

Post-cardiac arrest care: targeted temperature management and prognostication

2015 AHA Guidelines for CPR & ECC

ACLS 聯委會麻醉重症委員
義大醫院 醫療品質部/重症醫學部
王義明 醫師



中華民國急救加護醫學會

SOCIETY OF EMERGENCY & CRITICAL CARE MEDICINE, TAIWAN, R.O.C.

● 祝賀2016大吉大利大好年～

最新消息 HOT NEWS



猴塞插 Happy New Year

更新日期：2016/03/16

2015 AHA Guidelines for CPR & ECC NEW

購書請洽金名圖書參閱線上購書

- 2015 AHA Guidelines Update for CPR & ECC NEW
- 2015 AHA Handbook of Emergency Cardiovascular Care for Healthcare Provider NEW

2015 ERC Guidelines NEW

首頁

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ACLS聯委會

相關資源

訪客留言

重症專醫聯合甄審委員會



<https://eccguidelines.heart.org/index.php/circulation/cpr-ecc-guidelines-2/>

2015

American Heart Association Guidelines for CPR & ECC

NEW Web-Based Integrated Guidelines

This site blends the 2015 and 2010 AHA Guidelines for CPR & ECC into a new online interface.

Explore and search all guidelines from your desktop or mobile device.



IHCA



OHCA



Overview

- The hypoxemia, ischemia, and reperfusion
→ damage to multiple organ systems
(Circulation. 2008;118:2452–2483.)
- Effective care consists of identification and treatment of the precipitating cause combined with organ supportive treatment.

The initial objectives

- Optimize cardiopulmonary function and vital organ perfusion.
- Perform a comprehensive system of care for both IHCA and OHCA
- Try to identify and treat the precipitating causes of the arrest and prevent recurrent arrest.

Subsequent objectives of post– cardiac arrest care

- Control body temperature to optimize survival and neurological recovery
- Identify and treat acute coronary syndrome
- Optimize mechanical ventilation to minimize lung injury
- Reduce the risk of multiorgan injury and support organ function
- Objectively assess prognosis for recovery
- Assist survivors with rehabilitation services when required

Systems of care for improving post–cardiac arrest outcomes

- ***A comprehensive, structured, multidisciplinary system of care should be implemented*** *in a consistent manner for the treatment of post-cardiac arrest patients. (Class I, LOE B 2010)*

2015 guideline → systems of care

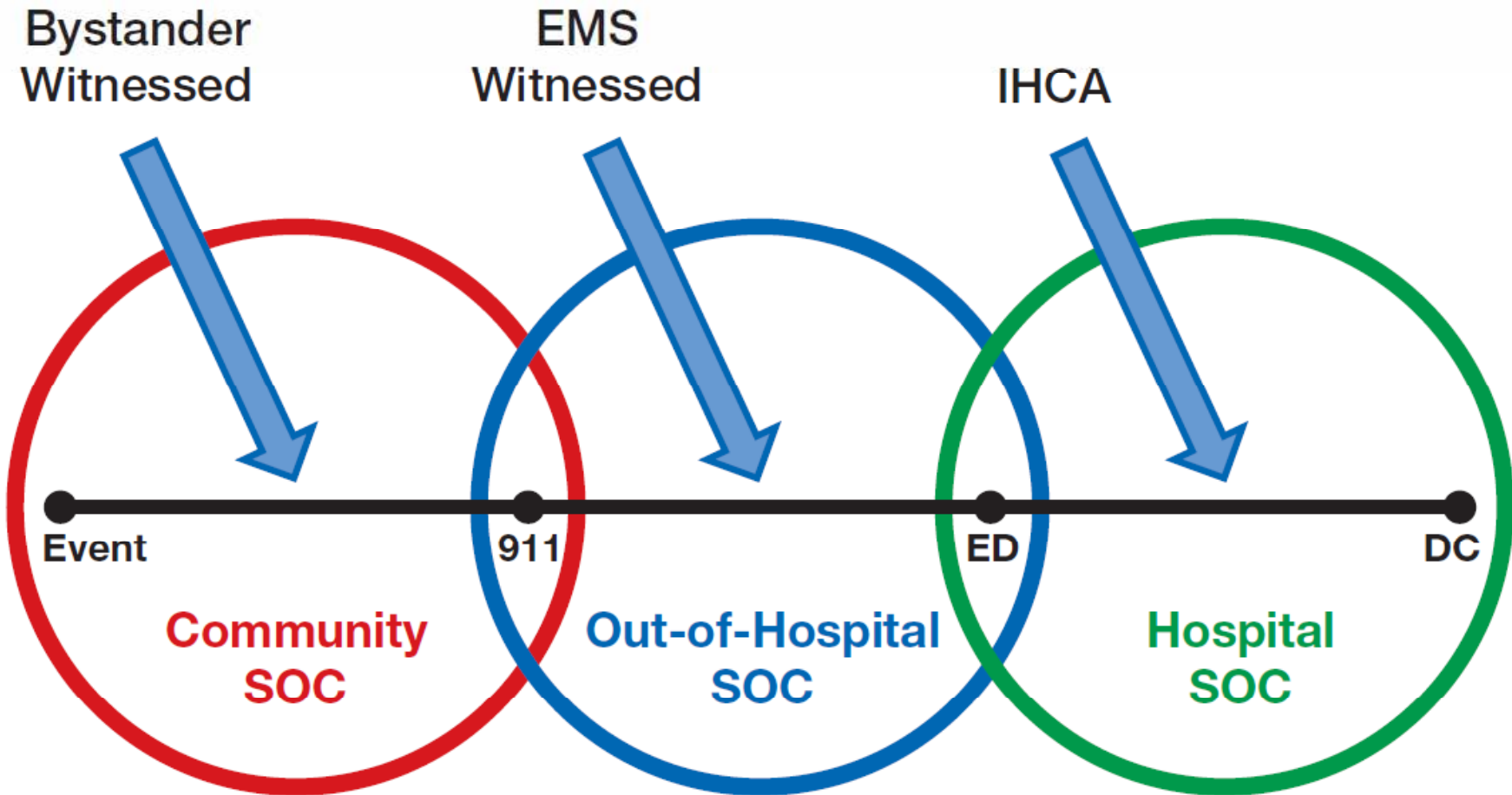


Figure 3. Patient's point of entry.

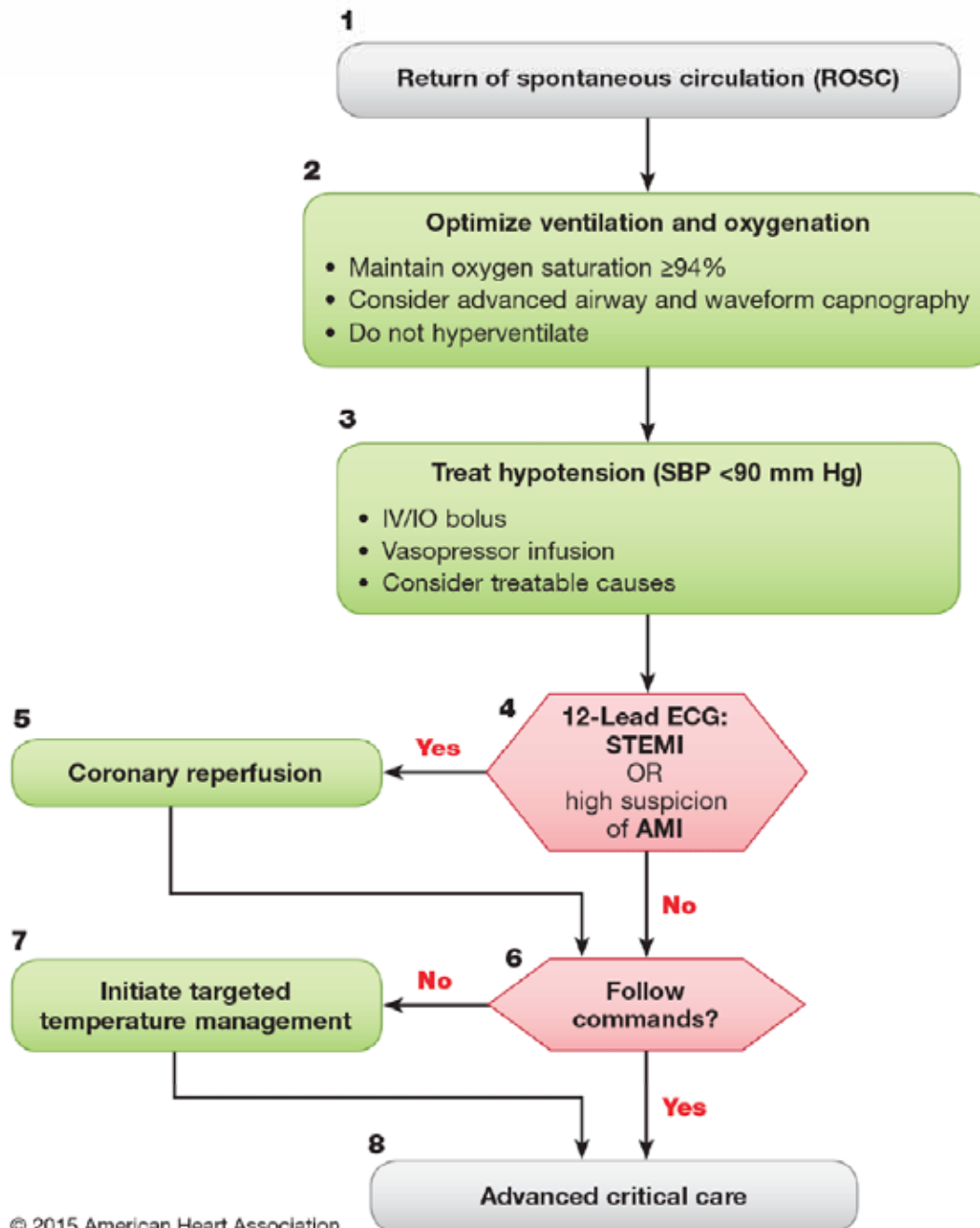
Programs should include:

- Targeted temperature management (TTM)
- Optimization of hemodynamics and gas exchange
- Immediate coronary reperfusion when indicate for restoration of coronary blood flow
- Modifying outcomes → glycemic control, steroid, hemofiltration
- Neurological diagnosis, management, and prognostication

Overview of post–cardiac arrest care

- Post-cardiac arrest care is a critical component of advanced life support.
- Identifying and optimizing practices that are likely to improve outcomes.
- System-wide plans for proactive treatment will benefit because multiple organ systems are affected.

