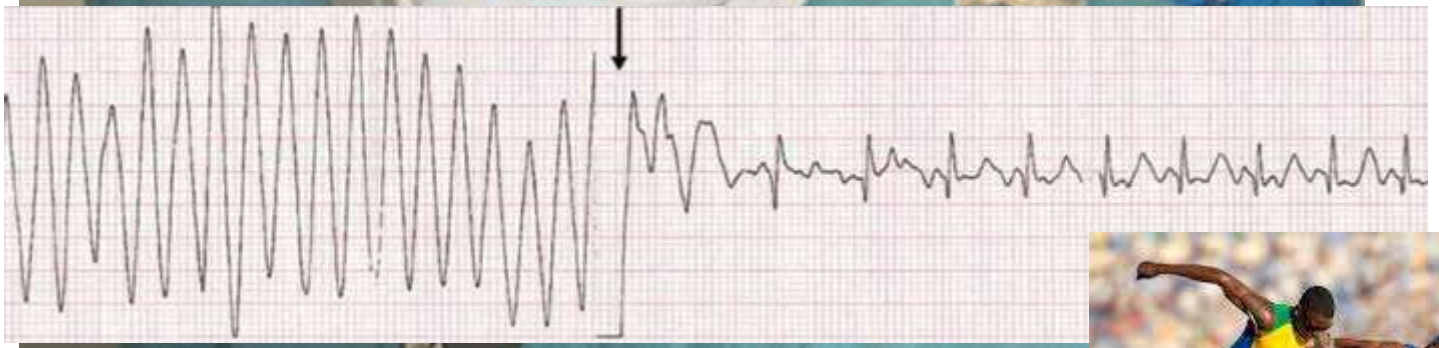
Post cardiac arrest care

Dr Ali Ashraf

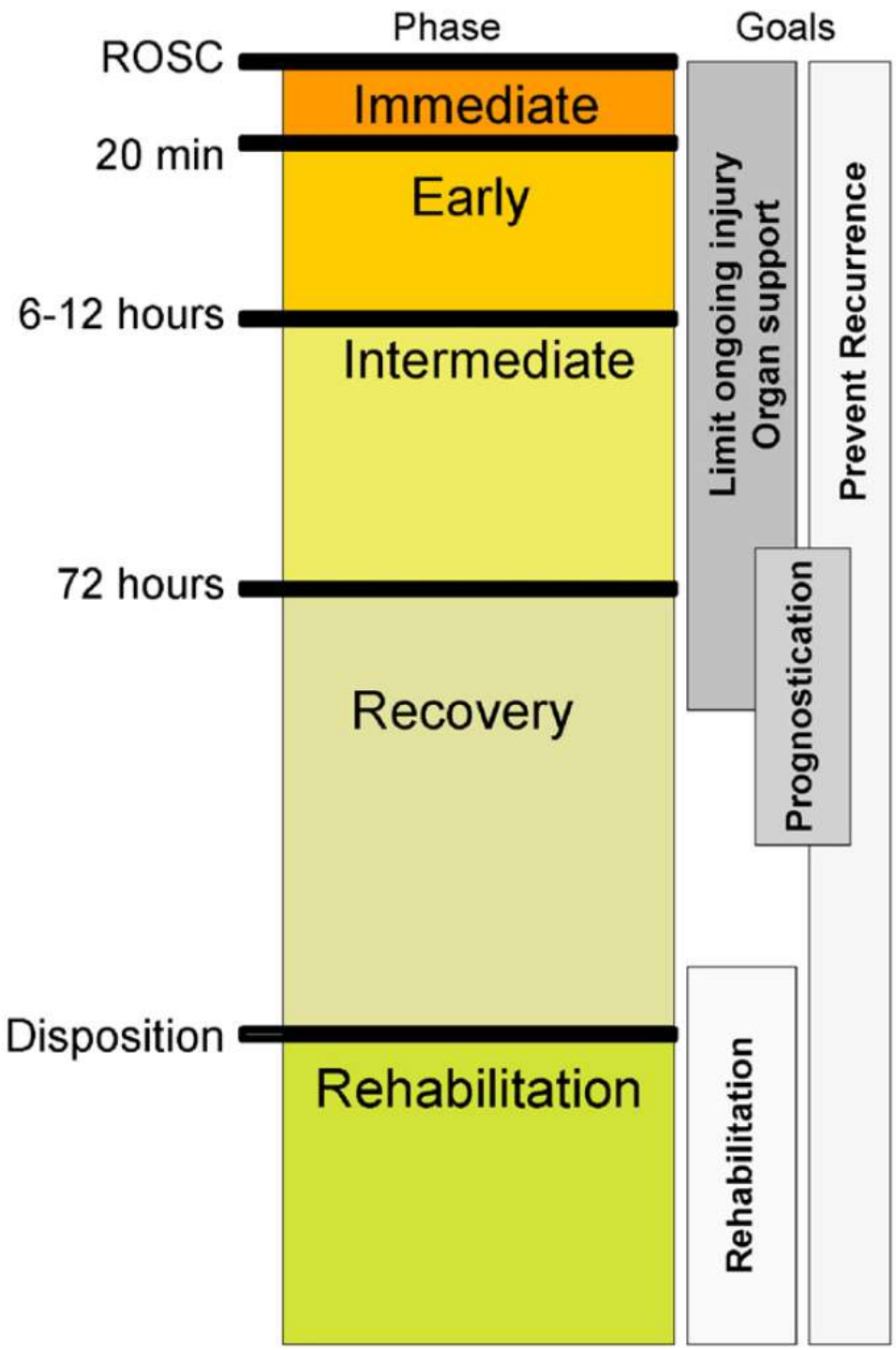
Intensivist

GUMS porsina H


CODE BLUE!!



Phases of post cardiac arrest syndrome

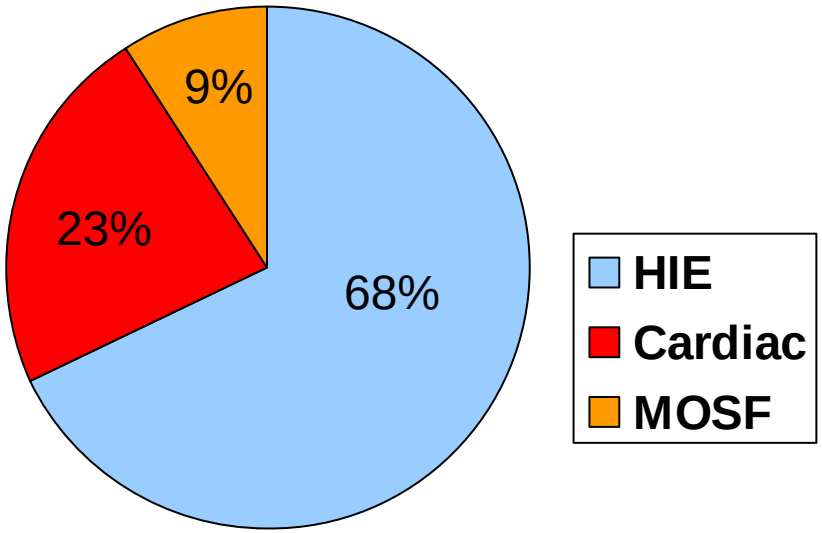


Critical Interventions Save Lives in Cardiac Arrest

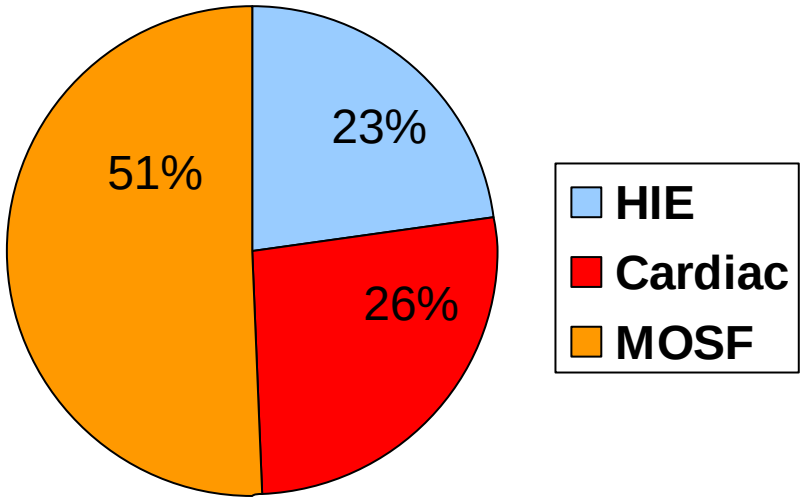
- **Bystander CPR**
  - Chest compressions only
- **Minimally interrupted CPR by EMS**
  - "Cardiocerebral resuscitation"
    - Ventilation kills
- **Modern post-resuscitation care**
  - Therapeutic hypothermia
  - Cardiac and hemodynamic support

From what do they die…?

Cause of Death in OHCA



Cause of death in IHCA



Laver. Intensive Care Med 2004;30:2126

The post-cardiac arrest syndrome

- post-cardiac arrest brain injury
- post-cardiac arrest myocardial dysfunction
- systemic ischaemia/reperfusion response
- persistent precipitating pathology.

The principal objective of postresuscitation care is the

- re-establishment of effective perfusion of organs and tissue.

In humans, hypotension after CA is poorly tolerated

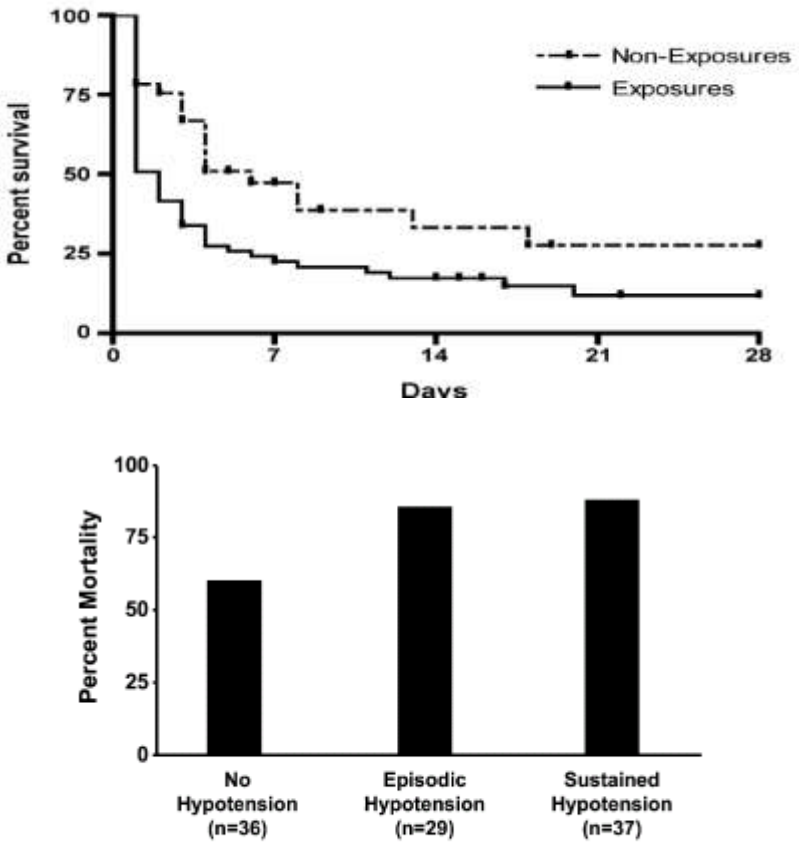


Figure 3 Percent in-hospital mortality for subjects with no hypotension, episodic hypotension, and sustained hypotension as defined by the criteria from Jones et al.[14,22]

- 102 patients/1 year with ROSC after CA
  - Retrospective evaluation
- SBP < 100mmHg on two episodes within first 6 hours independently associated with death
  - Careful correction for SOI
  - OR 3.5 poor outcome

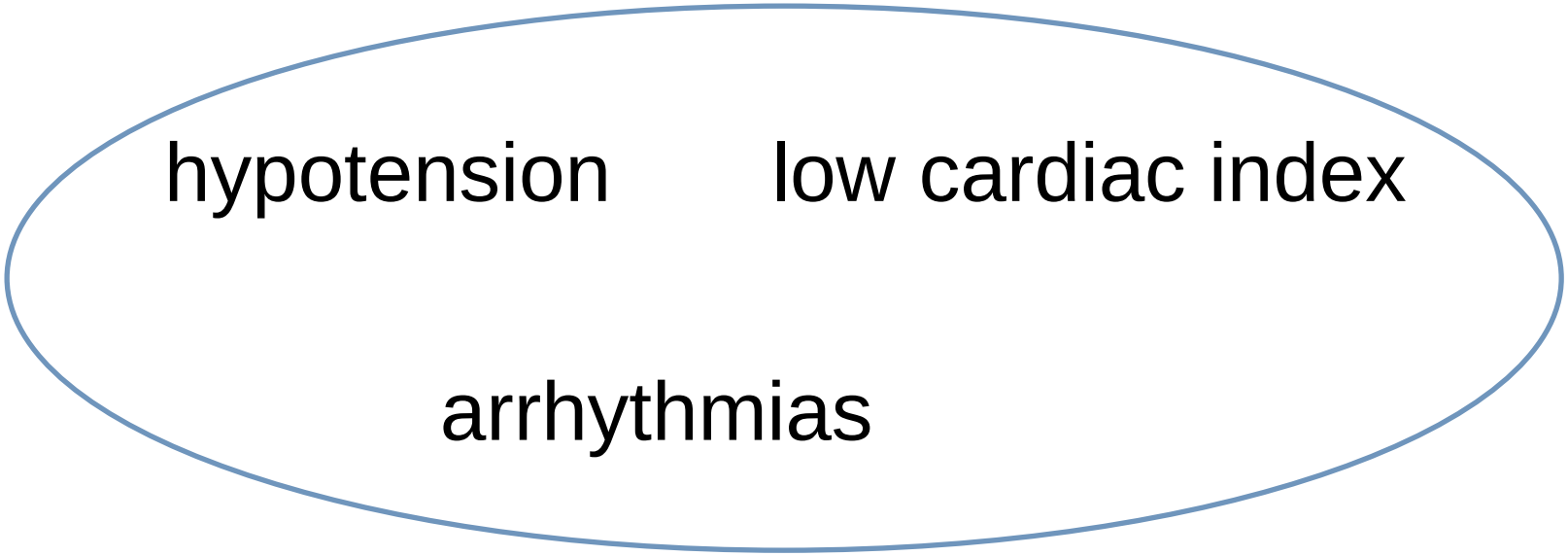
Resuscitation 2008;79:410-6.

