

Pitfalls in management of post cardiac arrest

CARDIOLOGY
on the move

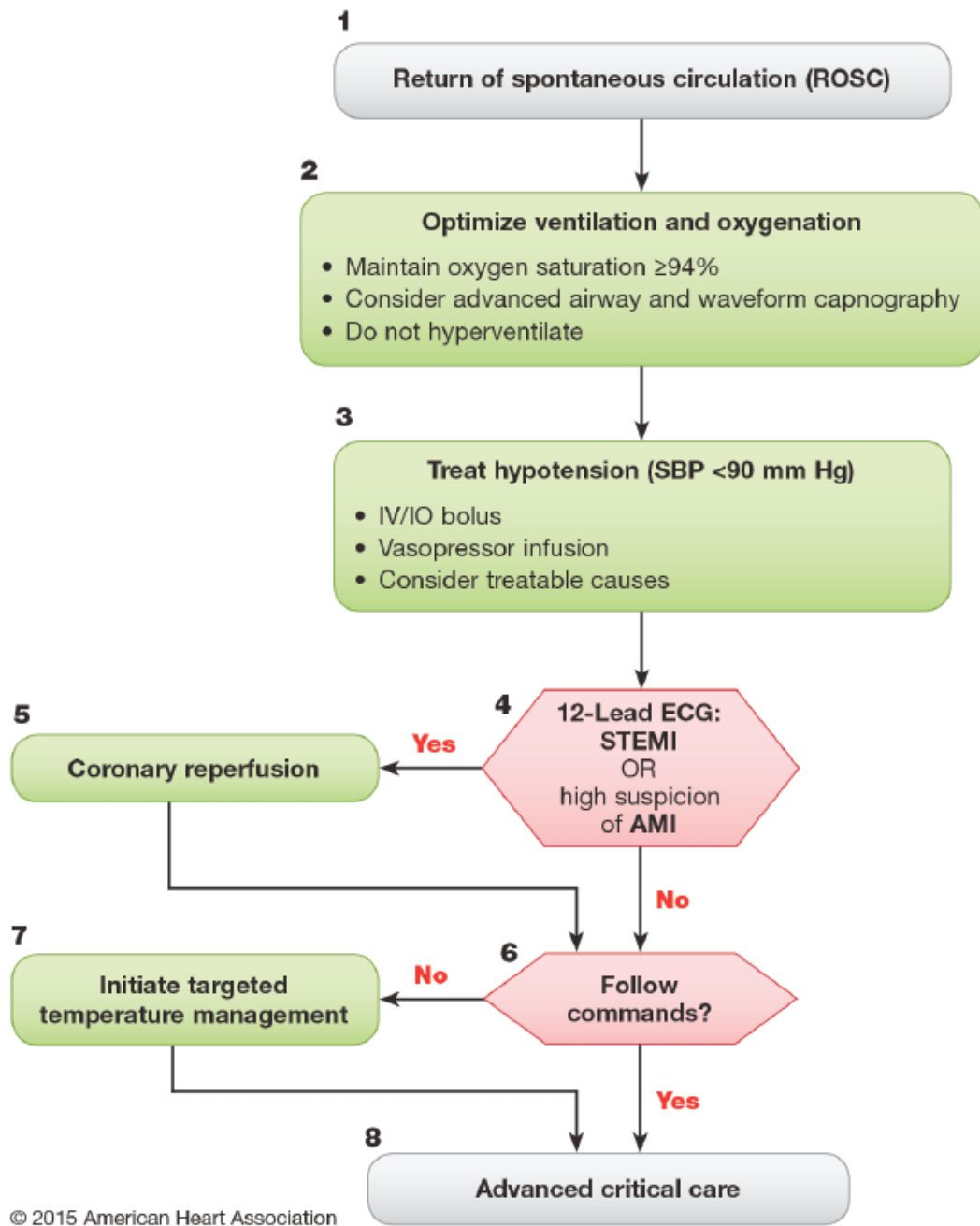


Ratchanee Lee, MD.

CPR training center, Cardiology unit, Department of Internal
Medicine

Faculty of Medicine, Ramathibodi hospital
Mahidol University.

Adult Immediate Post-Cardiac Arrest Care Algorithm—2015 Update

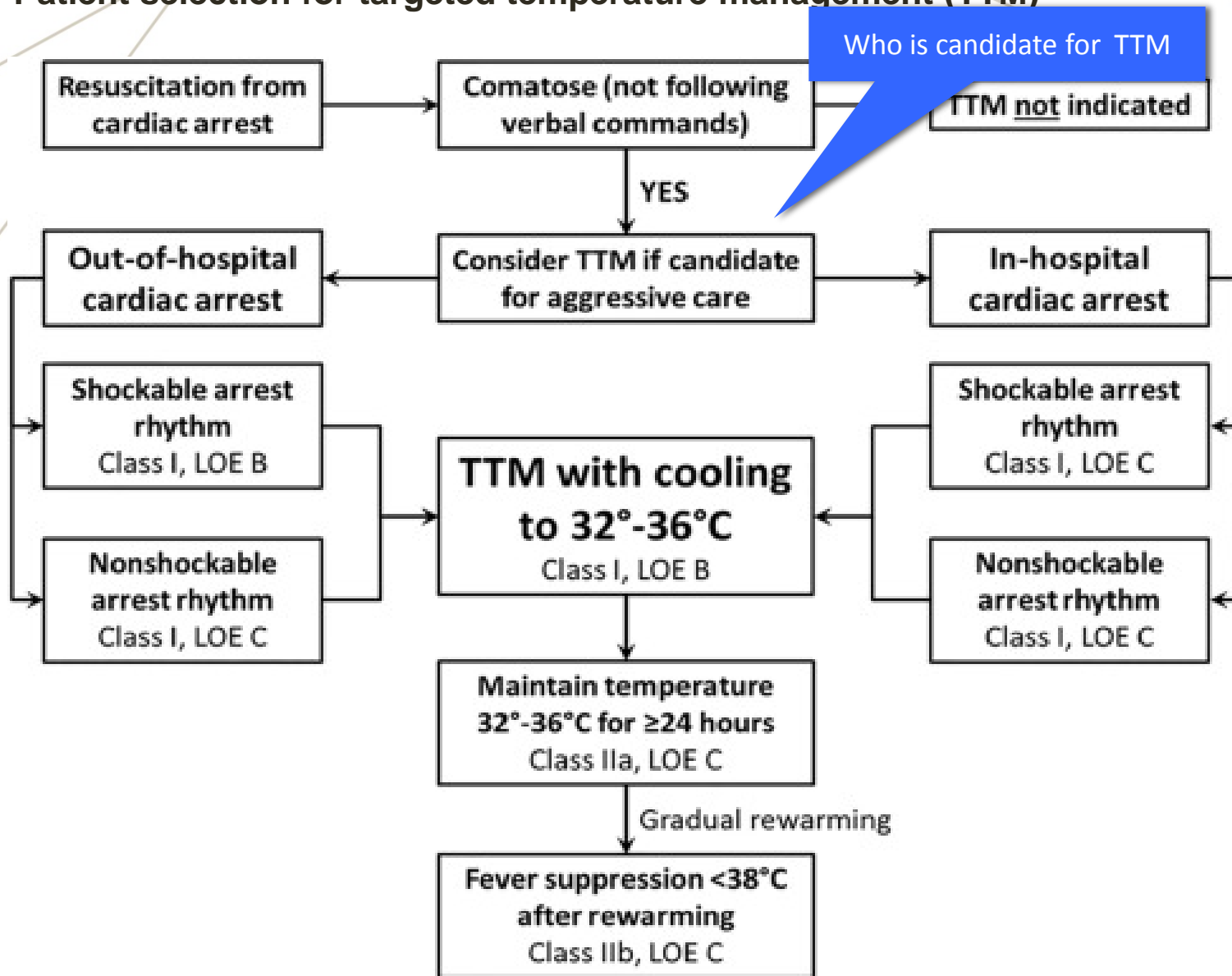


Doses/Details
<p>Ventilation/oxygenation: Avoid excessive ventilation. Start at 10 breaths/min and titrate to target PETCO₂ of 35-40 mm Hg. When feasible, titrate FIO₂ to minimum necessary to achieve SpO₂ ≥94%.</p>
<p>IV bolus: Approximately 1-2 L normal saline or lactated Ringer's</p>
<p>Epinephrine IV infusion: 0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)</p>
<p>Dopamine IV infusion: 5-10 mcg/kg per minute</p>
<p>Norepinephrine IV infusion: 0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)</p>
Reversible Causes
<ul style="list-style-type: none"> • Hypovolemia • Hypoxia • Hydrogen ion (acidosis) • Hypo-/hyperkalemia • Hypothermia • Tension pneumothorax • Tamponade, cardiac • Toxins • Thrombosis, pulmonary • Thrombosis, coronary

Targeted temperature management



Patient selection for targeted temperature management (TTM)



The New England Journal of Medicine

Copyright © 2002 by the Massachusetts Medical Society

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*

In patients who have been successfully resuscitated after cardiac arrest due to ventricular fibrillation, therapeutic mild hypothermia increased the rate of a favorable neurologic outcome and reduced mortality.

IHCA

Table 1. Nonrandomized Comparison of Therapeutic Hypothermia for In-Hospital Cardiac Arrest

Author	Year	Reference	Study Type	Patients (n)		Hospital Survival %		Good Neuro Outcomes %	
				TH	C	TH	C	TH	C
Arrich	2007	50	Multicenter observational European registry	43	59	39	60	28	29
Rittenberger	2004	51	Retrospective observational Univ. of Pitt	13	27	14	14	8	7
Kony	2012	52	Retrospective historic Beth Israel	17	16	29	31	24	31
Nichol	2013	53	Multicenter prospective observational USA	214	8102	27.1	31.0	18.7	20.1

Abbreviations: C, control group (did not receive therapeutic hypothermia); n, number; TH, received therapeutic hypothermia.

Non shockable rhythm

Table 2. Effect of Therapeutic Hypothermia on Patients After Cardiac Arrest Associated With Nonshockable Rhythms

Author	Year	Ref	Study			Site & Years of Study	Location of Arrest	# of Patients		Hospital Survival (%) [*]			Good Neuro Outcome (%) [†]		
			Type of Control	Design	Characteristics			TH	C	TC	C	SS	TC	C	SS
Hachimi-Idrissi	2001	69	RCT	P	Single Center	Before 2001 Belgium	OOH	16	14	19 [‡]	7 [‡]	NS [§]	12.5	0	NS
Kim	2007	70	RCT	P	Single Center Pre Hospital Cooling	2004-2006 Seattle	OOH	34	40	6	20	NS [§]	6	18	NS
Arrich	2007	50	Conc	P	Multicenter (19) Euro Registry	2003-2005 Europe	Both	124	73	36	19	S	28	19	NS
Bro-Jeppesen	2009	71	Hx	P	Single Center for City	2004-2006 v 2002-2004 Copenhagen	OOH	13	21	54	24	NS [§]	—	—	—
Don	2009	72	Hx	R	Single Center	2000-2004 Seattle	OOH	122	191	21	19	NS	11	9	NS
Dumas	2011	48	Hx	P	Single Center	2000-2009 Paris	OOH	261	176	—	—	—	15	17	NS
Lindner	2013	73	Conc	P	3 Hospitals	2004-2008 Norway	OOH	143	120	17	9	NS	14	8	NS
Lundbye	2012	74	Hx	R	Single Center	2007-2010 v 2004-2007 USA	Both	52	48	38	19	S [§]	29	13	S
Oddo	2006	75	Hx	R	Single Center	2002-2004 v 1999-2002 Switzerland	OOH	12	11	17	9	NS [§]	17	0	NS
Rittenberger	2008	51	Conc	R	Single Center	2005-2007 USA	Both	60	60	15	17	NS	8	10	NS
Storm	2012	76	Hx	P	Single Center	2007-2010 v 2002-2007 Berlin	OOH	87	88	41	37	NS [§]	27.5	18.2	NS
Testori	2011	39	Conc	R	Single Center	1992-2009 Vienna	OOH	135	239	39 [‡]	25 [‡]	S	35 [‡]	23 [‡]	S
Vaahersalo	2013	77	Conc	P	Multi-Center (21)	2010-2011 Finland	OOH	70	153	37	31	NS [§]	19 [†]	16 [†]	NS
Whitfield	2009	78	Conc	R	Single Center	2001-2007 Australia	OOH	15	13	†	†	NS	†	†	NS

Abbreviations: both, OOH and in-hospital; C, controls (ie, no TH); Conc, concurrent; Hx, historical; NS, not significant; OOH, out-of-hospital; P, prospective; R, retrospective; RCT, randomized control trial; S, significant; SS, statistical significance; TH, therapeutic hypothermia.

* Survival at time of hospital discharge unless otherwise stated in comments.

† At hospital discharge unless otherwise noted in comments.

‡ Outcome at 6 months.

§ Per Fisher's exact test by author of this essay.

|| Outcome at 1 year.

¶ Raw data not in primary reference but authors stated that these outcomes were not different in the 2 groups.

RESEARCH ARTICLE

Outcomes of Adult In-Hospital Cardiac Arrest Treated with Targeted Temperature Management: A Retrospective Cohort Study

Chih-Hung Wang^{1,2}^e, Chien-Hua Huang¹^e, Wei-Tien Chang¹, Min-Shan Tsai¹, Ping-Hsun Yu³, Yen-Wen Wu^{4,5,6}, Wen-Jone Chen^{1,7} *

1 Department of Emergency Medicine, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan, **2** Graduate Institute of Clinical Medicine, College of Medicine, National Taiwan University, Taipei, Taiwan, **3** Department of Emergency Medicine, Taipei Hospital, Ministry of Health Affairs, Taipei, Taiwan, **4** Department of Internal Medicine, National Taiwan University College of Medicine, Taipei, Taiwan, **5** Division of Geriatric Medicine, National Taiwan University College of Medicine, Taipei, Taiwan, **6** Division of Geriatric Medicine, Taipei Medical Center, Taipei, Taiwan, **7** Division of Geriatric Medicine, Taipei Medical Center, Taipei, Taiwan

Methods

We conducted a retrospective observational study in a single medical centre. We included 678 adult patients suffering IHCA between 2000 and 2014. We used multivariate logistic regression analysis to evaluate associations between TTM use and neurological outcomes.

Results

We included a total of 678 patients in the study. Most (81.1%) patients met at least one of the proposed exclusion criteria. Among patients who survived to hospital discharge, favourable neurological status at discharge was associated with TTM use (OR: 3.74, 95% CI: 1.03–13.7; *p* = 0.043), but it was not associated with survival in the emergency department (OR: 0.84, 95% CI: 0.40–1.74; *p* = 0.64) and having vasopressors in place at the time of arrest was inversely associated with TTM use (OR: 0.08, 95% CI: 0.004–0.42; *p* = 0.02).

Conclusion

TTM might be associated with favourable neurological outcome of IHCA patients, irrespective of arrest rhythms. The prevalence of proposed exclusion criteria for TTM was high among IHCA patients, but these factors did not influence the use of TTM in clinical practice or neurological outcomes after IHCA.


