

THERAPEUTIC HYPOTHERMIA (TH) AFTER CARDIAC ARREST

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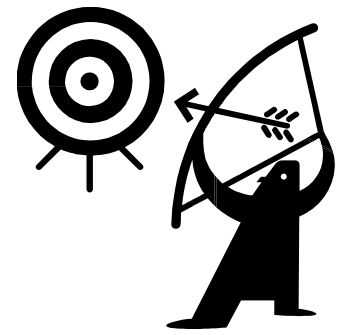


Wright State University

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NUR 7201- Summer 2013

OBJECTIVES



- Analyze the goal of TH
- Examine the uses of therapeutic hypothermia
- Evaluate indications and contraindications of therapeutic hypothermia
- Synthesize management techniques during therapeutic hypothermia
- Analyze the effectiveness of therapeutic hypothermia in patients after cardiac arrest
- Demonstrate efficacy of therapeutic hypothermia by reviewing literature
- Incorporate learned information on therapeutic hypothermia in clinical practice



THE HISTORY BEHIND HYPOTHERMIA



THE HISTORY BEHIND HYPOTHERMIA

- The actual use of hypothermia to benefit patient survival goes back relatively far in history
- In 1814, Baron Larrey, a French surgeon in the service of Napoleon's army recognized benefits
- He observed improved survival of injured soldiers left in the snow compared with those treated with warm blankets and heated drinks
- Hippocrates advocated packing bleeding patients in snow



THE HISTORY BEHIND HYPOTHERMIA

- The first reported use of induced hypothermia was in the setting of cancer treatment. A decade later, the induction of hypothermia in the setting of cardiac surgery was done with the goal of cerebral protection.
- Two other studies using hypothermia as therapy for cardiac arrest were published as well. Both these early cardiac arrest studies used moderate hypothermia of 30 to 34°C in patients after resuscitation from cardiac arrest.



THE HISTORY BEHIND HYPOTHERMIA

- During the 1960s and 1970s, the field of induced hypothermia lay relatively dormant for reasons that remain unclear
- It was understood from military combat experience that medical care for penetrating trauma was often delayed for practical reasons
- Suspended animation: Preservation of viability of the patient post cardiac arrest, which allows time for transport and repair and is followed by delayed resuscitation, hopefully to survive without brain damage



SO HOW AND WHY DOES THERAPEUTIC HYPOTHERMIA WORK?



REPERFUSION INJURY

- Brain Injury
 - Small oxygen store
 - Oxygen is depleted within 20 seconds
 - Glucose and ATP levels are depleted within 5 minutes if return of blood is not achieved
 - Turns to anaerobic metabolism to sustain function



REPERFUSION INJURY

- Initiates a chemical process that lead to inflammation and continued injury in the brain
- Injury is related to release of neurotransmitters (dopamine, glutamate), free radicals, nitric oxide, catecholamine's, cytokines and calcium shifts. This leads to mitochondrial damage and cell death.
- May last 24-48 hours



REPERFUSION INJURY

- Reoxygenation promotes high concentration of reactive oxygen species (free radicals specifically involving O₂)
- Reactive oxygen species combine with other inflammatory processes further exacerbate endothelial dysfunction, vasomotor dysfunction, edema, tissue-level hypoxia despite adequate arterial oxygenation and subsequent neurological damage



GOAL OF THERAPEUTIC HYPOTHERMIA

- To lower the brain temperature to 32 to 34 degree C during the first few hours after cardiac arrest
- Temper the post-cardiac arrest syndrome inflammatory cascade
- Prevent neurological injury
- Decrease the risk of death associated with cardiac arrest



THE MECHANISM OF NEUROPROTECTION

- For every 1 degree C decrease in brain temperature cerebral metabolic rate is decreased by 6-7%
- Decreases oxygen consumption
- Protects ATP stores necessary for energy provision
- Decreases disruption in blood brain barrier
- Decreases cerebral blood volume
- Decreases intracranial pressure
- Thereby improving the oxygen supply-and-demand mismatch

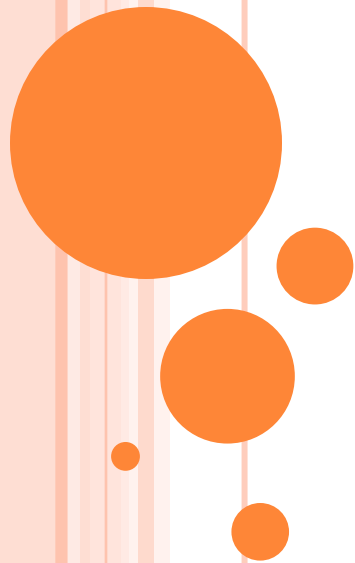


THE MECHANISM OF NEUROPROTECTION

- Suppresses many of the chemical reactions such as free radical production, excitatory neurotransmitters especially glutamate and dopamine and calcium shifts
- Prevents mitochondrial damage
- Aborts activated programmed cell death pathways (apoptosis)



THERAPEUTIC HYPOTHERMIA INDICATIONS & CONTRAINDICATIONS

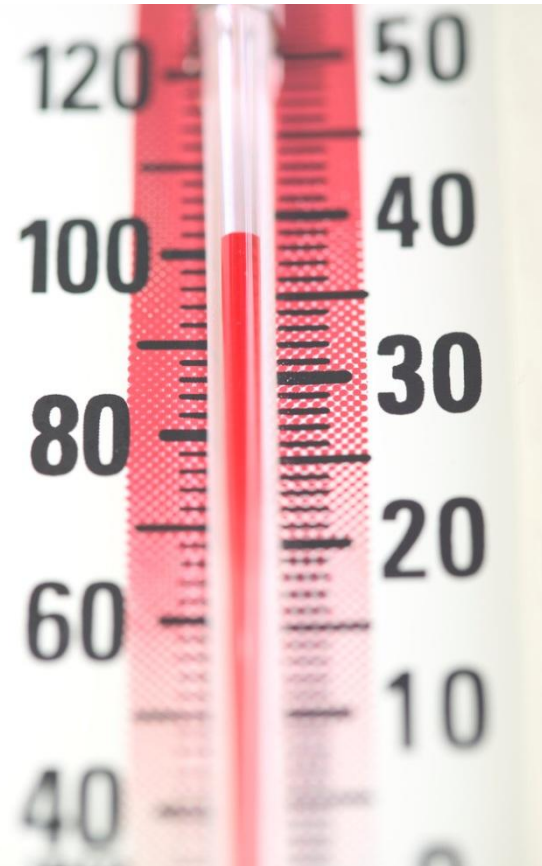


THERAPEUTIC HYPOTHERMIA USES

- Cardiac Arrest
- Myocardial Infarction
- Stroke

Additional Applications

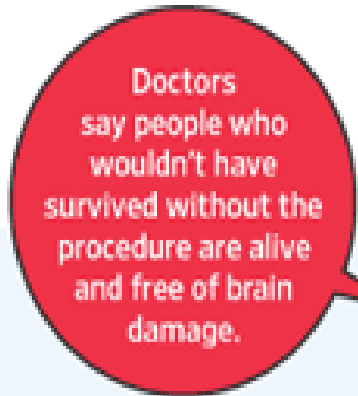
- Traumatic Brain Injury
- Neurogenic Fever
- Acute Liver Failure
- Aortic Arch Repair
- Cardiac Bypass Surgery



CARDIAC ARREST

- Cardiac arrest leads to at least 300,000 deaths each year in the United States
- Major complication: lack of perfusion
- Lack of cardiac output= O_2 supply cannot meet O_2 demand
- Most common causes of death from cardiac arrest outside of hospital is from neurological injury





About 300,000 Americans suffer cardiac arrest outside of a hospital each year.

CPR is attempted in about 40% of cases.

Normally, less than half of those survive.

Therapeutic hypothermia is an attempt to increase those odds.

After about 24 hours rewarming begins.

Deaths

Cold Comfort for Heart Cases

Improving the chances of survival

HOW IT WORKS:



A.



B.



C.