Adult Immediate Post-Cardiac Arrest Care Algorithm—2015 Update



Doses/Details

Ventilation/oxygenation:
 Avoid excessive ventilation. Start at 10 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:
 Approximately 1-2 L normal saline or lactated Ringer's

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

COR

建議類別 (強度)

第 I 級 (強烈)

效益 >>> 風險

建議用詞：

- 建議
- 適用 / 有用 / 有效 / 有益
- 應執行 / 施行 / 其他
- 比較療效用詞†：
 - 相對於治療 B，建議 / 適合採用治療 / 策略 A
 - 相對於治療 B，應優先選擇治療 A

第 IIa 級 (一般)

效益 >> 風險

建議用詞：

- 適當
- 可能有用 / 有效 / 有益
- 比較療效用詞†：
 - 相對於治療 B，可能建議 / 適用治療 / 策略 A
 - 相對於治療 B，選擇治療 A 是適當的做法

第 IIb 級 (薄弱)

效益 ≥ 風險

建議用詞：

- 或許 / 也許適當
- 或許 / 也許可以考慮
- 實用性 / 療效未知 / 不明 / 不確定或尚未確立

第 III 級：無效益 (一般)

效益 = 風險

(一般而言，僅適用 LOEA 或 B)

建議用詞：

- 不建議
- 不適用 / 無用 / 無效 / 無益
- 不應執行 / 施行 / 其他

第 III 級：傷害 (強烈)

風險 > 效益

建議用詞：

- 可能有害
- 引起傷害
- 與發病率 / 死亡率增加有關
- 不應執行 / 施行 / 其他

證據等級 (品質) ‡

A 級

- 高品質證據‡ (來自 1 項以上 RCT)
- 高品質 RCT 的統合分析
- 一項以上經高品質登錄研究確證的 RCT

B-R 級

(隨機分配)

- 中度品質證據‡ (來自 1 項以上 RCT)
- 中度品質 RCT 的統合分析

B-NR 級

(非隨機分配)

- 中度品質證據‡ (來自 1 項以上設計周延且完善執行的非隨機分配研究、觀察研究或登錄研究)
- 此類研究的統合分析

C-LD 級

(有限資料)

- 隨機分配或非隨機分配之觀察或登錄研究，有設計或執行方面的限制
- 此類研究的統合分析
- 人體的生理或機制研究

C-EO 級

(專家意見)

基於臨床經驗的專家共識

LOE

Targeted temperature management (TTM): Induced hypothermia

- For protection of the brain and other organs **who remain comatose**
- Questions :
 - specific **indications and populations**
 - **timing and duration** of therapy
 - **methods** for induction, maintenance, and subsequent reversal of hypothermia

Targeted temperature management

Benefit (ERC 2015 guideline)

- Animal and human data → neuroprotective and improves outcome after a period of global cerebral hypoxia-ischaemia
- Cooling suppresses many of the pathways leading to apoptosis.
- ↓ the cerebral metabolic rate for oxygen by about 6% for each 1 °C reduction in core temperature → ↓ excitatory amino acids and free radicals
- Blocks the intracellular consequences of excitotoxin exposure (high calcium and glutamate concentrations) and reduces the inflammatory response.

Timing of initiating hypothermia

- not completely understood
- Animal models: short-duration hypothermia (≤ 1 hour) achieved < 10 to 20 minutes after ROSC.
- Within 2 hours or at a median of 8 hours after ROSC both demonstrated better outcome. (prospective clinical trials)

Optimal duration

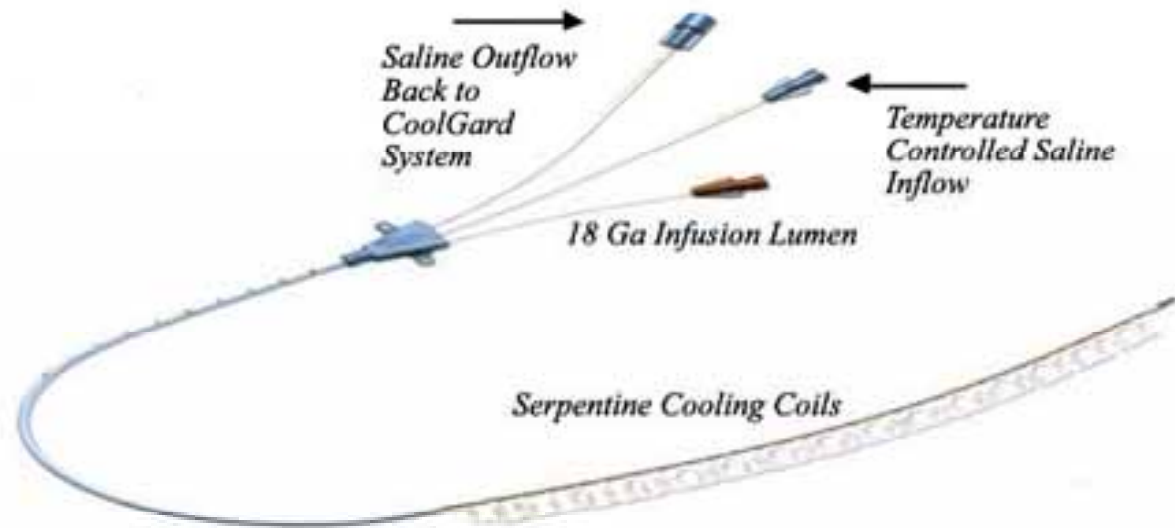
- **2015 updated:**
- It is reasonable that TTM be **maintained for at least 24 hours** after achieving target temperature (Class IIa, LOE C-EO).
 - The largest trials and studies of TTM maintained temperatures for **24 hours** (N Engl J Med. 2002;346:549–556.) or **28 hours** (N Engl J Med. 2013;369:2197–2206.) followed by a gradual (approximately 0.25°C/hour) return to normothermia.

Methods for inducing hypothermia

- Feedback-controlled endovascular catheters and surface cooling devices are available.

ERC
guideline

- If a target temperature of 36 °C is chosen, for the many patients who arrive in hospital with a temperature less than 36 °C, a practical approach is to let them rewarm spontaneously and to activate a TTM-device when they have reached 36 °C.
- If a lower target temperature, e.g., 33 °C is chosen, an infusion of 30 ml/kg of 4 °C saline or Hartmann's solution will decrease core temperature by approximately 1.0–1.5 °C. → in one prehospital RCT :
↑ pulmonary edema and ↑ rate of re-arrest



PRODUCT INTEGRITY



FREQUENTLY ASKED QUESTIONS

Methods for inducing hypothermia

ERC 2015 guideline

- As yet, there are **no data indicating that any specific cooling technique increases survival** when compared with any other cooling technique; however, **internal devices enable more precise temperature control** compared with external techniques.
- Water or air circulating blankets.^{7,8,10,182,226,228–234}
- Water circulating gel-coated pads.^{7,224,226,233,235–238}
- Transnasal evaporative cooling²⁰⁹ – this technique enables cooling before ROSC and is undergoing further investigation in a large multicentre randomised controlled trial.²³⁹
- Intravascular heat exchanger, placed usually in the femoral or subclavian veins.^{7,8,215,216,226,228,232,240–245}
- Extracorporeal circulation (e.g., cardiopulmonary bypass, ECMO).^{246,247}

Monitor of temperature

- continuously monitor the core temperature using an esophageal thermometer, bladder catheter, or pulmonary artery catheter
- axillary and oral temperatures are inadequate
- true tympanic temperature probes are rarely available
- rectal temperatures may differ from brain or core temperature

Potential complications

- Coagulopathy
- Arrhythmias
- Hyperglycemia
- Pneumonia and sepsis may increase
- Decrease immune function
- Ongoing bleeding should be controlled before decreasing temperature

Physiological effects and side effects of hypothermia (ERC 2015 guideline)

- **Shivering** → ↑ metabolic and heat production, ↓ cooling rates, **associated with a good neurological outcome** → **MgSO₄**, a NMDA receptor antagonist, ↓ **shivering** threshold slightly
- **↑ SVR and causes arrhythmias (usually bradycardia)** → may be beneficial, ↓ diastolic dysfunction, **associated with good neurological outcome**
- **Diuresis and electrolyte abnormalities** such as **hypo-P_i, K_i, Mg_i, Ca_i**
- ↓ insulin sensitivity and insulin secretion, and causes **hyperglycaemia**

Physiological effects and side effects of hypothermia (ERC 2015 guideline)

- Impairs coagulation and may increase bleeding → effect seems to be negligible and not confirmed in clinical studies
- Impair the immune system and increase infection rates → increased incidence of pneumonia
- ↑ serum amylase → the significance of this unclear
- The clearance of sedative drugs and neuromuscular blockers is reduced by up to 30% at a core temperature of 34 C.