Therapeutic Hypothermia after Prolonged Cardiac Arrest: Case Report with Review of Literature

[Gautam Rawal](#),¹ [Sankalp Yadav](#),² and [Nitin Garg](#)³

¹ Attending Consultant-Critical Care, Rockland Hospital, Qutab Institutional Area, New Delhi, India.

² General Duty Medical Officer-II, Chest Clinic, Moti Nagar, New Delhi, India.

³ Senior Consultant and Head-Critical Care, Rockland Hospital, Qutab Institutional Area, New Delhi, India.

 Corresponding author.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR: Dr. Gautam Rawal, Flat No. 417, Dhruva Apartments, Plot No. 4, I P Extension, Patparganj, Delhi-110092, India. E-mail: drgautamrawal@hotmail.com

[Author information](#) ▼ [Article notes](#) ► [Copyright and License information](#) ►

Abstract

[Go to:](#) 

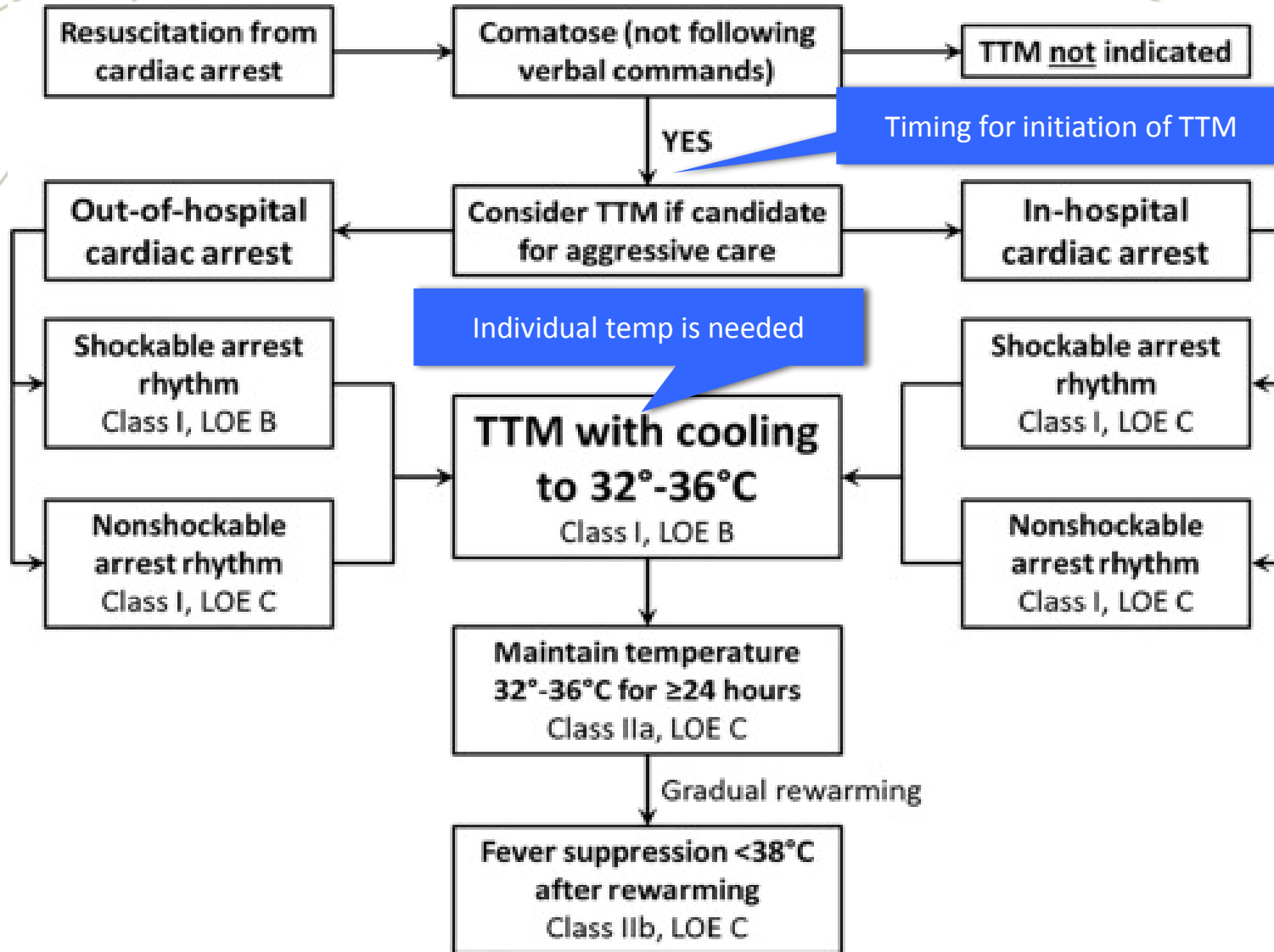
Patients who survive cardiac arrest often develop severe neurological dysfunction due to the hypoxic brain injury and reperfusion induced cell death. Therapeutic hypothermia (TH) has become a standard therapy of cerebral protection following the successful return of spontaneous circulation in patients of out-of-hospital cardiac arrest, according to American heart association guidelines. This is a case report of a 30-year-old patient who developed in-hospital cardiac arrest and was revived after prolonged cardiopulmonary resuscitation (CPR) and also required primary angioplasty. TH was then established with local measures for 24 hours for cerebral protection. The patient was gradually and successfully weaned off from ventilator with no neurological impairment. There is an increasing evidence of TH and its protective mechanisms in patients with non-shockable arrest rhythms with particular emphasis on neurological outcomes. This article emphasizes the role of TH in every successful CPR irrespective of the cardiac rhythm.

Keywords: Asystole, Cardiopulmonary resuscitation, Neurological outcome, Return of spontaneous circulation

Good candidate for TTM post cardiac arrest tend to get better result

- Shockable rhythm OHCA (data from HACA 2002)
- Young age
- ACS caused cardiac arrest with successful PCI
- Short CPR time, Short time to begin CPR
- Less co-morbidities
- Witness arrest with bystander CPR
- **Data TTM in IHCA is not RCT outcome depend on other prognostic factors**

Patient selection for targeted temperature management (TTM)



Cardiac Arrest

Pilot Trial Comparing 2 Levels of Target Temperature

Esteban Lopez-de-Sa, MD, FESC; Juan R. Rey, MD; Eduardo Armada, MD; Pablo Salinas, MD; Ana Viana-Tejedor, MD; Sandra Espinosa-Garcia, MD; Mercedes Martinez-Moreno, MD; Ervigio Corral, MD; Jose Lopez-Sendon, MD, PhD, FESC

Background—It is recommended that comatose survivors of out-of-hospital cardiac arrest should be cooled to 32° to 34°C for 12 to 24 hours. However, the optimal level of cooling is unknown. The aim of this pilot study was to obtain initial data on the effect of different levels of hypothermia. We hypothesized that deeper temperatures will be associated with better survival and neurological outcome.

Methods and Results—Patients were eligible if they had a witnessed out-of-hospital cardiac arrest from March 2008 to August 2011. Target temperature was randomly assigned to 32°C or 34°C. Enrollment was stratified on the basis of the initial rhythm as shockable or asystole. The target temperature was maintained during 24 hours followed by 12 to 24 hours of controlled rewarming. The primary outcome was survival free from severe dependence (Barthel Index score ≥ 60 points) at 6 months. Thirty-six patients were enrolled in the trial (26 shockable rhythm, 10 asystole), with 18 assigned to 34°C and 18 to 32°C. Eight of 18 patients in the 32°C group (44.4%) met the primary end point compared with 2 of 18 in the 34°C group (11.1%) (log-rank $P=0.12$). All patients whose initial rhythm was asystole died before 6 months in both groups. Eight of 13 patients with initial shockable rhythm assigned to 32°C (61.5%) were alive free from severe dependence at 6 months compared with 2 of 13 (15.4%) assigned to 34°C (log-rank $P=0.029$). The incidence of complications was similar in both groups except for the incidence of clinical seizures, which was lower (1 versus 11; $P=0.0002$) in patients assigned to 32°C compared with 34°C. On the contrary, there was a trend toward a higher incidence of bradycardia (7 versus 2; $P=0.054$) in patients assigned to 32°C. Although potassium levels decreased to a greater extent in patients assigned to 32°C, the incidence of hypokalemia was similar in both groups.

Conclusions—The findings of this pilot trial suggest that a lower cooling level may be associated with a better outcome in patients surviving out-of-hospital cardiac arrest secondary to a shockable rhythm. The benefits observed here merit further investigation in a larger trial in out-of-hospital cardiac arrest patients with different presenting rhythms.

Clinical Trial Registration—URL: <http://www.clinicaltrials.gov>. Unique identifier: NCT01155622.

(*Circulation*. 2012;126:2826-2833.)

Circulation. 2012;126:2826-2833

ORIGINAL ARTICLE

Targeted Temperature Management at 33°C versus 36°C after Cardiac Arrest

Niklas Nielsen, M.D., Ph.D., Jørn Wetterslev, M.D., Ph.D., Tobias Cronberg, M.D., Ph.D., David Erlinge, M.D., Ph.D., Yvan Gasche, M.D., Christian Hassager, M.D., D.M.Sci., Janneke Horn, M.D., Ph.D., Jan Hovdenes, M.D., Ph.D., Jesper Kjaergaard, M.D., D.M.Sci., Michael Kuiper, M.D., Ph.D., Pascal Stammet, M.D., Michael Wanscher, M.D., Ph.D., Anders Åneman, M.D., Ph.D., Nawaf Al-Sayid, M.D., Søren Boesgaard, M.D., D.M.Sci., John Bro-Jeppesen, M.D., Ph.D., Jan Frederik Bugge, M.D., Ph.D., Christopher D. Devereaux, M.D., Ph.D., Nicole P. Juffermans, M.D., Ph.D., Matty Koopmans, M.D., Ph.D., Lars Køber, M.D., D.M.Sci., Jørund Langørgen, M.D., Ph.D., Jacob Eifer Møller, M.D., D.M.Sci., Malin Rundgren, M.D., Ph.D., Christian Rylander, M.D., Ph.D., Ondrej Smid, M.D., Ph.D., Per Winkel, M.D., D.M.Sci., and Hans Friberg, M.D., Ph.D., for the TTM Trial Investigators

ABSTRACT

BACKGROUND

Unconscious survivors of out-of-hospital cardiac arrest have a high risk of death or poor neurologic function. Therapeutic hypothermia is recommended by international guidelines, but the supporting evidence is limited, and the target temperature associated with the best outcome is unknown. Our objective was to compare two target temperatures, both intended to prevent fever.

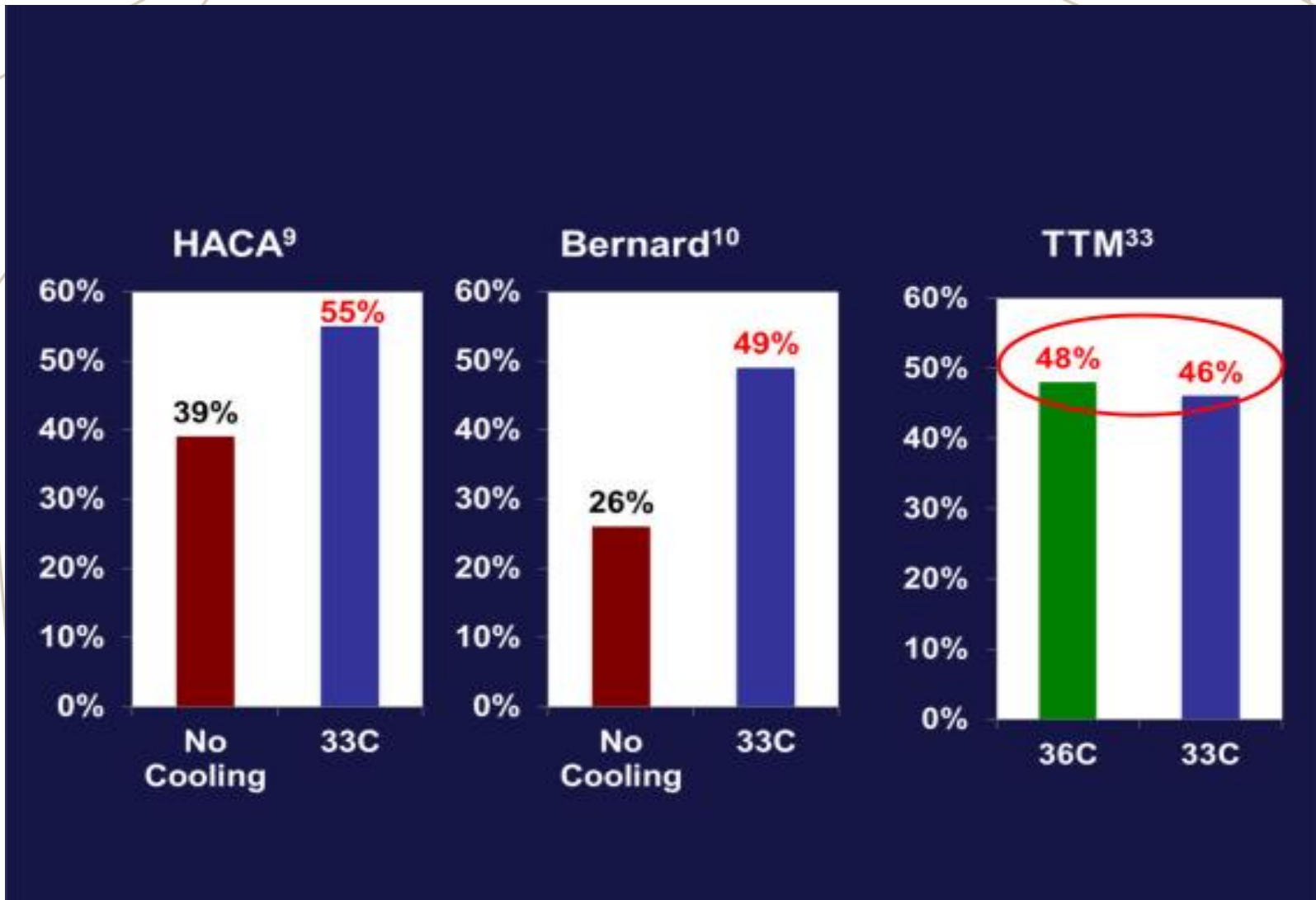
METHODS

In an international trial, we randomly assigned 950 unconscious adults after out-of-hospital cardiac arrest of presumed cardiac cause to targeted temperature management at either 33°C or 36°C. The primary outcome was all-cause mortality through the end of the trial. Secondary outcomes included a composite of poor neurologic function or death at 180 days, as evaluated with the Cerebral Performance Category (CPC) scale and the modified Rankin scale.

