HİPOTERMİ
LİPİT TEDAVİSİ
TROMBOLİTİK TEDAVİ
Güncel Literatür

N. Belgin Akıllı
S.B.Ü. Konya Eğitim ve Araştırma Hastanesi

Normotermi/Hipotermi?...

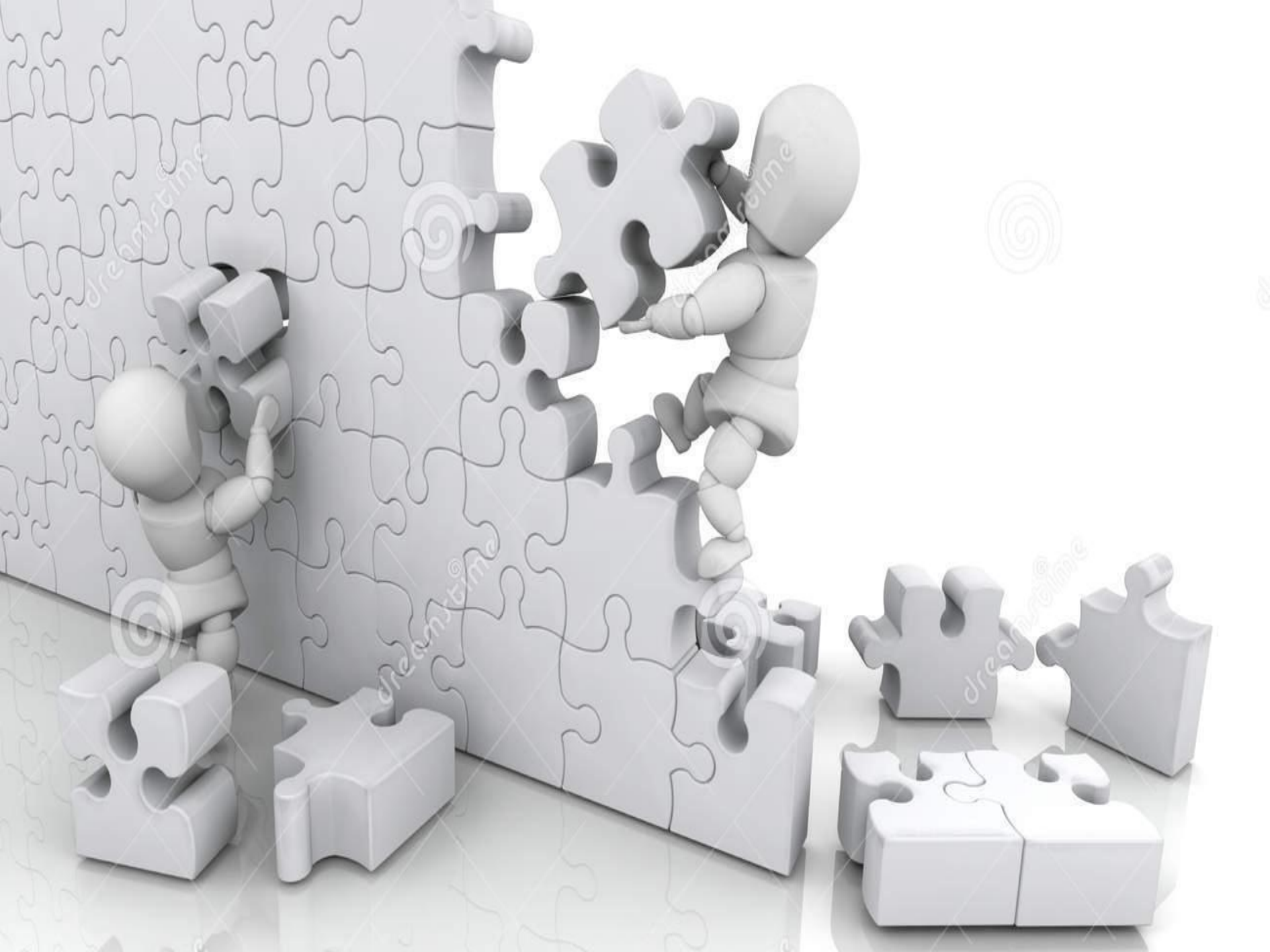
Systemic Ischemia/Reperfusion Injury
Systemic inflammatory response
Vasoplegia
Hypercoagulability
Microcirculatory dysfunction
Relative adrenal suppression
Immunosuppression

Hypoxic Brain Injury
Impaired or right-shifted autoregulation
Preserved CO_2 cerebrovascular sensitivity
Cerebral edema
Seizures / malignant EEG patterns
Coma

HİPOTERMİ

Myocardial Dysfunction
Transient global hypokinesis
Reduced cardiac output
Cardiogenic shock
Dysrhythmias
Superimposed acute coronary syndrome

Underlying Etiology of Cardiac Arrest
Cardiovascular disease (ACS, dysrhythmia)
Pulmonary disease / Hypoxia
Hypovolemia / Hemorrhage
Pulmonary embolism
Electrolyte disturbance
Sepsis
Poisoning



TREATMENT OF COMATOSE SURVIVORS OF OUT-OF-HOSPITAL CARDIAC ARREST WITH INDUCED HYPOTHERMIA

STEPHEN A. BERNARD, M.B., B.S., TIMOTHY W. GRAY, M.B., B.S., MICHAEL D. BUIST, M.B., B.S., BRUCE M. JONES, M.B., B.S., WILLIAM SILVESTER, M.B., B.S., GEOFF GUTTERIDGE, M.B., B.S., AND KAREN SMITH, B.SC.

ABSTRACT

Background Cardiac arrest outside the hospital is common and has a poor outcome. Studies in laboratory animals suggest that hypothermia induced shortly after the restoration of spontaneous circulation may improve neurologic outcome, but there have been no conclusive studies in humans. In a randomized, controlled trial, we compared the effects of moderate hypothermia and normothermia in patients who remained unconscious after resuscitation from out-of-hospital cardiac arrest.

Methods The study subjects were 77 patients who were randomly assigned to treatment with hypothermia (with the core body temperature reduced to 33°C within 2 hours after the return of spontaneous circulation and maintained at that temperature for 12 hours) or normothermia. The primary outcome measure was survival to hospital discharge with sufficiently good neurologic function to be discharged to home or to a rehabilitation facility.

Results The demographic characteristics of the patients were similar in the hypothermia and normothermia groups. Twenty-one of the 43 patients treated with hypothermia (49 percent) survived and had a good outcome — that is, they were discharged home or to a rehabilitation facility — as compared with 9 of the 34 treated with normothermia (26 percent, P=0.046). After adjustment for base-line differences in age and time from collapse to the return of spontaneous circulation, the odds ratio for a good outcome with hypothermia as compared with normothermia was 5.25 (95 percent confidence interval, 1.47 to 18.76; P=0.011). Hypothermia was associated with a lower cardiac index, higher systemic vascular resistance, and hyperglycemia. There was no difference in the frequency of adverse events.

Conclusions Our preliminary observations suggest that treatment with moderate hypothermia appears to improve outcomes in patients with coma after resuscitation from out-of-hospital cardiac arrest. (N Engl J Med 2002;346:557-63.)