Hemodynamic targets

- SBP > 90 mmHg
- MAP 70 – 75 mmHg
- CVP > 10 – 12 mmHg

Haemodynamic management

- Post-resuscitation myocardial dysfunction causes haemodynamic instability,



Haemodynamic management

- myocardial dysfunction often requires inotropic support
- Vasoplegia and severe vasodilation

Haemodynamic management

- Noradrenaline, with or without Dobutamine, and
- Fluid

most effective treatment

Airway and breathing
Control of oxygenation

- titrate the inspired oxygen concentration to arterial blood oxygen saturation in the range of 94–98%.

Obtunded cerebral function.

- Tracheal intubation,
- Sedation
- Controlled ventilation

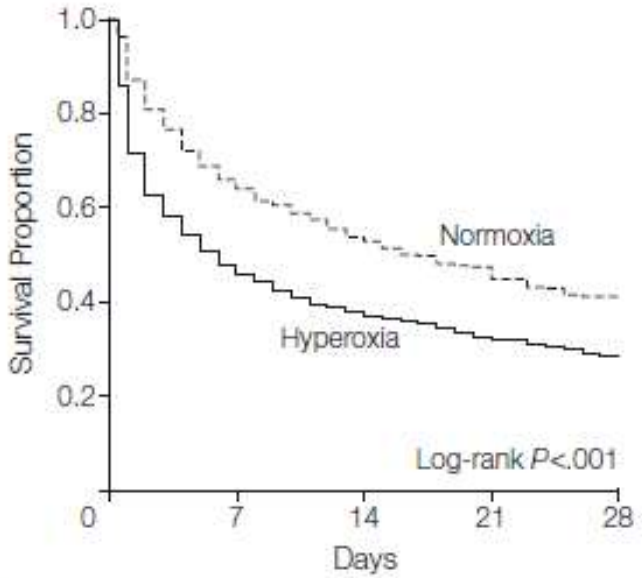
Airway and breathing

- Insert a gastric tube
- adequate doses of sedative
- neuromuscular blocking drug
- Continuous electroencephalography (EEG)
- chest radiograph

3 groups…

- *First* ABG
- Hypoxia:
  - PaO2<60mmHg
- Normoxia
  - PaO2 60-299mmHg
- Hyperoxia
  - PaO2≥300mmHg

Figure. In-Hospital Death Between Hyperoxia and Normoxia



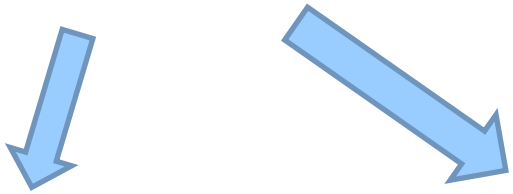
No. at risk

Normoxia	1171	514	236	129	83
Hyperoxia	1156	406	211	115	70

JAMA 2010;303:2165

Control of ventilation

- Hypocarbia ⇨ cerebral vasoconstriction, it
- adjust ventilation to achieve normocarbia



- end-tidal CO_2 arterial blood gas

Hyperoxia in post-resuscitation CA care questioned!

- “Normoxic resuscitation”
- “Lowest FiO2 to generate an SpO2 >94%
- Pao2 < 300 mmHg
- Paco2 35 -45 mmHg

apply protective lung ventilation:

- tidal volume 6–8 mL kg^{-1} ideal body weight
- positive end expiratory pressure 4–8 cm H_2O

Circulation, Coronary reperfusion

- Acute coronary syndrome (ACS) is a frequent cause of out-of-hospital cardiac arrest (OHCA)

PCI following ROSC with ST-elevation

- post-ROSC electrocardiogram (ECG) more than 80% will have an acute coronary lesion
- ST segment elevation (STE)
- Left bundle branch block (LBBB)
- Early invasive management is beneficial in STE patients.

PCI following ROSC with ST-elevation

- Immediate angiography and PCI

  in resuscitated OHCA patients whose initial ECG shows ST-elevation, even if they remain comatose

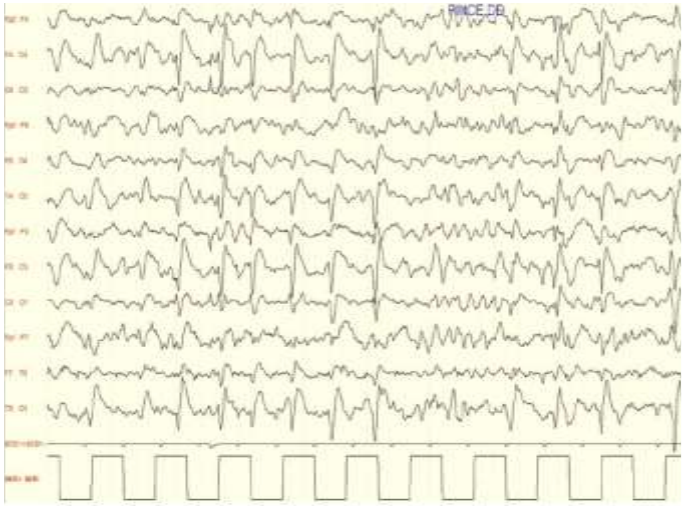
- Do not use level of consciousness after cardiac arrest caused by suspected acute STEMI to determine whether a person is eligible for coronary angiography

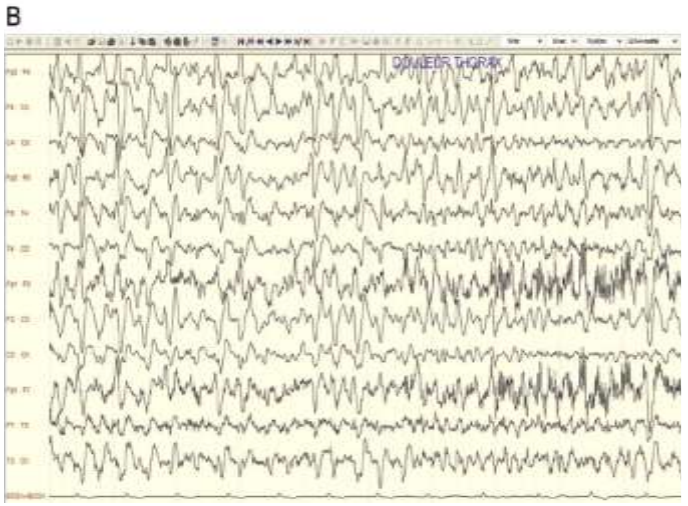
Percutaneous coronary intervention following ROSC without ST-elevation

- patient age
- duration of CPR
- haemodynamic instability
- presenting cardiac rhythm
- neurological status upon hospital arrival
- likelihood of cardiac aetiology

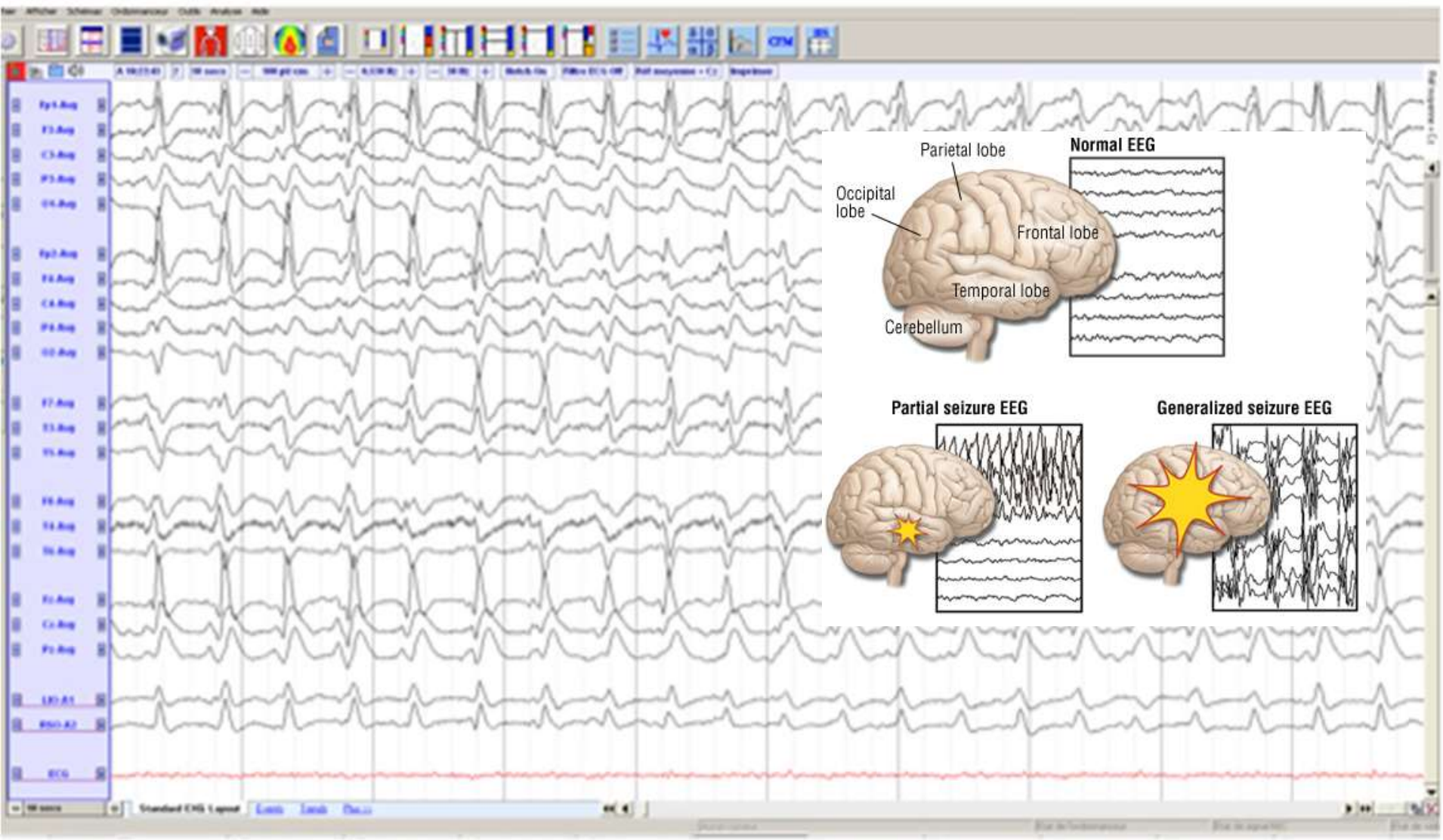
Other secondary injury…

- Uncontrolled seizure activity
- Hypotension, hypoperfusion
  - Postresuscitation syndrome
  - ICP crisis
  - Autoregulatory failure
- Fever
- Re-arrest
- Hypoxia
- Derangements of glucose metabolism



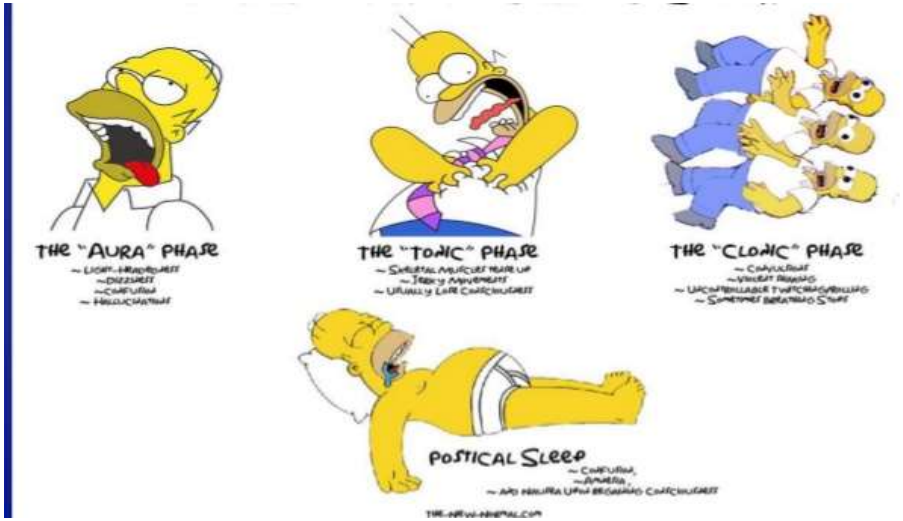


Neurology 2008;72:744


Normal EEG
Parietal lobe
Occipital lobe
Frontal lobe
Temporal lobe
Cerebellum
Partial seizure EEG
Generalized seizure EEG

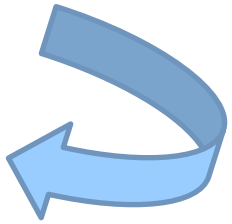
Control of seizures

- Non convulsive seizure should be diagnosed
- Serial EEG
- Routine Prophylaxy??