RESULTS

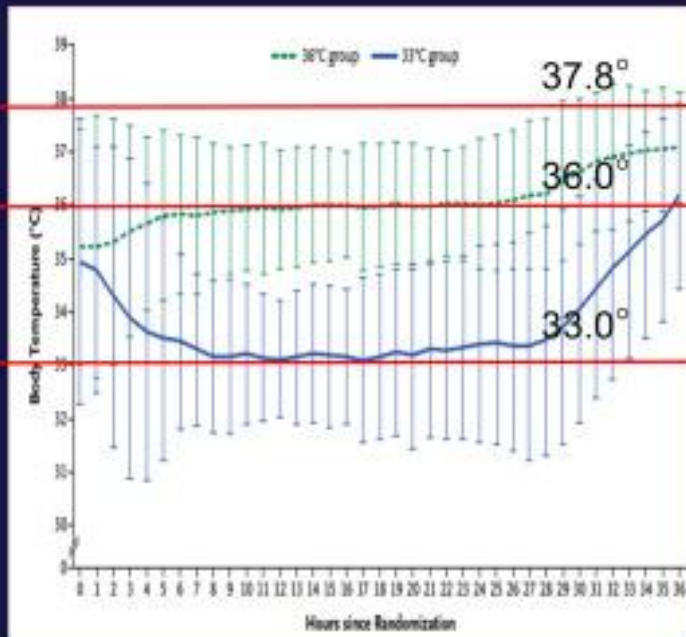
In total, 950 patients were included in the trial. At the end of the trial, 475 patients

In unconscious survivors of out-of-hospital cardiac arrest of presumed cardiac cause, hypothermia at a targeted temperature of 33°C did not confer a benefit as compared with a targeted temperature of 36°C.

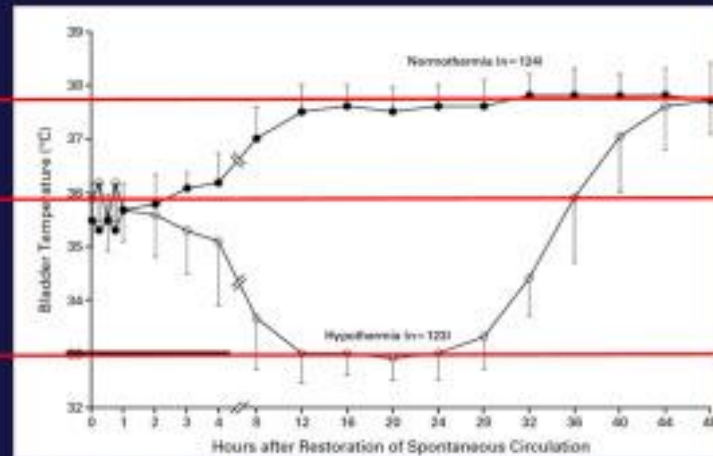


Good Neurologic Outcomes in Major Temperature Management Trials. (Modified from Abella B. at <https://www.med.upenn.edu/resuscitation/ttm-video.shtml>).

TTM Trial³³



HACA Trial⁹



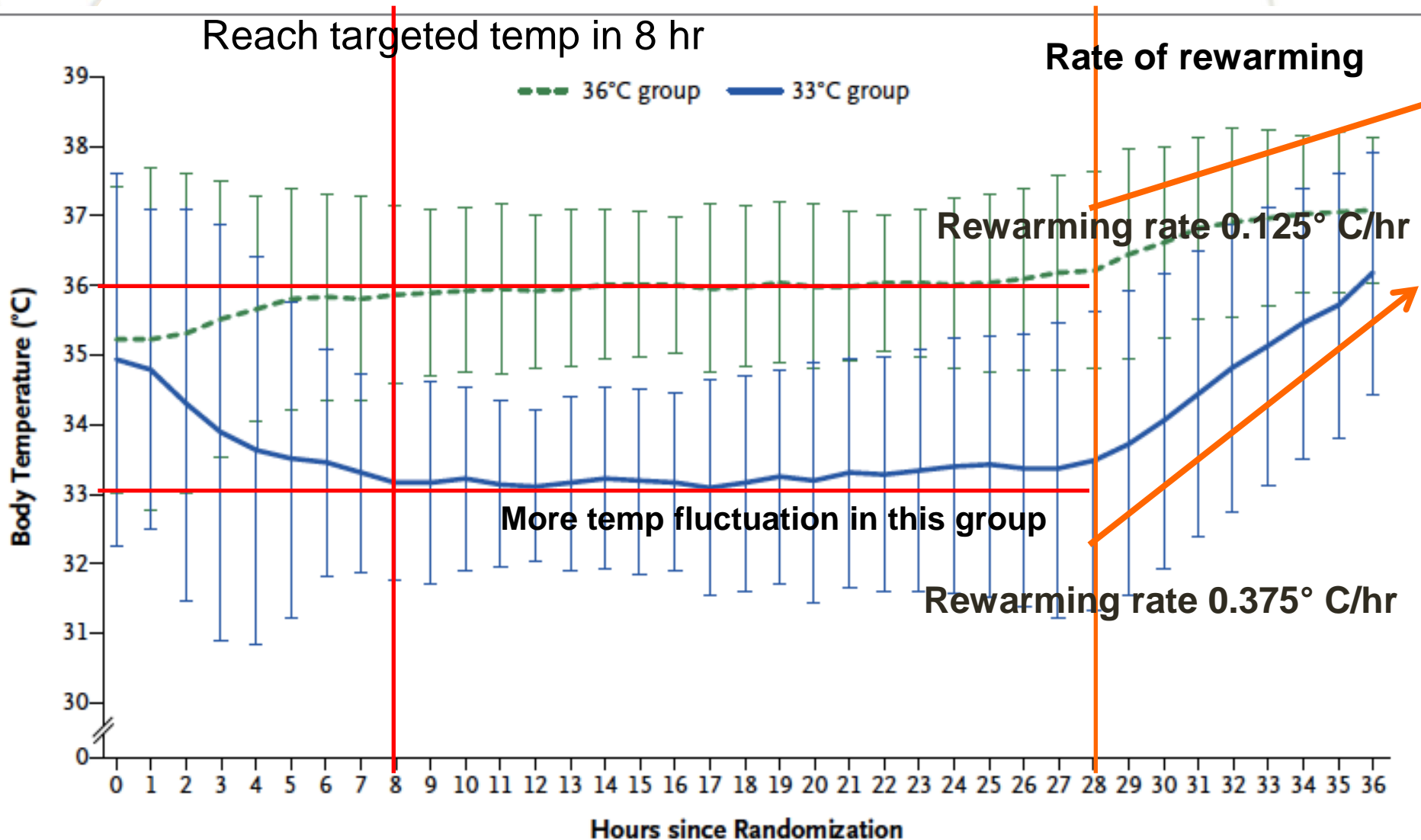
- *TTM Trial does not test same hypothesis as HACA⁹/Bernard¹⁰ Trials*
- *36° is very much "active" temperature management*

TTM Trial in Context With Prior Studies. (Modified from Abella B. at <https://www.med.upenn.edu/resuscitation/ttm-video.shtml>).

Table 1. Characteristics of the Modified Intention-to-Treat Population before Randomization.*

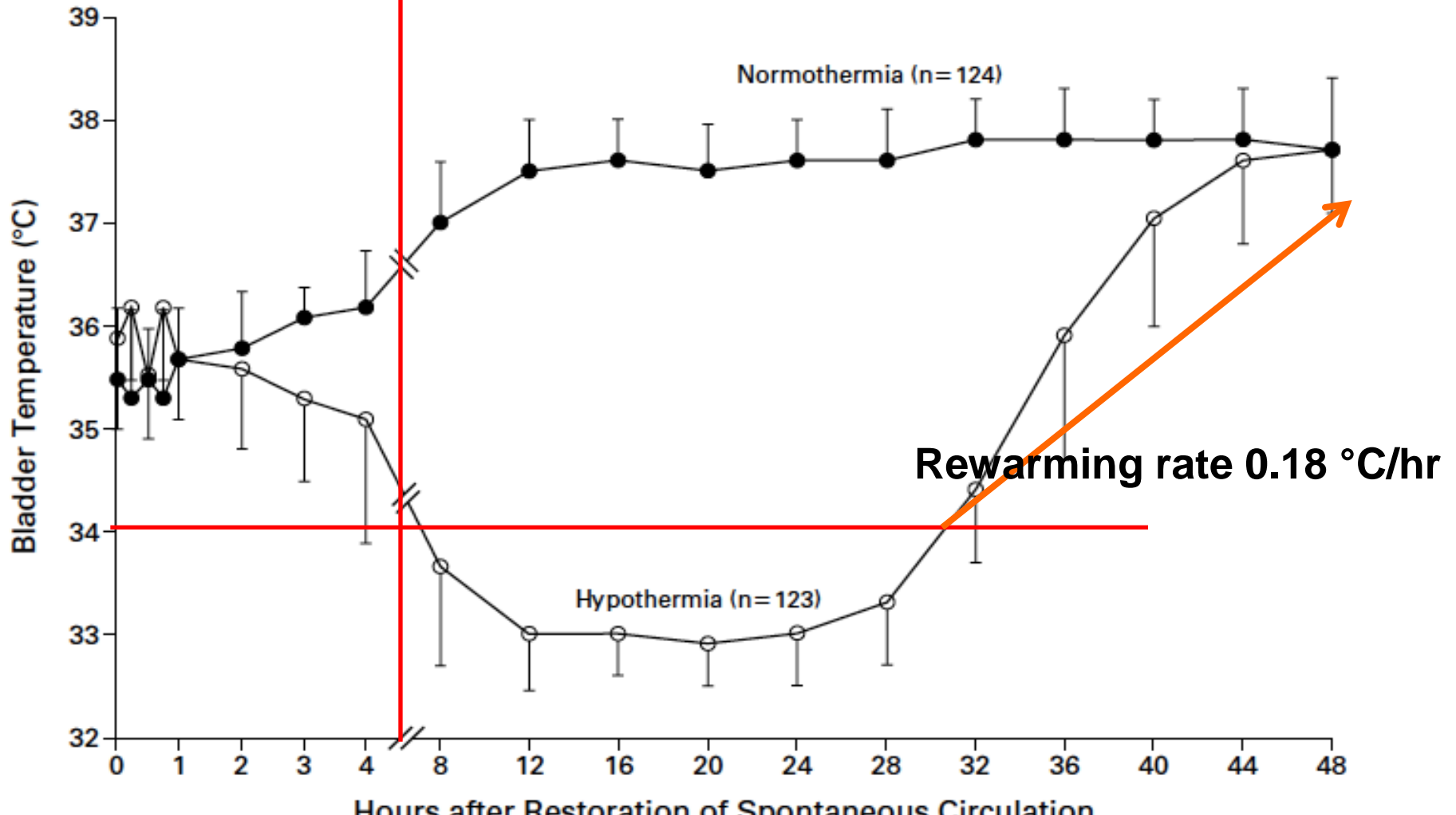
Characteristic	33°C Group (N=473)	36°C Group (N=466)
Demographic characteristics	More comorbidity	
Age — yr	64±12	64±13
Male sex — no. (%)	393 (83)	368 (79)
Medical history — no. (%)		
Chronic heart failure	32 (7)	29 (6)
Previous AMI	107 (23)	86 (18)
Ischemic heart disease	145 (31)	115 (25)
Previous cardiac arrhythmia	87 (18)	79 (17)
Arterial hypertension	193 (41)	181 (39)
Previous TIA or stroke	35 (7)	38 (8)
Diabetes mellitus	61 (13)	80 (17)
Asthma or COPD	48 (10)	49 (11)
Previous percutaneous coronary intervention	58 (12)	50 (11)
Previous coronary-artery bypass grafting	47 (10)	42 (9)