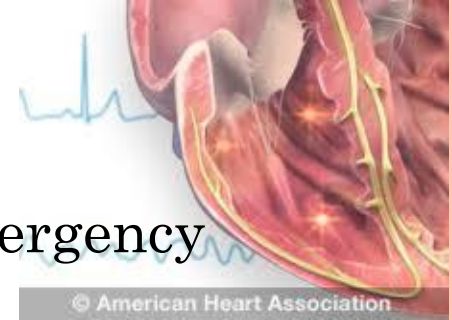
Cold prevents damage to the brain that can result from blood flow restarting.



The body is rewarmed in about 8 hours.

INDICATIONS

AHA Cardiopulmonary Resuscitation and Emergency Cardiovascular Care (2010)



- Comatose adult patients with return of spontaneous circulation (ROSC) after out-of-hospital VF cardiac arrest should be cooled to 32°C–34°C (89.6°F–93.2°F) for 12 to 24 h (Class I; Level of Evidence: 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; Level of Evidence: B).
- Active rewarming should be avoided in comatose patients who spontaneously develop a mild degree of hypothermia (>32°C [89.6°F]) after resuscitation from cardiac arrest during the first 48 h after ROSC (Class III; Level of Evidence: C).



OTHER INDICATIONS

- Witnessed out-of-hospital cardiac arrest
- Initial rhythm of ventricular fibrillation or pulseless ventricular tachycardia
- Successfully resuscitated
- Hemodynamically stable
- Comatose GCS < 8
- Lack of verbal response



CONTRAINDICATIONS

- DNR
- Active uncontrolled bleeding
- Hemorrhagic stroke
- Glasgow Coma Scale >8
- Cardiac Arrest due to drug overdose/trauma
- Preexisting hypothermia below 30 deg C
- Pregnant patients
- Terminally ill
- Inherited blood coagulation disorders



RELATIVE CONTRAINDICATIONS

- Baseline coagulopathy
- Severe hypotension (MAP < 60 mm Hg) that is not correctable by fluid infusion, vasopressors, or invasive hemodynamic support
- Patients who were comatose before the cardiac arrest
- Patients who are terminally ill or for whom intensive care does not seem to be appropriate

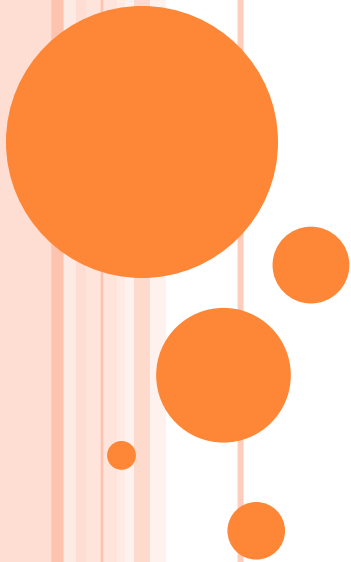


PCI + TH FOR ACS

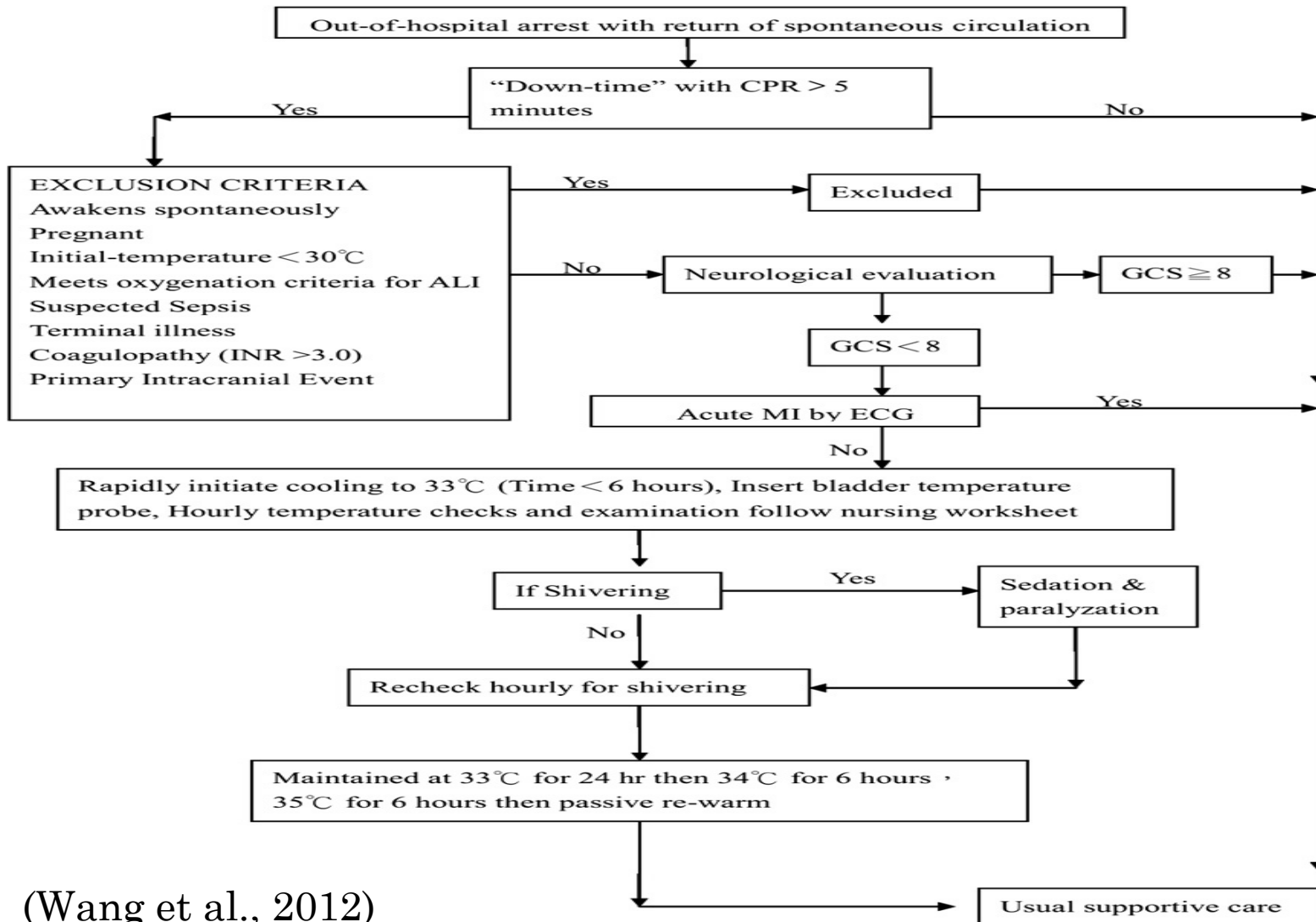
- AHA & ILCOR (2010): CA patients to undergo immediate coronary angiography if STEMI or high suspicion of ACS
- 25% mortality with PCI + TH vs. 66% mortality with only PCI (Knafelj et al., 2007)
- F/u in 6 months: 78% of PCI + TH had good neuro outcomes vs. only 6% with only PCI (Knafelj et al., 2007)
- Mortality decreased from 72% to 44% with introduction of PCI + TH protocol, and >90% of survivors were neurologically intact (Sunde et al., 2007)