Glucose control

- high blood glucose after resuscitation

  Poor neurological outcome 

- Do not implement strict glucose control in adult patients with ROSC after cardiac arrest

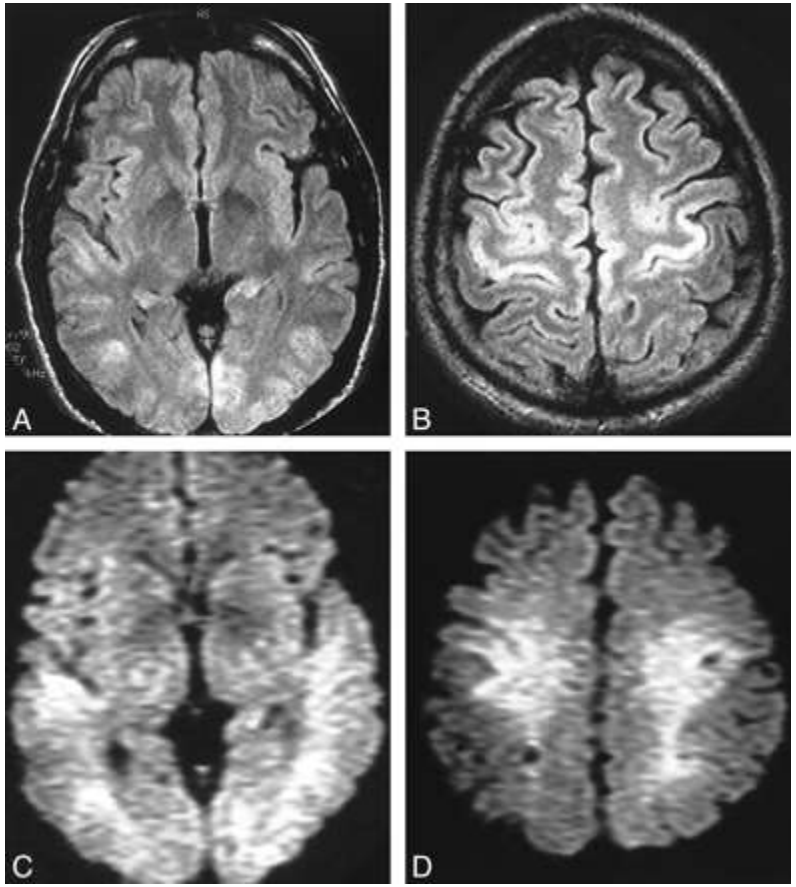
Mechanisms of brain injury in circulatory arrest

- Primary Injury:
  - "Energy failure" due to ATP depletion
- Secondary injury:
  - Loss of transcellular electrolyte gradients
    - Ca^{+}, Na^{+}, Cl^{-} enter, K^{+} exits cell
    - Water follows Na^{+} into cells causing cytotoxic edema
  - Lipid peroxidases damage membranes
  - Neurotransmitter release causes excitotoxicity
  - Activation of apoptotic pathways
  - Microvascular thrombosis
  - Reperfusion injury

6-72h

optimising neurological recovery

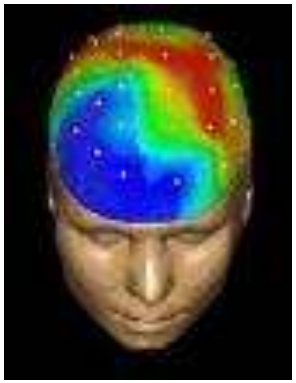
Cardiac arrest associated brain injury “CAABI”

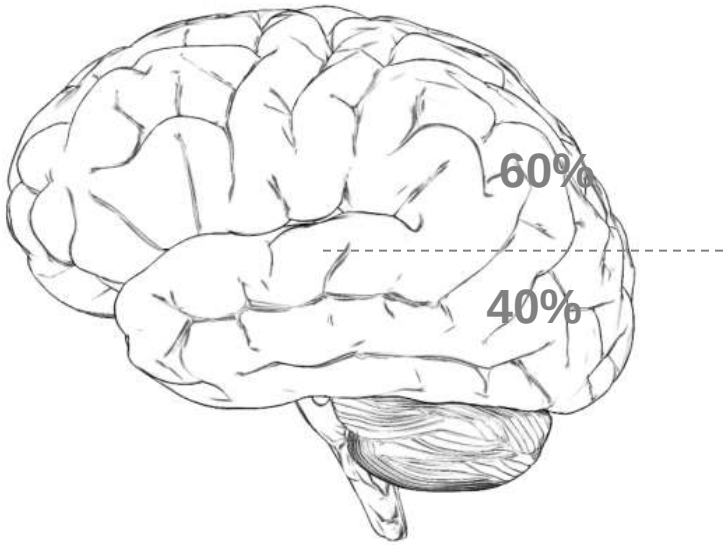


- “No flow” affects the most metabolically active areas of brain
  - Cortex
  - Basal ganglia
  - Cerebellum
- “Low flow” affects the watershed areas between vascular territories

Cerebral Energy Requirements

- CMRO2: 3.5 cc O2/min/100 g
- CMR glucose: 5.5 mg/min/100 g
  - Activation Metabolism
    - 55-60%
  - Residual Metabolism
    - 40-45%





Barash, Cullen, Stoelting. Clinical Anethesia, 5th Ed. Lippincott Williams & Wilkins, 2006; p 751.

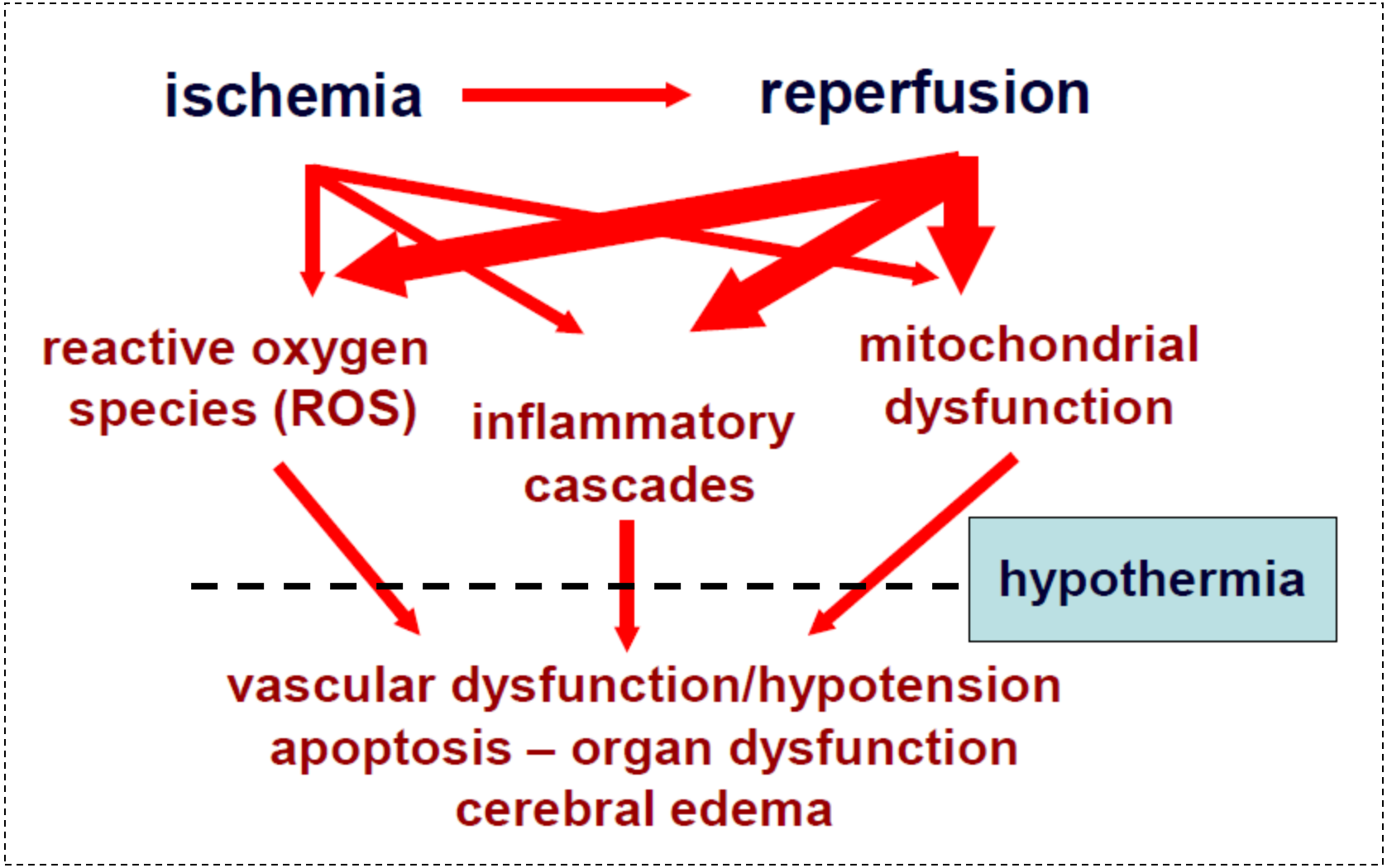
Neuro-Protection?

- Maintain blood flow
- Maintain ATP levels by reducing CMR
- Blocking Na and Ca influx
- Scavenge free radicals
- Block release/receptors for excitatory amino acids
- Delay membrane failure & apoptotic processes
- Inhibiting proteins that activate or contribute to damage (proteases, phospholipases, certain kinases)
- Activating proteins that induce repair or rescue

Barash, Cullen, Stoelting. Clinical Anethesia, 5th Ed. Lippincott Williams & Wilkins, 2006; p 751.

Hypothermia

therapeutic hypothermia in survivors of cardiac arrest


ischemia
reperfusion
reactive oxygen species (ROS)
inflammatory cascades
mitochondrial dysfunction
hypothermia
vascular dysfunction/hypotension
apoptosis – organ dysfunction
cerebral edema

Rationale for cooling

- Reduces the cerebral metabolic rate for oxygen (CMRO2)
    - ↓6% for every 1°C reduction in brain temperature >28°
- Suppresses chemical reactions associated with reperfusion injury
    - Inhibits excitatory amino acid release (eg glutamate)
    - Decreases free radical production
    - Limits calcium shifts that cause mitochondrial damage and apoptosis
- Decreases intracranial pressure

Historical context

- Hypothermia used in various scenarios
  - Described by ancient Greeks and Romans
  - Treatment of tetanus in 4th century BC
  - Treatment of trauma, Hippocrates
  - Treatment of typhoid fever, Osler 19th century

Historical context

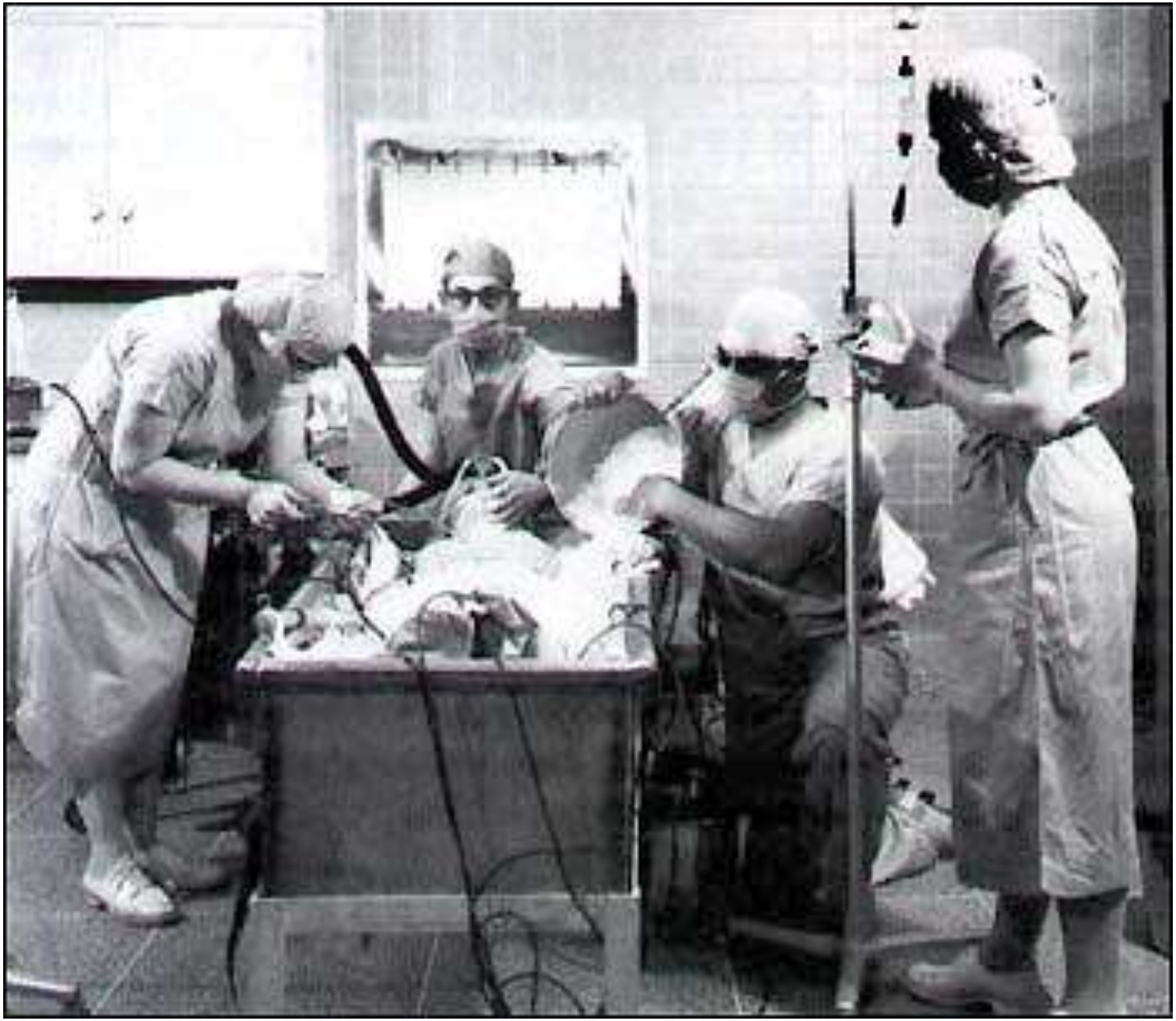
- Neurosurgeon Temple Fay advocated local and generalized refrigeration in 1930s-1950s
  - intractable headache pain
  - traumatic brain injury
  - abscess
  - cerebritis, seizures
  - malignancy

Fay T, Early experiences with local and generalized refrigeration of the human brain, Journal of Neurosurgery, May, 1959, number 3; 16:239-260,