Currently, the treatment of patients with coma after resuscitation from out-of-hospital cardiac arrest is largely supportive. Because cerebral ischemia may persist for some hours after resuscitation,[5] the use of induced hypothermia to decrease cerebral oxygen demand has been proposed as a treatment option.[6] Although this suggestion has been supported by studies in animal models,[7-12] the studies in humans that have been reported to date have been uncontrolled or retrospective.[13-18]

After a pilot study that suggested the feasibility, safety, and possible efficacy of this treatment,[16] we conducted a prospective, controlled trial comparing moderate induced hypothermia with normothermia in comatose survivors of out-of-hospital cardiac arrest.

METHODS

Study Design

The study was performed in Melbourne, Australia, between September 1996 and June 1999. The ambulance service has treatment protocols that follow the recommendations of the Australian Resuscitation Council.[19] Patients were enrolled in the study when the following criteria were fulfilled: an initial cardiac rhythm of ventricular fibrillation at the time of arrival of the ambulance, successful return of spontaneous circulation, persistent coma after the return of spontaneous circulation, and transfer to one of four participating emergency departments. The exclusion criteria were an age of less than 18 years for men, an age of less than 50 years for women (because of the possibility of pregnancy), cardiogenic shock (a systolic blood pressure of less than 90 mm Hg despite epinephrine infusion), or possible causes of coma other than cardiac arrest (drug overdose, head trauma, or cerebrovascular accident). Patients were also excluded if an intensive care bed was not available at a participating institution.

After the return of spontaneous circulation had been accomplished outside the hospital, eligible patients were randomly assigned to hypothermia or normothermia according to the day of the month, with patients assigned to hypothermia on odd-numbered days. For these patients, the paramedics began measures in the field to initiate hypothermia by removing the patient's clothing and applying cold packs (CoolCare, Cheltenham, Victoria, Australia) to the patient's head and torso. The treatment of patients assigned to normothermia followed usual prehospital treatment protocols.

On arrival at a participating emergency department, the patients underwent routine initial assessment and treatment, includ-

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MILD THERAPEUTIC HYPOTHERMIA TO IMPROVE THE NEUROLOGIC OUTCOME AFTER CARDIAC ARREST

THE HYPOTHERMIA AFTER CARDIAC ARREST STUDY GROUP*

ABSTRACT

Background Cardiac arrest with widespread cerebral ischemia frequently leads to severe neurologic impairment. We studied whether mild systemic hypothermia increases the rate of neurologic recovery after resuscitation from cardiac arrest due to ventricular fibrillation.

Methods In this multicenter trial with blinded assessment of the outcome, patients who had been resuscitated after cardiac arrest due to ventricular fibrillation were randomly assigned to undergo therapeutic hypothermia (target temperature, 32°C to 34°C, measured in the bladder) over a period of 24 hours or to receive standard treatment with normothermia. The primary end point was a favorable neurologic outcome within six months after cardiac arrest; secondary end points were mortality within six months and the rate of complications within seven days.

Results Seventy-five of the 136 patients in the hypothermia group for whom data were available (55 percent) had a favorable neurologic outcome (cerebral-performance category, 1 [good recovery] or 2 [moderate disability]), as compared with 54 of 137 (39 percent) in the normothermia group (risk ratio, 1.40; 95 percent confidence interval, 1.08 to 1.81). Mortality at six months was 41 percent in the hypothermia group (56 of 137 patients died), as compared with 55 percent in the normothermia group (76 of 138 patients; risk ratio, 0.74; 95 percent confidence interval, 0.58 to 0.95). The complication rate did not differ significantly between the two groups.

Conclusions 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. (N Engl J Med 2002;346:549-56.)

AN estimated 375,000 people in Europe undergo sudden cardiac arrest yearly.[1] Recovery without residual neurologic damage after cardiac arrest with global cerebral ischemia is rare. After cardiac arrest with no blood flow for more than five minutes, the generation of free radicals, together with other mediators, during reperfusion creates chemical cascades that result in cerebral injury.[2] Until recently, there was no therapy with documented efficacy in preventing brain damage after cardiac arrest.

Several studies have shown that moderate systemic hypothermia (30°C)[3] or mild hypothermia (34°C)[4-8] markedly mitigates brain damage after cardiac arrest in dogs. The exact mechanism for this cerebral resuscitative effect is not clear. A reduction in cerebral oxygen consumption[9,10] and other multifactorial chemical and physical mechanisms during and after ischemia have been postulated.[11-16] These include retardation of destructive enzymatic reactions, suppression of free-radical reactions, protection of the fluidity of lipoprotein membranes, reduction of the oxygen demand in low-flow regions, reduction of intracellular acidosis, and inhibition of the biosynthesis, release, and uptake of excitatory neurotransmitters.

Preliminary clinical studies have shown that patients treated with mild hypothermia after cardiac arrest have an improved neurologic outcome, without important side effects, as compared with the outcome in historical controls.[17-20]

We compared mild hypothermia with standard normothermia in patients who had had cardiac arrest due to ventricular fibrillation. The primary end point

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

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

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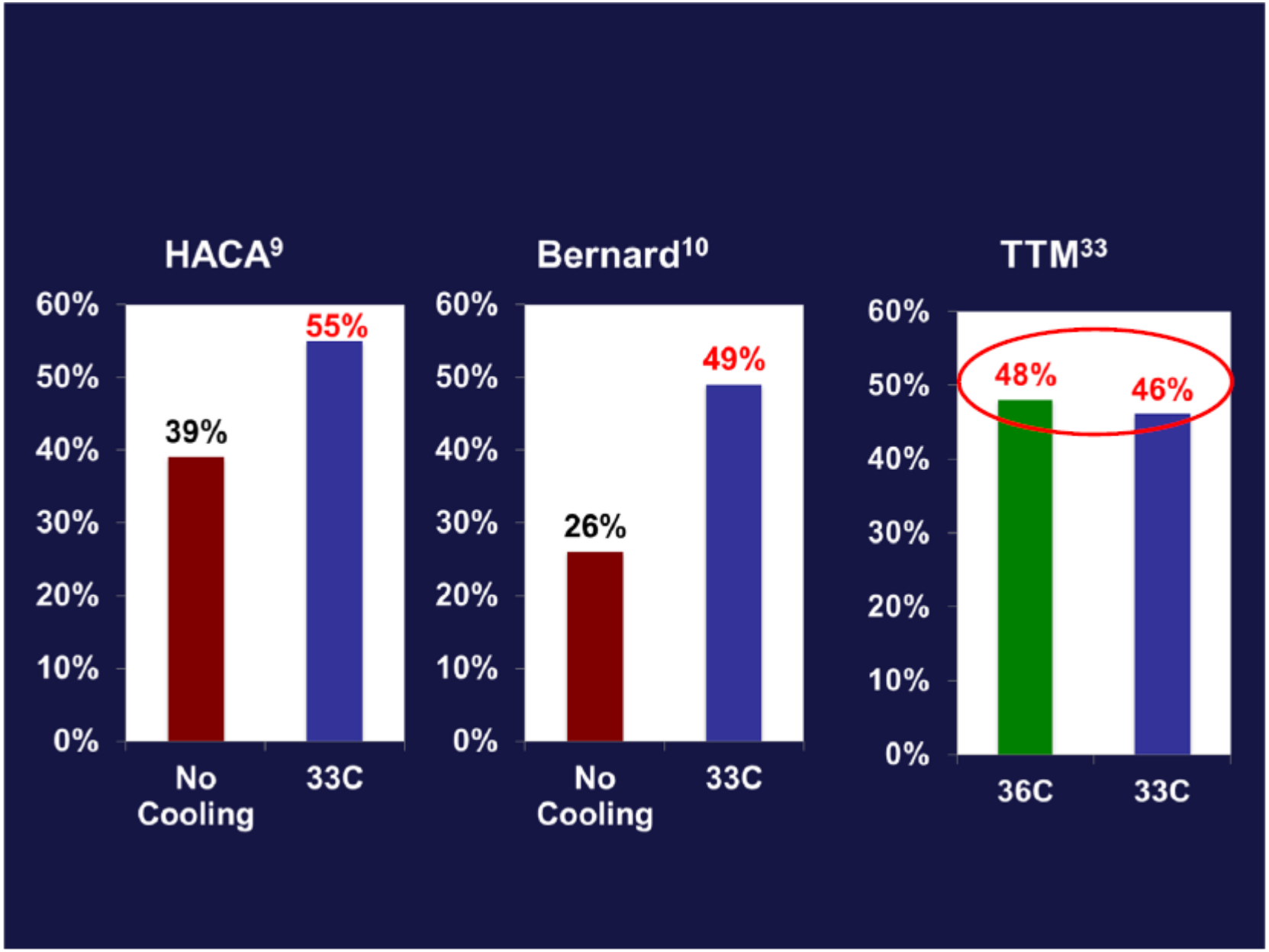
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No Cooling
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TTM[33]
60%
50%
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30%
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48%
46%
36C
33C