Temperature curve in TTM trial



Temperature curve in HACA

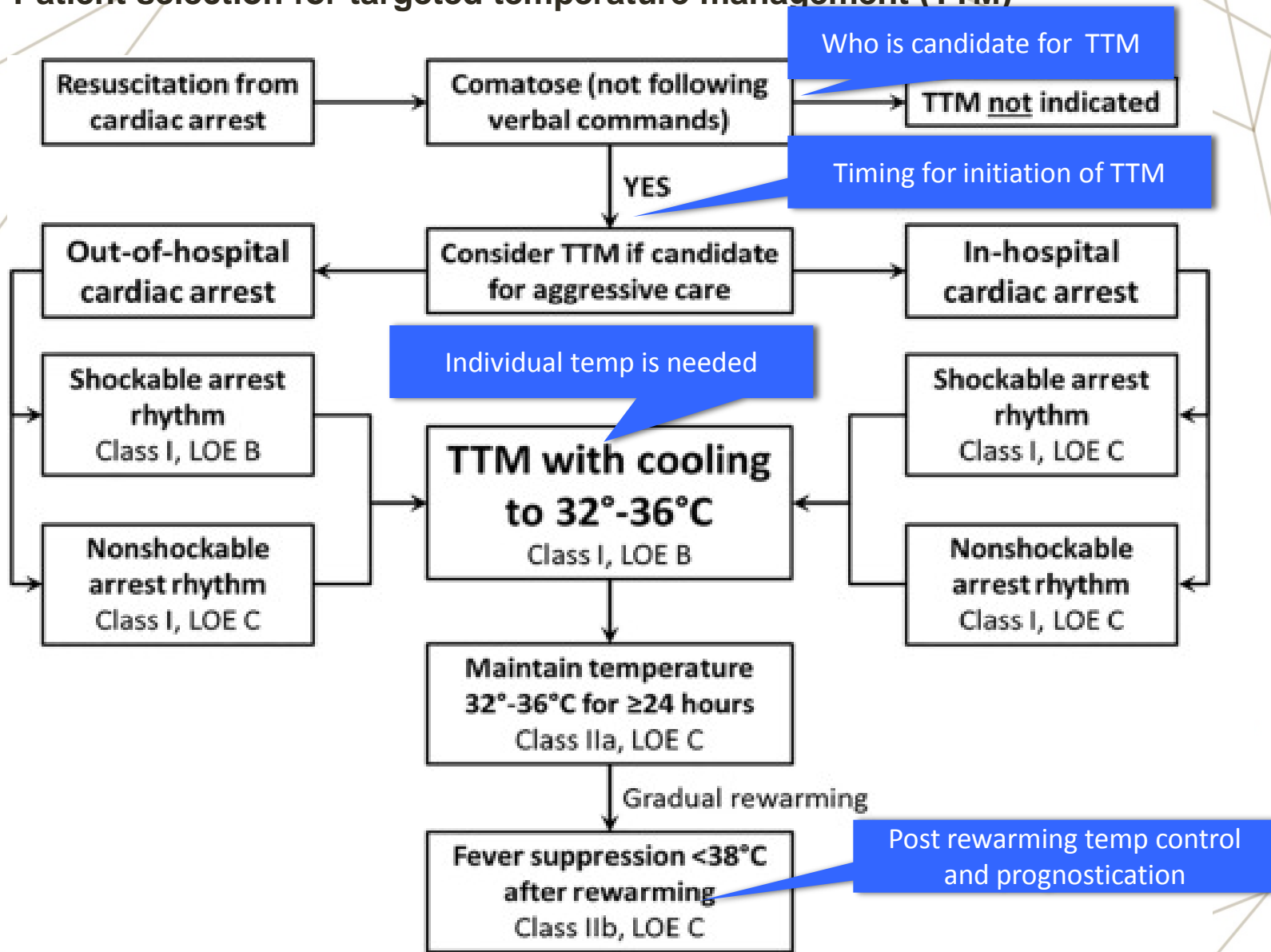
Reach targeted temp in 5 hr

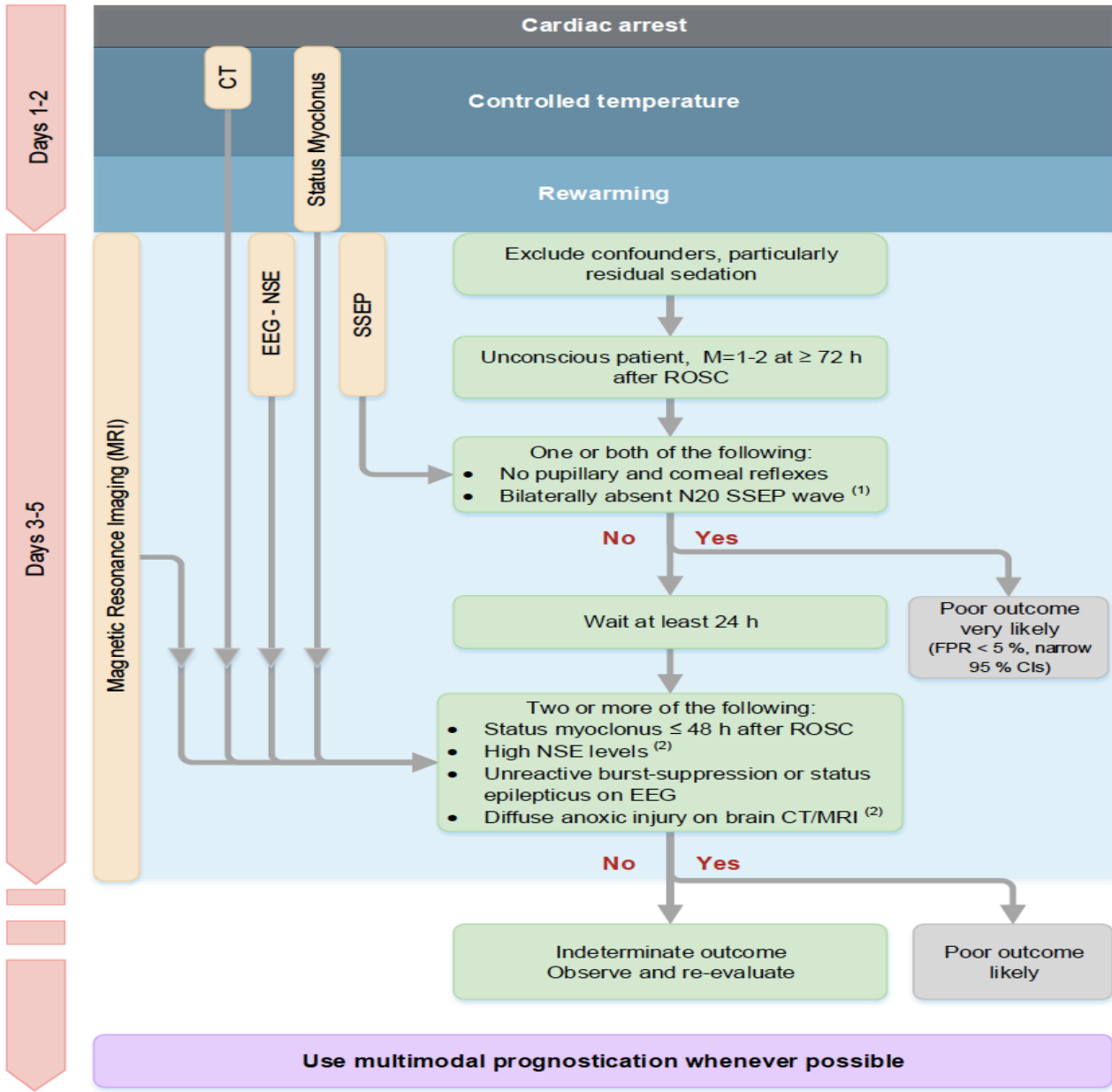


Comment for TTM trial

- Patients requiring chest compressions for >20 minutes were excluded, select less likely benefit from lower temp, and uncommon in the real world
- In TTM trial start cooling up to 4 hr time to targeted temp 8 hr (total 12 hr, quite long) compare to HACA 105 min in average, slow cooling rate may affect some patient with severe brain injury
- TTM trail has rapid rewarm rate (6 hr) ?? Rebound hyperthermia in 33° C group
- **Pending the results of further studies which should compare different temperature levels (32° C, 34° C, and 36° C) and determine optimal duration (24, 48, or 72 hours) of TH**

Patient selection for targeted temperature management (TTM)





Days 1-2

Days 3-5

Cardiac arrest

Controlled temperature

Rewarming

Magnetic Resonance Imaging (MRI)

CT

Status Myoclonus

EEG - NSE

SSEP

Exclude confounders, particularly residual sedation

Unconscious patient, M=1-2 at ≥ 72 h after ROSC

One or both of the following:
• No pupillary and corneal reflexes
• Bilaterally absent N20 SSEP wave ⁽¹⁾

No Yes

Wait at least 24 h

Poor outcome very likely (FPR < 5 %, narrow 95 % CIs)

Two or more of the following:
• Status myoclonus ≤ 48 h after ROSC
• High NSE levels ⁽²⁾
• Unreactive burst-suppression or status epilepticus on EEG
• Diffuse anoxic injury on brain CT/MRI ⁽²⁾

No Yes

Indeterminate outcome
Observe and re-evaluate

Poor outcome likely

Use multimodal prognostication whenever possible

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

TTM post cardiac arrest

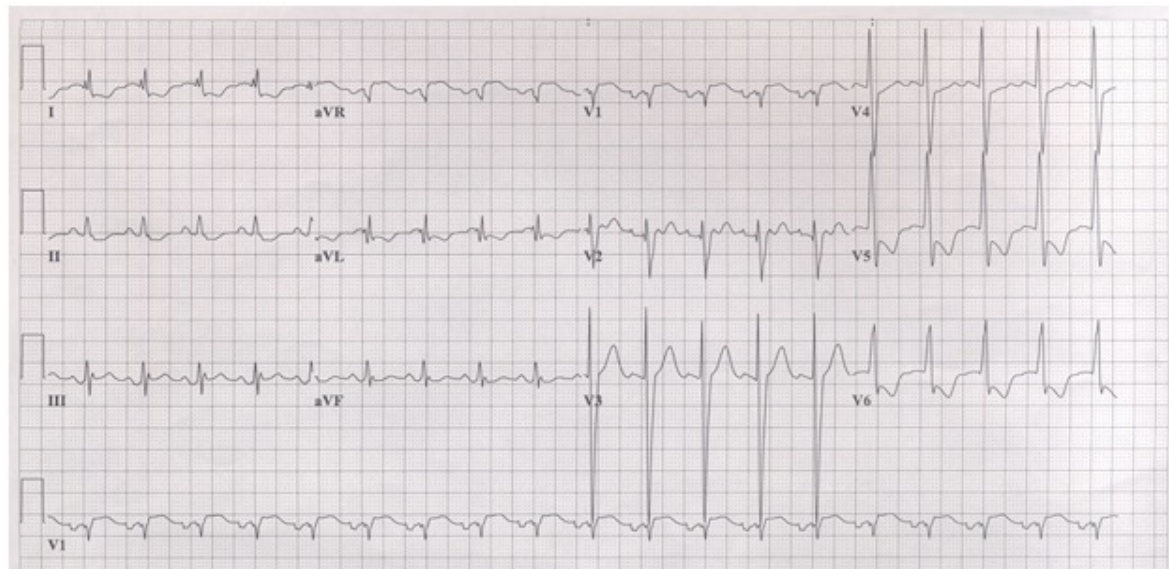
- All comatose post cardiac arrest should be sent to TTM, choose the good candidate is challenging issue for cost effectiveness.
- Timing to initiate TTM is no data usually less than 12 hr
- TTM range is 32-36 ° C each patient required difference temp for brain recovery
- After rewarming should control temp at 37 at least 24-48 hr
- Prognostication should be done after 72 hr but MAKE SURE that no sedative effect.

Coronary revascularization in post cardiac arrest

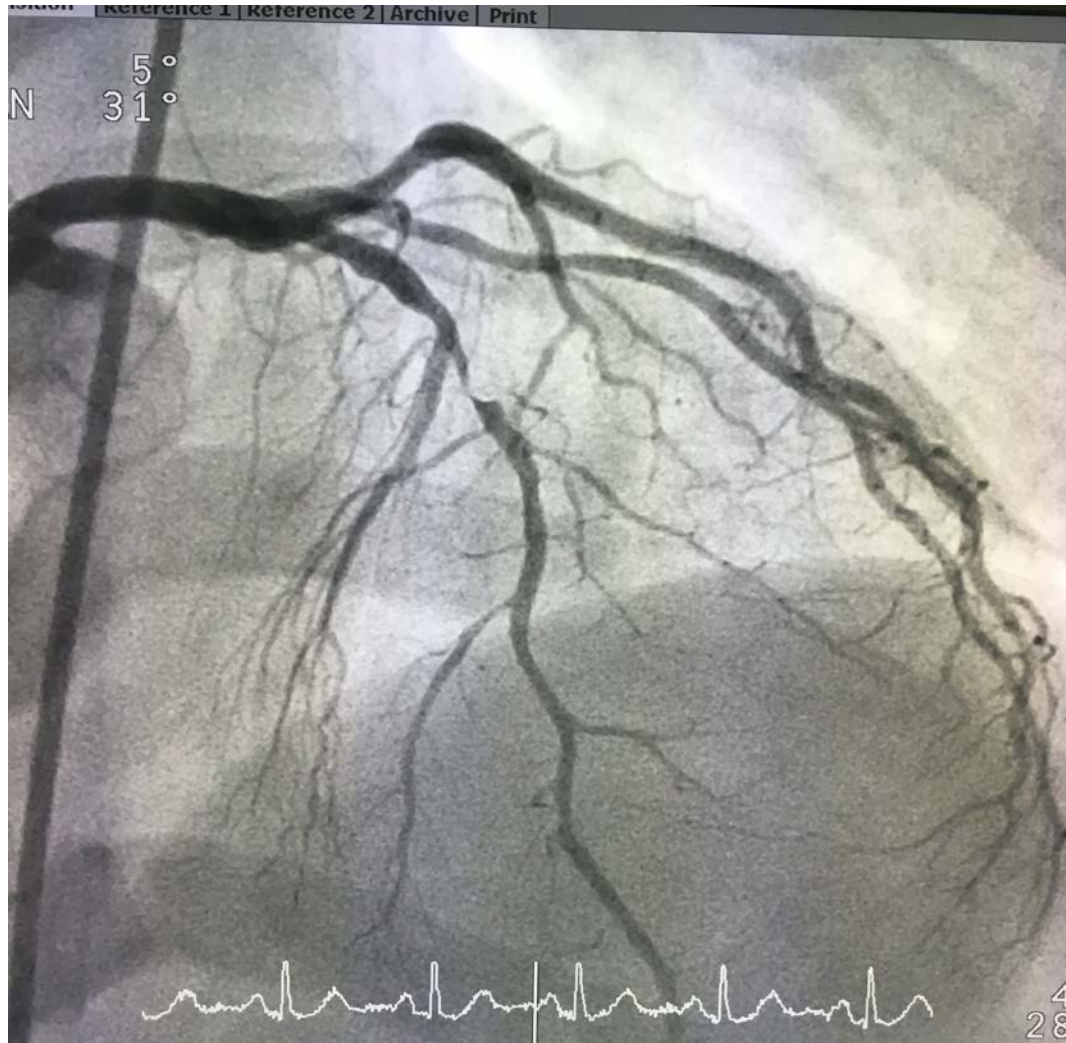
2015 Guideline Acute Cardiovascular Interventions

- CAG 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).
- Emergency CAG 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).
- CAG 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).

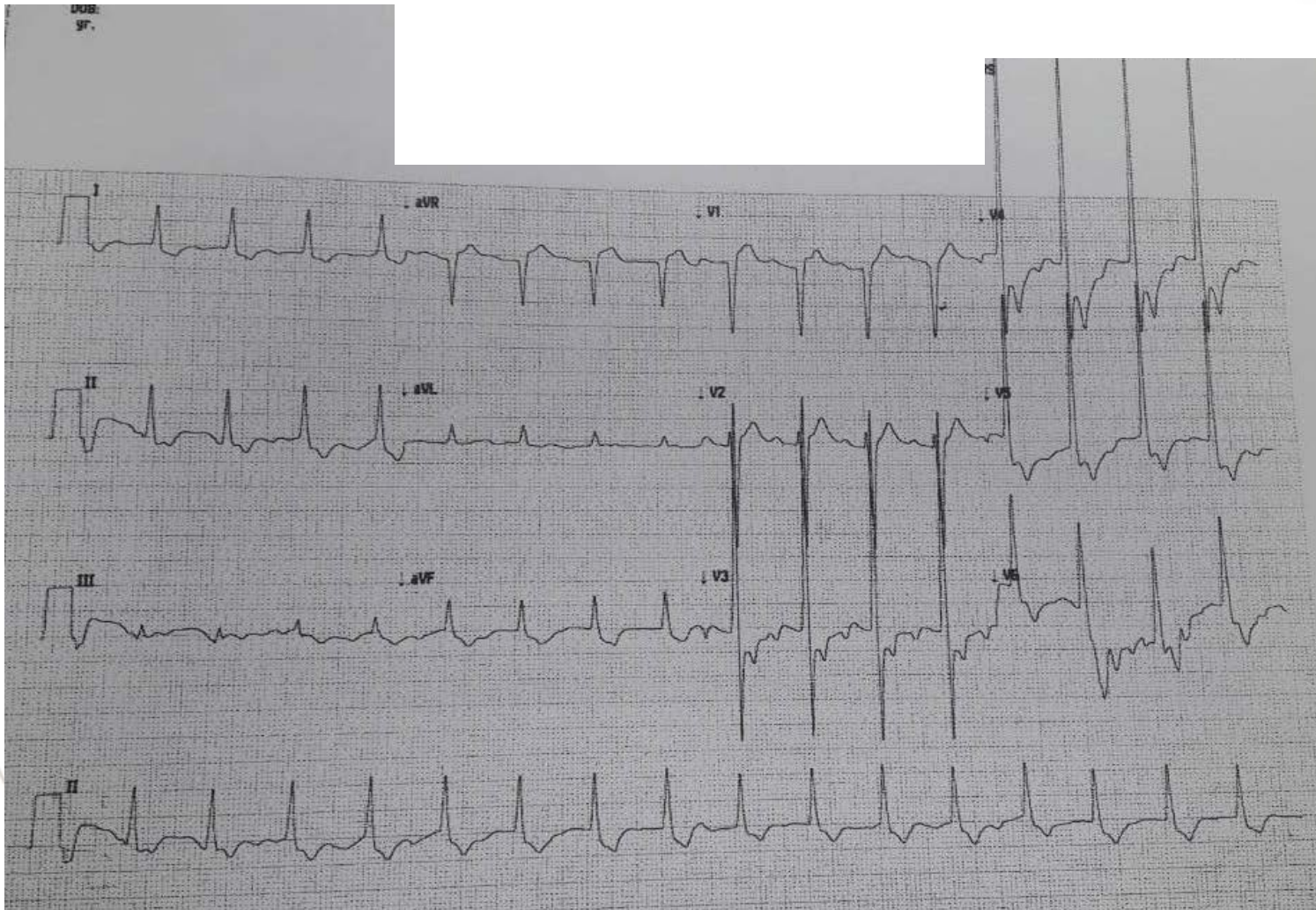
- 72 male unknown underlying, present with acute pulmonary edema, cardiogenic shock and post cardiac arrest from PEA.