THE USE OF HYPOTHERMIA AFTER CARDIAC ARREST

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

Anesthesia and Analgesia 1959;38 (6): 423



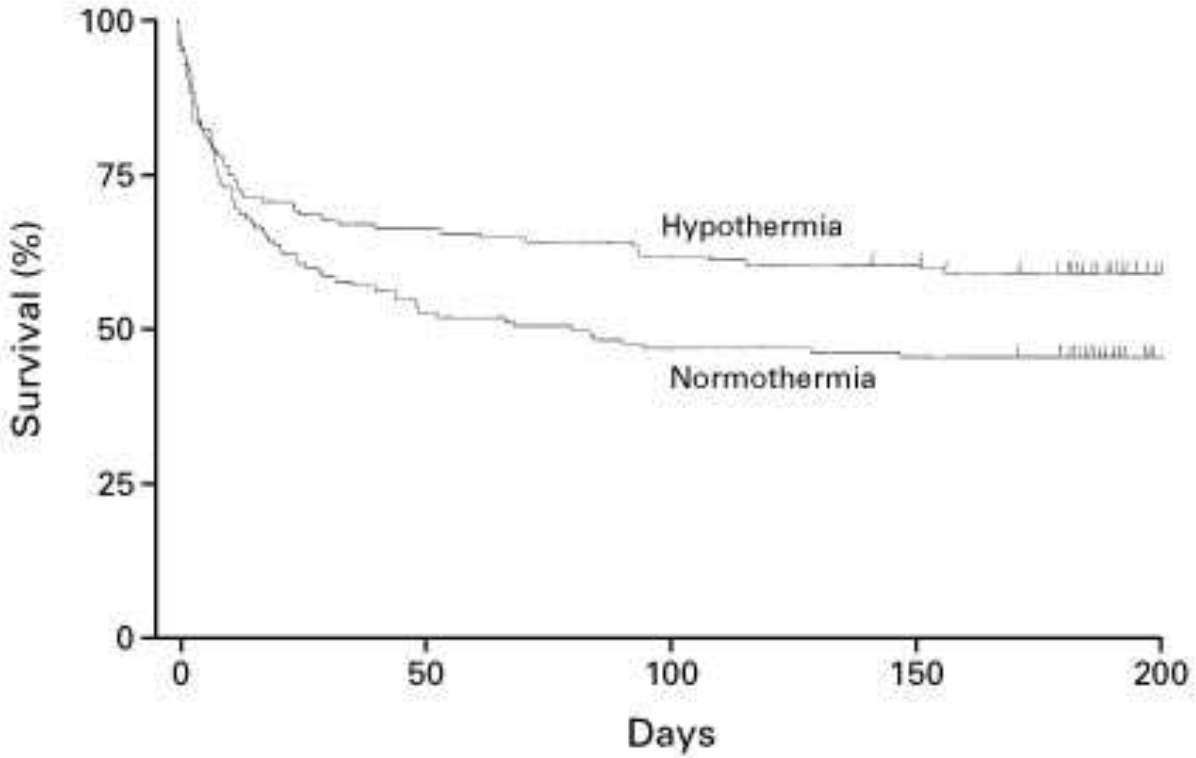
The New England
Journal of Medicine

VOLUME 346 FEBRUARY 21, 2002 NUMBER 8

MILD THERAPEUTIC HYPOTHERMIA TO IMPROVE THE NEUROLOGIC OUTCOME AFTER CARDIAC ARREST

THE HYPOTHERMIA AFTER CARDIAC ARREST STUDY GROUP*

Cumulative Survival



No. at Risk					
Hypothermia	137	92	86	83	11
Normothermia	138	74	66	64	9

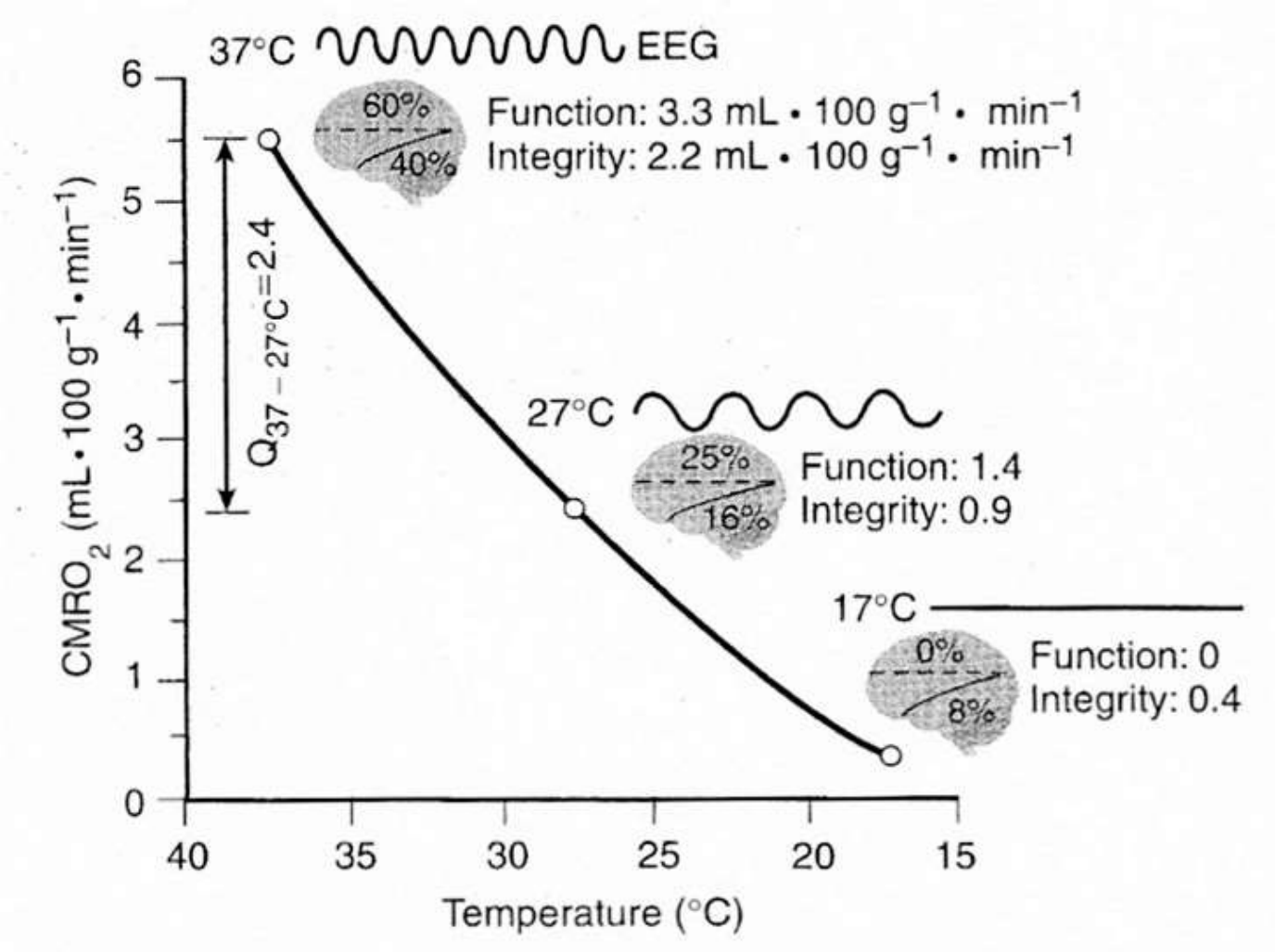
The Hypothermia after Cardiac Arrest Study Group. N Engl J Med 2002;346:549-556

The NEW ENGLAND JOURNAL of MEDICINE

Beneficial Effects of Hypothermia

- Decrease in cerebral metabolism
- Maintains integrity of membranes
- Preserves ion homeostasis
- Decreases excitatory AA release
- Decrease Ca influx
- Decrease lipid peroxidation
- Decrease free radical formation
- Decrease nitric oxide synthase activity

CMRO2 & Temperature



Miller. Miller's Anesthesia, 6th Ed, Vol 1. Elsevier Churchill Livingstone, 2005; p 816.

Only 10% patients with OHCA will meet RCT criteria for TH

Patients were excluded if they met any of the following criteria: a tympanic-membrane temperature below 30°C on admission, a comatose state before the cardiac arrest due to the administration of drugs that depress the central nervous system, pregnancy, response to verbal commands after the return of spontaneous circulation and before randomization, evidence of hypotension (mean arterial pressure, less than 60 mm Hg) for more than 30 minutes after the return of spontaneous circulation and before randomization, evidence of hypoxemia (arterial oxygen saturation, less than 85 percent) for more than 15 minutes after the return of spontaneous circulation and before randomization, a terminal illness that preceded the arrest, factors that made participation in follow-up unlikely, enrollment in another study, the occurrence of cardiac arrest after the arrival of emergency medical personnel, or a known preexisting coagulopathy.

- **The decision to initiate TH is usually based on clinical judgement of risk and benefit, not on proof!**

Risks

- Infections
- Bleeding
- Need for sedation

Benefits

- Strongly neuroprotective
- Decreased mortality
- Better neurological outcome

N Engl J Med 2002;346:549-56

TH after Cardiac Arrest

- **Clinical criteria for therapeutic hypothermia**
  - No more than **8 hours** have elapsed since the return of spontaneous circulation.
  - **Encephalopathy** is present, typically defined as the patient being unable to follow verbal commands.
  - There is **no life-threatening infection or bleeding**.
  - **Aggressive care is warranted** and desired by the patient or the patient's surrogate decision-maker
    - Terminal underlying disease
    - Impending cardiopulmonary collapse

What do I treat with therapeutic hypothermia?

- Cardiac Arrest
- Hepatic encephalopathy with cerebral edema
- Near hanging
- Neonatal asphyxia
- Elevated ICP, all causes
- Severe (Hunt and Hess IV-V) SAH with cerebral edema

What are the risks?

TABLE 4. COMPLICATIONS DURING THE FIRST SEVEN DAYS AFTER CARDIAC ARREST.*

COMPLICATION	NORMOTHERMIA	HYPOTHERMIA
	no./total no. (%)	
Bleeding of any severity†	26/138 (19)	35/135 (26)
Need for platelet transfusion	0/138	2/135 (1)
Pneumonia	40/137 (29)	50/135 (37)
Sepsis	9/138 (7)	17/135 (13)
Pancreatitis	2/138 (1)	1/135 (1)
Renal failure	14/138 (10)	13/135 (10)
Hemodialysis	6/138 (4)	6/135 (4)
Pulmonary edema	5/133 (4)	9/136 (7)
Seizures	11/133 (8)	10/136 (7)
Lethal or long-lasting arrhythmia	44/138 (32)	49/135 (36)
Pressure sores	0/133	0/136

*None of the comparisons between the two groups, performed with the use of Pearson's chi-square test, indicated significant differences.

†The sites of bleeding were mucous membranes, the nose, the urinary tract, the gastrointestinal tract, subcutaneous tissue, and skin, as well as intracerebral and intraabdominal sites.

- More infections
  - Lung
- Trends toward more bleeding*
- Electrolyte shifts
- Clinically insignificant bradycardia
- Changes in drug metabolism

HCASG. NEJM 2002;346:549-56

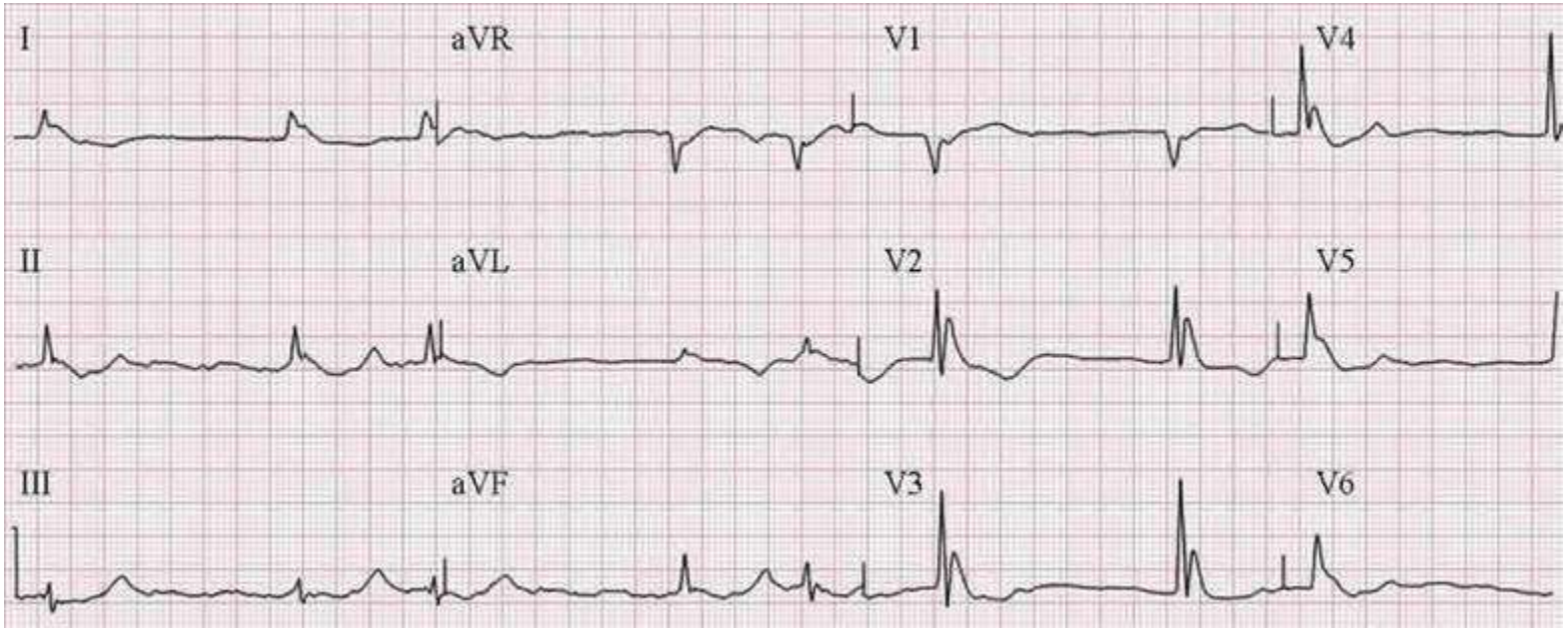
Physiologic effects of cooling

Physiologic effects of cooling

- Cardiovascular
  - Initially: cold vasoconstriction → ↑ venous return→ reflex tachycardia
    - ↑SVR, ↑BP, ↑CVP
  - <35 degrees: bradycardia, ↓ CO 25-40%
    - MVO2 ~same (↓ O2 consumption)
  - Hypovolemia due to “cold diuresis”
    - Vasoconstriction, hyperglycemia, ↓ renal tubular absorption, increased ADH
  - Lactate levels elevated (but stable)

Physiologic effects of cooling

- Cardiovascular
  - HR 40-60
  - ↑PR, ↑QTc, widened QRS
  - Osborne waves



Physiologic effects of cooling

- Cardiovascular
  - Arrhythmias:
    - Sinus brady
    - Afib→VT→VF (particularly <28C)
    - Myocardium less sensitive to anti-arrhythmics
  - Ischemia prevented/mitigated?

Physiologic effects of cooling

- Hepatic
  - ↓ drug metabolism
    - Paralytics
    - Opioids
    - Benzodiazepines
    - AEDs
    - propofol
    - cephalosporins

Physiologic effects of cooling

- Metabolic derangements
  - intracellular shift (K+, magnesium) and ↑ renal excretion
  - Hyperglycemia
    - ↓ insulin sensitivity in tissues
    - ↓ insulin secretion by islet cells
  - Metabolic acidosis
    - ↑FFA, ketones, lactate
    - Intracellular pH is actually higher
  - ↓ O2 consumption/CO2 production

Physiologic effects of cooling

- Hematologic
  - ↑ bleeding time
  - platelet dysfunction, mild thrombocytopenia
  - clotting factor dysfunction
  - activation of fibrinolysis cascade
- no significant increase in bleeding risk unless already bleeding (eg trauma)

Physiologic effects of cooling

Study of 31 comatose patients post-cardiac arrest due to AMI treated with hypothermia 2005-2006

- Historical matched controls
- Goal temp 33°C
- 11 pts received thrombolysis in the field
- 25 pts underwent PCI and stenting
- All pts got clopidogrel 600mg load/75mg daily, heparin gtt
- 16 PCI pts received IIb/IIIa inhibitor

Int J Cardiology.132;2009:387-391.

Physiologic effects of cooling

Study of 31 comatose patients post-cardiac arrest due to AMI treated with hypothermia 2005-2006

- Favorable neuro outcome in TH (19/31 had CPC 1 or 2 vs control 6/31)
- ***No difference in ICU mortality (10/31 in both groups)***
- ***No difference in incidence of clinically overt blood loss (6/31 in both groups)***
  - Trend towards increase in # units transfused in TH

Int J Cardiology.132;2009:387-391.