ILCOR Advisory Statement

Temperature Management After Cardiac Arrest

An Advisory Statement by the Advanced Life Support Task Force of the International Liaison Committee on Resuscitation and the American Heart Association Emergency Cardiovascular Care Committee and the Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation

Michael W. Donnino, MD; Lars W. Andersen, MD; Katherine M. Berg, MD;
Joshua C. Reynolds, MD, MS; Jerry P. Nolan, FRCA, FRCP, FFICM, FCEM (Hon);
Peter T. Morley, MBBS, FRACP, FANZCA, FCICM, FERC; Eddy Lang, MD;
Michael N. Cocchi, MD; Theodoros Xanthos, MD, Pg Dip (Ed), MSc, PhD, FHEA, FAcadMEd;
Clifton W. Callaway, MD, PhD*; Jasmeet Soar, FRCA, FFICM, FRCP*;
and the ILCOR ALS Task Force

Abstract—For more than a decade, mild induced hypothermia (32°C–34°C) has been standard of care for patients remaining comatose after resuscitation from out-of-hospital cardiac arrest with an initial shockable rhythm, and this has been extrapolated to survivors of cardiac arrest with initially nonshockable rhythms and to patients with in-hospital cardiac arrest. Two randomized trials published in 2002 reported a survival and neurological benefit with mild induced hypothermia. One recent randomized trial reported similar outcomes in patients treated with targeted temperature management at either 33°C or 36°C. In response to these new data, the International Liaison Committee on Resuscitation Advanced Life Support Task Force performed a systematic review to evaluate 3 key questions: (1) Should mild induced hypothermia (or some form of targeted temperature management) be used in comatose post–cardiac arrest patients? (2) If used, what is the ideal timing of the intervention? (3) If used, what is the ideal duration of the intervention? The task force used Grading of Recommendations Assessment, Development and Evaluation methodology to assess and summarize the evidence and to provide a consensus on science statement and treatment recommendations. The task force recommends targeted temperature management for adults with out-of-hospital cardiac arrest with an initial shockable rhythm at a constant temperature between 32°C and 36°C for at least 24 hours. Similar suggestions are made for out-of-hospital cardiac arrest with a nonshockable rhythm and in-hospital cardiac arrest. The task force recommends against prehospital cooling with rapid infusion of large volumes of cold intravenous fluid. Additional and specific recommendations are provided in the document. (*Circulation*. 2015;132:2448-2456. DOI: 10.1161/CIR.0000000000000313.)

Soru 1

Hafif hipotermi, hedeflenmiş sıcaklık yönetimi (TTM) uygulanmamış olan hastalarla karşılaştırıldığında sonuçları iyileştirir mi?

Soru 2

Hedeflenmiş sıcaklık yönetiminin erken (hastane öncesi) indüksiyonu sonuçları etkiler mi?

Soru 3

Hedeflenmiş sıcaklık yönetiminin süresi sonuçları etkiler mi?

- Şoklanabilir ritimle başvuran ve ROSC sonrası cevapsız kalan hastalarda hedeflenmiş sıcaklık yönetimi (32-36 C) önerilmektedir..
- Şoklanamaz ritimle başvuranlarda da uygulanabilir.
- HİKA hastalarında da ROSC sonrası bilinçsiz hastalarda uygulanabilir.
- Hastane öncesi soğuk hızlı salin uygulamasını rutin önerilmemektedir.
- Hedeflenmiş sıcaklık yönetimi uygulanacaksa en az 24 saat uygulanmalıdır.

Kılavuz Önerileri:

1-Başlangıç ritmi VF veya pVT olduğunda HDKA sonrası ROSC bilinçsiz erişkin hastalarda hipotermi uygulanabilir. (sınıf I, KD: B-R)

2-Nonşokabl ritimlerde de hipotermi faydalı olabilir (sınıf I, KD: C-EO)

3-Sıcaklık 32°C ile 36°C arasında olmalıdır (sınıf I, KD: B-R).

4-TTM'yi en az 24 saat süreyle sürdürmek mantıklıdır (sınıf IIa, KD: C-EO).

5- Rutin olarak ROSC lu hastalarda hastane öncesi IV hızlı infüzyon ile hipotermi önerilmemektedir (sınıf III: yarar yok; KD-A)

6-TTM sonrası komadaki hastalarda ateşi önlemek makuldür (sınıf IIb, KD C-LD).

7-Kardiyak arrest nedeniyle resüsitasyon sonrası spontan hafif hipotermi (> 33 ° C) olan hemodinamik olarak stabil olan hastalar aktif olarak yeniden ısıtılmamalıdır.