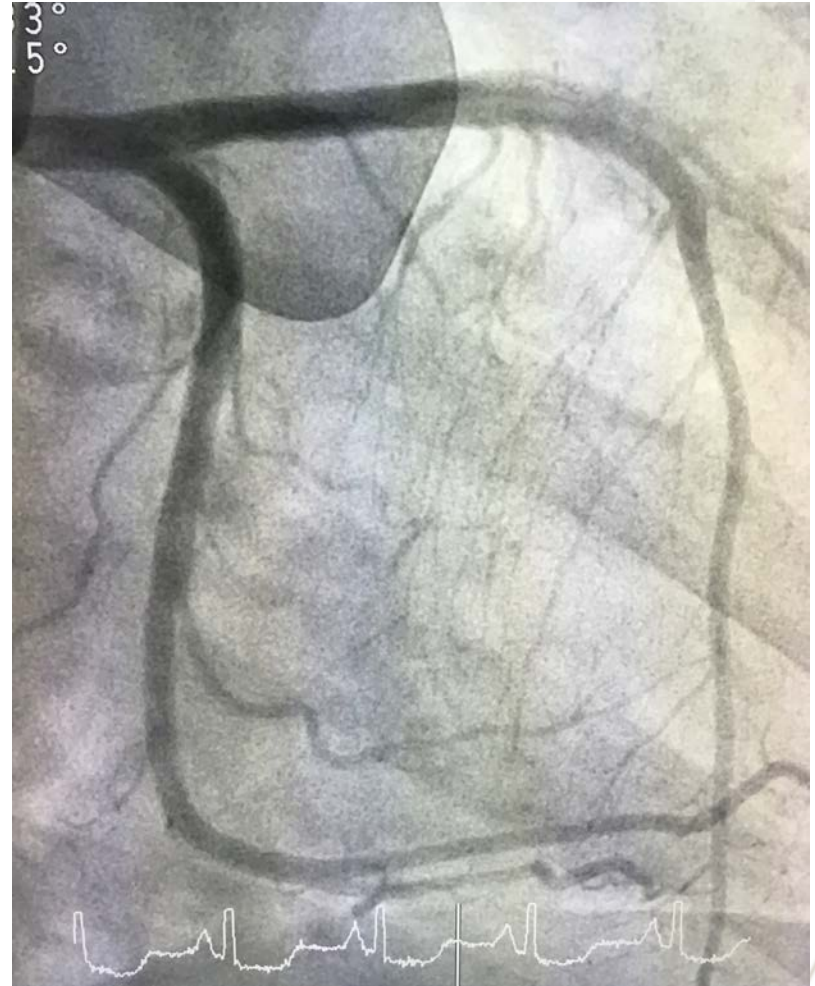
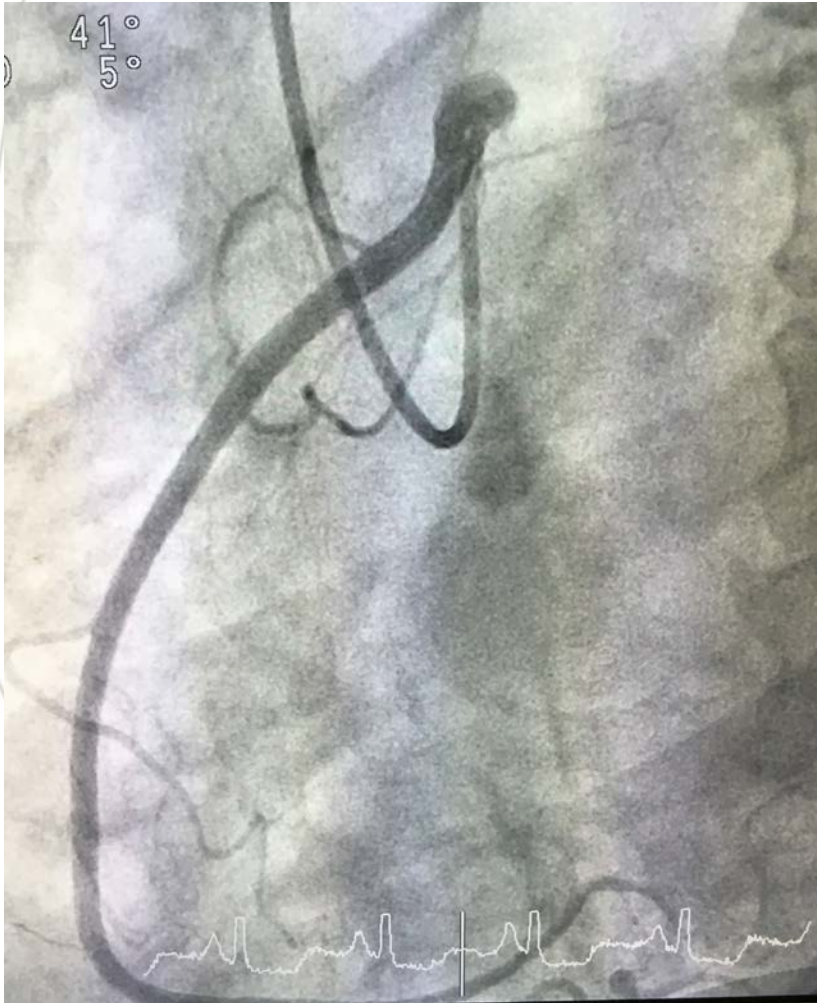
CAG



73 yr female with pVT arrest, CPR 10min x 2 episodes, troponin-T 2000+, ECG post arrest



CAG result



How can we identify the real ACS?

ECG matter??

TABLE 2 Angiographic Findings in Patients With Cardiac Arrest and No ST-Segment Elevation on ECG

First Author, Year (Ref. #)	Acute Occlusion	Culprit Lesion*	Significant CAD†
Merchant et al., 2008 (55)	6/17 (35)	—	10/17 (55)
Reynolds et al., 2009 (14)	—	—	31/54 (57)
Anyfantakis et al., 2009 (56)	—	—	27/44 (61)
Radsel et al., 2011 (31)	4/54 (7)	13/54 (24)	32/54 (59)
Bro-Jeppesen et al., 2012 (30)	—	—	43/82 (52)
Dumas et al., 2010 (3)	—	—	176/301 (58)
Hollenbeck et al., 2014 (25)	44/163 (27)	—	—
Kern et al., 2015 (52)	23	33	—
Total (%)	23	29	58

Values are n/N (%) or %. *Defined as acute occlusion or irregular plaque morphology with or without thrombus. †Defined according to the definition used in each study.

CAD = coronary artery disease; ECG = electrocardiogram.

TABLE 3 Outcomes of Patients With Cardiac Arrest Referred for Coronary Angiography

First Author, Year (Ref. #)	Early CAG Group (Includes Patients With and Without STE, Unless Otherwise Indicated)				No/Late CAG Group			Comments
	No STE	Early CAG	PCI	Survival/Good Outcome	No/Late CAG	PCI	Survival/Good Outcome	
			(% of CAG)	(% of CAG)				
Dumas et al., 2010 (3)	301 (69)	435 (100)	202 (46)	171 (39)	—	—	—	All patients studied got CAG; successful PCI was an independent predictor of survival, adjusted OR: 2.06; 95% CI: 1.16–3.66; p = 0.013.
Nanjayya et al., 2011 (59)	NA	35 (50)	21 (60)	18 (51)	35 (50)	1 (2)	12 (34)	Adjusted odds for survival with good neurological outcome with CAG: 1.32; 95% CI: 0.26–7.37; p = 0.78.
Mooney et al., 2011 (12)	72 (51)	101 (72)	56 (55)	63 (62)	39 (29)	—	15 (38)	It is not indicated whether the use of CAG was included in the multivariate analysis of factors related to survival.
Aurore et al., 2011 (61)	367 (82)	133 (30)	71 (53)	30 (23)	312 (70)	—	30 (9.6)	p values for comparisons not provided.
Tømte et al., 2011 (37)	NA	145 (83)	80 (55)	76 (52)	29 (17)	—	9 (31)	Adjusted odds for good outcome with emergency CAG: 11.21; 95% CI: 2.96–42.49; p < 0.001.
Stub et al., 2011 (28)	46 (37)	82 (66)	41 (50)	—	43 (34)	—	—	Also included conscious patients; unadjusted OR for survival with CAG: 7.6; 95% CI: 3.2–17.5; p = 0.01. Adjusted analysis not performed.
Strote et al., 2012 (58)	158 (66)	61 (25)	38 (62)	44 (72)	179 (75)	13 (30)†	87 (49)	Assessed survival by tertiles of likelihood of getting CAG. In highest tertile group, survival was higher with early CAG (73% vs. 33%; p = 0.001).
Zanuttini et al., 2012 (10)	61 (66)	48 (52)	25 (52)	29 (60)	45 (48)	6 (33)†	21 (47)	Adjusted odds for survival overall with successful PCI: 2.32; 95% CI: 1.23–4.38; p = 0.009.
Cronier et al., 2011 (11)	61 (55)	91 (82)	46 (51)	NA	—	—	—	Only patients without hemodynamic instability got CAG; adjusted OR for death with PCI: 0.30; 95% CI: 0.11–0.79; p = 0.01.
Bro-Jeppesen et al., 2012 (30)	244 (68)	82 (34)*	24 (29)*	54 (66)*	162 (66)*	—	83 (53)*	Adjusted HR for mortality in with emergency CAG in group without STE: 0.69; 95% CI: 0.4–1.2; p = 0.18.
Søholm et al., 2013 (60)	1,020 (84)	128 (13)*	NA	NA	892 (87)*	NA	NA	Univariate OR for 30-day mortality with early CAG: 0.35; 95% CI: 0.18–0.70; p = 0.003; early CAG was not an independent predictor of mortality.
Hollenbeck et al., 2014 (25)	269 (100)	122 (45)	40 (33)	80 (66)	147 (55)	16 (11)†	71 (49)	Adjusted OR for mortality with CAG: 0.35; 95% CI: 0.18–0.70; p = 0.003.
Callaway et al., 2014 (43)	3,408 (86)	765 (19)	705 (92)	495 (65)	3,216 (81)	—	871 (27)	Adjusted OR for survival with early CAG: 1.69; 95% CI: 1.06–2.70

Values are n (%). *Among those without STE. †Among those getting CAG.

CAG = coronary angiography; CI = confidence interval; HR = hazard ratio; NA = not available; OR = odds ratio; PCI = percutaneous coronary intervention; STE = ST-segment elevation; STEMI = ST-segment elevation myocardial infarction.

TABLE 1 28 Clinical Reports of Combining TTM and Early Coronary Angiography in Resuscitated, But Comatose Patients With STEMI on the ECG

First Author, Date (Ref. #)	Survivors to DC (n = 2,687/4,510 [60%])	Good Neuro Among Survivors (n = 2,090/2,426 [86%])
Hovdenes et al., 2007 (17)	41/50	34/41
Richling et al., 2007 (33)	24/46	22/24
Knafelj et al., 2007 (18)	30/40	22/30
Wolfrum et al., 2008 (22)	12/16	11/12
Peels et al., 2008 (104)	22/44	NA
Schefold et al., 2009 (34)	NA	19/31
Reynolds et al., 2009 (14)	52/96	NA
Nielsen et al., 2009 (35)	303/479	278/303
Batista et al., 2010 (27)	8/20	6/8
Dumas et al., 2010 (3)	171/435	160/171
Koeth et al., 2010 (105)	114/143	NA
Stub et al., 2011 (28)	52/81	46/52
Laish-Farkash et al., 2011 (36)	69/110	59/69
Tømte et al., 2011 (37)	140/252	132/140
Radsel et al., 2011 (31)	154/212	128/154
Mooney et al., 2011 (12)	78/140	72/78
Cronier et al., 2011 (11)	60/111	54/60
Gräsner et al., 2011 (90)	143/183	118/143
Bro-Jeppesen et al., 2012 (30)	211/360	207/219
Zanuttini et al., 2012 (10)	29/48	NA
Liu et al., 2012 (106)	36/81	NA
Nanjayya et al., 2012 (59)	18/35	14/18
Strote et al., 2012 (58)	44/61	34/44
Waldo et al., 2013 (107)	57/84	NA
Velders et al., 2013 (32)	187/222	168/183
Callaway et al., 2014 (43)	495/765	413/495
Thomas et al., 2014 (108)	168/348	115/168
Sideris et al., 2014 (88)	97/300	80/97

Values are n/N.

DC = discharge; ECG = electrocardiogram; neuro = neurological function; TTM = targeted temperature management.

Outcomes of Comatose Cardiac Arrest Survivors With and Without ST-Segment Elevation Myocardial Infarction

Importance of Coronary Angiography

ABSTRACT

OBJECTIVES The aim of this study was to compare outcomes and coronary angiographic findings in post-cardiac arrest patients with and without ST-segment elevation myocardial infarction (STEMI).

BACKGROUND The 2013 STEMI guidelines recommend performing immediate angiography in resuscitated patients whose initial electrocardiogram shows STEMI. The optimal approach for those without STEMI post-cardiac arrest is less clear.

METHODS A retrospective evaluation of a post-cardiac arrest registry was performed.

RESULTS The database consisted of 746 comatose post-cardiac arrest patients including 198 with STEMI (26.5%) and 548 without STEMI (73.5%). Overall survival was greater in those with STEMI compared with those without (55.1% vs. 41.3%; $p = 0.001$), whereas in all patients who underwent immediate coronary angiography, survival was similar between those with and without STEMI (54.7% vs. 57.9%; $p = 0.60$). A culprit vessel was more frequently identified in those with STEMI, but also in one-third of patients without STEMI (80.2% vs. 33.2%; $p = 0.001$). The majority of culprit vessels were occluded (STEMI, 92.7%; no STEMI, 69.2%; $p < 0.0001$). An occluded culprit vessel was found in 74.3% of STEMI patients and in 22.9% of no STEMI patients. Among cardiac arrest survivors discharged from the hospital who had presented without STEMI, coronary angiography was associated with better functional outcome (93.3% vs. 78.7%; $p < 0.003$).

CONCLUSIONS Early coronary angiography is associated with improved functional outcome among resuscitated patients with and without STEMI. Resuscitated patients with a presumed cardiac etiology appear to benefit from immediate coronary angiography. (J Am Coll Cardiol Intv 2015;8:1031-40) © 2015 by the American College of Cardiology Foundation.