SCREENING FOR THERAPEUTIC HYPOTHERMIA



SCREENING FOR TH

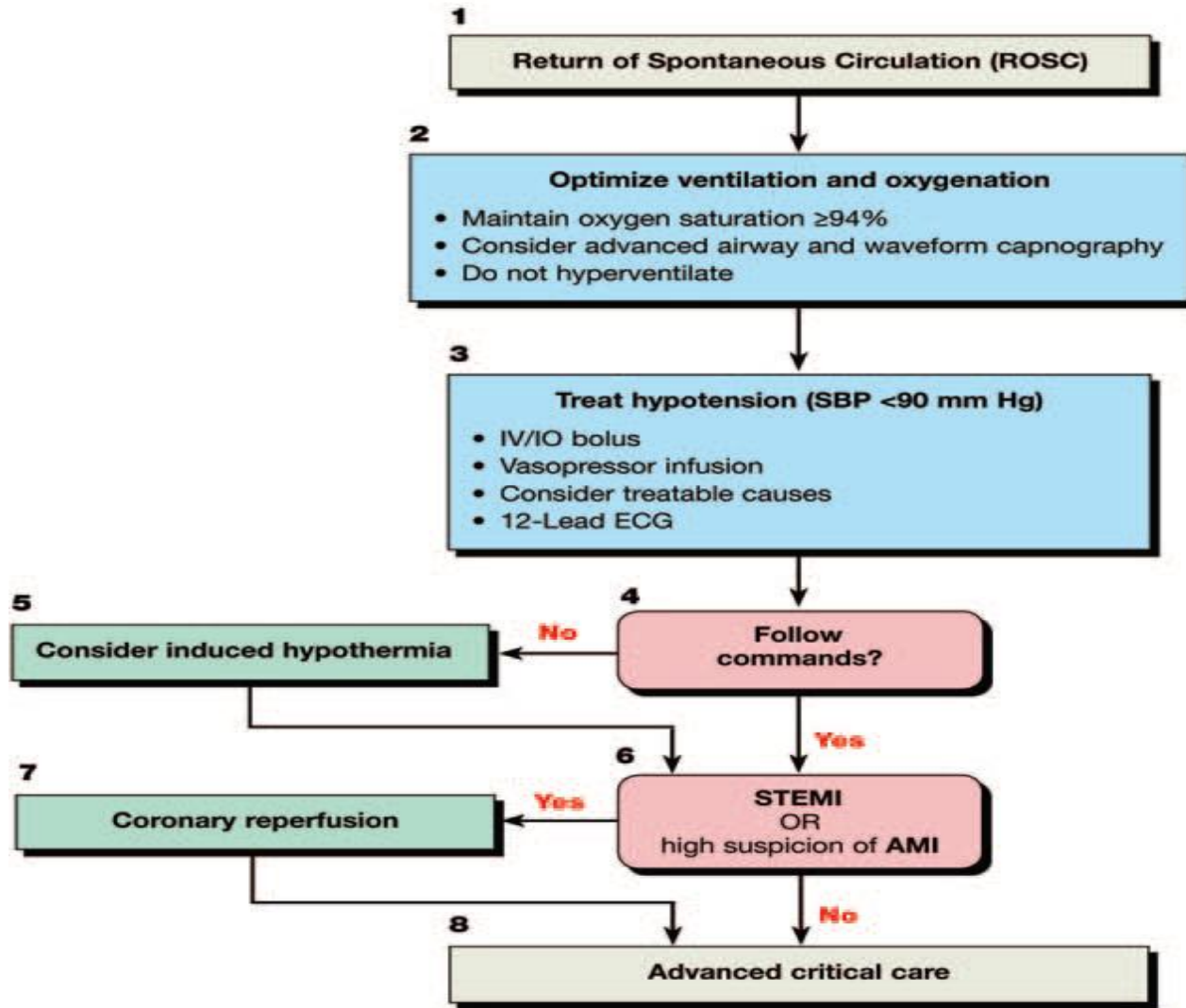


(Wang et al., 2012)



SCREENING FOR TH

Adult Immediate Post-Cardiac Arrest Care



Doses/Details

Ventilation/Oxygenation
 Avoid excessive ventilation. Start at 10-12 breaths/min and titrate to target PETCO₂ of 35-40 mm Hg. When feasible, titrate FIO₂ to minimum necessary to achieve SpO₂ ≥94%.

IV Bolus
 1-2 L normal saline or lactated Ringer's. If inducing hypothermia, may use 4°C fluid.

Epinephrine IV Infusion:
 0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)

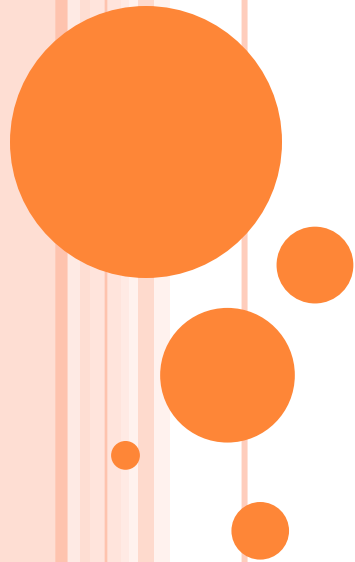
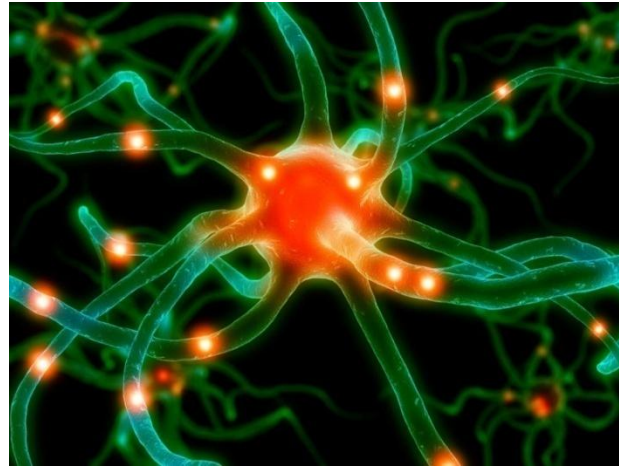
Dopamine IV Infusion:
 5-10 mcg/kg per minute

Norepinephrine IV Infusion:
 0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)