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Canadian Guidelines for the use of targeted temperature management (therapeutic hypothermia) after cardiac arrest: A joint statement from The Canadian Critical Care Society (CCCS), Canadian Neurocritical Care Society (CNCCS), and the Canadian Critical Care Trials Group (CCCTG)

Daniel Howes[a,b,*], Sara H. Gray[d], Steven C. Brooks[a,p], J. Gordon Boyd[b,e], Dennis Djogovic[f], Eyal Golan[g,h], Robert S. Green[i], Michael J. Jacka[j], Tasnim Sinuff[k,l], Timothy Chaplin[a], Orla M. Smith[m], Julian Owen[n], Adam Szulewski[a], Laurel Murphy[i], Stephanie Irvine[b], Draga Jichici[o], John Muscedere[b,c]

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Endikasyonlar:

1. TTM resüsitasyon sonrası uygun erişkin hastalara nöral koruma amacıyla uygulanmalıdır.(öd güçlü, ks yüksek)

2. Resüsitasyon sonrası komatöz ve sözlü komutlara yanıt vermeyen vakalar için TTM düşünülmelidir.(öd güçlü, ks düşük)

öd güçlü, ks yüksek

- Şoklanabilir ritim
- HDKA
- Kardiyak nedenli ya da bilinmeyen kardiyak nedenli arrestler

öd şartlı, ks düşük

- Şoklanamaz ritimler
- HİKA
- Nonkardiyak nedenler

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Association Between Therapeutic Hypothermia and Survival After In-Hospital Cardiac Arrest:

Hypothermia after in-hospital cardiac arrest

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Saint Luke's Mid America Heart Institute, Kansas City, Missouri (P.S.C., Y.T., J.A.S.) and the University of Missouri-Kansas City (P.S.C., J.A.S.); Children's Hospital of Philadelphia, PA (R.A.B), and the Duke Clinical Research Institute and Duke University School of Medicine, Durham, North Carolina (L.H.C)

Özel Durumlar:

- Gebe hastalar TTM için uygundur.(öd şartlı, ks düşük)
- İleri yaş TTM için kontrendikasyon değildir. (öd güçlü, ks düşük)
- Acil koroner anjiografi veya PCI gereksinimi olan hastalar içinTTM kontrendike değildir. (öd güçlü, ks düşük)
- Rekürren VF/VT kontrendikasyon değildir.(öd şartlı, ks düşük)

Kontrendikasyonlar:

- Kontrolsüz kanama ve dirençli şok tablosu TTM için kontrendikasyon olarak düşünülmelidir. (öd güçlü, ks düşük)
- Ağır enfeksiyonu olan hastalara TTM uygulanmamalıdır.(öd şartlı, ks düşük)
- Hipotansiyon (MAP<60 mmHg) TTM için mutlak kontrendikasyon değildir. MAP ve perfüzyonu iyileştirmek amacıyla agresif resüsitasyon uygulanmalıdır.

TTM'nin hastane öncesi, acil servis ve kritik bakım ünitesi dahil olmak üzere gerekli desteklerle herhangi tıbbi bir ortamda başlatılmasını tavsiye ediyoruz.

(öd güçlü, ks orta)

Induction of Therapeutic Hypothermia During Out-of-Hospital Cardiac Arrest Using a Rapid Infusion of Cold Saline

The RINSE Trial (Rapid Infusion of Cold Normal Saline)

BACKGROUND: Patients successfully resuscitated by paramedics from out-of-hospital cardiac arrest often have severe neurologic injury. Laboratory and observational clinical reports have suggested that induction of therapeutic hypothermia during cardiopulmonary resuscitation (CPR) may improve neurologic outcomes. One technique for induction of mild therapeutic hypothermia during CPR is a rapid infusion of large-volume cold crystalloid fluid.

METHODS: In this multicenter, randomized, controlled trial we assigned adults with out-of-hospital cardiac arrest undergoing CPR to either a rapid intravenous infusion of up to 2 L of cold saline or standard care. The primary outcome measure was survival at hospital discharge; secondary end points included return of a spontaneous circulation. The trial was closed early (at 48% recruitment target) due to changes in temperature management at major receiving hospitals.

RESULTS: A total of 1198 patients were assigned to either therapeutic hypothermia during CPR (618 patients) or standard prehospital care (580 patients). Patients allocated to therapeutic hypothermia received a mean (SD) of 1193 (647) mL cold saline. For patients with an initial shockable cardiac rhythm, there was a decrease in the rate of return of a spontaneous circulation in patients who received cold saline compared with standard care (41.2% compared with 50.6%, P=0.03). Overall 10.2% of patients allocated to therapeutic hypothermia during CPR were alive at hospital discharge compared with 11.4% who received standard care (P=0.71).

CONCLUSIONS: In adults with out-of-hospital cardiac arrest, induction of mild therapeutic hypothermia using a rapid infusion of large-volume, intravenous cold saline during CPR may decrease the rate of return of a spontaneous circulation in patients with an initial shockable rhythm and produced no trend toward improved outcomes at hospital discharge.

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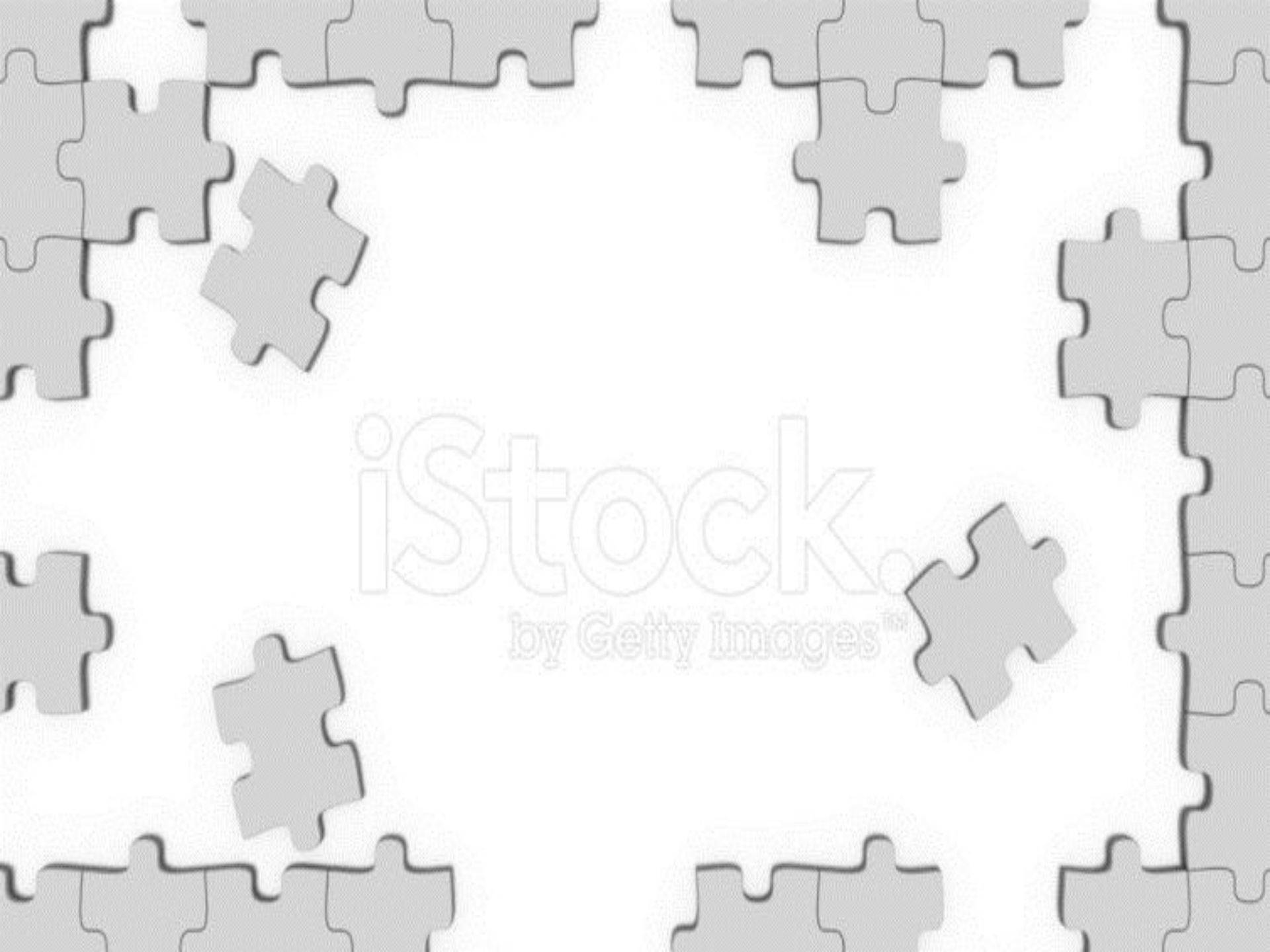
Zamanlama:

- Klinisyenler hedef sıcaklığa olabildiğince erken ulaşmalıdır.(öd güçlü, ks düşük)
- Önlenemeyen bir gecikmenin yaşandığı durumlarda , TTM ROSC'tan 8 veya daha fazla saat sonra da faydalı olabilir. Hastaların soğutulmaması gereken bir süre belli değildir. Ancak zaman geçtikçe faydanın azalma olasılığı artar.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Soğutma İşlemi:

TTM uygulanan hastaların 32-34 derece bir hedef sıcaklığa soğutulması önerilir. (ÖD koşullu, KD yüksek)

Contents lists available at ScienceDirect

Resuscitation

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

Review article

Therapeutic hypothermia after cardiac arrest: A systematic review/meta-analysis exploring the impact of expanded criteria and targeted temperature☆

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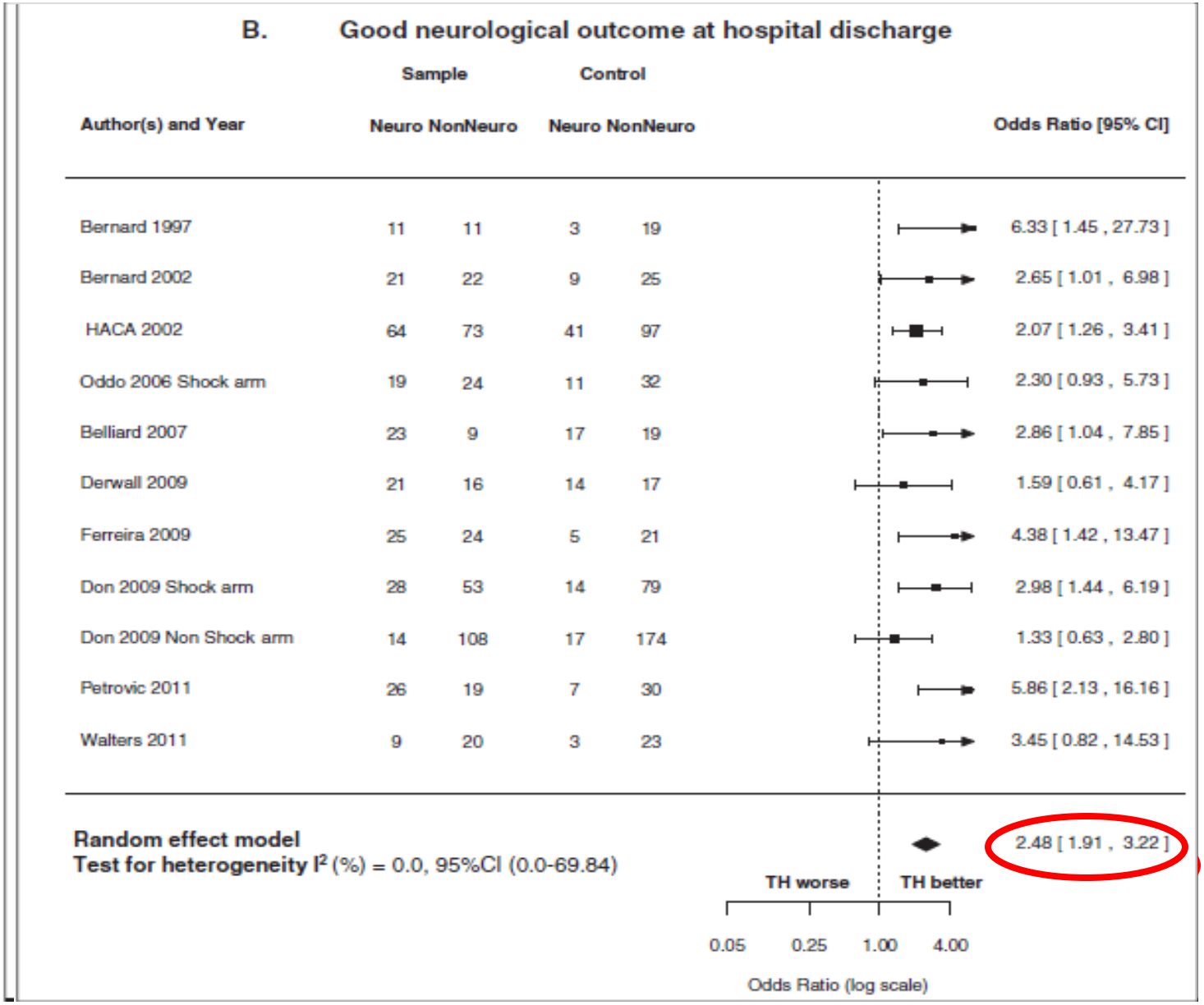
Targeted temperature management
Expanded hypothermia use

ABSTRACT

Aims of the study: We aimed to determine the benefit of an expanded use of TH. We also described the impact of a targeted temperature management on outcomes at discharge.
Data sources: We identified studies by searching MEDLINE, EMBASE and Cochrane Library databases. We included RCTs and observational studies restricted to those reporting achieved temperature during TH after OHCA. No other patient, cardiac arrest or hypothermia protocol restrictions were applied. Outcomes of interest were hospital mortality and neurological outcome at discharge. Appropriate risk of bias assessment for meta-analyzed studies was conducted. Studies contrasting hypothermia and normothermia outcomes were meta-analyzed using a random-effect model. Outcomes of cooling arms, obtained from enrolled studies, were pooled and compared across achieved temperatures.
Results: Search strategy yielded 32,275 citations of which 24 articles met inclusion criteria. Eleven studies were meta-analyzed. The use of TH after OHCA, even within an expanded use, decreased the mortality (OR 0.51, 95%CI [0.41–0.64]) and improved the odds of good neurological outcome (OR 2.48, 95%CI [1.91–3.22]). No statistical heterogeneity was found for either mortality ($I^2 = 4.0\%$) or neurological outcome ($I^2 = 0.0\%$). No differences in hospital mortality ($p = 0.86$) or neurological outcomes at discharge ($p = 0.32$) were found when pooled outcomes of 34 hypothermia arms grouped by cooling temperature were compared.
Conclusion: The use of TH after OHCA is associated with a survival and neuroprotective benefit, even when including patients with non-shockable rhythms, more lenient downtimes, unwitnessed arrest and/or persistent shock. We found no evidence to support one specific temperature over another during hypothermia.