Contents lists available at [ScienceDirect](#)

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation



Clinical paper

Cardiac catheterization is associated with superior outcomes for survivors of out of hospital cardiac arrest: Review and meta-analysis[☆]



Anthony C. Camuglia^{a,b,c,*}, Varinder K. Randhawa^d, Shahar Lavi^d, Darren L. Walters^{c,e}

^a Mater Health Cardiovascular Unit, South Brisbane, QLD, Australia

^b Princess Alexandra Hospital, Department of Cardiology, Brisbane, QLD, Australia

^c University of Queensland, Brisbane, QLD, Australia

^d London Health Sciences Centre, University of Western Ontario, London, ON, Canada

^e The Prince Charles Hospital, Chermside, Brisbane, QLD, Australia

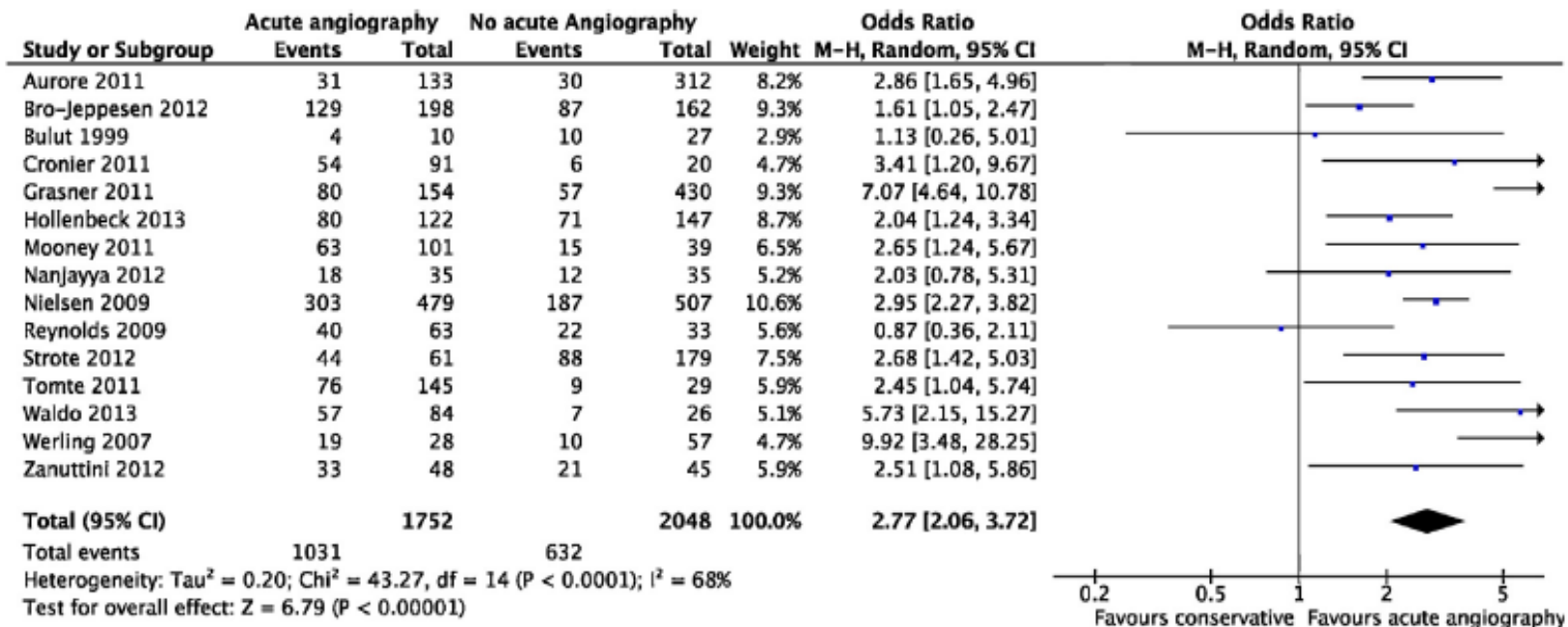


Fig. 2. Weighted hazard effects model of the relationship between acute coronary angiography and survival after OHCA.

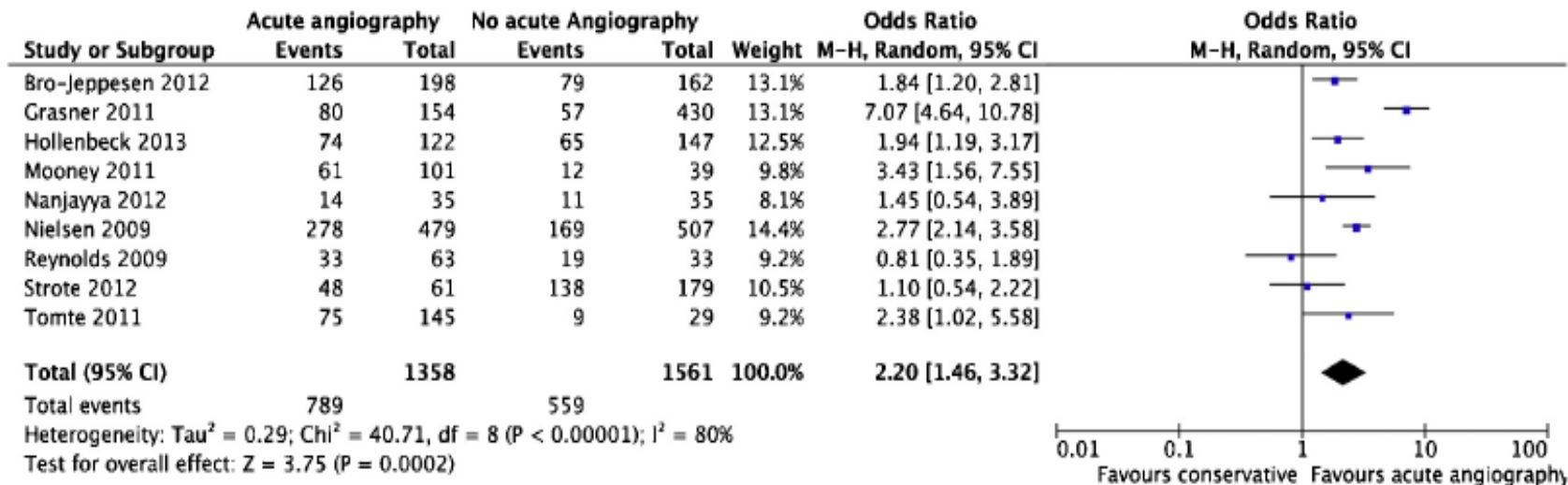
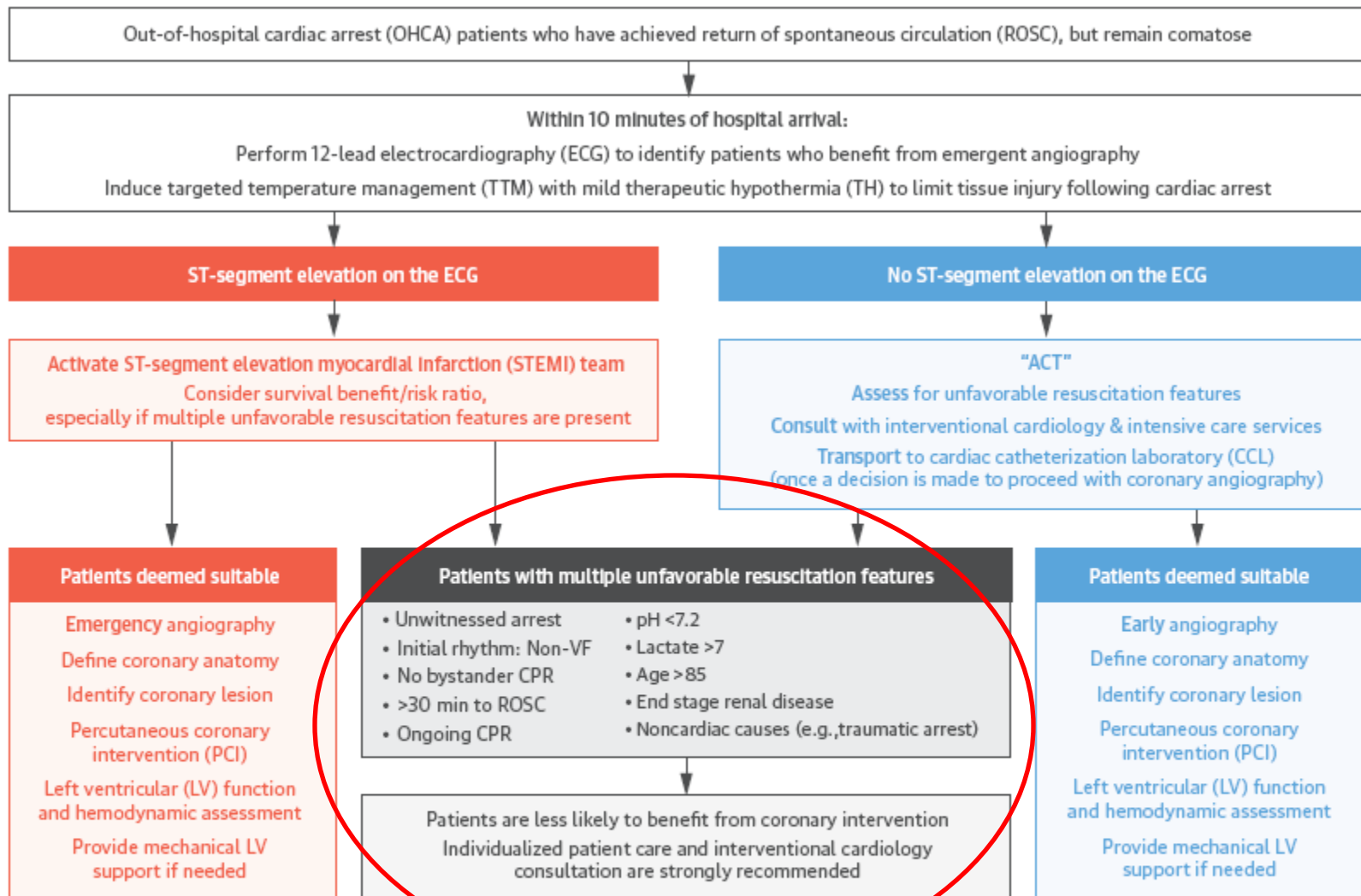


Fig. 3. Weighted hazard effects model of the relationship between acute coronary angiography and good neurological outcome after OHCA.

Which patient worth for
revascularization?

CENTRAL ILLUSTRATION Algorithm for Risk Stratification of Comatose Cardiac Arrest Patients



Rab, T. et al. J Am Coll Cardiol. 2015; 66(1):62-73.

ACT = assessment, consultation, transport; CCL = cardiac catheterization laboratory; CPR = cardiopulmonary resuscitation; ECG = electrocardiography; LV = left ventricular; OHCA = out-of-hospital cardiac arrest; PCI = percutaneous coronary intervention; ROSC = return of spontaneous circulation; STEMI = ST-segment elevation myocardial infarction; TH = therapeutic hypothermia; TTM = targeted temperature management; VF = ventricular fibrillation.

Table 3 Factors predictive of a positive coronary angiography in univariate analysis

	Patients, <i>n</i> (%)		OR (95% CI)	<i>P</i> value
	Positive ^a (<i>n</i> = 98)	Negative (<i>n</i> = 35)		
Initial rhythm VF or VT	73 (74)	17 (49)	2.9 (1.3–6.6)	0.008
Semiautomatic defibrillator on	61 (62)	13 (37)	2.7 (1.2–5.9)	0.02
Public place	48 (49)	6 (17)	4.6 (1.7–12.0)	0.001
History of coronary disease	34 (35)	3 (9)	5.5 (1.6–19.3)	0.004
History of diabetes	22 (22)	2 (6)	4.6 (1.0–20.9)	0.03
ST segment depression (ECG)	40 (41)	8 (23)	2.2 (0.9–5.5)	0.07
Male	86 (88)	23 (66)	3.7 (1.4–9.7)	0.005
ST segment elevation (ECG)	51 (52)	14 (40)	1.6 (0.7–3.6)	0.3

CI, confidence interval; ECG, electrocardiography; OR, odds ratio; VF, ventricular fibrillation; VT, ventricular tachycardia.

^aCoronary angiography revealing at least one significant coronary lesion or percutaneous coronary intervention.

Table 4 Independent factors predictive of a positive coronary angiography in multivariate analysis

	Odds ratio (95% CI)
Positive coronary angiography ^a	
History of diabetes	7.1 (1.4–36.6)
ST segment depression (out-of-hospital ECG)	5.4 (1.1–27.8)
History of coronary disease	5.3 (1.4–20.1)
Cardiac arrest in a public place	3.7 (1.3–10.7)
Initial rhythm VF or VT	3.1 (1.1–8.6)

CI, confidence interval; ECG, electrocardiography; VF, ventricular fibrillation; VT, ventricular tachycardia.

^aCoronary angiography revealing at least one significant coronary lesion. The independent variables of the multiple logistic regression were factors with $P < 0.10$ in the univariate analysis. The *P* value in the Hosmer–Lemeshow goodness-of-fit test was 0.83, indicating good model fit.

Guideline-Recommended Indications for Emergent Coronary Angiography After OHCA

STE on ECG (class I)

Hemodynamic instability or shock (class IIa)

Recurrent VF (class IIa)

Suspected acute MI (class IIa)

Additional Suggested Indications for Coronary Angiography After OHCA

Shockable arrest rhythm (ie, VF)

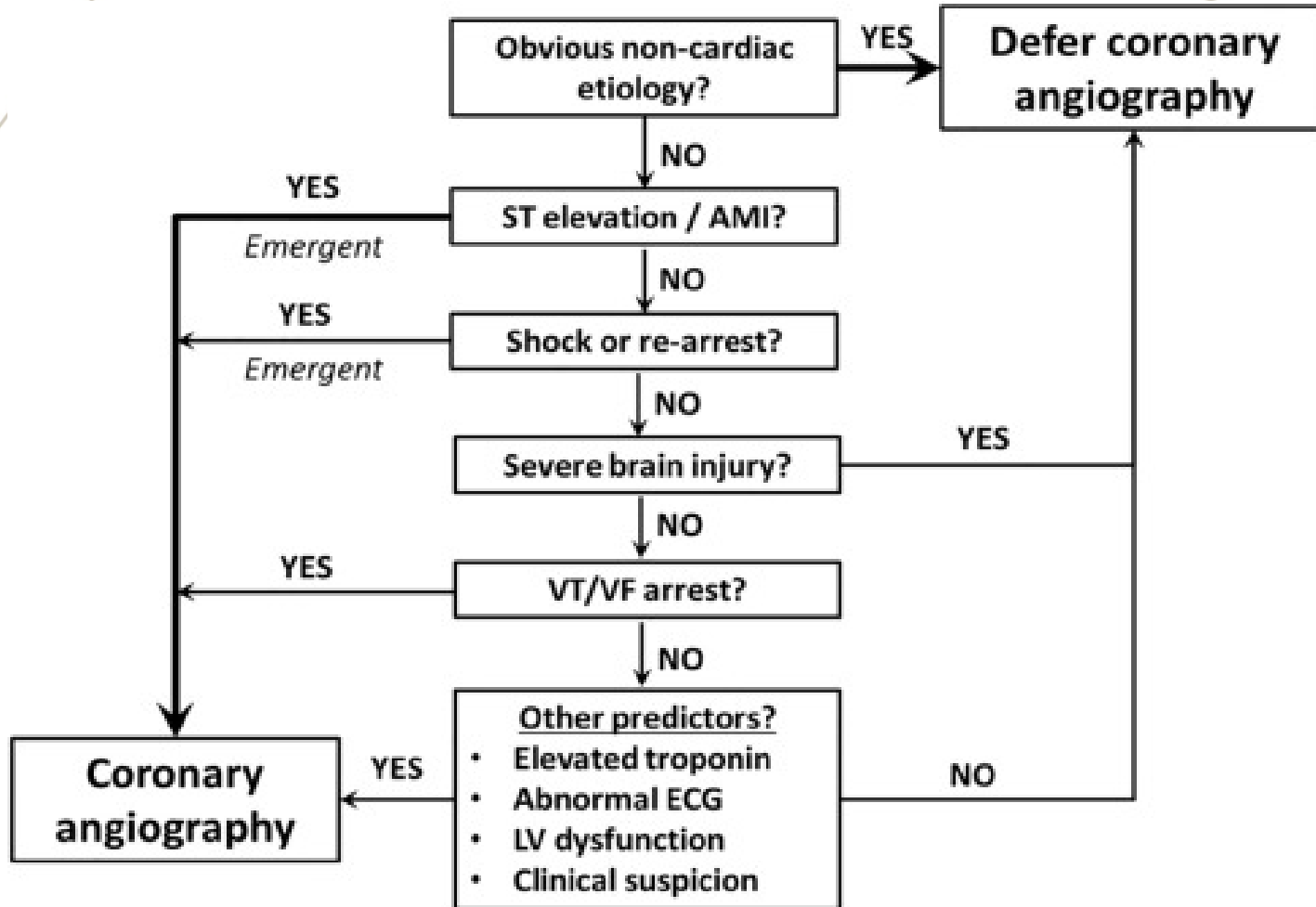
Markedly elevated cardiac troponin levels

Ischemic ECG changes (other than STE)

Left ventricular systolic dysfunction

High pretest probability of CAD

Patient selection for coronary angiography after out-of-hospital cardiac arrest. Emergent coronary angiography is defined as within 2 hours, and routine coronary angiography is defined as within 24 hours. AMI indicates acute myocardial infarction; ECG,...