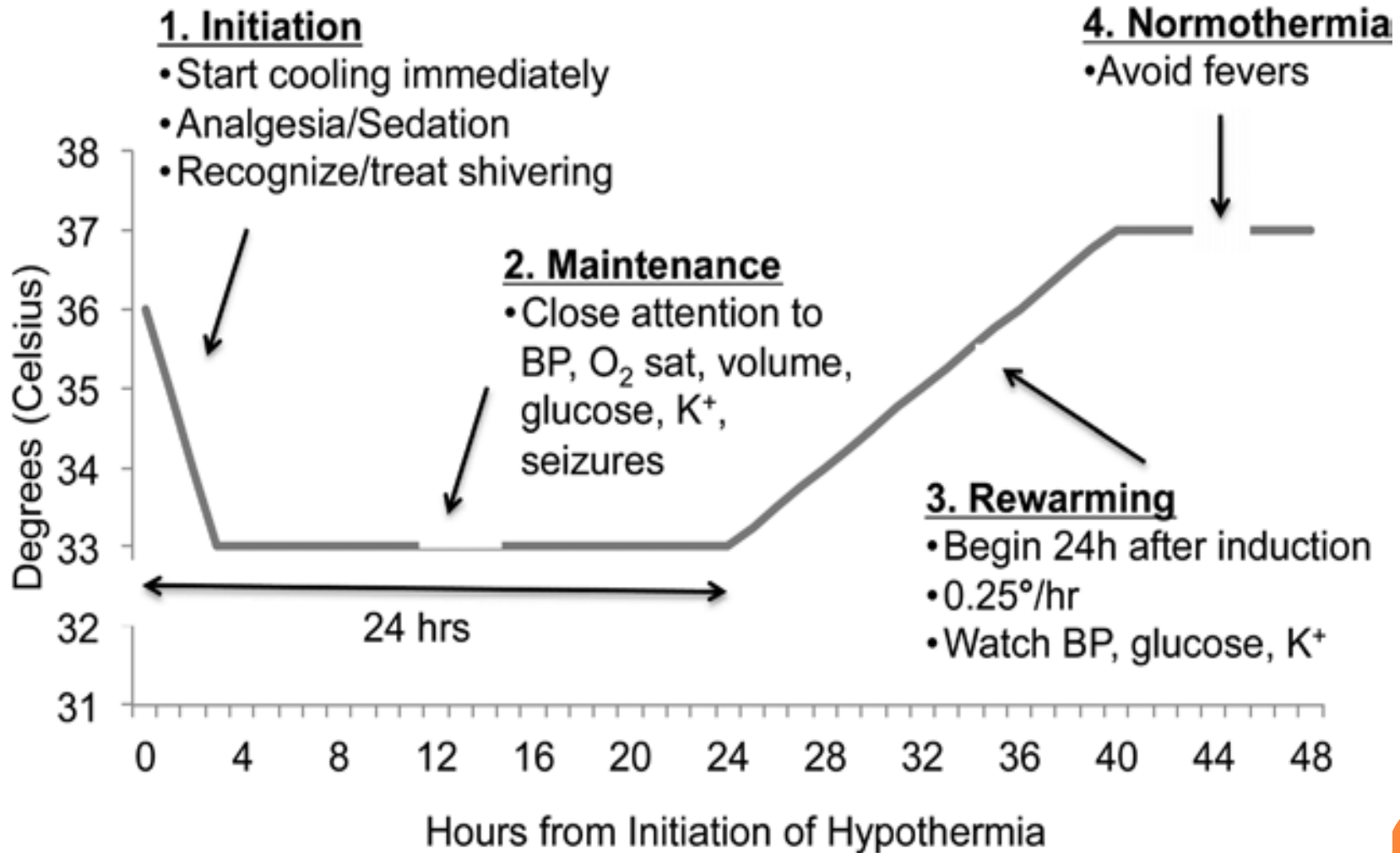
Reversible Causes

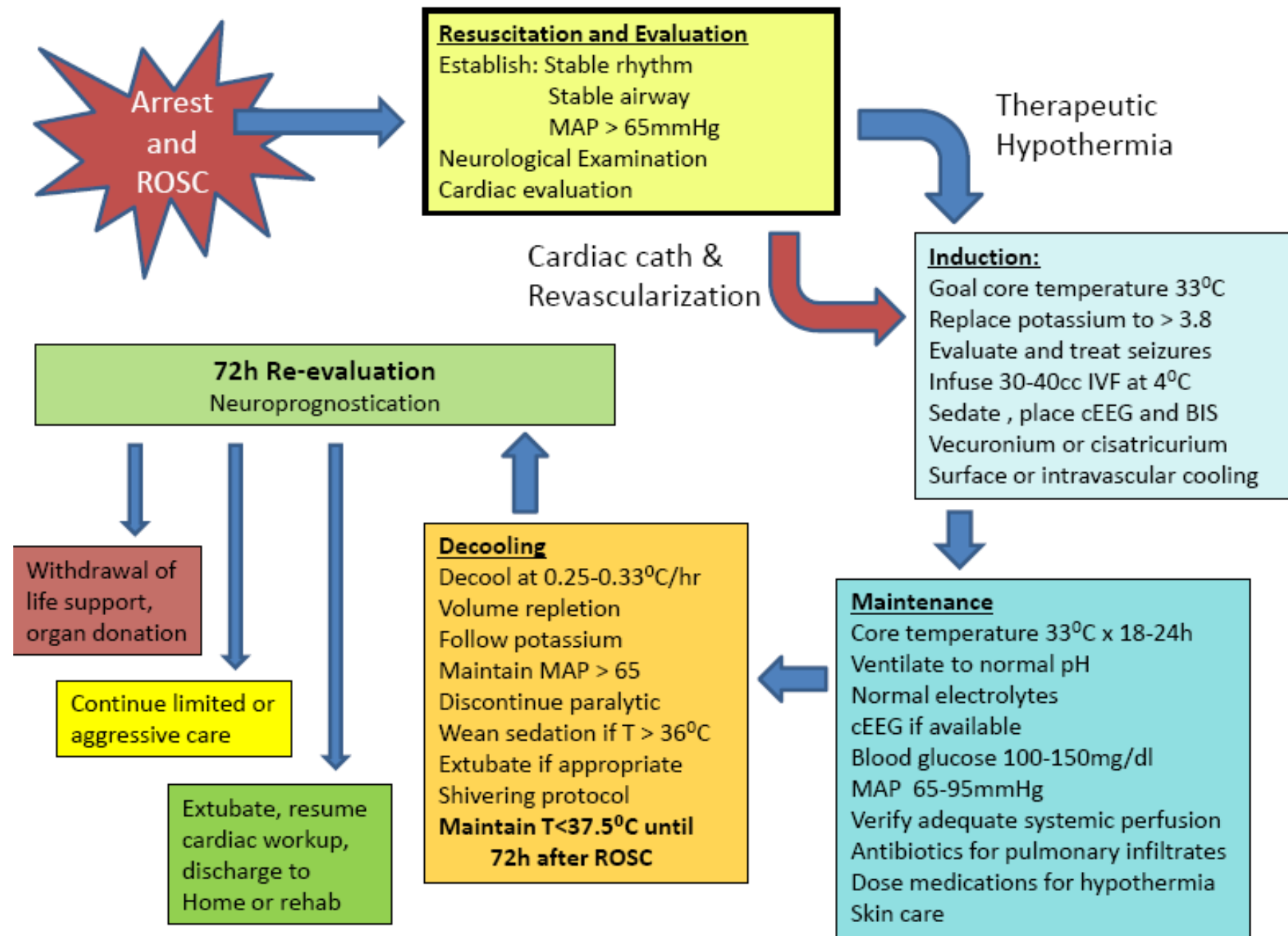
- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo-/hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

INITIATION OF THERAPEUTIC HYPOTHERMIA



INITIATION OF TH: 4 STAGES





STAGE I- INITIATION

- Achieve temperature between 32-34 C within 6 hours post cardiac arrest
- Goal temperature should be reached within 3 hours of initiation of hypothermia
- Evidence suggests maintaining TH for 12 to 24 hours
- Treatment instituted sooner than 6 hours is associated with improved neurologic outcomes



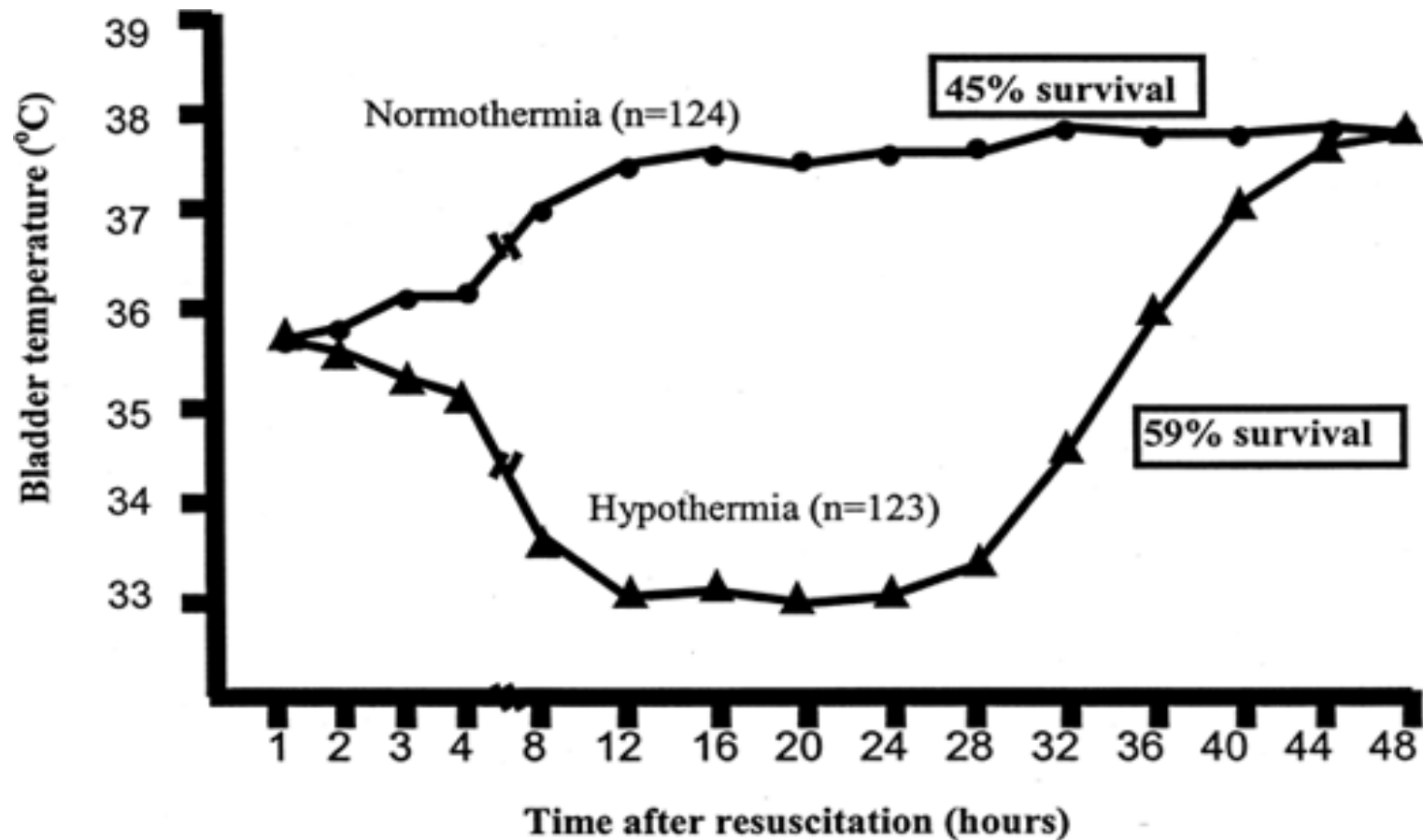
EVIDENCE EARLY VS. LATE COOLING

- If time lapsed between ROSC and cooling >2.5 hrs, patients 63% less likely to survive to discharge than <1.5 hours
- There is a 20% increase in mortality for every hour of delay in the initiation of TH