Contraindications to TTM

- **ERC 2015** guideline
- Contraindications to TTM at 33 C, but which are not applied universally, include: **severe systemic infection and pre-existing medical coagulopathy** (fibrinolytic therapy is not a contraindication to mild induced hypothermia)

TTM: optimal temperature, how deep

- For patients with VF/pVT OHCA, RCT reported increased survival and increased functional recovery with 32°C to 34°C. (N Engl J Med. 2002;346:549–556., N Engl J Med. 2002;346:557–563.)
- One well-controlled RCT found that neurologic outcomes and survival at 6 months after OHCA were not superior when temperature was controlled at 36°C versus 33°C. (N Engl J Med. 2013;369:2197–2206.)

Table 2. Outcomes.

Outcome
Primary outcome: death
Secondary outcomes
Neurologic function
CPC of 3–5
Modified Rankin Scale
Deaths at 180 days

Ratio (95% CI)*	P Value
1.28)	0.51
1.16)	0.78
1.14)	0.87
1.15)	0.92

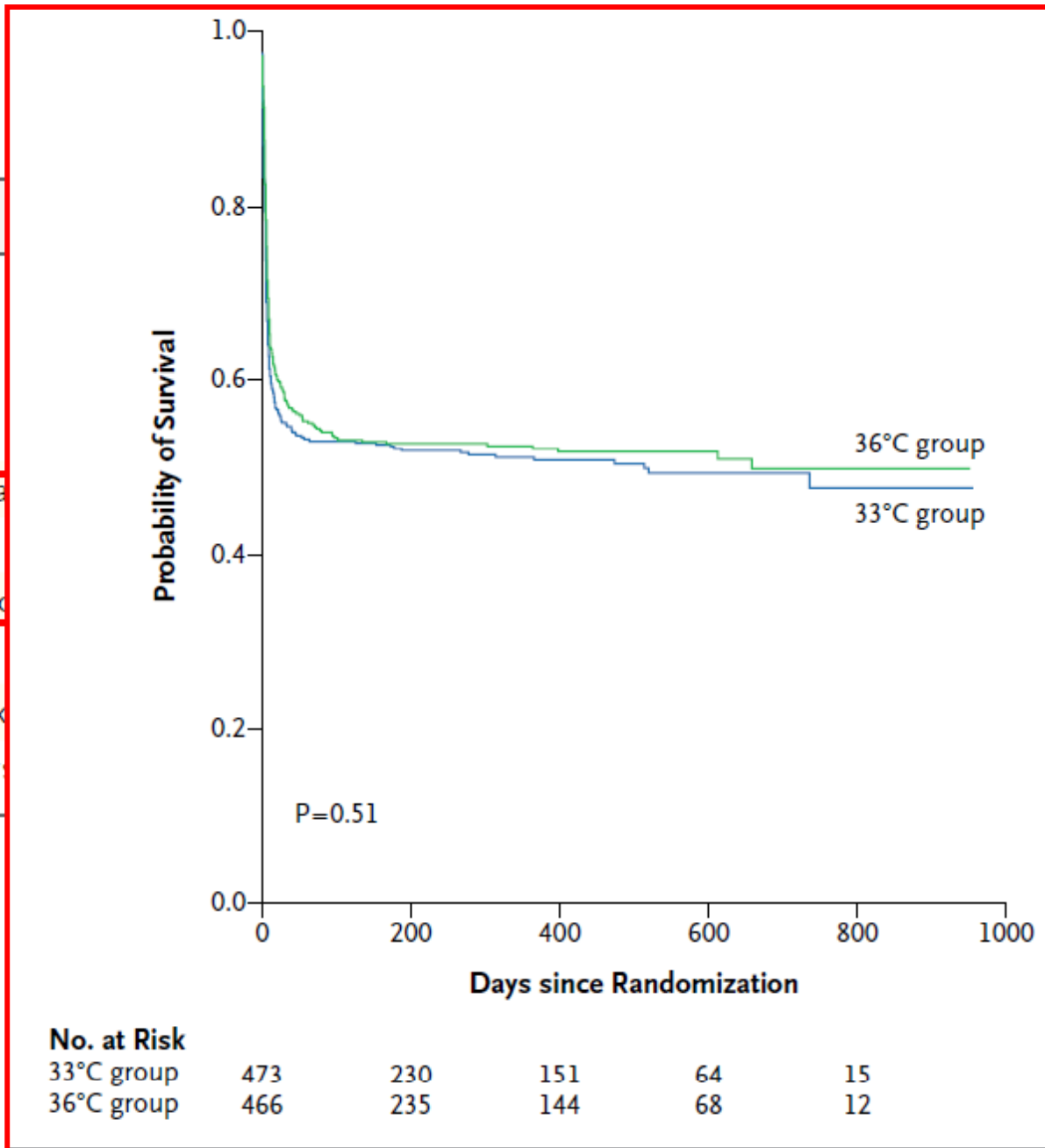


Figure 2. Probability of Survival through the End of the Trial.

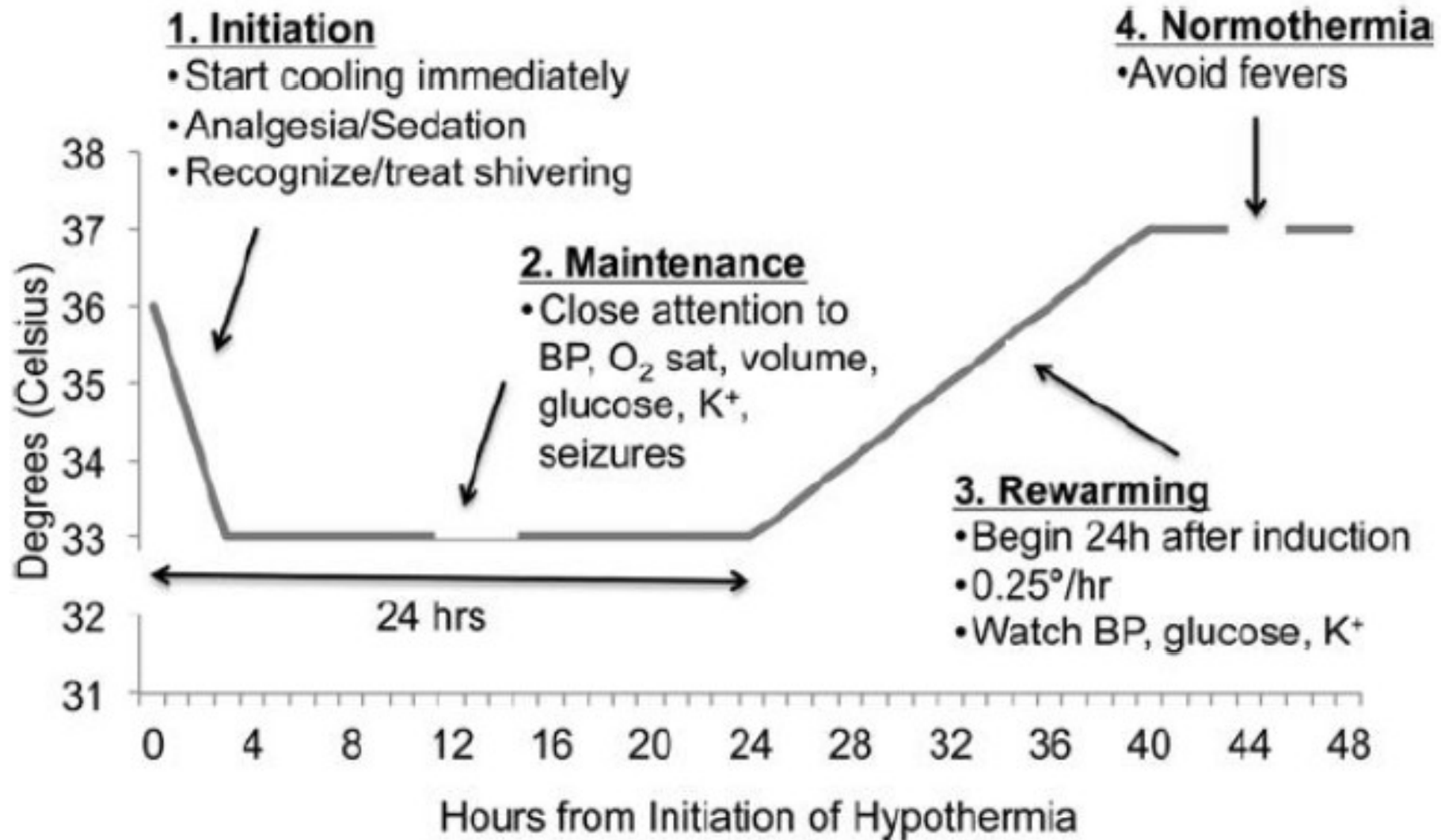
TTM: optimal temperature, how deep

- Higher temperatures might be preferred for patients with some risk (eg, bleeding)
- Lower temperatures might be preferred for patients with worsened clinical features (eg, seizures, cerebral edema)
- While it is stated that choosing a temperature within the 32°C to 36°C range is acceptable, actively or rapidly warming patients is not suggested.