Jacob C. Jentzer, Casey M. Clements, Joseph G. Murphy, R. Scott Wright

Recent Developments in the Management of Patients Resuscitated From Cardiac Arrest ☆☆☆

Journal of Critical Care, 2017, Available online 16 February 2017

<http://dx.doi.org/10.1016/j.jcrc.2017.02.011>

Pitfalls in post cardiac arrest CAG

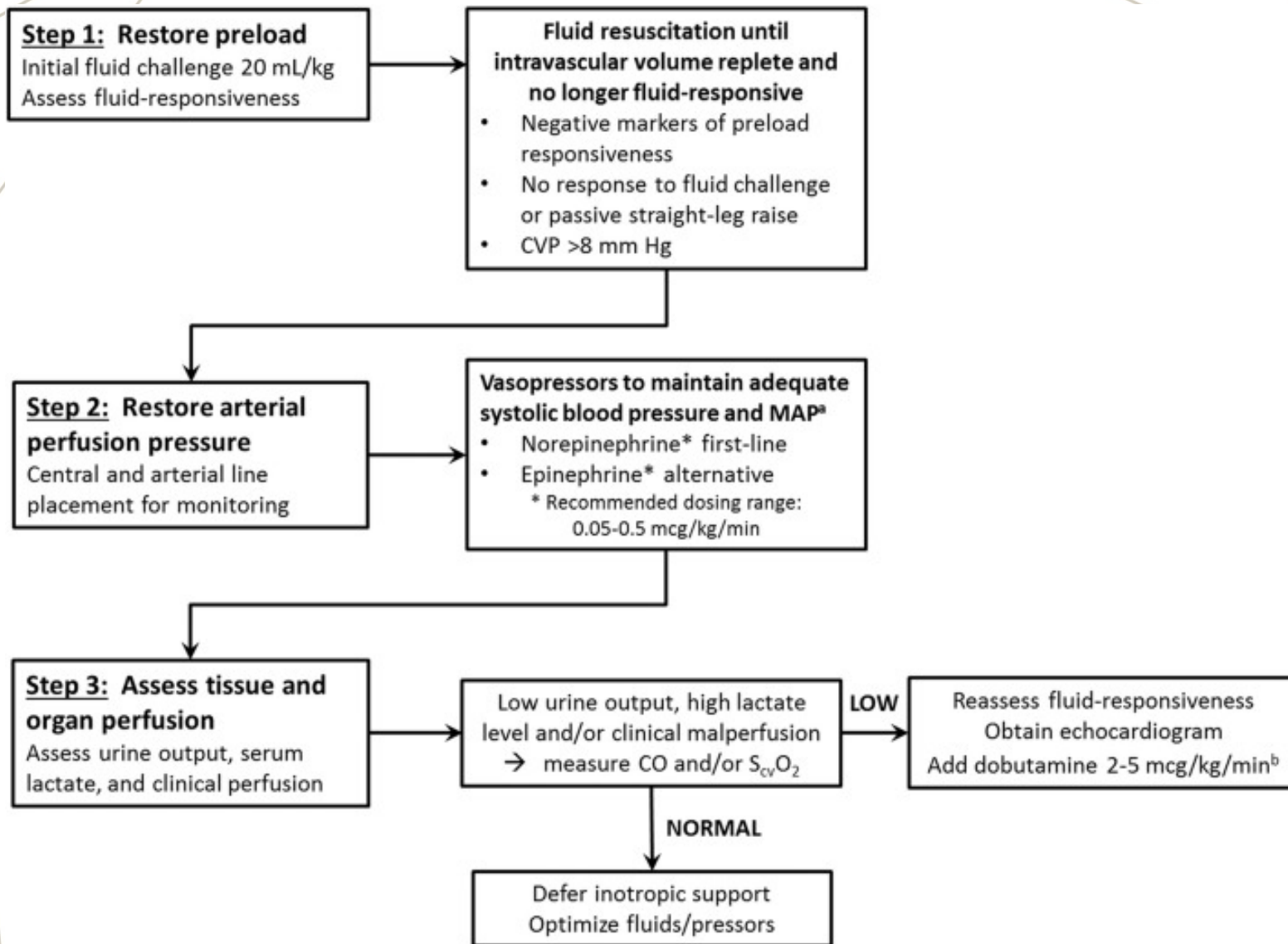
- Don't rely on ECG post cardiac arrest or cardiac troponin.
- Always looking for other cause of CA presenting with similar picture of ACS esp PE, Aortic dissection.
- Common misunderstand is CAG improve survival of post cardiac arrest, must be PCI successful case!!
- Challenging is to select the pt who worth for revascularization.

Points of concern

- Cost VS expected result/ overall prognosis!!
(Regardless of consciousness but if poor overall prognosis CAG should be delayed)
- Cath lab facilities, transferred time and interventionist skill.
- Other non cardiac cause of cardiac arrest and delayed management due to aggressive D2B time.
- ECPR may play role in the future to improve outcome

Hemodynamic in post cardiac arrest

Suggested early hemodynamic optimization strategy.



Clinical paper

Impact of mean arterial pressure on clinical outcomes in comatose survivors of out-of-hospital cardiac arrest: Insights from the University of Ottawa Heart Institute Regional Cardiac Arrest Registry (CAPITAL-CARe)[☆]



Juan J. Russo, Tyler E. James, Benjamin Hibbert, Altayyeb Yousef, Christina Osborne, George A. Wells, Michael P.V. Froeschl, Derek Y. So, Aun Yeong Chong, Marino Labinaz, Chris A. Glover, Jean-François Marquis, Alexander Dick, Jordan Bernick,

Mich

Departm

ABSTRACT

Aim of the study: We sought to assess the relationship between mean arterial pressure (MAP) and clinical outcomes in comatose survivors of out-of-hospital cardiac arrest (OHCA).

Methods: We identified consecutive comatose survivors of OHCA with an initial shockable rhythm treated with targeted temperature management. We examined clinical outcomes in relation to mean MAP (measured hourly) during the first 96 h of hospitalization. Co-primary outcomes were the rates of death and severe neurological dysfunction at discharge.

Results: In 122 patients meeting inclusion criteria, death occurred in 29 (24%) and severe neurological dysfunction in 39 (32%). Higher mean MAPs were associated with lower odds of death (OR 0.55 per 5 mmHg increase; 95%CI 0.38–0.79; $p=0.002$) and severe neurological dysfunction (OR 0.66 per 5 mmHg increase; 95%CI 0.48–0.90; $p=0.01$). After adjustment for differences in patient, index event, and treatment characteristics, higher mean MAPs remained associated with lower odds of death (OR 0.60 per 5 mmHg increase; 95%CI 0.40–0.89; $p=0.01$) but not severe neurological dysfunction (OR 0.73 per 5 mmHg increase; 95%CI 0.51–1.03; $p=0.07$). The relationship between mean MAP and the odds of death (p -interaction=0.03) and severe neurological dysfunction (p -interaction=0.03) was attenuated by increased patient age.

Conclusion: In comatose survivors of OHCA treated with target temperature management, a higher mean MAP during the first 96 h of admission is associated with increased survival. The association between mean MAP and clinical outcomes appears to be attenuated by increased age.

© 2017 Elsevier B.V. All rights reserved.

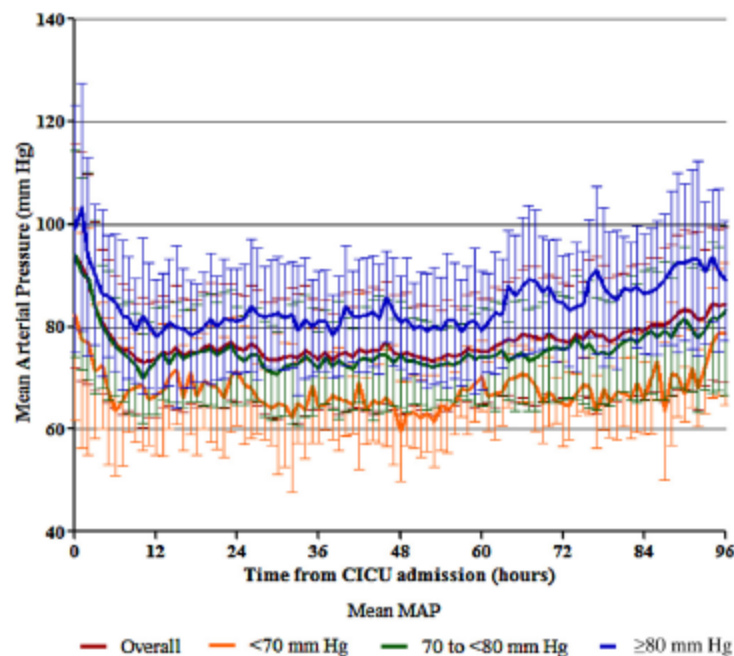


Fig. 1. Hourly mean MAP during first 96 h of CICU admission. MAP = mean arterial pressure; CICU = cardiac intensive care unit.

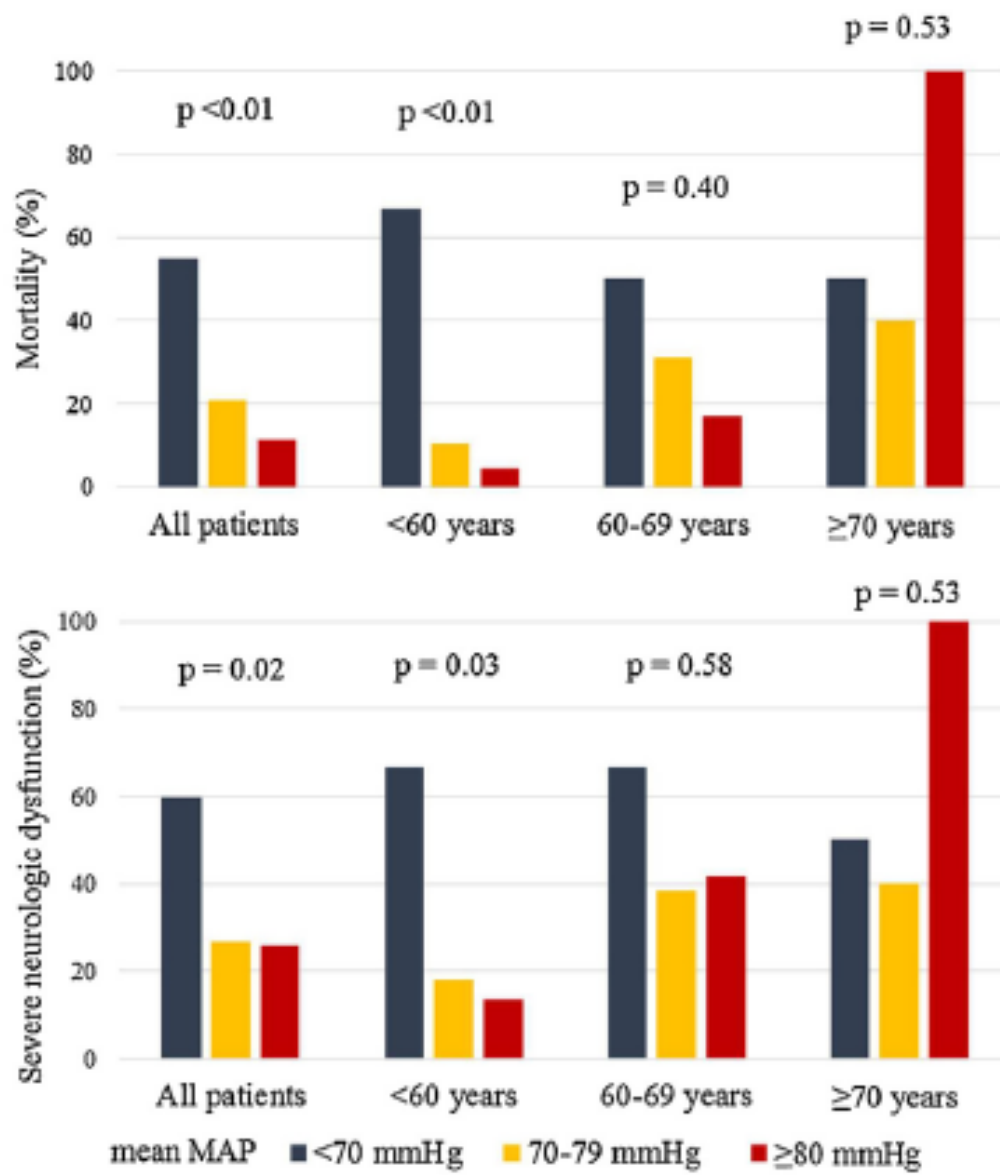


Fig. 2. Clinical outcomes stratified by mean MAP and age. MAP= mean arterial pressure.