(Mooney et al., 2011)



TIME COMPARED TO SURVIVAL



Source: Hall JB, Schmidt GA, Wood LDH: *Principles of Critical Care*, 3rd Edition: <http://www.accessmedicine.com>

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STAGE I- INITIATION

- Monitor vitals at baseline and q15 x 4, q30 x 2, then hourly
- Neuro checks every hour
- Baseline skin assessment
- Labs obtained at baseline and every 4-6 hours (BMP, Coag profile)
- Baseline blood cultures, urinalysis, and chest x-ray
- Baseline non-contrast head CT to r/o ICH



STAGE I- INITIATION

- All patients require protection of their airway and mechanical ventilation
- Blood gases should be measured at least every 4 hours to allow for adjustment of respiratory therapy
- Sedation, analgesia, and/or paralysis should be initiated to prevent shivering and minimize discomfort
- Hemodynamic support with IV fluids, inotropic agents or vasopressors
- Acute MI patients- revascularization should be considered
- Hemodialysis may be necessary for patients with renal injury
- Blood glucose monitoring and management
- Antibiotic therapy for infection



METHODS FOR IMPLEMENTING TH

Core cooling

○ IV infusion of cold fluids

- 30 ml/kg of cold (4 C/39 F) isotonic saline or Lactated Ringer's solution
- May use pressure bag to increase the rate of infusion
- 1L over 15 minutes decreases temp by 1C. 70kg person will receive 2.1L
- Reduces the core temperature by >2 C per hour

○ Endovascular cooling catheters

- Metal coated tube or balloon filled with cold saline lowers the temperature of the patient's blood

- <http://www.youtube.com/watch?v=xJgwhrU2Nsg>



METHODS FOR IMPLEMENTING TH

○ **Surface cooling methods**

- Ice packs around head, neck, torso and limbs
- Water circulating cooling blankets
- Cooling vests
- Cool caps
- Leg wraps
- Cold water immersion



COMBINATION REGIMEN- IV + SURFACE

- Infuse cold saline using pressure while simultaneously implementing surface cooling blankets above and below the patient and ice packs applied to the axillae, groin, and neck
- Thermostatically controlled devices provide the most precise minute-to-minute temperature regulation



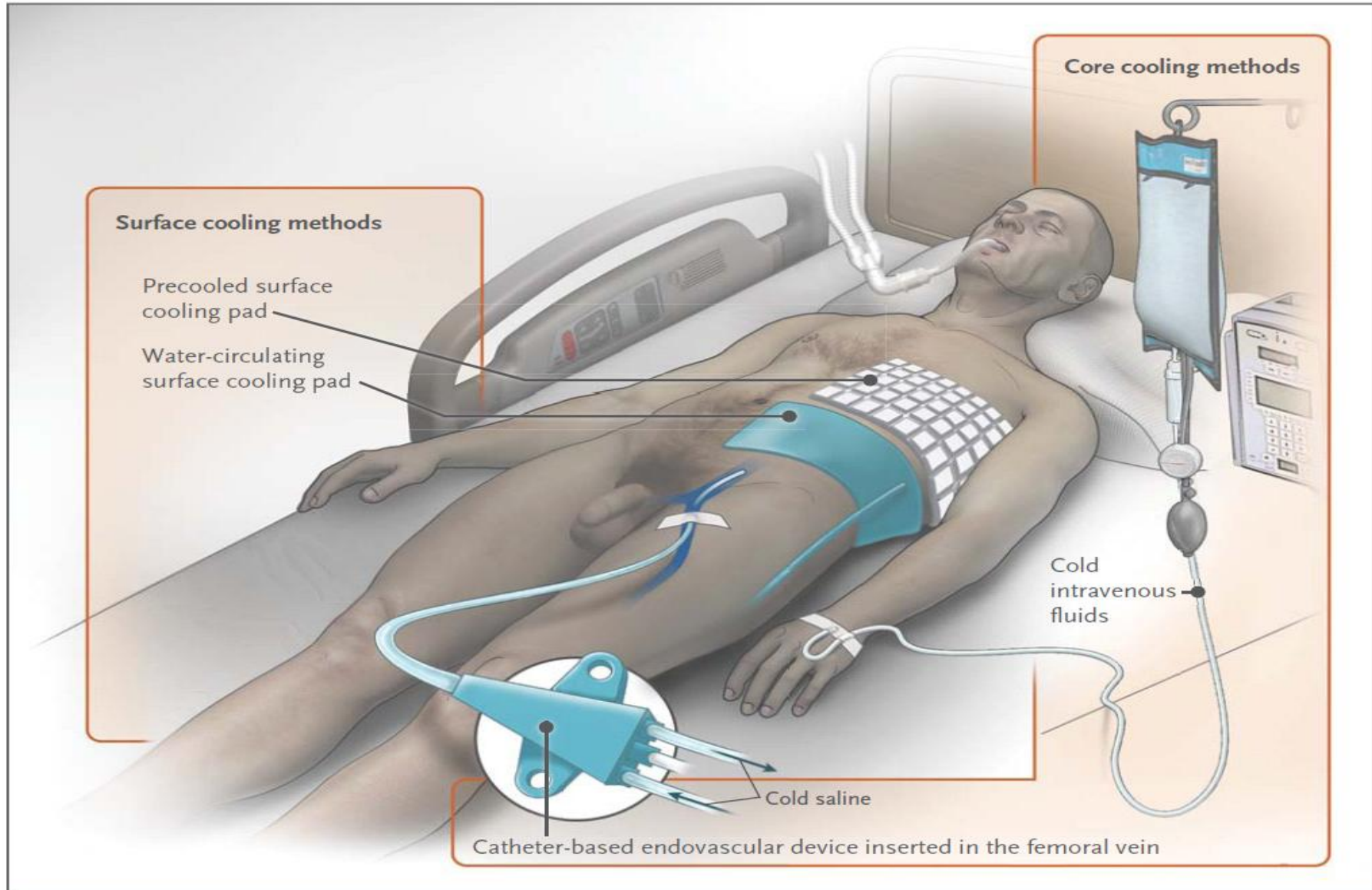


Figure 1. Cooling Methods Used in Clinical Practice. Surface cooling methods include the use of precooled (refrigerated) surface cooling pads and water-circulating surface cooling pads. Core cooling methods include the infusion of cold intravenous fluids and the use of catheter-based endovascular devices.