TTM: optimal temperature, how deep

- **2015 Updated**
- Comatose (ie, lack of meaningful response to verbal commands) adult patients with ROSC after cardiac arrest have TTM (**Class I, LOE B-R for VF/pVT OHCA; Class I, LOE C-EO for non-VF/pVT (ie, “nonshockable”) and IHCA**).
- Selecting and maintaining a constant temperature between **32⁰C and 36⁰C** during TTM (Class I, LOE B-R).

Phases of Therapeutic Hypothermia



Relative or absolute Contra-indication



Therapeutic Hypothermia after Cardiac Arrest Guidelines of Care

Inclusion Criteria:

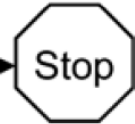
- Within 6 hrs following cardiac arrest (up to 12 hours at attending physician's discretion)
- Successful restoration of a perfusing rhythm and the ability to maintain a blood pressure with/without inotropes or vasopressors
- Comatose state – *Lack of meaningful response to verbal commands*

Yes

Exclusion Criteria:

- Major head trauma
- Major surgery within prior 14 days
- Systemic infection/sepsis
- Patients with clinically significant bleeding / risk of bleeding

Yes

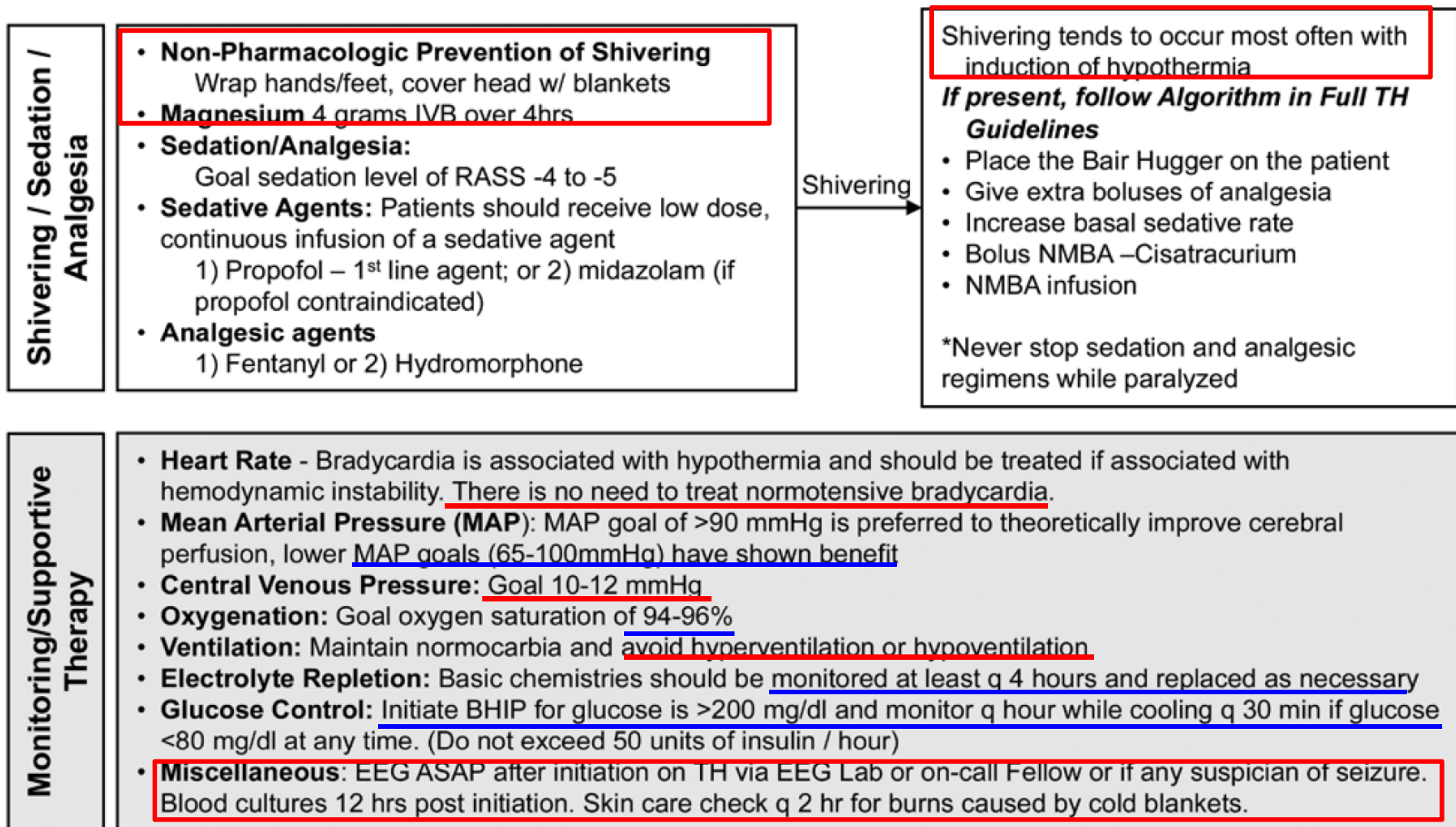


No

Initiation of Cooling

- Initiate cooling as rapidly as possible.
- Either method of cooling – ice packs/cooling blankets or Arctic Sun System – can be used to initiate cooling and should be started as soon as possible.
- Remove ice packs once the [Arctic Sun] system initiated to prevent overcooling of the patient
- Defibrillator pads may be placed under the Arctic Sun gel pads. It is safe to defibrillate the patient with the Arctic Sun pads on the patient.
- Ensure two methods (bladder, esophageal, core, rectal, groin, axillary) of measuring patient temperature

Benjamin M. Scirica, *Circulation*. 2013;127:244-250.



Benjamin M. Scirica, *Circulation*. 2013;127:244-250.

Rewarming

- Begin re-warming 24 hrs after initiation of cooling at a rate of 0.25°C (0.5° F) every hour until the patient returns to normothermia (37°C/98.6° F).
- Keep Artic Sun pads on for 48hr and set temperature at 37°C/98.6° F to maintain normothermia.
- Maintain paralytic (if started) and sedation until temperature of 36C/96.8F degrees is reached.
- Hypotension, hyperkalemia, hypoglycemia, and hyperthermia may occur during and after the re-warming.
- Stop IV insulin when glucose <200 mg/dl, unless T1DM.
- Testing to assess for neurologic prognosis should be delayed to at least 72 hours after the return to normothermia as patients who have TH have delayed neurologic recovery

Benjamin M. Scirica, *Circulation*. 2013;127:244-250.

全民健康保險醫療服務給付項目及支付標準 部分診療項目修正草案總說明

依據全民健康保險法第四十一條規定及本署於一百零四年九月十日召開之一百零四年度第三次「全民健康保險醫療服務給付項目及支付標準共同擬訂會議」會議結論，爰配合修正本支付標準。

第六節 治療處置 Therapeutic Treatment

第一項 處置費

一、一般處置 General Treatment (47001~47100)

編號	診療項目	基層院所	地區醫院	區域醫院	醫學中心	支付點數
47094B	<u>心跳停止之低溫療法</u> — 第一天 (≤24小時)		√	√	√	9068
47095B	— 第二天 (>24小時~≤48小時)		√	√	√	1500
47096B	— 第三天 (>48小時)		√	√	√	1500
	1.適應症： <u>心跳停止患者，進行復甦急救後意識仍不清者(GCS<8)或無遵循口頭醫囑 (motor<6)。</u>					
	2.排除條件： <u>(1)恢復自發性循環大於12小時</u> <u>(2)腦出血</u> <u>(3)收縮血壓<90mmHg</u> <u>(4)大量活動性出血</u> <u>(5)無法終止的致命性心律不整</u> <u>(6)在心跳停止前即有失智或長期意識障礙</u> <u>(7)末期疾病。</u>					
	3.支付規範： <u>(1)總治療療程為3天。</u> <u>(2)不得同時申報項目：47037B「冰毯-12小時以內」、47038B「冰毯-12-24小時以內」、47049B「自動體溫控制床使用費」。</u> <u>(3)特材另計。</u>					

	<u>週產期新生兒低溫療法</u>				
<u>47097B</u>	<u>— 第一天 (≤24小時)</u>	<u>v</u>	<u>v</u>	<u>v</u>	<u>10000</u>
<u>47098B</u>	<u>— 第二天 (>24小時~≤48小時)</u>	<u>v</u>	<u>v</u>	<u>v</u>	<u>3994</u>
<u>47099B</u>	<u>— 第三天 (>48小時~≤72小時)</u>	<u>v</u>	<u>v</u>	<u>v</u>	<u>3000</u>
<u>47100B</u>	<u>— 第四天 (>72小時)</u>	<u>v</u>	<u>v</u>	<u>v</u>	<u>3000</u>
	<u>1.適應症，須符合以下3項：</u>				
	<u>(1)出生週數≥36週</u>				
	<u>(2)事件發生後6小時內實施</u>				
	<u>(3)出生後有「中等嚴重度」至「重度」</u>				
	<u>腦病變之證據。且有下列任一項之情形：</u>				
	<u>I.出生後一小時內嚴重酸血症(severe acidosis)，血液PH值≤7或base deficit≥16mmol/L(採血來源：動脈血或靜脈血均可)。</u>				
	<u>II.出生10分鐘時的Apgar分數≤5分。</u>				
	<u>III出生後持續急救至少10分鐘。</u>				
	<u>2.支付規範：</u>				
	<u>(1)總治療程為4天。</u>				
	<u>(2)不得同時申報47037B「冰毯-12小時以內」、47038B「冰毯-12-24小時以內」、47049B「自動體溫控制床使用費」。</u>				
	<u>(3)特材另計。</u>				

Hypothermia in the prehospital setting

- Neither survival nor neurologic recovery differed for any of 5 RCTs alone or when combined in a meta-analysis.
- One trial found an increase in pulmonary edema and rearrest among patients treated with a goal of prehospital infusion of 2 L of cold fluids. ([JAMA.2014](#);311:45–52)

Hypothermia in the prehospital setting

- **2015—New**
- We recommend **against the routine prehospital cooling** of patients after ROSC **with rapid infusion of cold intravenous fluids** (Class III: No Benefit, LOE A).