Review

The effect of hyperoxia on survival following adult cardiac arrest: A systematic review and meta-analysis of observational studies[☆]

Chih-Hung Wang^{a,b}, Wei-Tien Chang^a, Chien-Hua Huang^a, Min-Shan Tsai^a,
Ping-Hsun Yu^c, An-Yi Wang^a, Nai-Chuan Chen^d, Wen-Jone Chen^{a,e,*}

^a Department of Emergency Medicine, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan

^b Graduate Institute of Clinical Medicine, College of Medicine, National Taiwan University, Taipei, Taiwan

^c Department of Emergency Medicine, Taipei Hospital, Ministry of Health and Welfare, New Taipei City, Taiwan

^d Department of Emergency Medicine, Tao Yuan General Hospital, Ministry of Health and Welfare, Taoyuan, Taiwan

^e Department of Emergency Medicine, Lotung Poh-Ai Hospital, Yilan County, Taiwan



ARTICLE INFO

Article history:

Received 8 February 2014

Received in revised form 16 May 2014

Accepted 21 May 2014

Keywords:

Heart arrest

Cardiopulmonary resuscitation

Critical care

Meta-analysis

Hyperoxia

Emergency medicine

ABSTRACT

Objective: Studies have shown the detrimental effect of hyperoxia in animals with return of spontaneous circulation (ROSC) after cardiac arrest. To maximize the value of existing clinical studies, we performed the systemic review and meta-analysis of human observational studies to examine the effect of hyperoxia on outcomes of post-ROSC patients.

Methods: We searched PubMed and Embase from the inception to October 2013. We selected adult observational studies that compared different levels of partial pressure of arterial oxygen (PaO₂) in post-ROSC patients with mortality or neurological status at hospital discharge as outcome. Studies comparing hypoxia with normoxia only were excluded.

Results: Fourteen studies were identified from 2982 references. Odds ratio (OR) was used as effect estimate. OR was reconstructed if not provided in original articles. Hyperoxia was defined as a PaO₂ >300 mmHg. Meta-analysis indicated that hyperoxia appeared to be correlated with increased in-hospital mortality (OR, 1.40; 95% CI, 1.02–1.93; I², 69.27%; 8 studies) but not worsened neurological outcome (OR, 1.62; 95% CI, 0.87–3.02; I², 55.61%; 2 studies). However, the results were inconsistent in subgroup and sensitivity analyses.

Conclusions: Hyperoxia appears to be correlated with increased in-hospital mortality of post-ROSC patients. This result should be interpreted cautiously because of the significant heterogeneity and limited number of studies analyzed. However, because exposure to hyperoxia had no obvious benefits, clinicians should monitor PaO₂ closely and titrate oxygen administration cautiously.

Forest Plot for Odds Ratio of In-hospital Mortality

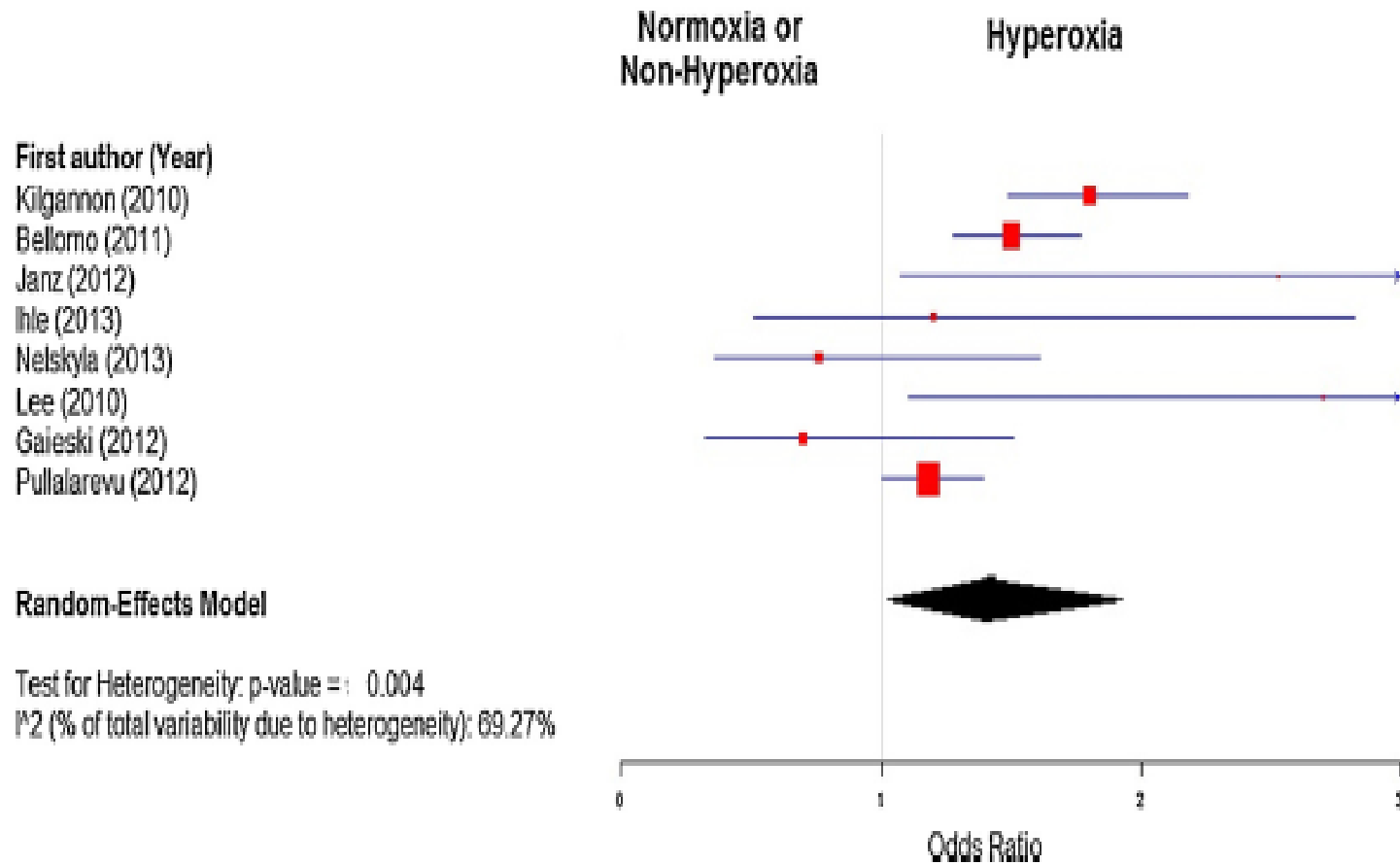


Fig. 2. Forest plot for odds ratios of in-hospital mortality.

RESEARCH

Open Access



Associations of arterial carbon dioxide and arterial oxygen concentrations with hospital mortality after resuscitation from cardiac arrest

Hendrik J. F. Helmerhorst^{1,2*}, Marie-José Roos-Blom^{3,4}, David J. van Westerloo¹, Ameen Abu-Hanna³, Nicolette F. de Keizer^{3,4} and Evert de Jonge^{1,4}

Abstract

Introduction: Arterial concentrations of carbon dioxide (PaCO_2) and oxygen (PaO_2) during admission to the intensive care unit (ICU) may substantially affect organ perfusion and outcome after cardiac arrest. Our aim was to investigate the independent and synergistic effects of both parameters on hospital mortality.

Methods: This was a cohort study using data from mechanically ventilated cardiac arrest patients in the Dutch National Intensive Care Evaluation (NICE) registry between 2007 and 2012. PaCO_2 and PaO_2 levels from arterial blood gas analyses corresponding to the worst oxygenation in the first 24 h of ICU stay were retrieved for analyses. Logistic regression analyses were performed to assess the relationship between hospital mortality and both categorized groups and a spline-based transformation of the continuous values of PaCO_2 and PaO_2 .

Results: In total, 5,258 cardiac arrest patients admitted to 82 ICUs in the Netherlands were included. In the first 24 h of ICU admission, hypocapnia was encountered in 22 %, and hypercapnia in 35 % of included cases. Hypoxia and hyperoxia were observed in 8 % and 3 % of the patients, respectively. Both PaCO_2 and PaO_2 had an independent U-shaped relationship with hospital mortality and after adjustment for confounders, hypocapnia and hypoxia were significant predictors of hospital mortality: OR 1.37 (95 % CI 1.17–1.61) and OR 1.34 (95 % CI 1.08–1.66). A synergistic effect of concurrent derangements of PaCO_2 and PaO_2 was not observed ($P = 0.75$).

Conclusions: The effects of aberrant arterial carbon dioxide and arterial oxygen concentrations were independently but not synergistically associated with hospital mortality after cardiac arrest.

Survival curve of PaCO₂

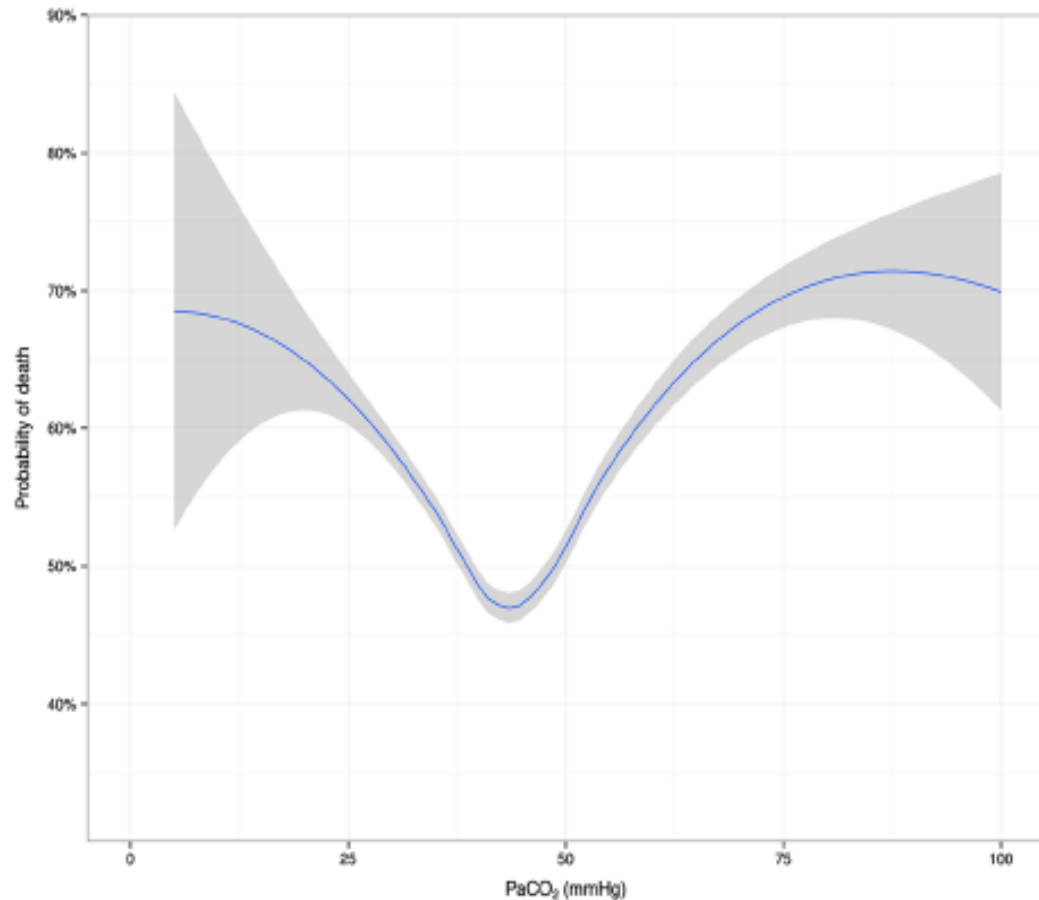


Fig. 1 Adjusted probability of in-hospital death by arterial carbon dioxide levels. Loess smoothing curve predicted from logistic regression model adjusted for spline functions of age, lowest glucose, AP4-adj and PaO₂. Grey zones represent 95 % confidence intervals

Survival curve of PaO₂

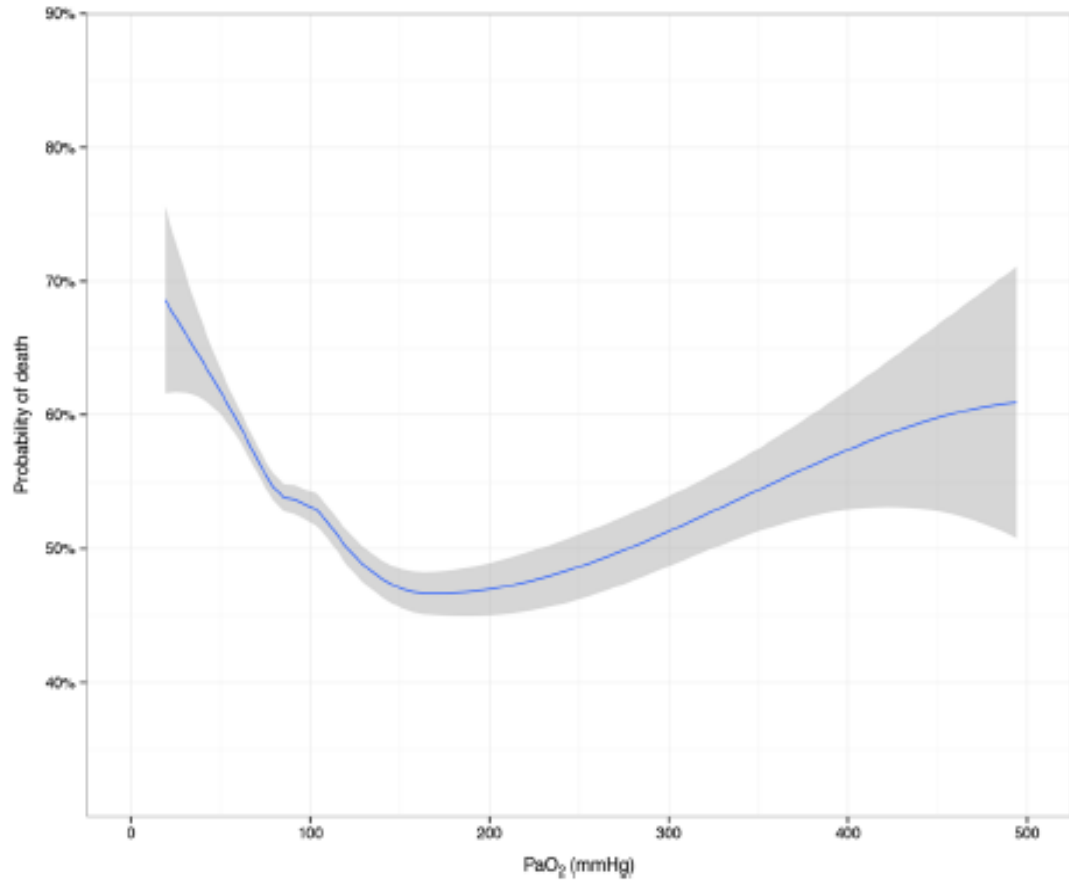


Fig. 2 Adjusted probability of in-hospital death by arterial oxygen levels. Loess smoothing curve predicted from logistic regression model adjusted for spline functions of age, lowest glucose, AP4-adj and PaCO₂. Grey zones represent 95 % confidence intervals

Hemodynamic and ventilation in post cardiac arrest

- If possible MAP should be 70-80 after ROSC to improve survival and neurological outcome
- Optimum PaO₂(100-200) and PaCO₂ (35-45) improve survival
- Most common complication after ROSC is infection and multiorgan failure that TTM may help to decrease multiorgan failure but not to be zero

Q & A