FACTORS AFFECTING THE RATE OF EXTERNAL COOLING

- Obesity
- Higher core body temperature at admission
- Early coronary angiography, delayed arrival to ICU
- Delay between collapse and the start of cooling
- Male sex and hemodialysis positively associated with cooling success
- Severe neurological injury and an inability to autoregulate body temp- achieve goal rapidly



STAGE II- MAINTENANCE

- During the maintenance stage temperature fluctuations should be minimized to 0.5 degree C.
- Core body temperature should be monitored continuously during TH
- Pulmonary artery catheters and central venous catheters are considered the gold standard for core temperature monitoring
- In practice, many patients admitted to the coronary intensive care unit after out-of-hospital cardiac arrest require pulmonary artery catheterization anyway for other indications



STAGE II- MAINTENANCE

- Surrogate methods- Esophageal, bladder, or rectal probes (in order of preference)
- Esophageal-most accurate surrogate method
- Esophageal monitoring is relatively noninvasive and reliable as long as the probe is placed about 45 cm from the nose and is not affected by the location to the trachea
- Bladder- not effective if urine output is less than 0.5ml/kg/hr
- Rectal- may lag behind by up to 1.5 C
- Ensure two methods of measuring temperature



STAGE II- PHYSIOLOGICAL CONSEQUENCES

- Complications can occur
 - Shivering
 - Hemodynamics: Heart rate, BP
 - Oxygenation/ventilation
 - Glucose control
 - Electrolyte imbalance- hypokalemia
 - Infection
 - Skin breakdown r/t peripheral vasoconstriction
 - Seizures



STAGE II-PHYSIOLOGIC CONSEQUENCES

Shivering

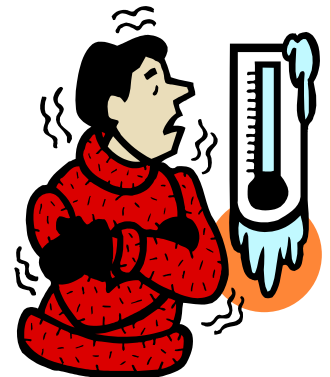
- Shivering is natural body response to hypothermia
- Shivering should be recognized early and treated aggressively because it raises body temperature, increases metabolic rate and oxygen consumption
- S/S of shivering
 - Decreased mixed venous O₂ saturation
 - Increased respiratory rate
 - Facial tensing
 - Static tracing on EKG
 - Palpation of muscle fasciculation of the face and chest
- Occurs mainly during induction phase (35 C-37 C) and less likely during maintenance and warming phases



MANAGEMENT OF SHIVERING

Sedative agents

- 1st choice Propofol- Bolus (optional) 0.3-0.5 mg/kg; infusion of 5-80 mcg/kg/min
- If ineffective add fentanyl bolus 1-2 mcg/kg or as a continuous infusion starting at 0.5-2.0 mcg/kg per hour
- 2nd Choice Midazolam 0.01-0.05 mg/kg; infusion 0.02-0.1 mg/kg/hr.
- MgSO₄ 4gm IV to raise shivering threshold.
- Titrate sedation to shivering suppression
- RASS -4 to -5



MANAGEMENT OF SHIVERING

Neuromuscular Blockade

- Cisatracurium (Nimbex) 150 mcg/kg boluses or maintenance dose 1-2 mcg/kg/min can suppress shivering, but can mask seizures (3-44% post CA patients)
- Rocuronium 0.5 mg/kg bolus and then continuous 8-12 mcg/kg/min
- May need continuous EEG monitoring for the safe use of neuromuscular blockade
- Seizures can be treated with valproic acid, phenytoin, midazolam, phenobarbital
- Medication clearance is decreased 10% for every 1 degree C below 37 degree C. May use less of drug.



STAGE II- PHYSIOLOGIC CONSEQUENCES

○ Hemodynamics

- Initial tachycardia and HTN from vasoconstriction and shivering
- Once cool, bradycardia is most common, along with PR prolongation, junction or ventricular escape rhythm, QT interval prolongation
- No need to treat normotensive bradycardia
- Hypotension can occur due to vasodilatation
- Map goal of 80-100 mmHg
- CVP goal of 10-12 mmHg



STAGE II- PHYSIOLOGIC CONSEQUENCES

○ **Oxygenation/Ventilation**

- Start 10-12 breaths/minute
- Avoid excessive ventilation
- Titrate FiO₂ to minimum, to achieve SpO₂ >94%
- Reduce FIO₂ ASAP to prevent neurological damage from reactive oxygen production
- High levels of PaO₂ are associated with increased in-hospital mortality and poor neurological status (198 vs 254 mmHg) (Janz et al., 2012)
- Maintain PaCO₂ 40-45 mmHg to prevent hypocapnia induced cerebral vasoconstriction
- Obtain ABGs q 4-6 hours during TH for adjustment of respiratory therapy



STAGE II- MANAGEMENT STAGE

○ **Hyperglycemia**

- Hyperglycemia is common during TH
- Low temperatures decrease insulin secretion and increases insulin resistance
- Typically no treatment until BS >200 mg/mL
- If >200 mg/mL, put on insulin drip and monitor blood glucose hourly



STAGE II-MAINTENANCE STAGE

○ **Electrolyte Imbalance**

- Cold diuresis occurs during hypothermia r/t decreased reabsorption of solute in the ascending limb of the loop of Henle
 - Causes hypovolemia, hypokalemia, hypomagnesemia, and hypophosphatemia
 - Hypothermia causes K to shift into the cells and hypokalemia may occur. Maintain 3.5-4.5
 - Caution: Rewarming reverses K flux so do not replete K 4 hours prior to rewarming!
- Monitor BMP Q 4-6 hrs



STAGE II-MAINTENANCE STAGE

○ Infection

- Common in CA and with TH
 - Cellular and antibody immunity suppressed
 - Pulmonary and blood stream infections are most common
 - Incidence increases if TH >24 hours
- Monitor CBC daily
 - Surveillance cultures
 - Prompt broad-spectrum antibiotics if infection is suspected



STAGE II-MAINTENANCE

○ Impaired Coagulation

- Slow clotting enzyme function & less effective platelets
- Bleeding is seen in 20% of the patient
- If significant bleeding, rewarm $>35\text{ C}$

○ Other Effects of TH

- Metabolism and excretion decreased
- Medication clearance is decreased 10% for every 1 degree C below 37 C
- Serum troponin is measured q 8 hrs for 24 hours to detect myocardial injury



INCIDENCE OF ADVERSE EFFECTS

765 patients in 22 hospitals from 2004-2008

- Pneumonia – 48%
- Metabolic and electrolyte disorders – 37%
- Seizures – 24%
- Arrhythmias – 7-14%
- Bleeding – 6%
- Sepsis – 4%
- Sustained hyperglycemia ($> 8\text{mmol/L}$) & seizures increased mortality ($p<0.001$)
- Bleeding & sepsis not associated with increased mortality ($p=0.01$)

(Nielsen et al., 2011)



ADVERSE EVENTS

Adverse Event	Targeted Temperature Management (N = 300)	Standard Treatment (N = 285)
	<i>no. (%)</i>	
Arrhythmia	55 (18)	47 (16)
Hemodynamic instability	14 (5)	15 (5)
Bleeding	26 (9)	19 (7)
Pneumonia	37 (12)	29 (10)
Sepsis	13 (4)	7 (2)
Electrolyte disorder	17 (6)	0
Renal failure	17 (6)	19 (7)
Seizures	10 (3)	11 (4)
Pancreatitis	1 (<1)	2 (1)
Pulmonary edema	33 (11)	52 (18)

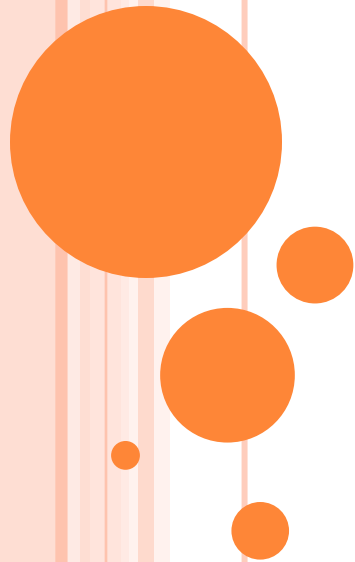
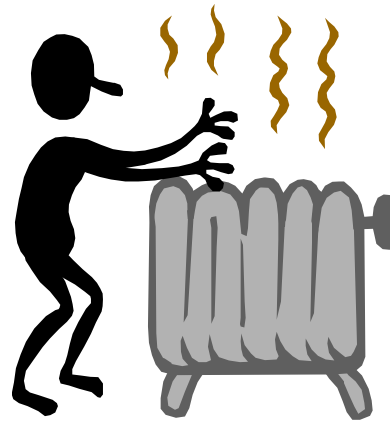
* Data are from major clinical trials comparing targeted temperature management with standard therapy after cardiac arrest.^{26,27,45-47}

MAINTENANCE STAGE

- Basic Interventions
 - Raising HOB to 30 deg. to prevent aspiration
 - Stress ulcer prophylaxis
 - DVT prophylaxis
 - Early PT and OT
 - Enteral feeding- resume after TH since bowel motility is suppressed during TH
 - Glycemic control-maintain BS <200 mg/mL
 - Skin care checks, frequent turns
 - Oral care