Physiologic effects of cooling

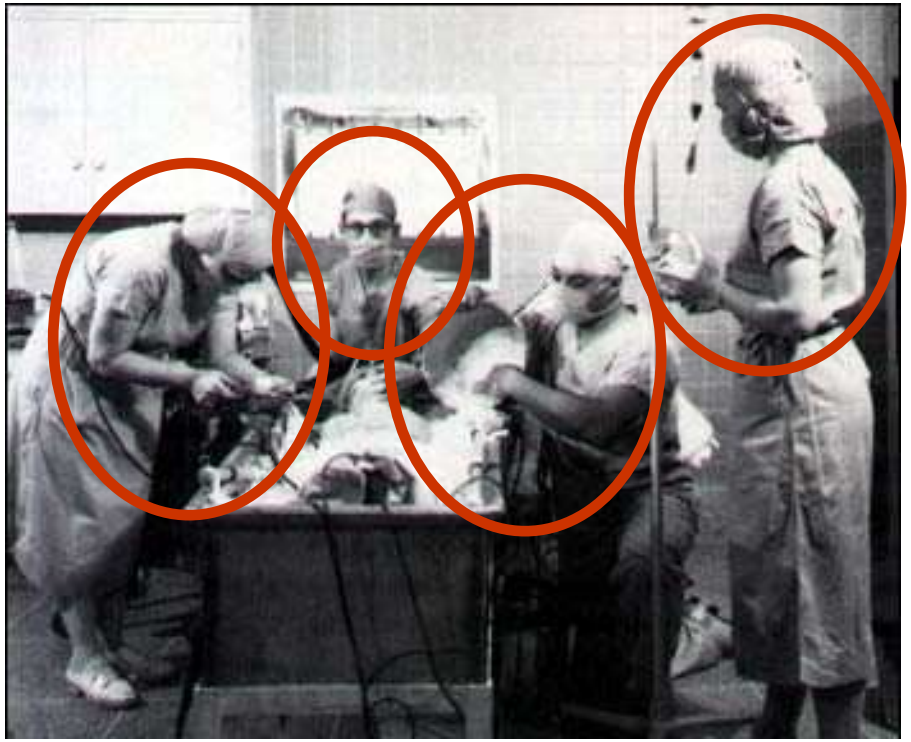
- Immunologic
  - Inhibits leukocyte migration, function
  - Inhibits secretion of inflammatory cytokines
  - Increased risk of wound infection
    - Related to vasoconstriction to skin, hyperglycemia
  - Already at-risk population (higher rates of aspiration, more ventilator days, higher rates of VAP, sepsis in post-cardiac arrest patients)

Resuscitation 2004;60:65-69.
Intensive Care Med 2005;31:621-626

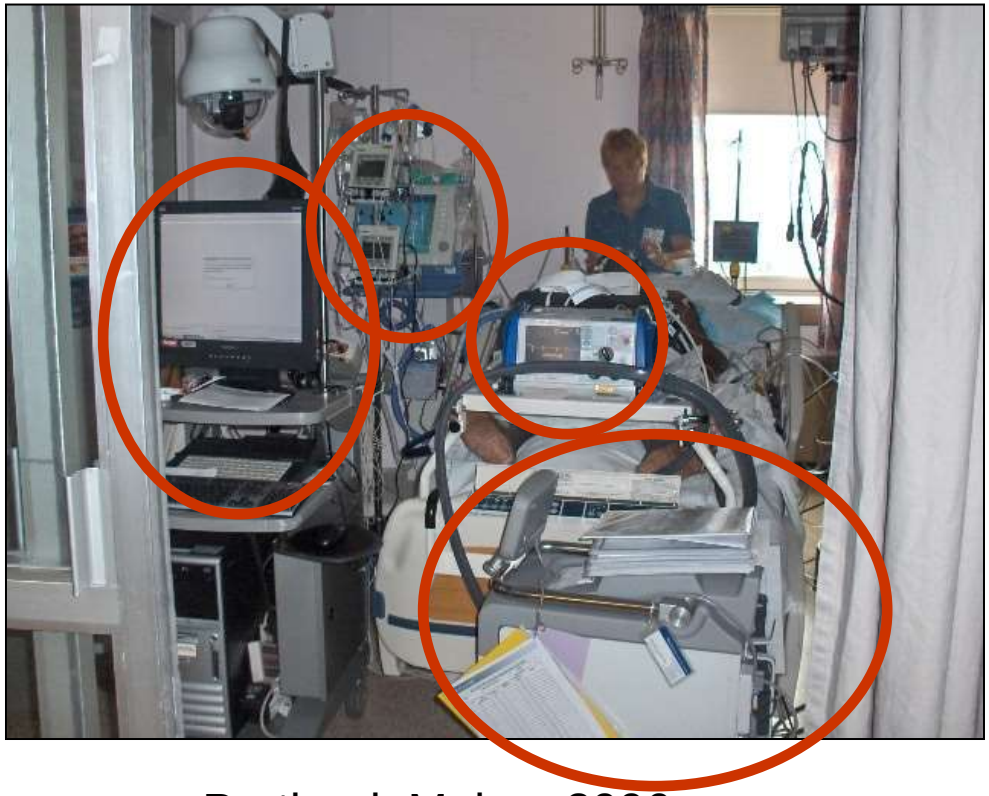
3 stages of therapeutic hypothermia

- Induction phase
  - Goal <34°C ?as quickly as possible
  - Highest risk of acute instability (electrolytes, hemodynamics)
- Maintenance phase
  - 24h tight core temp control
  - Highest risk of long-term complications (infections, skin breakdown)
- Rewarming phase
  - Slow, controlled
  - 0.2-0.5 degree/hour
  - Some acute instability but more easily managed

How to cool…



Baltimore, 1955



Portland, Maine, 2006

How to Cool: Four Modes of Heat Transfer

- **Conduction**
  - Cold water immersion
- **Radiation**
  - Cold room
- **Convection**
  - Fans (do not use for infection control purposes)
- **Evaporation**
  - Sweating

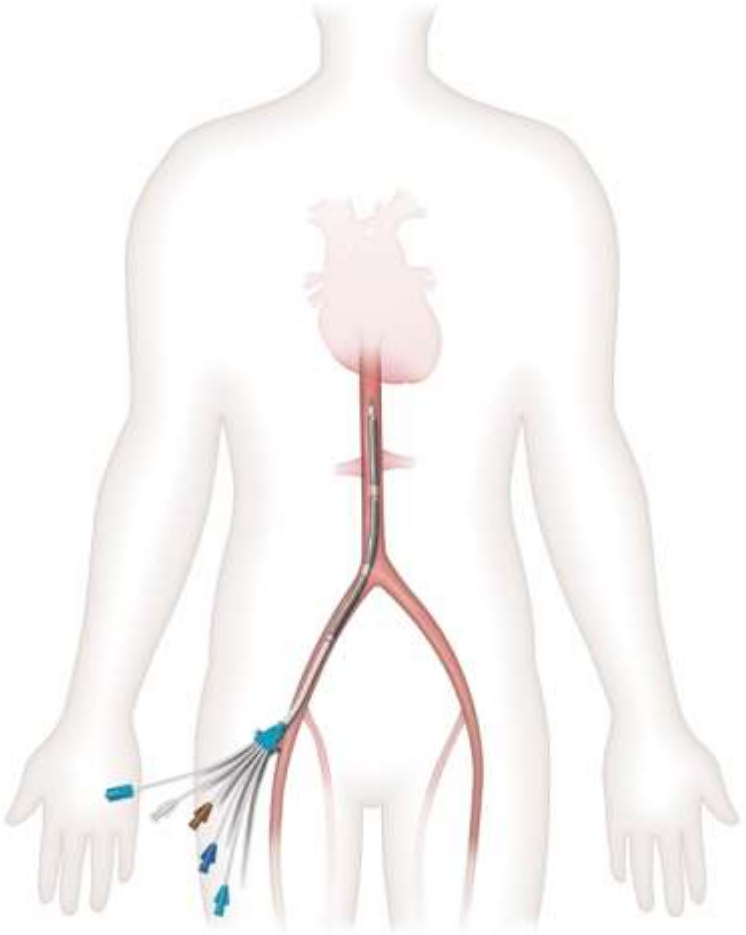
Cooling techniques

- Ice packs
- Iced saline infusion
- Iced nasogastric or bladder lavage
- Cooling blankets
- Fans
- ECMO
- Cooling devices

Induced hypothermia after out-of-hospital cardiac arrest

- Randomization **in the field**
  - Cooling measures (ice packs) instituted in ambulance
- Cooled to 33°C, maintained for 12 hours
- At 18 hours, active rewarming for 6 hours with heated-air blanket
- Lower rate of successful ROSC
- Start TTM on hospital arrival

Cool Gard with Icy Femoral Catheter



Arctic Sun


A Patented 3-Layer Construction
B Leak Proof Technology™
Simple, Safe and Effective C
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Arctic Sun


A Patented 3-Layer Construction
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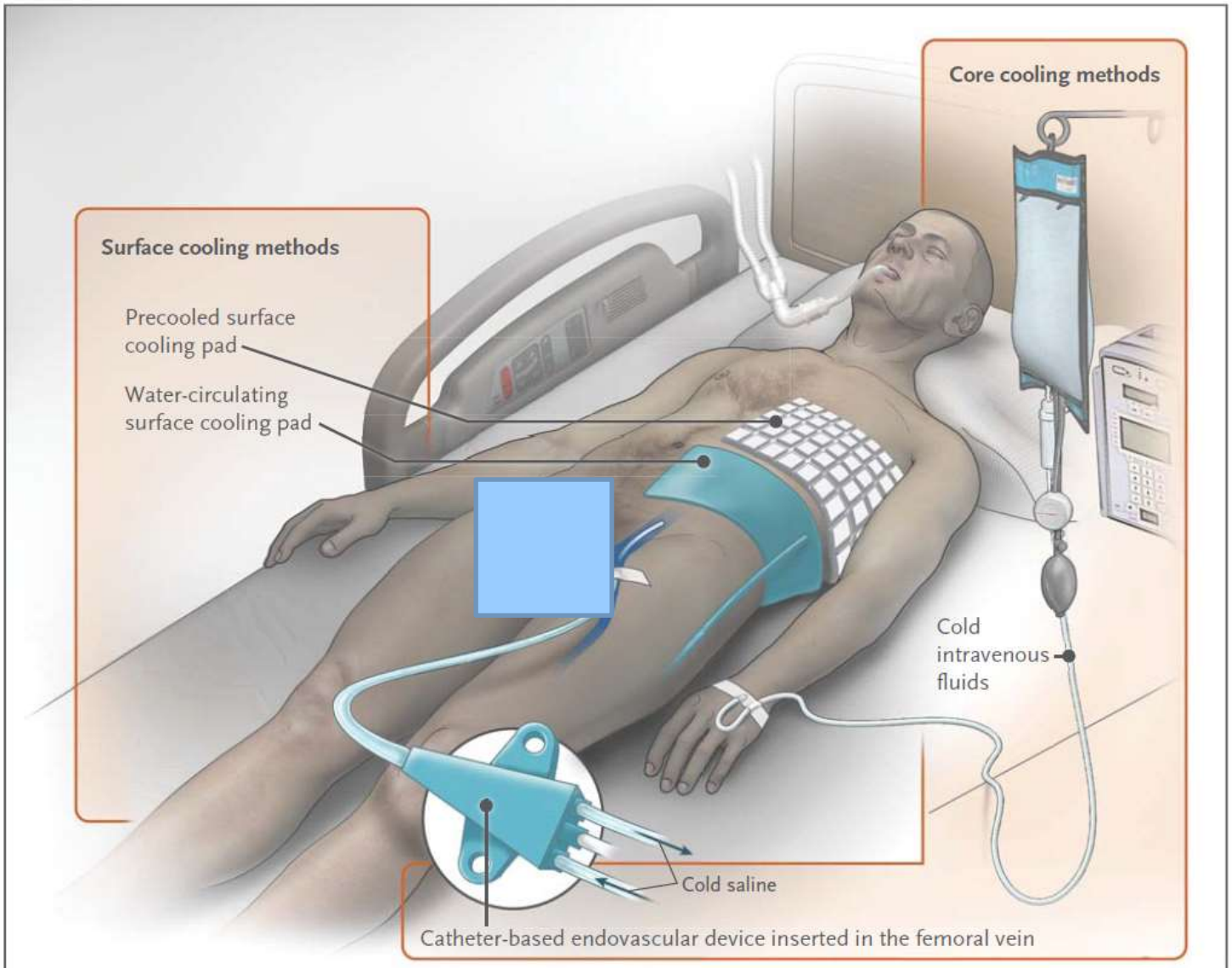


Figure 1. Cooling Methods Used in Clinical Practice.

Surface cooling methods include the use of precooled (refrigerated) surface cooling pads and water-circulating surface cooling pads. Core cooling methods include the infusion of cold intravenous fluids and the use of catheter-based endovascular devices.

Holzer. New Engl J Med 2010;363:1256-64

Basics of Therapeutic Hypothermia

- There are 3 phases of treatment:
  - **Induction**
    - Rapidly bring the temperature to 32-34C
    - Sedate with propofol or midazolam during TH
    - Paralyze to suppress heat production
  - **Maintenance**
    - maintain the goal temperature at 33C
    - Standard 12-24 hours (optimal duration is unknown)
    - Suppress shivering
  - **De-cooling (rewarming)**
    - Most dangerous period: hypotension, cerebral edema, seizures
    - Goal is to reach normal body temperature over 12-24h
    - Stop sedation when normal body temperature is achieved

Pilot Randomized Clinical Trial of Prehospital Induction of Mild Hypothermia in Out-of-Hospital Cardiac Arrest Patients With a Rapid Infusion of 4°C Normal Saline

Francis Kim, MD; Michele Olsufka, RN; W.T. Longstreth, Jr, MD; Charles Maynard, PhD; David Carlbom, MD; Steven Deem, MD; Peter Kudenchuk, MD; Michael K. Copass, MD; Leonard A. Cobb, MD

- 125 patients randomized to prehospital vs ED cooling
- Good outcome in VT pts cooled in the field
  - 20/29 vs 10/22 (P=0.15)
- No safety concerns
- Average temp at ED arrival differed by only 1ºC

TABLE 1. Baseline Characteristics of Patients

	Randomized to Field Cooling (n=63)	Not Randomized to Field Cooling (n=62)	P*
Age, mean±SD, y	67±15	65±17	0.55
Men	42 (67)	46 (74)	0.36
Witnessed cardiac arrest	42 (67)	46 (74)	0.64
CPR before paramedic arrival	27 (43)	27 (43)	0.94
Initial rhythm	...	...	0.45
Asystole	18 (29)	21 (34)	...
Pulseless electrical activity	15 (24)	19 (31)	...
VF	29 (46)	22 (36)	...
Unknown	1 (2)	0 (0)	...