B. Good neurological outcome at hospital discharge
Sample
Control
Author(s) and Year
Neuro NonNeuro
Neuro NonNeuro
Odds Ratio [95% CI]
Bernard 1997 11 11 3 19 6.33 [1.45 , 27.73]
Bernard 2002 21 22 9 25 2.65 [1.01 , 6.98]
HACA 2002 64 73 41 97 2.07 [1.26 , 3.41]
Oddo 2006 Shock arm 19 24 11 32 2.30 [0.93 , 5.73]
Belliard 2007 23 9 17 19 2.86 [1.04 , 7.85]
Derwall 2009 21 16 14 17 1.59 [0.61 , 4.17]
Ferreira 2009 25 24 5 21 4.38 [1.42 , 13.47]
Don 2009 Shock arm 28 53 14 79 2.98 [1.44 , 6.19]
Don 2009 Non Shock arm 14 108 17 174 1.33 [0.63 , 2.80]
Petrovic 2011 26 19 7 30 5.86 [2.13 , 16.16]
Walters 2011 9 20 3 23 3.45 [0.82 , 14.53]
Random effect model
Test for heterogeneity I² (%) = 0.0, 95%CI (0.0-69.84)
2.48 [1.91 , 3.22]
TH worse
TH better
0.05
0.25
1.00
4.00
Odds Ratio (log scale)

Contents lists available at ScienceDirect

Resuscitation

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

Clinical paper

Changing target temperature from 33 °C to 36 °C in the ICU management of out-of-hospital cardiac arrest: A before and after study☆

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Keywords:
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Resuscitation
Therapeutic hypothermia

ABSTRACT

Introduction: In December 2013, our institution changed the target temperature management (TTM) for the first 24 h in ventricular fibrillation out-of-hospital cardiac arrest (VF-OHCA) patients from 33 °C to 36 °C. This study aimed to examine the impact this change had on measured temperatures and patient outcomes.

Methods: We conducted a retrospective cohort study of consecutive VF-OHCA patients admitted to a tertiary referral hospital in Melbourne (Australia) between January 2013 and August 2015. Outcomes were adjusted for age and duration of cardiac arrest.

Results: Over the 30-month period, 76 VF-OHCA cases were admitted (24 before and 52 after the TTM change). Patient demographics, cardiac arrest features and hospital interventions were similar between the two periods. After the TTM change, less patients received active cooling (100% vs. 70%, $p < 0.001$), patients spent less time at target temperature (87% vs. 50%, $p < 0.001$), and fever rates increased (0% vs. 19%, $p = 0.03$). During the 36 °C period, there was a decrease in the proportion of patients who were discharged: alive (71% vs. 58%, $p = 0.31$), home (58% vs. 40%, $p = 0.08$); and, with a favourable neurological outcome (cerebral performance category score 1-2: 71% vs. 56%, $p = 0.22$).

Conclusion: After the change from a TTM target of 33 °C to 36 °C, we report low compliance with target temperature, higher rates of fever, and a trend towards clinical worsening in patient outcomes. Hospitals adopting a 36 °C target temperature to need to be aware that this target may not be easy to achieve, and requires adequate sedation and muscle-relaxant to avoid fever.

Table 3
Comparison of temperatures and patient outcomes for patients admitted to ICU for the 33 °C and 36 °C TTM periods.

Outcome variables	33 °C N = 24	36 °C N = 52	Unadjusted p-value	Adjusted p-value[a]
Temperatures				
Temperature on arrival at ED, mean (SD)	34.5 (1.4)	35.2 (1.5)	0.07	–
Average temperature across first 24 h, median (IQR)	33.4 (33.1–33.5)	36.2 (36.1–36.5)	<0.001	–
Time at target temperature in first 24-h (%), mean (SD)	87 (15)	50 (31)	<0.001	–
All temperatures at or below target in first 24-h, n (%)				
Temperature ≥38.0 in first 24 h, n (%)	0 (0)	10 (19)	0.03	–
Temperature ≥38.0 in first 36 h, n (%)	2 (8)	16 (31)	0.04	–
Outcomes				
Discharge within 24 h of ICU, n (%)	0 (0)	2 (4)	0.99	–
Died within 24-h of ICU, n (%)	0 (0)	7 (14)	0.09	–
ICU survival, n (%)	17 (71)	30 (58)	0.32	0.31
Hospital survival, n (%)	17 (71)	30 (58)	0.32	0.31
Day treatment withdrawn, median (IQR)	3 (3–16)	4 (3–18)	0.99	
CPC score 1–2, n (%)	17 (71)	29 (56)	0.31	0.22
Discharged home, n (%)	14 (58)	21 (40)	0.22	0.08
Discharged to rehabilitation, n (%)	3 (18)	8 (27)	0.48	0.55
Survivors CPC 1, n (%)	<16 (94)	19 (63)		–
Survivors CPC 2, n (%)	1 (6)	10 (33)	0.02	
Survivors CPC 3, n (%)	0 (0)	1 (3)		

Hipotermi Süresi ve Yeniden Isınma:

- Yeniden ısınmanın hedef sıcaklığa ulaşmasından 24 saat sonra başlamalıdır.
- Hastalar satte 0.25-0.5 derece hızda yeniden ısıtılmalıdır.
- Hipertermi (çekirdek sıcaklığın 37.5 derece üzerinde olması) arrest sonrası en az 72 saat süre ile önlenmelidir.

Research

JAMA | Original Investigation | CARING FOR THE CRITICALLY ILL PATIENT

Targeted Temperature Management for 48 vs 24 Hours and Neurologic Outcome After Out-of-Hospital Cardiac Arrest

A Randomized Clinical Trial

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Table 3. Primary and Secondary Outcomes After Targeted Temperature Management