Hyperthermia

- One of the etiology of fever after cardiac arrest may be **related to inflammatory cytokines**
- After resuscitation, temperature elevation above normal may **impair brain recovery and associate with poor outcome** → Pyrexia $\geq 37.6^{\circ}\text{C}$
- **Late hyperthermia** (rewarming posthypothermia) **occur frequently** should also be identified and treated
- **Closely monitor core temperature** after ROSC and actively intervene to **avoid hyperthermia** (Class I, LOE C 2010).

Avoidance of hyperthermia

- **2015 —New**
- It may be reasonable to actively prevent fever in comatose patients after TTM (Class IIb, LOE C-LD).

Organ-specific evaluation and support

- Cardiovascular system
- Pulmonary system
- Central nervous system
- **Modifying outcomes**: glucose control, steroid, hemofiltration, sedation
- **Prognostication** of neurological outcome
- **Organ donation** after cardiac arrest

心、肺、腦

Cardiovascular care

Acute cardiovascular interventions

- **Post–cardiac arrest patients** with suspected cardiovascular cause were taken to coronary angiography, a coronary artery lesion amenable to emergency treatment was found in **96% of patients with ST elevation and in 58% of patients without ST elevation.** (*Circ Cardiovasc Interv.* 2010;3:200–207.)

Cardiovascular care

Acute cardiovascular interventions

- A 12-lead ECG should be obtained as soon as possible after ROSC to determine whether acute ST elevation is present. (Class I, LOE B 2010)
- **2015– Updated**
- Coronary angiography should be performed emergently (rather than later in the hospital stay or not at all) for OHCA patients with suspected cardiac etiology of arrest and ST elevation on ECG. (Class I, LOE B-NR)

Cardiovascular care

Acute cardiovascular interventions

- **2015 – Updated**
- Emergency coronary angiography is reasonable for select (eg, **electrically or hemodynamically unstable**) adult patients who are comatose after OHCA of suspected cardiac origin but **without ST elevation** on ECG. (**Class IIa**, LOE B-NR)
- Coronary angiography is reasonable in post-cardiac arrest patients for whom **coronary angiography is indicated regardless of whether the patient is comatose or awake**. (**Class IIa**, LOE C-LD)

Vasoactive drugs

- improve heart rate (**chronotropic effects**)
- myocardial contractility (**inotropic effects**)
- arterial pressure (**vasoconstrictive effects**)
- reduce afterload (**vasodilator effects**)

Adverse effects:

- many adrenergic drugs are not selective
- create a mismatch between myocardial oxygen demand and delivery
- may also have metabolic effects

Vasoactive drugs

- Vasoactive drugs must be titrated
- Aware of the concentrations delivered and concurrently administered drugs
- Adrenergic drugs should not be mixed with sodium bicarbonate or other alkaline solutions in the IV line (*Crit Care Med.* 1995;23:1061–1066. ; *Ann Emerg Med.* 1990;19:1242–1244.)
 - → adrenergic agents are inactivated in alkaline solutions
- Norepinephrine and other catecholamines may produce tissue necrosis if extravasation
- Infiltrate 5 to 10 mg of phentolamine diluted in 10 to 15 mL of saline into the site of extravasation as soon as possible

Table 2. Common Vasoactive Drugs

Drug	Typical Starting Dose (Then Titrate to Effect)
Epinephrine	<p data-bbox="712 268 1693 320">0.1–0.5 mcg/kg/min (In 70-kg adult, 7–35 mcg/min)</p> <ul data-bbox="763 347 1731 778" style="list-style-type: none"><li data-bbox="763 347 1731 507">● Useful for symptomatic bradycardia if atropine and transcutaneous pacing fail or if pacing is not available<li data-bbox="763 539 1731 639">● Used to treat severe hypotension (eg, systolic blood pressure <70 mm Hg)<li data-bbox="763 671 1731 778">● Useful for anaphylaxis associated with hemodynamic instability or respiratory distress¹⁵⁸
Norepinephrine	<p data-bbox="712 799 1693 852">0.1–0.5 mcg/kg/min (In 70-kg adult, 7–35 mcg/min)</p> <ul data-bbox="763 879 1731 1513" style="list-style-type: none"><li data-bbox="763 879 1731 1038">● Used to treat severe hypotension (eg, systolic blood pressure <70 mm Hg) and a low total peripheral resistance<li data-bbox="763 1070 1731 1278">● Relatively contraindicated in patients with hypovolemia. It may increase myocardial oxygen requirements, mandating cautious use in patients with ischemic heart disease<li data-bbox="763 1310 1731 1513">● Usually induces renal and mesenteric vasoconstriction; in sepsis, however, norepinephrine improves renal blood flow and urine output^{159,160}

Phenylephrine	<p>0.5–2.0 mcg/kg/min (In 70-kg adult, 35–140 mcg/min)</p> <ul style="list-style-type: none"> ● Used to treat severe hypotension (eg, systolic blood pressure <70 mm Hg) and a low total peripheral resistance
Dopamine	<p>5–10 mcg/kg/min</p> <ul style="list-style-type: none"> ● Used to treat hypotension, especially if it is associated with symptomatic bradycardia ● Although low-dose dopamine infusion has frequently been recommended to maintain renal blood flow or improve renal function, more recent data have failed to show a beneficial effect from such therapy^{161,162}
Dobutamine	<p>5–10 mcg/kg/min</p> <ul style="list-style-type: none"> ● The (+) isomer is a potent beta-adrenergic agonist, whereas the (–) isomer is a potent alpha-1-agonist¹⁶³ ● The vasodilating beta₂-adrenergic effects of the (+) isomer counterbalance the vasoconstricting alpha-adrenergic effects, often leading to little change or a reduction in systemic vascular resistance
Milrinone	<p>Load 50 mcg/kg over 10 minutes then infuse at 0.375 mcg/kg/min</p> <ul style="list-style-type: none"> ● Used to treat low cardiac output ● May cause less tachycardia than dobutamine

Use of vasoactive drugs after cardiac arrest

Hemodynamic instability is common after cardiac arrest:

- Persistently low cardiac index during the first 24 hours
- Vasodilation may occur from loss of sympathetic tone and from metabolic acidosis
- Ischemia/reperfusion of cardiac arrest and electric defibrillation both can cause transient myocardial stunning and dysfunction

- Echocardiographic evaluation within the first 24 hours
- Invasive monitoring may be necessary
- Mechanical circulatory support after cardiac arrest is not recommended
- Fluid administration as well as vasoactive, inotropic, and inodilator agents should be titrated as needed to optimize blood pressure, cardiac output, and systemic perfusion (Class I ,LOE B 2010)

Hemodynamic goals

- **2015 —New**
- **Avoiding** and immediately correcting hypotension (**SBP less than 90 mm Hg, MAP less than 65 mm Hg**) during post-resuscitation care may be reasonable (**Class IIb, LOE C-LD**).

Other neurologic care

Brain injury

- A common cause of morbidity and mortality after cardiac arrest
- The cause of death in 68% of OHCA and in 23% of IHCA
- The pathophysiology involves a complex cascade of molecular events.
- **Clinical manifestations**: coma, **seizures**, myoclonus, various degrees of neurocognitive dysfunction and brain death.

Seizure management

- **2015 evidence summary**
- **The prevalence** of seizures, nonconvulsive status epilepticus, and other epileptiform activity among patients who are comatose after cardiac arrest is estimated to be **12% to 22%**.
- Three case series looked at 47 post–cardiac arrest patients who were treated for seizures or status epilepticus and found that **only 1 patient survived with good neurologic function**.
- Available evidence does **not support prophylactic administration of anticonvulsant drugs**.

Seizure management

- **2015—Updated**
- An **EEG** for the diagnosis of seizure should be promptly performed and interpreted, and then should be **monitored frequently or continuously in comatose** patients after ROSC (**Class I**, LOE C-LD).
- The same **anticonvulsant regimens for the treatment of status epilepticus** caused by other etiologies may be considered after cardiac arrest (**Class IIb**, LOE C-LD).

Neuroprotective drugs

- Broad therapeutic window for neuroprotective drug therapy
- **No neuroprotection benefit** was observed when patients (without hypothermia) were treated with thiopental, glucocorticoids, nimodipine, lidoflazine, diazepam, and magnesium sulfate
- The routine use of coenzyme Q10 in patients treated with hypothermia is uncertain (Class IIb, LOE B 2010).

Pulmonary dysfunction after cardiac arrest

Etiologies include:

- Hydrostatic pulmonary edema
- Noncardiogenic edema from inflammatory, infective, or physical injuries
- Severe pulmonary atelectasis
- Aspiration
- Regional mismatch of ventilation and perfusion

Mechanical ventilatory support

Based on:

- measured oxyhemoglobin saturation
- blood gas values
- minute ventilation
- patient-ventilator synchrony

Chest radiograph should verify...