REWARMING STAGE OF TH



STAGE III-REWARMING STAGE

- Raise temperature gradually, not exceeding 0.5 C per hour until the patient returns to normothermia at 37°C/98.6°F
 - Closely monitor temperature
 - Automated devices
 - Takes 12-16 hrs to rewarm
- Rapid rewarming can cause electrolyte imbalance (hyperkalemia), cerebral edema, seizures, hypoglycemia, hyperthermia
- Animal studies have shown that rapid rewarming eliminated the benefits of TH



REWARMING STAGE

- Stop insulin infusion when BG <200mg/dL
- Maintain paralytic and/or sedation until normothermia (37°C/98.6°F) is reached
- If ice packs were used, remove few every hour
- Rate of rewarming increased by:
 - Raising the room temp
 - Applying convective heating devices
 - Heating lamps
 - Warming humidifier
 - Ventilator air

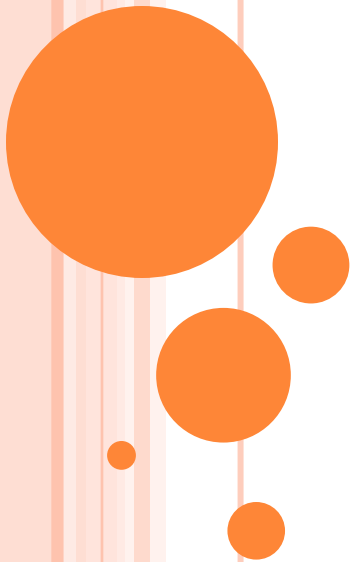


ACHIEVING NORMOTHERMIA

- Goal: maintain a temperature of 37°C (98.6°F) and avoid hyperthermia
- Post-CA fevers are associated with worse neurological outcomes
- Use cooling surfaces and Tylenol to reduce temp
- Continue continuous temp monitoring



PROGNOSIS



PROGNOSIS

- Meaningful neurological recovery in patients who received TH may occur late
- 2010 AHA Guidelines: recommend that neurological prognostication should be delayed until at least 72 hrs post normothermia (~ 5 days after CA)



PROGNOSIS

- Pre-CA Factors:
 - Advanced age
 - Pre-CA health/cardiac history
- Intra-CA Factors:
 - Asystole as the initial rhythm
 - Non-cardiac causes of arrest
 - Long interval between CA and start of CPR
 - $\text{ETCO}_2 < 10 \text{ mmHg}$
- Post-CA Factors:
 - Neurologic function