	48-Hour Group (n = 175)	24-Hour Group (n = 176)	Difference, % (95% CI)	Relative Risk or Ratio of Geometric Means (95% CI)	*P* Value
Primary outcome, No. (%)					
CPC score of 1 or 2 at 6 mo[a]	120 (69)	112 (64)	4.9 (−5 to 14.8)	1.08 (0.93 to 1.25)	.33
Secondary outcomes, No. (%)					
ICU mortality	26 (15)	30 (17)	−2.1 (−9.7 to 5.5)	0.88 (0.54 to 1.42)	.59
Hospital mortality	40 (23)	44 (25)	−2 (−10.9 to 6.9)	0.92 (0.63 to 1.34)	.66
Mortality at 6 mo	48 (27)	60 (34)	−6.5 (−16.1 to 3.1)	0.81 (0.59 to 1.11)	.19
Adverse events, No. (%)					
Any adverse event	169 (97)	161 (91)	5.6 (0.6 to 10.6)	1.06 (1.01 to 1.12)	.03
Pneumonia	86 (49)	76 (43)	6.2 (−4.2 to 16.6)	1.14 (0.91 to 1.44)	.24
Any bleeding	17 (10)	23 (13)	−3.3 (−9.9 to 3.3)	0.75 (0.41 to 1.35)	.33
Resource use, median (IQR)					
Time receiving mechanical ventilation, h[b,c]	120 (99 to 146)	87 (72 to 106)	26 (16 to 36)	1.37 (1.19 to 1.59)	<.001
Survivors, h	121 (98 to 149)	85 (69 to 105)	28 (18 to 38)	1.41 (1.22 to 1.64)	<.001
Nonsurvivors, h	107 (71 to 163)	151 (89 to 256)	−79 (−205 to 48)	0.71 (0.37 to 1.37)	.35
ICU length of stay, h[c]	151 (127 to 178)	117 (99 to 138)	28 (15 to 41)	1.3 (1.14 to 1.47)[c]	<.001
Survivors, h	184 (141 to 240)	134 (103 to 175)	30 (17 to 44)	1.37 (1.22 to 1.54)	<.001
Nonsurvivors, h	92 (66 to 129)	88 (64 to 121)	9 (−25 to 44)	1.04 (0.66 to 1.64)	.86
Hospital length of stay, d[c]	11.1 (9.3 to 13.3)	11.8 (9.9 to 14.1)	−0.85 (−2.6 to 0.9)	0.94 (0.79 to 1.11)	.50
Survivors, d	15.9 (12.6 to 20)	17.2 (13.7 to 21.6)	−1.15 (−3.1 to 0.8)	0.93 (0.8 to 1.06)	.28
Nonsurvivors, d	5.1 (3.6 to 7.1)	5.3 (3.8 to 7.5)	0 (−1.9 to 1.9)	0.95 (0.64 to 1.41)	.79


BERNARD
HACA
CAPITAL-CHILL
HYPERION
NIELSEN
THAPCA-OH

Kardiyak Arrestte Lipit Emülsiyon Tedavisi

- ILE tedavisi 1967 yılında ilk defa antidot olarak kullanılmış.
- 1997 yılında bupivakaine ait bir toksisite vaka raprunda yayınlanmış.
- LA toksisitesi ve lipofilik ilaçlar…

Etki Mekanizması:

- Emülsiyon lipofilik ilaç molekülünü çevreleyerek etkisiz hale getirir ve bir "lipid havuzu" olarak hareket eder.
- Lipid solüsyonu içinde bulunan uzun zincirli yağ asitlerinin myositlerdeki voltaj bağımlı kalsiyum kanallarını aktive ettikleri gösterilmiş. Bu sayede myositlere kalsiyum girişi artmakta ve kontraktilite artmakta.
- Kardiyak inotropiyi artırır.
- ILE'den gelen yağ asitlerinin miyokarda, hazır bir enerji kaynağı sağlaması ve böylece kardiyak fonksiyonu iyileştirmesidir

Uygulama:

- %20 lik uzun zincirli trigliserit emülsiyonu
- 1.5 ml/kg yağsız vücut kilosuna göre bolus aynı doz, üç üç dozda olmak üzere her üç ila beş dakikada bir kardiyak arrest durumunda tekrarlanabilir.
- 0.25-0.5 ml/kg/dk 30-60 dk'da verilmelidir.

Kılavuz:

- Lokal anestezik toksisitesine bağlı , özellikle bupivakain toksisitesine bağlı kardiyak arrest vakalarında resüsitatif bakıma ilavaten ILE uygulanabilir. (SınıfIIb-KD C-EO)
- Diğer ilaç toksisitelerine bağlı kardiyak arrestte ILE uygulaması makuldür. (SınıfIIb-KD C-EO)

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REVIEW

Evidence-based recommendations on the use of intravenous lipid emulsion therapy in poisoning*

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Bupivakain toksisitesine bağlı kardiyak arrest ACLS ile birlikte ILE kullanılması önerilir.(1D)

Amitriptilin ve TCA
Beta Blokerler
Kalsiyum kanal blokerleri NÖTR
Bupropion
Kokain
Difenhidramin
Lamotrijin

Online Review Article

OPEN

Experts Consensus Recommendations for the Management of Calcium Channel Blocker Poisoning in Adults

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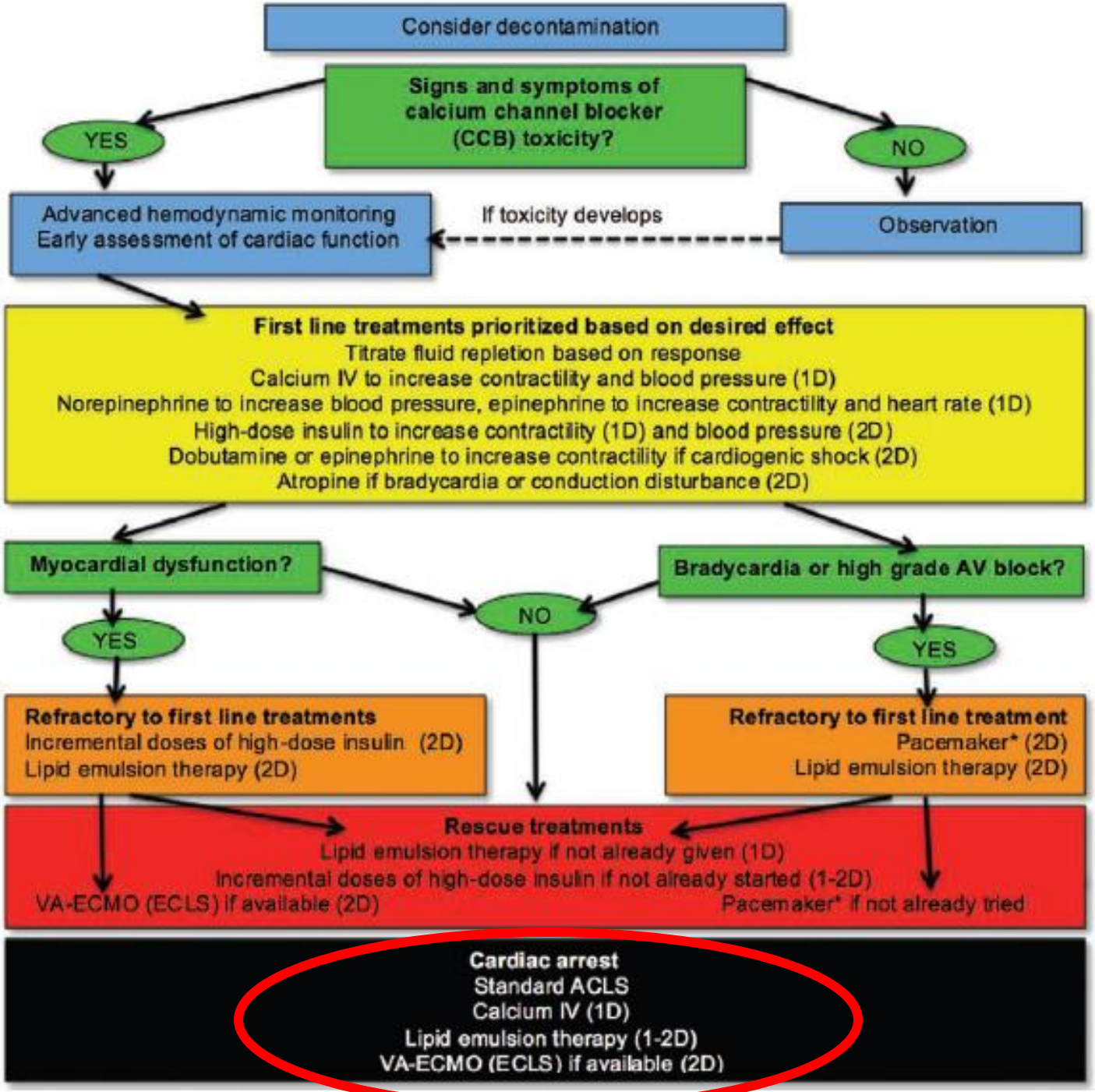