- correct position of the endotracheal tube
- the distribution of pulmonary infiltration or edema
- identify complications from chest compression
- pneumonia

Mechanical ventilatory support

- Positive end-expiratory pressure (PEEP)
- Lung-protective strategy
- Titrated FiO_2
- Lung “recruitment maneuver” procedures
- Level of support may be gradually decreased

Lung-protective strategy

- low-volume/high-rate ventilation: maintain VT of 6 to 8 mL/kg
- inspiratory plateau pressure ≤ 30 cm H₂O
- Avoiding auto-PEEP (*intrinsic PEEP* or *gas trapping*)

Optimal FiO_2 and SpO_2

- Optimal FiO_2 during the immediate period after cardiac arrest is still debated
- Once the circulation is restored, monitor systemic arterial oxyhemoglobin saturation

Hyperoxia

- generating oxygen-derived **free radicals** during the reperfusion phase
 - increase brain lipid peroxidation
 - Increase metabolic dysfunctions
 - increase neurological degeneration
 - **worsen short-term neurological outcome**

Hyperventilation may be detrimental

- sustained hypocapnia may cause cerebral vasoconstriction, reduce CBF and exacerbate cerebral ischemic injury
- may compromise systemic blood flow because of occult or auto-PEEP
- hyperventilation should be avoided, especially in hypotensive patients
- hypercapnia and outcome : no consistent association in observational trials

Respiratory care

- **Ventilation**
- ***2015—Updated***
- Maintaining the **PaCO₂ within a normal physiological range**, taking into account any temperature correction, may be reasonable (**Class IIb**, LOE B-NR).
 - Normocarbica (end-tidal CO₂ 30–40 mm Hg or PaCO₂ 35–45 mm Hg) may be a reasonable goal unless patient factors prompt more individualized treatment.

Respiratory care

- **Oxygenation**
- **2015—New and Updated**
- To avoid hypoxia in adults with ROSC after cardiac arrest, it is reasonable to use the highest available oxygen concentration until the SaO_2 or the PaO_2 can be measured (Class IIa, LOE C-EO).
- When resources are available to titrate the FiO_2 and to monitor oxyhemoglobin saturation, it is reasonable to decrease the FiO_2 when oxyhemoglobin saturation is 100%, provided the oxyhemoglobin saturation can be maintained at 94% or greater (Class IIa, LOE C-LD).

Treatment of pulmonary embolism after CPR

- In post–cardiac arrest patients with arrest due to presumed or known pulmonary embolism, fibrinolytics may be considered. (Class IIb, LOE C 2010)

Sedation after cardiac arrest

- Post–cardiac arrest cognitive dysfunction may display **agitation or delirium** with **purposeless movement and are at risk of self-injury**
- Opioids, anxiolytics, sedative-hypnotic agents, α_2 -adrenergic agonists, and butyrophenones can be used
- If agitation is life-threatening, neuromuscular blocking agents can be used
 - To facilitate induced hypothermia and to control shivering

Sedation after cardiac arrest

- Cautiously with **daily interruptions** and **titrated to the desired effect**
 - **Caution for patients at high risk of seizures unless continuous EEG**
- A number of sedation **scales** and motor activity scales were developed
- **Neuromuscular blocker should be minimized & monitored with a nerve twitch stimulator.**

Sedation after cardiac arrest

- It is reasonable to consider the titrated use of sedation and analgesia in critically ill patients who require mechanical ventilation or shivering suppression during induced hypothermia after cardiac arrest (Class IIb, LOE C 2010).
- Duration of neuromuscular blocking agents should be kept to a minimum or avoided altogether.

Modifying outcomes from critical illness

- Cardiac arrest involve multiorgan ischemic injury and microcirculatory dysfunction
- Implementing a **protocol for goal-directed therapy**
 - Glucose, steroids, hemofiltration

Glucose control : 2010 Recommendation

- Higher glucose levels → ↑ mortality or worse neurological outcomes
- Strategies to target moderate glycemic control (144 to 180 mg/dL) may be considered in adult patients with ROSC after cardiac arrest (**Class IIb**, LOE B).
- A lower range 80 to 110 mg/dL should not be implemented after cardiac arrest due to the increased risk of hypoglycemia (**Class III**, LOE B).

Glucose control:

2015 evidence summary

- **No new evidence** that a specific target range for blood glucose management improved relevant clinical outcomes after cardiac arrest
- **No data** suggest that the approach to glucose management chosen for other critically ill patients should be modified for cardiac arrest patients.

Glucose control: 2015—Updated

- The benefit of any specific target range of glucose management is **uncertain** in adults with ROSC after cardiac arrest (**Class IIb**, LOE B-R).
- **ERC 2015 guideline** → Based on the available data, following ROSC maintain the blood glucose at **≤180 mg/dl and avoid hypoglycaemia.**

Steroids : 2010 guideline

- Corticosteroids have an essential role in the physiological response to severe stress
- **Relative adrenal insufficiency** in the post-cardiac arrest phase was **associated with higher rates of mortality**
- **Routine use of corticosteroids for patients with ROSC following cardiac arrest is uncertain**

Hemofiltration : 2010 guideline

- A method to **modify** the humoral response to the **ischemic-reperfusion injury**
- **No difference** in 6-month survival
- Future investigations are required to determine whether hemofiltration will improve outcome

Prognostication of neurological outcome in comatose survivors

- Neurological Assessment
- EEG
- Evoked Potentials
- Neuroimaging
- Blood and Cerebrospinal Fluid Biomarkers

Prognostication of outcome: timing

- **2015—New and Updated**
- The earliest time for prognostication using clinical examination **in patients treated with TTM**, where sedation or paralysis could be a confounder, **may be 72 hours after return to normothermia** (Class IIb, LOE C-EO).

Prognostication of outcome: **timing**

- We recommend the earliest time to prognosticate a poor neurologic outcome using clinical examination **in patients not treated with TTM is 72 hours after cardiac arrest** (Class I, LOE B-NR).
- This time until prognostication can be even **longer than 72 hours** after cardiac arrest **if the residual effect of sedation or paralysis confounds** the clinical examination (Class IIa, LOE C-LD).
 - Operationally, the timing for prognostication is typically **4.5 to 5 days after ROSC for patients treated with TTM**. This approach minimizes the possibility of obtaining false-positive results (ie, inaccurately suggesting a poor outcome) because of drug-induced depression of neurologic function.

Clinical examination findings

- **2015—New and Updated**
- In comatose patients who are **not treated with TTM**, the **absence of pupillary reflex to light at 72 hours or more** after cardiac arrest is a reasonable exam finding with which to predict poor neurologic outcome (**FPR, 0%**; 95% CI, 0%–8%; **Class IIa**, LOE B-NR).
- In comatose patients who are **treated with TTM**, the **absence of pupillary reflex to light at 72 hours or more** after cardiac arrest is useful to predict poor neurologic outcome (**FPR, 1%**; 95% CI, 0%–3%; **Class I**, LOE B-NR).

Clinical examination findings

- We recommend that, given their unacceptable FPRs, the findings of either **absent motor movements or extensor posturing** *should not be used alone* for predicting a poor neurologic outcome (**FPR, 10%**; 95% CI, 7%–15% to **FPR, 15%**; 95% CI, 5%–31%; **Class III: Harm**, LOE B-NR).
- The **motor examination** may be a reasonable means to **identify the population who need further prognostic testing** to predict poor outcome (**Class IIb**, LOE B-NR).

Clinical examination findings

- We recommend that the presence of myoclonus, which is distinct from status myoclonus, *should not be used* to predict poor neurologic outcomes because of the high FPR (FPR, 5%; 95% CI, 3%–8% to FPR, 11%; 95% CI, 3%–26%; **Class III: Harm**, LOE B-NR).
- In combination with other diagnostic tests at 72 or more hours after cardiac arrest, the presence of status myoclonus during the first 72 to 120 hours after cardiac arrest is a reasonable finding to help predict poor neurologic outcomes (FPR, 0%; 95% CI, 0%–4%; **Class IIa**, LOE B-NR).

EEG findings

- **2015—Updated**
- In comatose post–cardiac arrest patients who are treated with TTM, it may be reasonable to consider persistent absence of EEG reactivity to external stimuli at 72 hours after cardiac arrest, and persistent burst suppression on EEG after rewarming, to predict a poor outcome (FPR, 0%; 95% CI, 0%–3%; Class IIb, LOE B-NR).
- Intractable and persistent (more than 72 hours) status epilepticus in the absence of EEG reactivity to external stimuli may be reasonable to predict poor outcome (Class IIb, LOE B-NR).

EEG findings

- In comatose post–cardiac arrest patients who are **not treated with TTM**, it may be reasonable to consider the **presence of burst suppression on EEG at 72 hours or more** after cardiac arrest, in combination with other predictors, to predict a poor neurologic outcome (FPR, 0%; 95% CI, 0%–11%; **Class IIb**, LOE B-NR).

Evoked potentials

- **2015—Updated**
- In patients who are comatose after resuscitation from cardiac arrest regardless of treatment with TTM, it is reasonable to consider bilateral absence of the N20 SSEP wave 24 to 72 hours after cardiac arrest or after rewarming a predictor of poor outcome (FPR, 1%; 95% CI, 0%–3%; **Class IIa, LOE B-NR**).

Imaging tests

- **2015—New**
- In patients who are comatose after resuscitation from cardiac arrest and **not treated with TTM**, it may be reasonable to use the presence of a **marked reduction of the GWR on brain CT** obtained within 2 hours after cardiac arrest to predict poor outcome (**Class IIb**, LOE B-NR).
- It may be reasonable to **consider extensive restriction of diffusion on brain MRI** at 2 to 6 days after cardiac arrest in combination with other established predictors to predict a poor neurologic outcome (**Class IIb**, LOE B-NR).