Values are expressed as n (%) unless otherwise noted. CPR indicates cardiopulmonary resuscitation.
*Based on χ^2 or t test as appropriate.

TABLE 3. Outcomes in 125 Patients Resuscitated From Out-of-Hospital Cardiac Arrest and Randomized to Standard Care With or Without Field Cooling

	Cooling (n=63), n (%)		No Cooling (n=62), n (%)	
	VF (n=29)	No VF (n=34)	VF (n=22)	No VF (n=40)
Deaths before hospital admission	3 (10)	11 (32)	3 (14)	11 (27)
In-hospital deaths	7 (24)	21 (62)	9 (41)	21 (52)
Discharged alive*	19 (66)	2 (6)	10 (45)	8 (20)

Values are expressed as n (%). Patients are grouped according to initial cardiac rhythm of VF or no VF. Deaths before hospital admission include deaths in the field and emergency department.
*Of patients discharged alive, 2 in each treatment group had severe neurological deficits: 1 in the no-cooling VF group, 1 in the no-cooling no-VF group, and 2 in the cooling VF group.

Circulation. 2007;115(24):3064-70.

Cold IVF

TABLE 3. Echocardiographic Measurements

	Baseline	1 Hour After Infusion	*P*
EF, %	34.1±18.6	39.6±20.6	0.09
E/E′	9.1±6	7.4±3.4	0.11
Pulmonary artery pressure, mm Hg	36.2±15	34.0±14	0.74
Central venous pressure, mm Hg	8.9±5.9	8.4±5.4	0.7

- 2-3L of Ringers or Saline at 4C decreases body temperature
  - No effect on LVEF by echo
  - Improved hemodynamic indices

Kim. Circulation 2005;112:715

Induction: how to cool

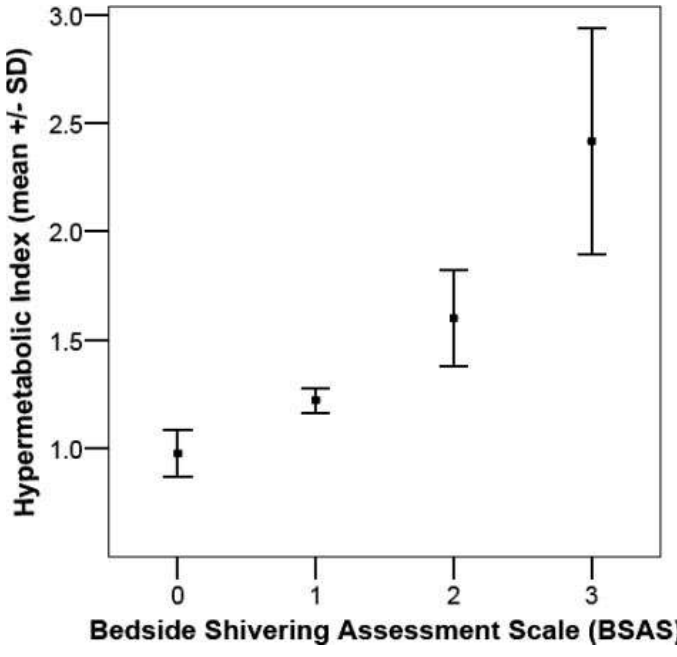
- **Monitor core temperature**
  - Bladder, esophagus, or central venous/pulmonary arterial
- **Cold fluid**
  - 30cc/kg LR or 0.9%NS over 30 minutes
    - 2-2.5C temperature reduction
  - No adverse cardiovascular results
  - Rare to cause pulmonary edema
- **Ice packs and cooling mats**
  - Effective, but difficult to control rate of temperature change
  - Overcooling is dangerous

Induction: how to cool

- **Commercial cooling devices**
  - Servo mechanism varies temperature of circulating water or air (prevents overcooling)
  - External (surface cooling) systems
    - Hydrogel heat exchange pads
    - Cold water circulating through plastic “suit”
    - Cold water immersion – awaiting safety data
  - Invasive (catheter based) systems
    - Heat exchange catheter in SVC or IVC
    - Plastic or metalic heat-exchange catheter

Shivering

- Drives up systemic metabolic rate
  - Increased CO2 production
  - Increased O2 consumption
  - Major cardiac stressor
- Drives up cerebral oxygen consumption
  - Favors ischemia
- Uncomfortable



Score	Definition
0	**None:** no shivering noted on palpation of the masseter, neck, or chest wall
1	**Mild:** shivering localized to the neck and/or thorax only
2	**Moderate:** shivering involves gross movement of the upper extremities (in addition to neck and thorax)
3	**Severe:** shivering involves gross movements of the trunk and upper and lower extremities

Stroke. 2008 Dec;39(12):3242-7.

Management of shivering

- Neuromuscular blockade
  - Must give sedation, first!!
  - Vecuronium bolus 0.1mg/kg prn for shivering
- Meperidine or fentanyl
- Propofol
- Alpha blockade
  - Dexmedetomidine infusion or clonidine
- Scheduled acetaminophen
- Focal counterwarming
- Magnesium infusion (serum level 3-4mg/dl)


I May Be Shakin' … But It Ain't A Seizure!

De-cooling

- 0.25 – 0.5 degree per hour
- Risk of
- hypoglycemia
- Hyperkalemia
- Hypotension

- Then after Prevent fever

De-cooling

- Vasodilation causes hypotension
  - May require several liters IVF
- More shivering during this phase
- Inflammation increases at higher temperature
  - “post-resuscitation” syndrome
- Increased ICP
- Watch for hyperkalemia
  - Primarily problematic in renal failure
- SEIZURES

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

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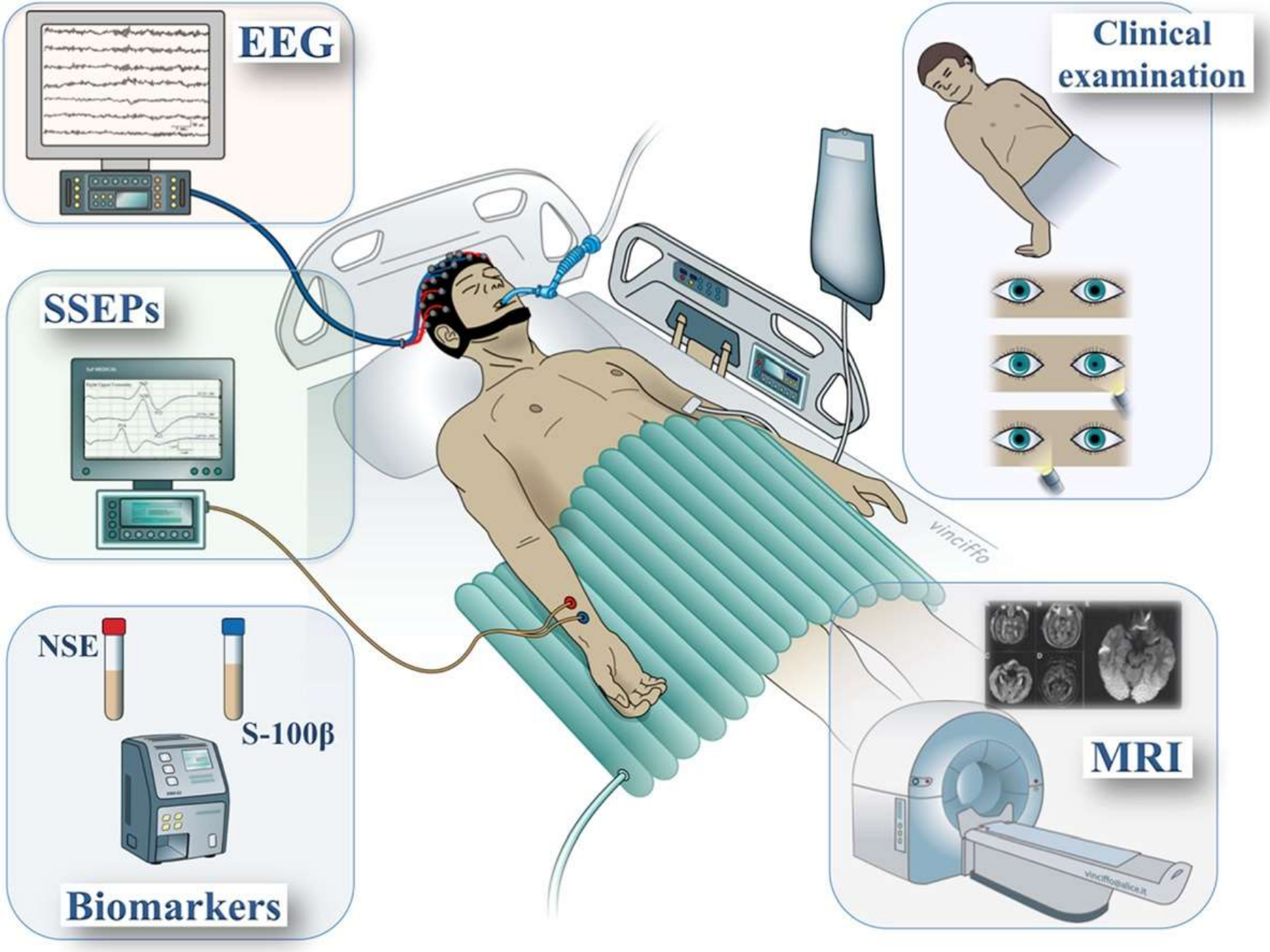
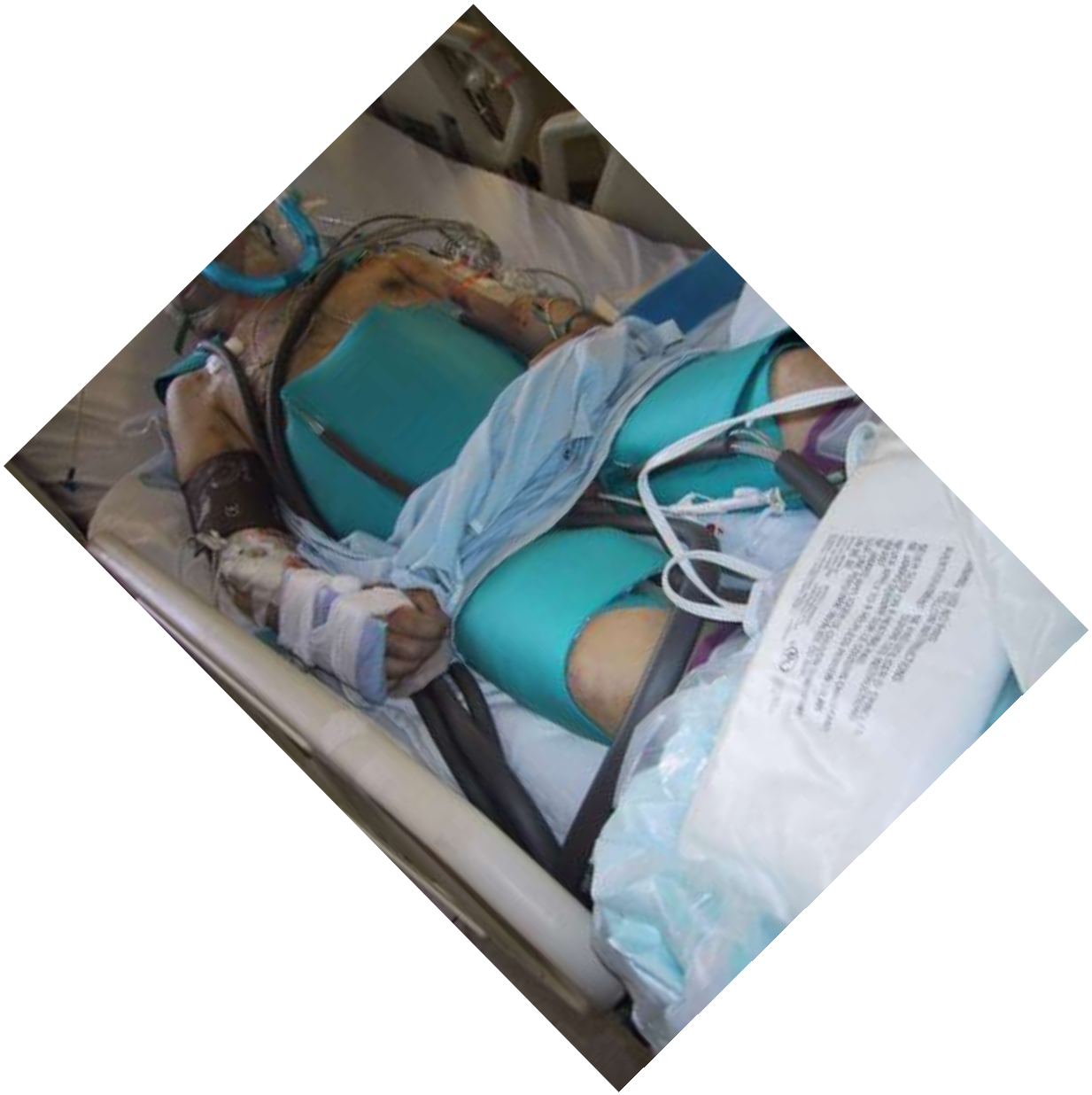
Regional Systems of Care for Out-of-Hospital Cardiac Arrest. A Policy Statement From the American Heart Association