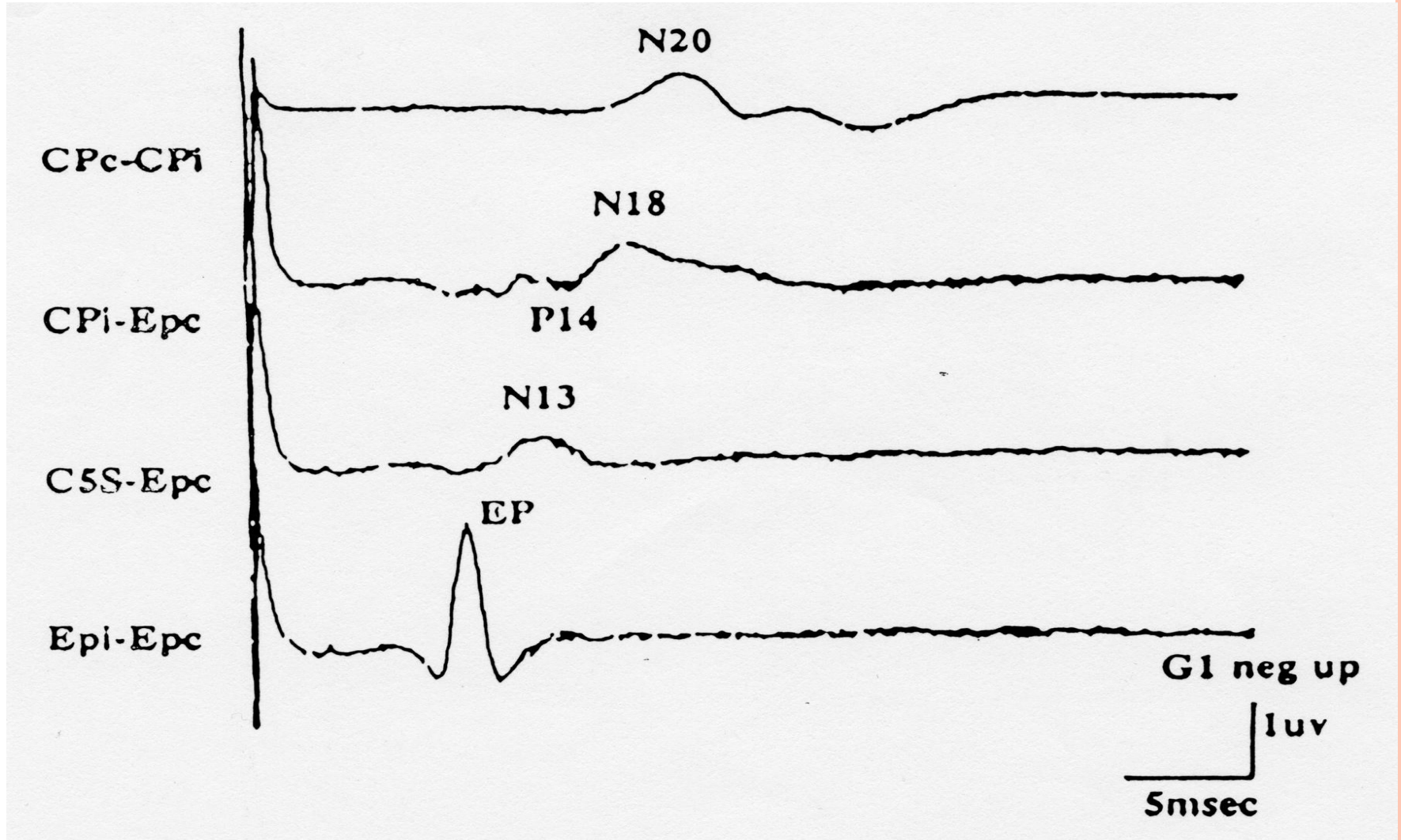


PROGNOSIS

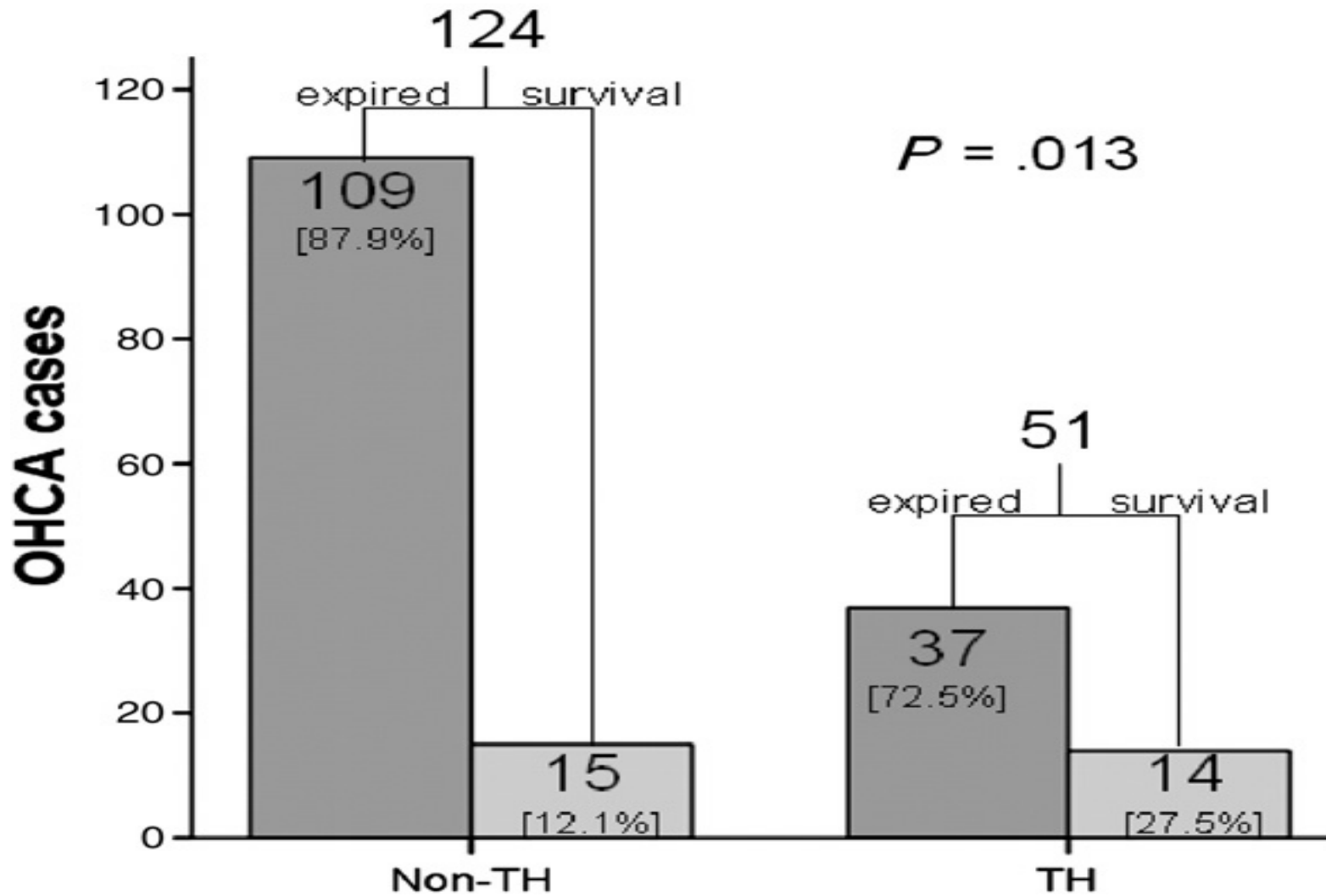
- Assessment of brain injury
 - Extensive midbrain injury = poor outcomes
- Illness severity scores- multiple organ systems assessment
- Neurological testing: physical examination, EEG, neuroimaging, sensory stimulatory evoked potentials
 - Two abnormal findings = higher specificity for poor neurological recovery



NORMAL SSEPs RESPONSE



SURVIVAL RATES



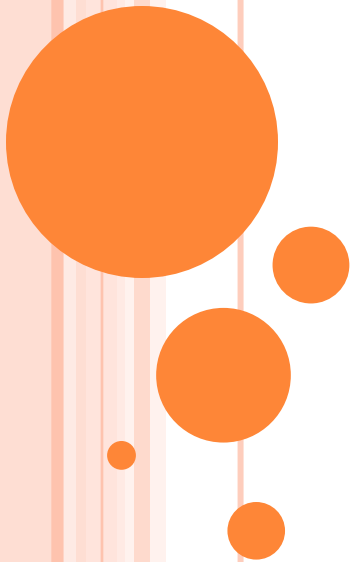
(Wang et al., 2012)

BARRIERS TO IMPLEMENTATION OF TH

- Structural barriers
 - Resources
 - Organizational
 - Scientific
- Environmental
 - Political
 - Economic
 - Cultural
- Personal barriers
 - Intellectual
 - Motivation



CASE STUDIES



CASE SCENARIO

- A 63-year-old man was at home watching television when his roommate noticed him gurgle and lose consciousness. He started CPR and called EMS. The initial rhythm VF. A perfusing rhythm was obtained within 15 minutes, but there were repeated episodes of VF requiring multiple defibrillations and repeated episodes of CPR. With restoration of circulation, he was not responsive, GCS 7. In the emergency department, initial ECG revealed inferior ST-segment elevations. VS: HR 110 bpm, BP 86/60 mmHg, RR 34/min, SpO2 85%
- *What would you do next?*
- *What orders will you initiate?*



CASE SCENARIO INTERVENTIONS

- Lab orders: PT/INR, BMP, CBC, BNP, Troponin x3, ABG
- Protect airway, intubate/ventilate to keep SpO₂ >92%
- Sedation with opioids (analgesia) and hypnotics (e.g. propofol) → use sedation scales to monitor
- Start IV iced saline and place ice packs
- STAT cardiac catheterization (or thrombolysis if non-PCI facility)
- Place central line access
- If neuromuscular blocking agents used → EEG
- Train of 4 (2/4) if on a neuromuscular blocking agent
- Vital signs and neuro assessments Q 1 hr, routine labs to check electrolytes – Q 4-6 hrs



CASE SCENARIO CONTINUED

- A stent was placed in a thrombotic RCA. On arrival at the cardiac intensive care unit, surface cooling pads were placed, and he received TH.



QUESTION #1

- What is the ideal time lapse between ROSC and cooling?
 - A – <1.5 hours
 - B – 2-3 hours
 - C – 3-4 hours
 - D – 5-6 hours



QUESTION #1

- What is the ideal time lapse between ROSC and cooling?
 - A – <1.5 hours
 - B – 2-3 hours
 - C – 3-4 hours
 - D – 5-6 hours

(Mooney et al., 2011)



QUESTION #2

- What is the duration of therapeutic hypothermia therapy?
 - A – 6 hours
 - B – 8 hours
 - C – 15 hours
 - D – 24 hours



QUESTION #2

- What is the duration of therapeutic hypothermia therapy?
 - A – 6 hours
 - B – 8 hours
 - C – 15 hours
 - D – 24 hours

(AHA, 2010)



QUESTION #3

- What is the mechanism of action of TH?
 - A – Releases catecholamines and neurotransmitters
 - B – Induces shivering response which decreases metabolic rate
 - C – Increases rate of apoptosis
 - D – Decreases oxygen consumption and inhibits the inflammatory cascade



QUESTION #3

- What is the mechanism of action of TH?
 - A – Releases catecholamines and neurotransmitters
 - B – Induces shivering response which decreases metabolic rate
 - C – Increases rate of apoptosis
 - D – Decreases oxygen consumption and inhibits the inflammatory cascade

(Arrich et al., 2012)



QUESTION #4

- What are the possible complications of TH?
 - A – Hypoglycemia and hypokalemia
 - B – Sepsis and hyperkalemia
 - C – Seizures and hypokalemia
 - D – Increased metabolism and hypoglycemia



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(ILCOR & AHA, 2008; Scirica, 2013)



QUESTION #5

- During the rewarming stage, the temperature should be:
 - A – Increased by 1°C every one hour.
 - B – Increased by 0.5°C every one hour.
 - C – Increased by 2°C every one hour.
 - D – Increased by $3\text{-}4^{\circ}\text{C}$ every one hour.



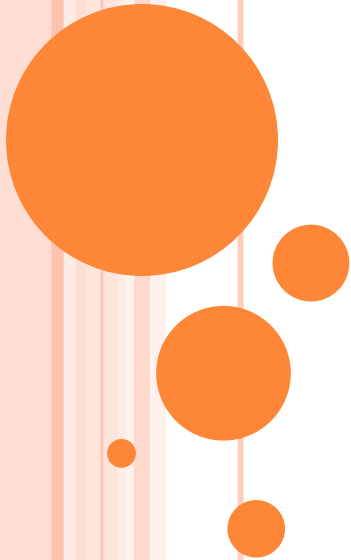
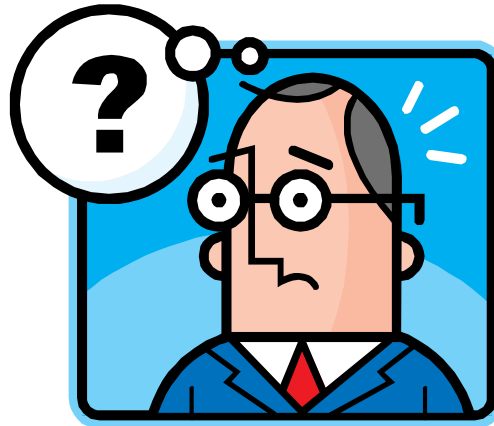
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(Arrich et al., 2012; ILCOR & AHA, 2008)



QUESTIONS?
COMMENTS/EXPERIENCES?




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