有助於判斷 神經系統結果不佳的 臨床症狀 *

- 心臟停止後 72 小時以上對光沒有瞳孔反射
- 心臟停止後前 72 小時內，出現持續性肌陣攣 (status myoclonus) (與單一肌陣攣發作不同)
- 心臟停止或回溫後 24 至 72 小時，仍未出現體感誘發電位的大腦皮質反應 N20
- 心臟停止後 2 小時內拍攝的腦部 CT 顯示，灰質白質比明顯降低
- 心臟停止後 2 至 6 天，腦部 MRI 出現廣泛性的擴散限制
- 心臟停止後 72 小時，對外界刺激仍持續缺乏 EEG 反應
- 回溫後 EEG 上仍出現持續性猝發抑制，或頑固型癲癇連續狀態

不應單獨使用缺乏運動動作、伸展肌姿勢或肌陣攣，預測病患的結果。

* 務必謹慎考慮休克、體溫、代謝失常、曾使用鎮靜劑或神經肌肉阻斷劑，以及其他臨床因素，因為可能影響部分檢驗的結果或判讀。

縮寫：CT (Computed Tomography，電腦斷層掃描)；

EEG (electroencephalogram，腦電波圖)；

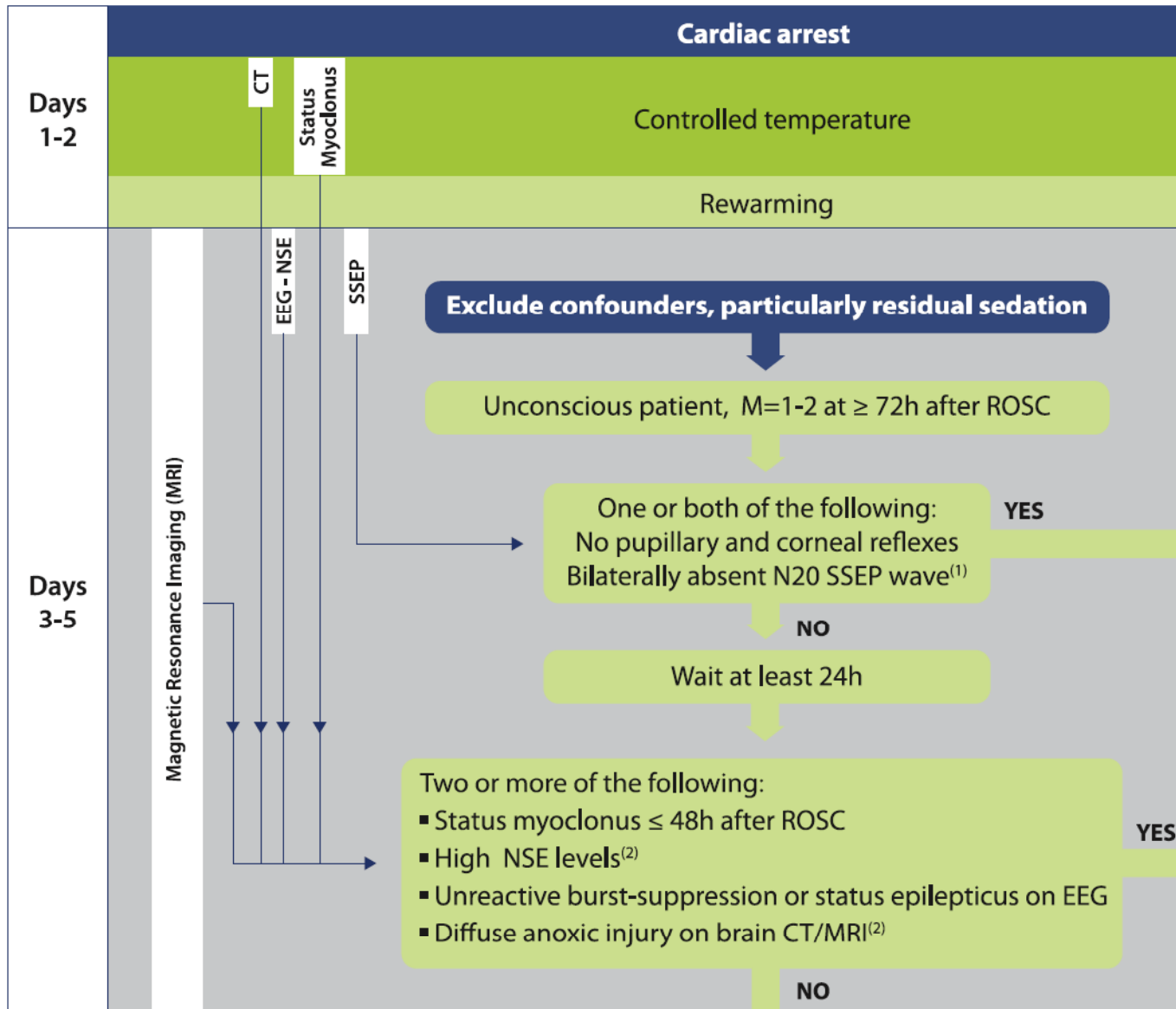
MRI (Magnetic Resonance Imaging，磁振造影)。

Blood markers

- **2015—Updated**
- Given the possibility of high FPRs, blood levels of NSE and S-100B **should not be used alone** to predict a poor neurologic outcome (**Class III: Harm**, LOE C-LD).
- When performed with other prognostic tests at 72 hours or more after cardiac arrest, it may be reasonable to consider **high serum values of NSE at 48 to 72 hours after cardiac arrest** to support the prognosis of a poor neurologic outcome (**Class IIb**, LOE B-NR), especially if **repeated sampling reveals persistently high values** (**Class IIb**, LOE C-LD).

2015 ERC Guideline

J.P. Nolan et al. /
Resuscitation 95 (2015)
202–222



Use multimodal prognostication whenever possible

⁽¹⁾ At ≥ 24 h after ROSC in patients not treated with targeted temperature

⁽²⁾ See text for details.

Organ donation

- **2015—Updated and New**
- We recommend that **all patients** who are **resuscitated from cardiac arrest** but who subsequently **progress to death or brain death** be **evaluated for organ donation** (**Class I**, LOE B-NR).
- Patients who **do not have ROSC** after resuscitation efforts and who would otherwise have termination of efforts **may be considered candidates for kidney or liver donation** in settings where programs exist (**Class IIb**, LOE B-NR).

Ventilation

Hemodynamics

Cardiovascular

Neurological

Metabolic

Summary of **multiple system approach** to **post-cardiac arrest care**

- Ventilation
- Hemodynamics
- Cardiovascular
- Neurological
- Metabolic

Table 1. Multiple System Approach to Post-Cardiac Arrest Care

Ventilation	Hemodynamics	Cardiovascular	Neurological	Metabolic
<ul style="list-style-type: none"> ● Capnography <ul style="list-style-type: none"> ● Rationale: Confirm secure airway and titrate ventilation ● Endotracheal tube when possible for comatose patients ● $P_{\text{etCO}_2} \sim 35\text{--}40$ mm Hg ● $P_{\text{aco}_2} \sim 40\text{--}45$ mm Hg ● Chest X-ray <ul style="list-style-type: none"> ● Rationale: Confirm secure airway and detect causes or complications of arrest: pneumonitis, pneumonia, pulmonary edema ● Pulse Oximetry/ABG <ul style="list-style-type: none"> ● Rationale: Maintain adequate oxygenation and minimize FiO_2 ● $\text{SpO}_2 \geq 94\%$ ● $\text{PaO}_2 \sim 100$ mm Hg ● Reduce FiO_2 as tolerated 	<ul style="list-style-type: none"> ● Frequent Blood Pressure Monitoring/Arterial-line <ul style="list-style-type: none"> ● Rationale: Maintain perfusion and prevent recurrent hypotension ● Mean arterial pressure ≥ 65 mm Hg or systolic blood pressure ≥ 90 mm Hg ● Treat Hypotension <ul style="list-style-type: none"> ● Rationale: Maintain perfusion <ul style="list-style-type: none"> ● Fluid bolus if tolerated ● Dopamine 5–10 mcg/kg per min ● Norepinephrine 0.1–0.5 mcg/kg per min ● Epinephrine 0.1–0.5 mcg/kg per min 	<ul style="list-style-type: none"> ● Continuous Cardiac Monitoring <ul style="list-style-type: none"> ● Rationale: Detect recurrent arrhythmia ● No prophylactic antiarrhythmics ● Treat arrhythmias as required ● Remove reversible causes ● 12-lead ECG/Troponin <ul style="list-style-type: none"> ● Rationale: Detect Acute Coronary Syndrome/ST-Elevation Myocardial Infarction; Assess QT interval ● Treat Acute Coronary Syndrome <ul style="list-style-type: none"> ● Aspirin/heparin ● Transfer to acute coronary treatment center ● Consider emergent PCI or fibrinolysis 	<ul style="list-style-type: none"> ● Serial Neurological Exam <ul style="list-style-type: none"> ● Rationale: Serial examinations define coma, brain injury, and prognosis ● Response to verbal commands or physical stimulation ● Pupillary light and corneal reflex, spontaneous eye movement ● Gag, cough, spontaneous breaths ● EEG Monitoring If Comatose <ul style="list-style-type: none"> ● Rationale: Exclude seizures ● Anticonvulsants if seizing ● Core Temperature Measurement If Comatose <ul style="list-style-type: none"> ● Rationale: Minimize brain injury and improve outcome ● Prevent hyperpyrexia $>37.7^\circ\text{C}$ ● Induce therapeutic hypothermia if no contraindications ● Cold IV fluid bolus 30 mL/kg if no 	<ul style="list-style-type: none"> ● Serial Lactate <ul style="list-style-type: none"> ● Rationale: Confirm adequate perfusion ● Serum Potassium <ul style="list-style-type: none"> ● Rationale: Avoid hypokalemia which promotes arrhythmias ● Replace to maintain $\text{K} > 3.5$ mEq/L ● Urine Output, Serum Creatinine <ul style="list-style-type: none"> ● Rationale: Detect acute kidney injury ● Maintain euvolemia ● Renal replacement therapy if indicated