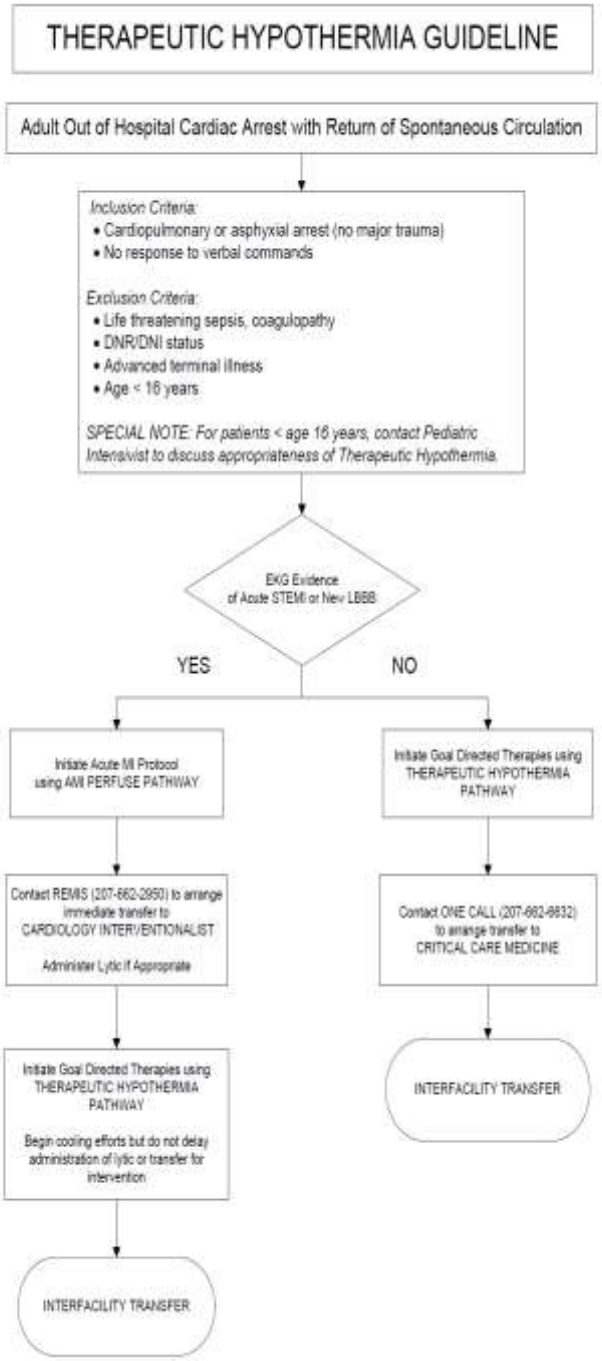
Graham Nichol, Tom P. Aufderheide, Brian Eigel, Robert W. Neumar, Keith G. Lurie, Vincent J. Bufalino, Clifton W. Callaway, Venugopal Menon, Robert R. Bass, Benjamin S. Abella, Michael Sayre, Cynthia M. Dougherty, Edward M. Racht, Monica E. Kleinman, Robert E. O'Connor, John P. Reilly, Eric W. Ossmann, Eric Peterson and on behalf of the American Heart Association Emergency Cardiovascular Care Committee; Council on Arteriosclerosis, Thrombosis, and Vascular Biology; Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation; Council on Cardiovascular Nursi

Circulation published online Jan 14, 2010;
DOI: 10.1161/CIR.0b013e3181cdb7db


EEG
Clinical examination
SSEPs
NSE
S-100β
Biomarkers
MRI



MaineHealth CA Guideline



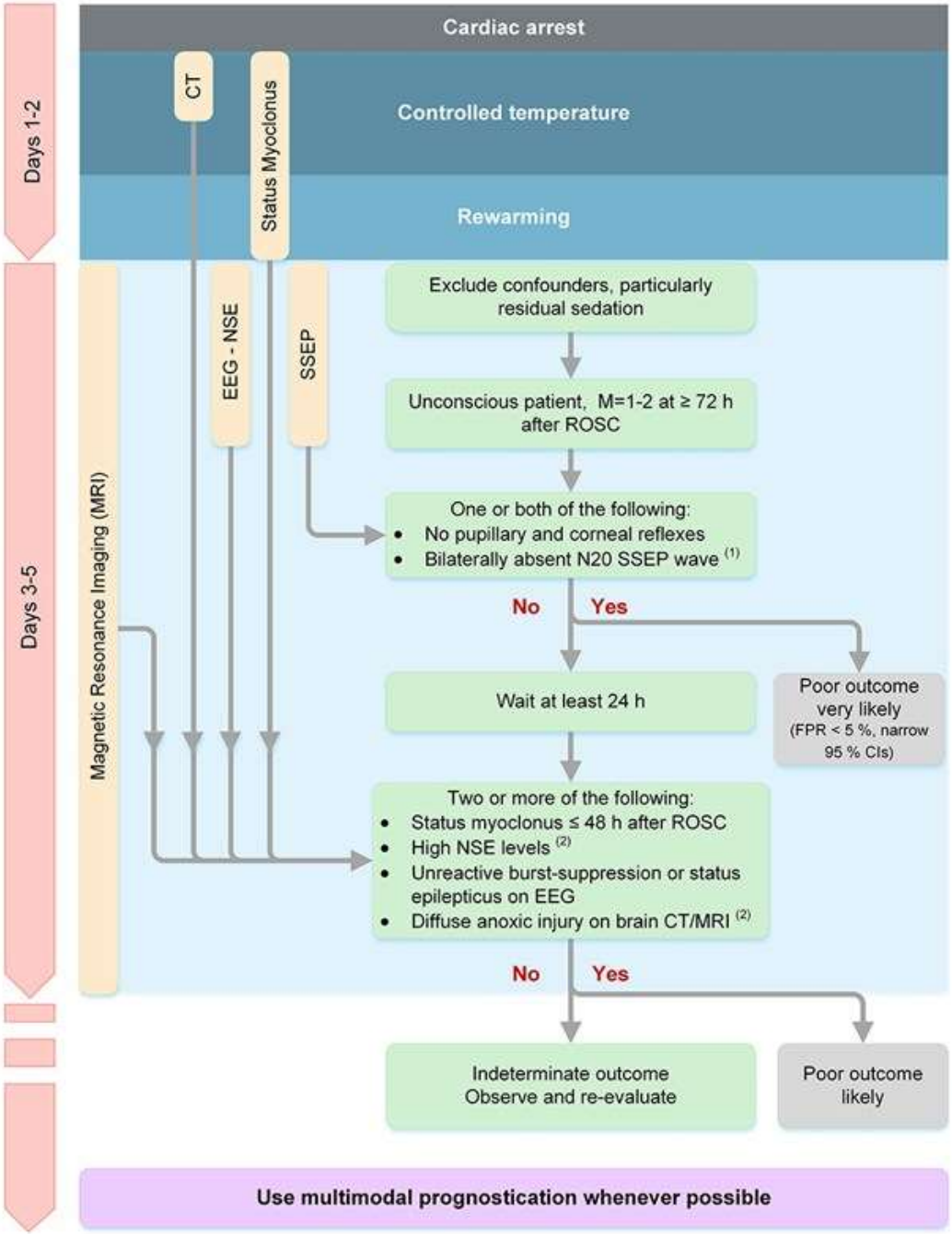
Emergency Department Order Set
Therapeutic Hypothermia for Adult Post Cardiac Arrest

ALLERGIES ☐ **None** ☐ **Yes, Drug/Reaction**_____

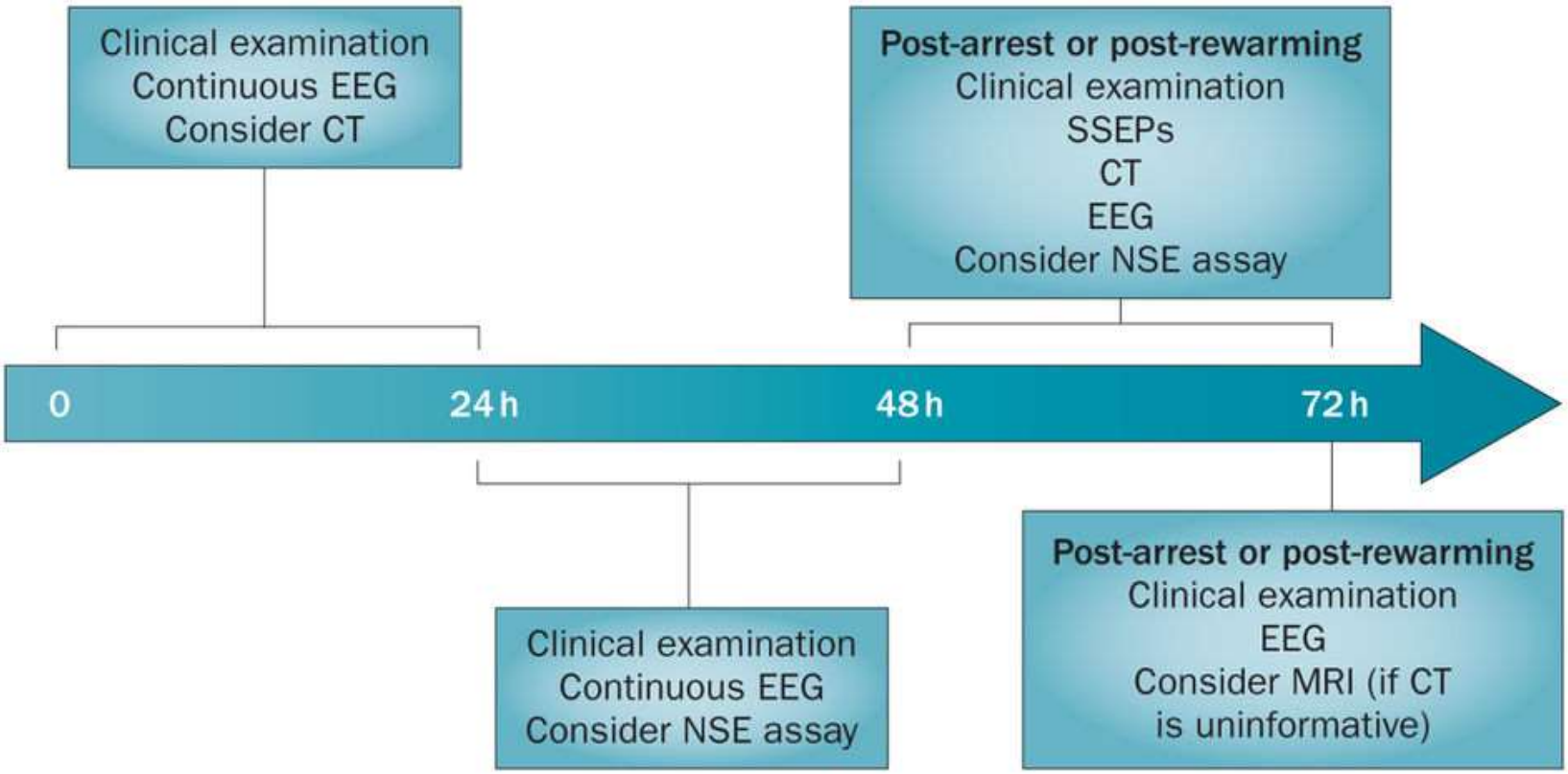
EMERGENCY DEPARTMENT TIMES and INFO TO DOCUMENT

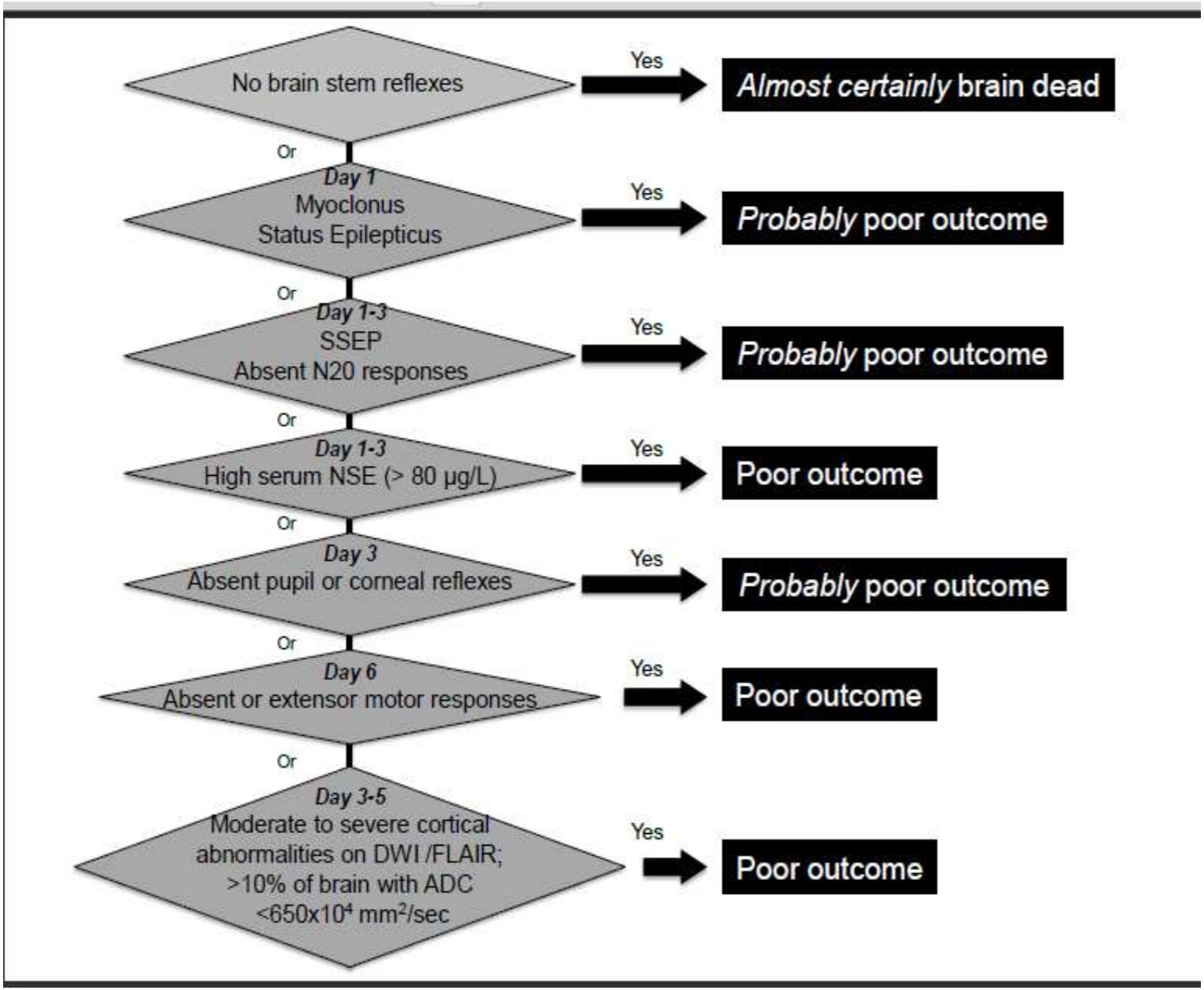
Time of Cardiac Arrest (if unknown, enter time EMS activation and check here ☐)		Total duration of re-arrest(s) after ROSC	
Witnessed arrest Yes/No		Time lytic administered	
Bystander CPR Yes/No		**Initial Core Temperature**	
Time of First CPR		**Time Hypothermia Initiated**	
Initial Rhythm		Time REMIS Called	
Time of Return of Spontaneous Circulation		Time Transport Called	
Time of ED Arrival		Time of Transport Arrival	
Time of 1st EKG		Time of ED Departure	
STEMI Diagnostic EKG Yes/No		**Core Temperature at ED Departure**	

