agents and Blood					93.8
Target Temp 32-34°C	5.6	11.1	44.4	55.6	61.1	66.7	77.8

4°C Chilled Saline; Cooling Wraps

New Strategy

	MAP	CVP	ScvO2	Input	Output
1 hour	84	6	78		
ED				3800	500
Balance					3300+
6 hours	75	17	87		
12 hours				5330	1400
Balance					4130+
24 hours	96	7	87		
24 hours				13546	6410
Balance					7136+

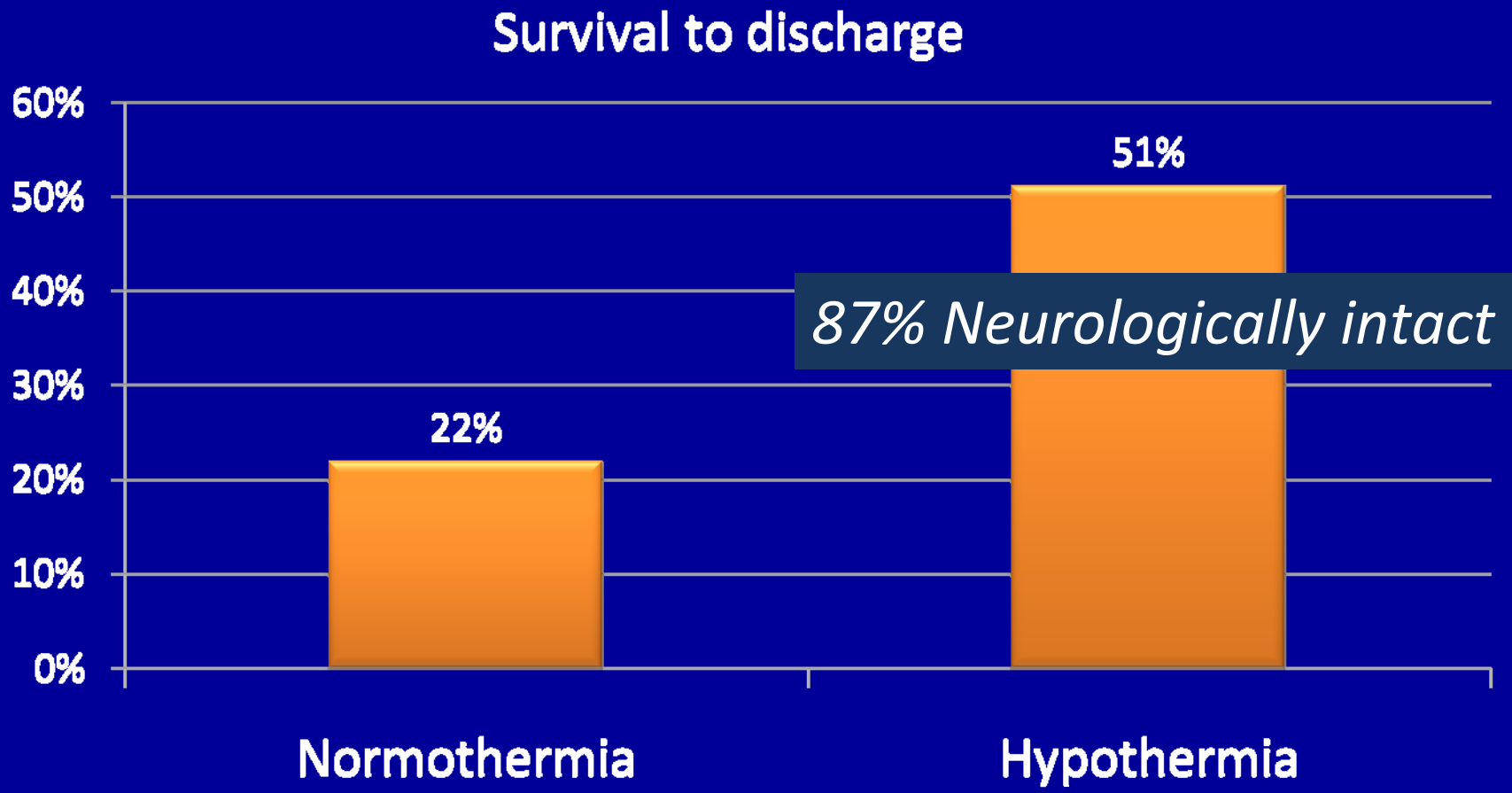
20 yo M, witnessed arrest, no bystander CPR
asystole first rhythm on monitor
ED ongoing CPR, ROSC @ minute 21

Dobutamine started @ hour 12

Rapidly Changing Hemodynamics

ECHO		EF
1 Hr	The LV is normal in size with moderate global systolic dysfunction	40-45%
12 Hr	The LV is mildly dilated with moderate global systolic dysfunction	40%
32 Hr	The LV is normal in size with normal systolic function	60%

HUP Data



Bernard's pilot study 26% vs 49%

Conclusions

- Multiple studies demonstrate that treatment of Post-Resuscitation Syndrome improves outcomes
- Implementation of Early Hemodynamic Optimization Strategy in the ED while inducing therapeutic hypothermia is feasible
- Further understanding of post-arrest hemodynamic changes is needed
- Optimal Post-Resuscitation Bundle not yet defined

Center for Resuscitation Science

Lance Becker
Bob Neumar
Vinay Nadkarni
Dave Gaieski
Munish Goyal
Raina Merchant
Ben Abella
Roger Band
Brendan Carr
Bob Berg

CRS*
Center for Resuscitation Science