<ul style="list-style-type: none"> ● Pulse Oximetry/ABG 	...	<ul style="list-style-type: none"> ● Treat Acute Coronary Syndrome 	<ul style="list-style-type: none"> ● Core Temperature Measurement If Comatose 	<ul style="list-style-type: none"> ● Urine Output, Serum Creatinine
<ul style="list-style-type: none"> ● Rationale: Maintain adequate oxygenation and minimize F_{iO_2} 	...	<ul style="list-style-type: none"> ● Aspirin/heparin 	<ul style="list-style-type: none"> ● Rationale: Minimize brain injury and improve outcome 	<ul style="list-style-type: none"> ● Rationale: Detect acute kidney injury
<ul style="list-style-type: none"> ● $Spo_2 \geq 94\%$...	<ul style="list-style-type: none"> ● Transfer to acute coronary treatment center 	<ul style="list-style-type: none"> ● Prevent hyperpyrexia $>37.7^\circ C$ 	<ul style="list-style-type: none"> ● Maintain euvolemia
<ul style="list-style-type: none"> ● $Pao_2 \sim 100$ mm Hg 	...	<ul style="list-style-type: none"> ● Consider emergent PCI or fibrinolysis 	<ul style="list-style-type: none"> ● Induce therapeutic hypothermia if no contraindications 	<ul style="list-style-type: none"> ● Renal replacement therapy if indicated
<ul style="list-style-type: none"> ● Reduce F_{iO_2} as tolerated 	...		<ul style="list-style-type: none"> ● Cold IV fluid bolus 30 mL/kg if no contraindication 	
<ul style="list-style-type: none"> ● Pao_2/F_{iO_2} ratio to follow acute lung injury 	...		<ul style="list-style-type: none"> ● Surface or endovascular cooling for $32^\circ C-34^\circ C \times 24$ hours 	
	...		<ul style="list-style-type: none"> ● After 24 hours, slow rewarming $0.25^\circ C/hr$ 	
<ul style="list-style-type: none"> ● Mechanical Ventilation 	...	<ul style="list-style-type: none"> ● Echocardiogram 	<ul style="list-style-type: none"> ● Consider Non-enhanced CT Scan 	<ul style="list-style-type: none"> ● Serum Glucose
<ul style="list-style-type: none"> ● Rationale: Minimize acute lung injury, potential oxygen toxicity 	...	<ul style="list-style-type: none"> ● Rationale: Detect global stunning, wall-motion abnormalities, structural problems or cardiomyopathy 	<ul style="list-style-type: none"> ● Rationale: Exclude primary intracranial process 	<ul style="list-style-type: none"> ● Rationale: Detect hyperglycemia and hypoglycemia
<ul style="list-style-type: none"> ● Tidal Volume 6–8 mL/kg 	...			<ul style="list-style-type: none"> ● Treat hypoglycemia (<80 mg/dL) with dextrose
<ul style="list-style-type: none"> ● Titrate minute ventilation to $Perco_2 \sim 35-40$ mm Hg $Paco_2 \sim 40-45$ mm Hg 	...			<ul style="list-style-type: none"> ● Treat hyperglycemia to target glucose 144–180 mg/dL
<ul style="list-style-type: none"> ● Reduce F_{iO_2} as tolerated to keep Spo_2 or $Sao_2 \geq 94\%$...			<ul style="list-style-type: none"> ● Local insulin protocols
	...	<ul style="list-style-type: none"> ● Treat Myocardial Stunning 	<ul style="list-style-type: none"> ● Sedation/Muscle Relaxation 	<ul style="list-style-type: none"> ● Avoid Hypotonic Fluids
	...	<ul style="list-style-type: none"> ● Fluids to optimize volume status (requires clinical judgment) 	<ul style="list-style-type: none"> ● Rationale: To control shivering, agitation, or ventilator desynchrony as needed 	<ul style="list-style-type: none"> ● Rationale: May increase edema, including cerebral edema
	...	<ul style="list-style-type: none"> ● Dobutamine 5–10 mcg/kg per min 		
	...	<ul style="list-style-type: none"> ● Mechanical augmentation (IABP) 		

Ventilation: Capnography

- Rationale: Confirm secure airway and titrate ventilation
- Endotracheal tube when possible for comatose patients
- PETCO₂ : 30 - 40 mm Hg
- PaCO₂ : 35 - 45 mm Hg

Ventilation: Chest X-ray

- Confirm secure airway
- Detect causes or complications of arrest: pneumonitis, pneumonia, pulmonary edema

Ventilation: Pulse oximetry/ABG

- Rationale: Maintain adequate oxygenation and minimize FiO_2
- After ROSC, use the highest available oxygen concentration until PaO_2 or SaO_2 available
- Reduce FiO_2 as tolerated to keep $\text{SpO}_2 \geq 94\%$
- $\text{PaO}_2/\text{FiO}_2$ ratio to follow acute lung injury

Mechanical ventilation

- Rationale: Minimize acute lung injury, potential oxygen toxicity
- Tidal Volume: 6 - 8 mL/kg
- Titrate minute ventilation to PETCO₂:30-40 mm Hg, PaCO₂: 35-45 mm Hg
- Reduce FiO₂ as tolerated to keep SaO₂ \geq 94%

Hemodynamics: Blood pressure monitoring/Arterial-line

- Rationale: maintain perfusion and prevent recurrent hypotension
- Mean arterial pressure \geq 65 mm Hg or systolic blood pressure \geq 90 mm Hg

Hemodynamics: Treat hypotension

- Rationale: Maintain perfusion
- Fluid bolus if tolerated
- Dopamine 5–10 mcg/kg per min
- Norepinephrine 0.1–0.5 mcg/kg per min
- Epinephrine 0.1–0.5 mcg/kg per min

Cardiovascular: Continuous cardiac monitoring

- Rationale: Detect recurrent arrhythmia
- No prophylactic antiarrhythmics
- Treat arrhythmias as required
- Remove reversible causes
- 12-lead ECG/Troponin: Detect acute coronary syndrome/ST elevation myocardial Infarction; assess QT interval

Cardiovascular: ACS

- Aspirin/heparin
- Transfer to acute coronary treatment center
- Consider emergent PCI or fibrinolysis

Cardiovascular: Treat myocardial stunning

- Echocardiogram: Detect global stunning, wall-motion abnormalities, structural problems or cardiomyopathy
- Fluids to optimize volume status (requires clinical judgment)
- Dobutamine 5–10 mcg/kg per min
- Mechanical augmentation (IABP)

Neurological: Serial neurological exam.

- Rationale: Serial examinations define coma, brain injury, and prognosis
- Response to verbal commands or physical stimulation
- Pupillary light and corneal reflex, spontaneous eye movement
- Gag, cough, spontaneous breaths

Neurological: EEG monitoring if comatose

- Rationale: Exclude seizures
- Anticonvulsants if seizing

Core temperature measurement

- Rationale: minimize brain injury and improve outcome
- Prevent hyperpyrexia
- *Targeted temperature management* if no contraindications
- Surface or endovascular cooling 32 ° C–36 ° C for at least 24 hours
- After 24 hours, slow rewarming 0.25 ° C/hr

Neurological

- Consider non-enhanced CT scan: exclude primary intracranial process
- Sedation/muscle relaxation: to control shivering, agitation, or ventilator desynchrony as needed

Metabolic

- Serial lactate: confirm adequate perfusion
- Serum potassium: avoid hypo- or hyperkalemia which promotes arrhythmias
- Urine output, serum creatinine: detect acute kidney injury & renal replacement therapy if indicated

Metabolic

- Serum Glucose:
 - Treat hypoglycemia (<80 mg/dL)
 - Target range of glucose management is uncertain in adults with ROSC after cardiac arrest
- Avoid hypotonic fluids: may increase edema, including cerebral edema

Summary

The goal of immediate post - cardiac arrest care:

- Optimize tissue perfusion
- Restore metabolic homeostasis
- Support organ system function
- Increase the likelihood of intact neurological survival

Multiple system approach

Summary of **key issues** and **major changes**

- **Emergency coronary angiography** → for STEMI and for non STEMI WITH hemodynamically or electrically unstable
- **TTM** → a range of temperatures may be **acceptable** to target in the post–cardiac arrest period.
- **After TTM** → the prevention of fever is considered benign and therefore is reasonable to pursue

Summary of **key issues** and **major changes**

- **Identification and correction of hypotension** → in the immediate post–cardiac arrest period.
- **Prognostication timing** → after the completion of TTM; for those who do not have TTM, 72 hours later after ROSC
- **All patients who progress to brain death or circulatory death** → should be considered potential organ donors

Thanks for your attention !!