	Insert ✔marks as needed to order tests/treatments not already preselected	Order Noted (Date/Time) (Initial)	
Initial Evaluation			
✔	Vital Signs: BP Pulse RR O_2 Sat Weight (kg)		
✔	Measure Rectal Temperature (record above)		
✔	Continuous Cardiac Monitoring		
✔	Baseline Neurologic Exam (please assess **prior** to paralysis): Following Commands? YES NO; Motor Function (circle) Spontaneous purposeful localizes withdraws flexion extension no motor response; Symmetrical? YES NO; Seizure Activity? YES NO Describe: ; Pupils mm R mm L React to light? YES NO N/A (atropine); Corneal reflex Present Absent		
Laboratory Tests			
✔	CBC and differential		
✔	Comprehensive Metabolic Profile, Mg^{++}, Phosphorus		
✔	INR/PTT		
✔	Lactate		
✔	CK, CK-MB, Troponin		
✔	ABG		
✔	Urinalysis (Cath)		
	Urine B-HCG in women of childbearing age		
Other Tests			
✔	12 Lead ECG Indication: Post Cardiac Arrest		
✔	Portable AP Chest X-Ray Indication: Intubation, Post OHCA		
	Head CT, non contrast Indication: Post cardiac arrest		
Airway/Breathing Order Set			
✔	Ventilator Settings: CMV mode, target pCO_2 35-45 mm Hg		
✔	FIO_2: **Start at FiO2 0.5** and titrate up as needed to SpO2 greater than 95%		
	PEEP: mg Hg (minimum 5 mm Hg)		
	Rate: breaths/minute (initial 12 breaths/min – higher if severe acidosis)		
	Tidal Volume: ml (recommend 6 to 8 ml/kg of Ideal Body Weight)		
	Place $ETCO_2$ monitor and titrate vent rate to maintain $ETCO_2$ 35-45 mm Hg		
Nursing Procedures			
✔	Expose patient		
	Insert rectal esophageal bladder (circle one) probe for temperature monitoring		
✔	Insert two peripheral IVs, 20 gauge or larger		
✔	Insert Nasogastric or Orogastric tube, intermittent suction		
✔	Insert Foley catheter		
✔	Elevate HOB to 30°		



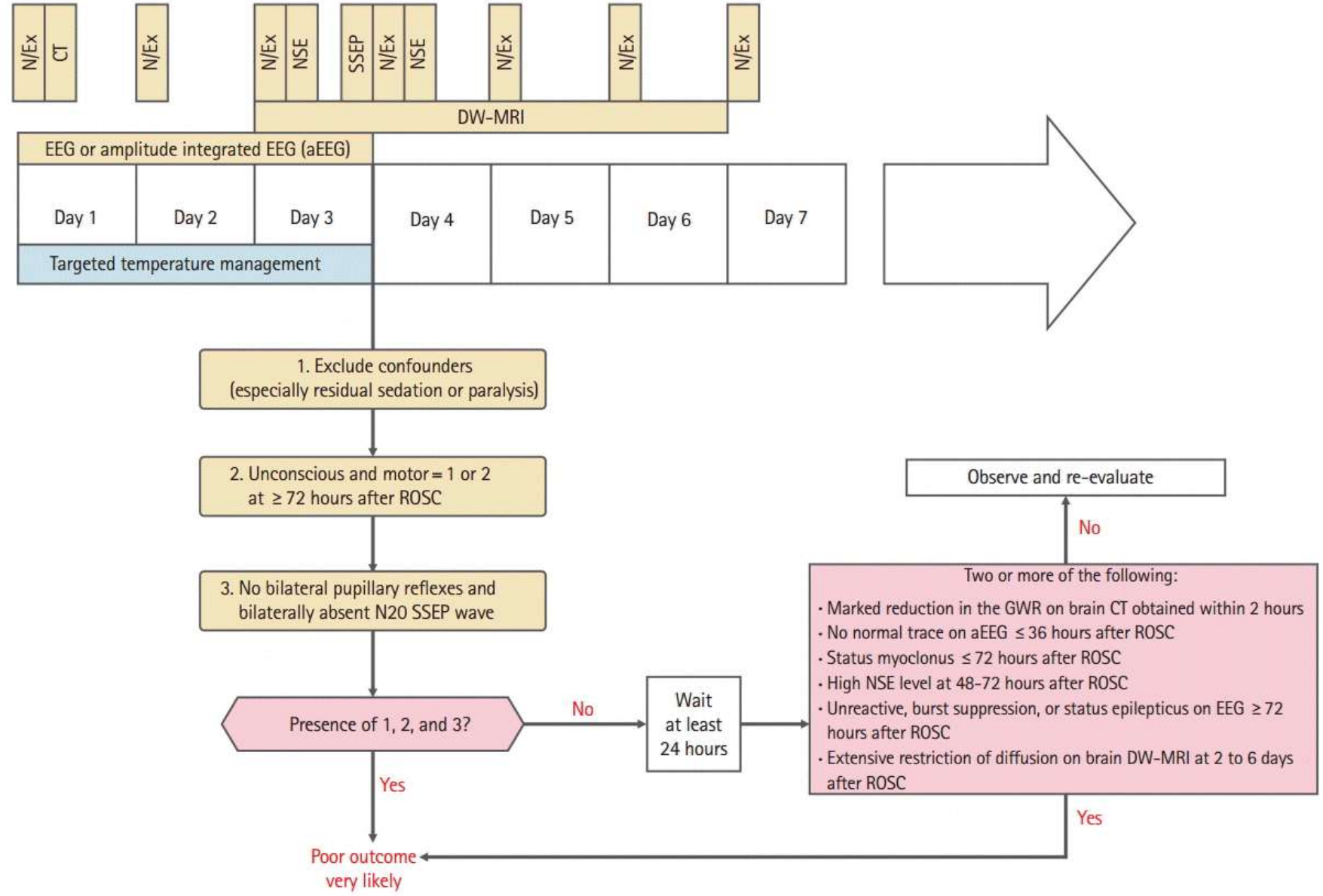
(1) At ≥ 24 h after ROSC in patients not treated with targeted temperature
(2) See text for details.

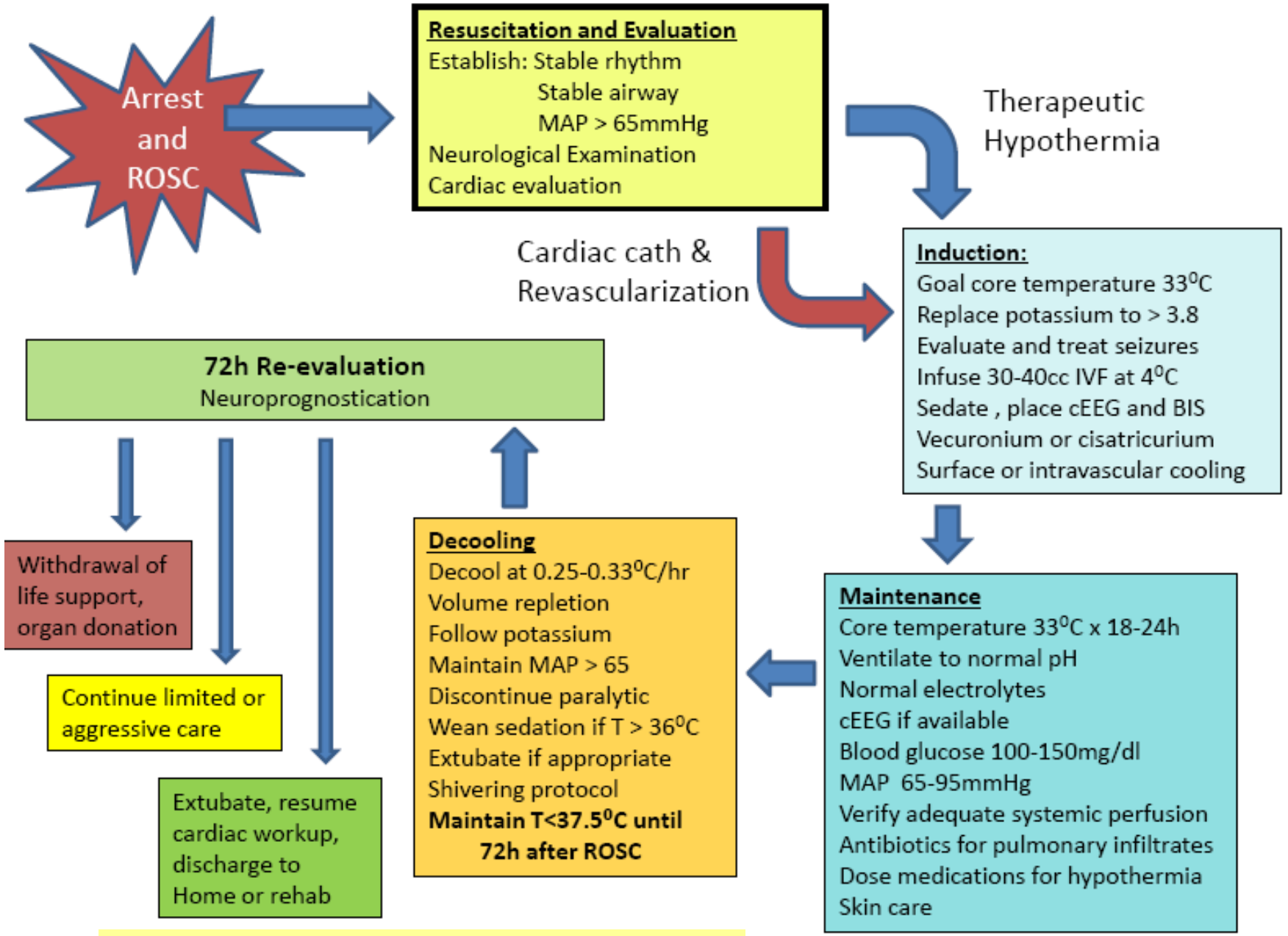

Clinical examination
Continuous EEG
Consider CT
Post-arrest or post-rewarming
Clinical examination
SSEPs
CT
EEG
Consider NSE assay
0
24 h
48 h
72 h
Clinical examination
Continuous EEG
Consider NSE assay
Post-arrest or post-rewarming
Clinical examination
EEG
Consider MRI (if CT
is uninformative)



From the 8th New York Symposium on Neurocritical Care, 2011

Recommended timing of prognostic tests





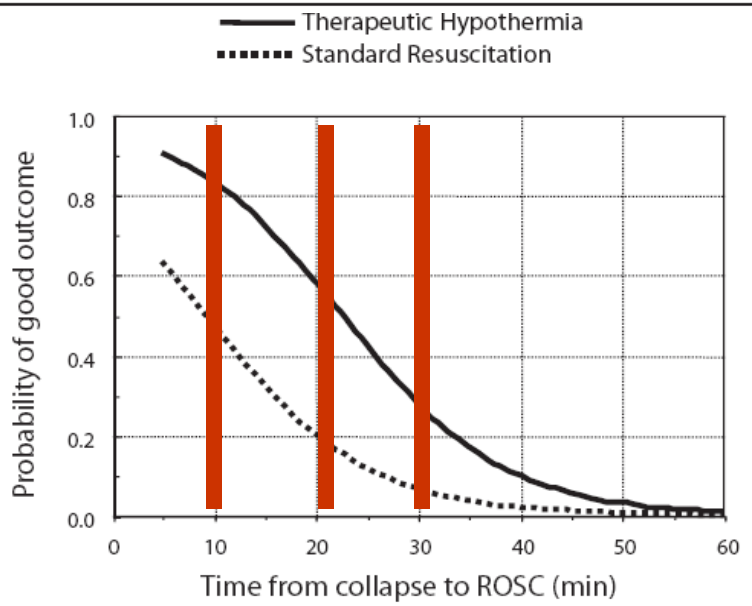
Crit Care Med 2009;37 (Suppl):S211-S222.

Lausanne

- 55 VT/VF OHCA treated with TH 2002-2004
- Compared to historical controls 1999-02
- Similar DT, severity of illness
- CPC 1-2: 56% vs. 26% pre-TH

Table 4. Outcome, at hospital discharge, of comatose patients with out-of-hospital cardiac arrest (initial rhythm: ventricular fibrillation)

	Outcome				
Treatment Group	CPC 1 Total Recovery	CPC 2 Moderate Disability	CPC 3 Severe Disability	CPC 4 Vegetative State	CPC 5 Death
Therapeutic hypothermia	18/43 (41.9)	6/43 (13.9)	2/43 (4.7)	0/43 (0)	17/43 (39.5)
Standard resuscitation	6/43 (14.0)	5/43 (11.6)	8/43 (18.6)	0/43 (0)	24/43 (55.8)



Effect of the implementation of a therapeutic hypothermia protocol on neurological outcome after out-of-hospital VF/VT arrest

-Crit Care Med 2006;34:1865

Summary

- Rapid consideration and early initiation of therapeutic hypothermia
- Aggressive hemodynamic support including PCI when appropriate
- Suppression of shivering
- Treatment in an experienced center with appropriate resources

Summary

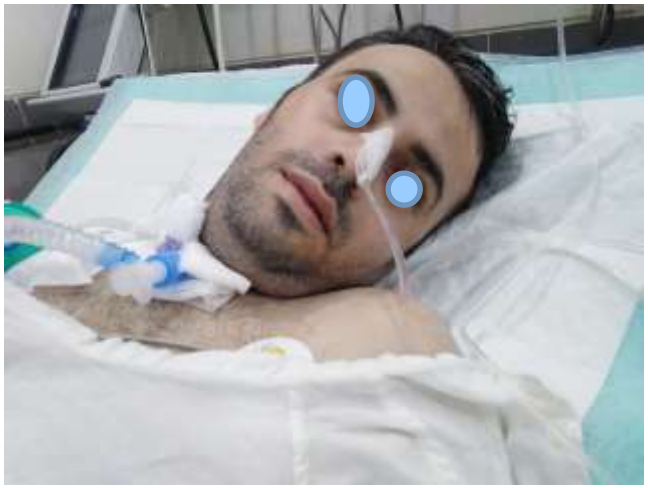
- therapeutic hypothermia, hemodynamic support, and quality ICU care, can expect better OHCA outcomes
  - All rhythms: 30-40% good neurological outcome
  - VT/VT: 50-65% good neurological outcome
  - PEA/Asystole 13-25% good neurological outcome

Take home messages

- Strong evidence that mild hypothermia is neuro-protective after return of spontaneous circulation.
- Fever is detrimental post resuscitation
- Hypothermia is underutilized so far but should be included in post resuscitaion care of cardiac arrest victims

كل نفس ذائقة الموت

سورة آل عمران - آية ١٨٥



Thanks for your attention