Definitions:

- Signs of CCB toxicity: Hemodynamics abnormalities such as low heart rate, low blood pressure, decreased contractility or abnormal peripheral vascular resistances
- First line treatment: treatment initially provided to a symptomatic CCB poisoned patient
- Rescue treatment: treatment provided to a CCB poisoned patient in refractory shock or peri-arrest
- Shock: state where there is an inadequate blood flow and oxygen delivery to organs and tissues
- Myocardial dysfunction: decreased myocardial contractility seen on the cardiac ultrasound or cardiac index of less than 2.2L/min/m^2

Dose regimens:

- Calcium chloride 10% IV: Adults: 10-20 ml (1-2 g) Q10-20 min or infusion at 0.2-0.4 ml/kg/h (0.02-0.04 g/kg/h)
- Calcium gluconate 10% IV: Adults: 30-60 ml (3-6 g) Q10-20 min or infusion at (0.6-1.2ml/kg/h (0.06-0.12 g/kg/h)
- Atropine: 0.02 mg/kg (min 0.1 mg and max 0.5 mg)
- High-dose insulin IV (regular): 1unit/kg bolus followed by an infusion at 1unit/kg/h (maintain euglycemia with dextrose)
- Incremental doses of high-dose insulin IV (regular): progressive increase of the infusion rate up to 10units/kg/h (maintain euglycemia with dextrose)

*: in the absence of myocardial dysfunction

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REVIEW

Systematic review of clinical adverse events reported after acute intravenous lipid emulsion administration

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ABSTRACT

Background: Intravenous lipid emulsions (ILEs) were initially developed to provide parenteral nutrition. In recent years, ILE has emerged as a treatment for poisoning by local anesthetics and various other drugs. The dosing regimen for the clinical toxicology indications differs significantly from those used for parenteral nutrition. The evidence on the efficacy of ILE to reverse acute toxicity of diverse substances consists mainly of case reports and animal experiments. Adverse events to ILE are important to consider when clinicians need to make a risk/benefit analysis for this therapy. **Methods:** Multiple publication databases were searched to identify reports of adverse effects associated with acute ILE administration for either treatment of acute poisoning or parenteral nutrition. Articles were selected based on pre-defined criteria to reflect acute use of ILE. Experimental studies and reports of adverse effects as a complication of long-term therapy exceeding 14 days were excluded. **Results:** The search identified 789 full-text articles, of which 114 met the study criteria. 27 were animal studies, and 87 were human studies. The adverse effects associated with acute ILE administration included acute kidney injury, cardiac arrest, ventilation perfusion mismatch, acute lung injury, venous thromboembolism, hypersensitivity, fat embolism, fat overload syndrome, pancreatitis, extracorporeal circulation machine circuit obstruction, allergic reaction, and increased susceptibility to infection. **Conclusion:** The emerging use of ILE administration in clinical toxicology warrants careful attention to its potential adverse effects. The dosing regimen and context of administration leading to the adverse events documented in this review are not generalizable to all clinical toxicology scenarios. Adverse effects seem to be proportional to the rate of infusion as well as total dose received. Further safety studies in humans and reporting of adverse events associated with ILE administration at the doses advocated in current clinical toxicology literature are needed.

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- Asistoli
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- Yağ embolisi, ARDS,ALI
- Priapizm, DVT, flebit
- Hipertrigliseritemi, pankreatit
- Hipersensitivite ve allerjik yan etkiler

TROMBOLİTİK TEDAVİ

MİYOKARD İNFARKTÜSÜ

STROKE

PULMONER EMBOLİ

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Thrombolysis during Resuscitation for Out-of-Hospital Cardiac Arrest

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TISSUE PLASMINOGEN ACTIVATOR IN CARDIAC ARREST WITH PULSELESS ELECTRICAL ACTIVITY

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ABSTRACT

Background Coronary thrombosis and pulmonary thromboembolism are common causes of cardiac arrest. We assessed whether the administration of tissue plasminogen activator (t-PA) during cardiopulmonary resuscitation would benefit patients with cardiac arrest and pulseless electrical activity of unknown or presumed cardiovascular cause.

Methods Patients who were older than 16 years of age and who had more than one minute of pulseless electrical activity that was unresponsive to initial therapy outside the hospital or in the emergency department were eligible. Patients were randomly assigned to receive 100 mg of t-PA or placebo intravenously over a 15-minute period in a double-blind fashion. Standard resuscitation was then continued for at least 15 minutes. The primary outcome was survival to hospital discharge.

Results During the study period, 1583 patients with cardiac arrest were treated and 233 patients were enrolled (117 in the t-PA group and 116 in the placebo group). The characteristics of the patients in the two groups were similar. One patient in the t-PA group survived to hospital discharge, as compared with none in the placebo group (absolute difference between groups, 0.9; 95 percent confidence interval, −2.6 to 4.8; P=0.99). The proportion of patients with return of spontaneous circulation was 21.4 percent in the t-PA group and 23.3 percent in the placebo group (absolute difference between groups, −1.9; 95 percent confidence interval, −12.6 to 8.8; P=0.85).

Conclusions We found no evidence of a beneficial effect of fibrinolysis in patients with cardiac arrest and pulseless electrical activity of unknown or presumed cardiovascular cause. Our study had limited statistical power, and it remains unknown whether there is a small treatment effect or whether selected subgroups may benefit. (N Engl J Med 2002;346:1522-8.)

severe shock, since some forward blood flow can occur.[4-6] This observation has prompted heightened vigilance for reversible causes during treatment of pulseless electrical activity.[7] That low-grade cerebral and coronary perfusion may persist during pulseless electrical activity supports the concept that the outcome can be good, even after prolonged cardiopulmonary resuscitation (CPR), if the cause of cardiac arrest is identified and addressed.

Few studies have characterized the causes of cardiac arrest, but acute coronary thrombosis and pulmonary thromboembolism are clearly important.[8,9] Given the benefit of fibrinolytic (thrombolytic) agents in myocardial infarction and pulmonary embolism, there is increasing interest in the potential role of these drugs in cardiac arrest. Both theoretical reasoning and studies in laboratory animals support the hypothesis that fibrinolytic agents could be effective during CPR.[10-14]

Many studies have suggested that the administration of fibrinolytic agents during CPR can have a dramatic effect,[15-25] but there have been no randomized trials of this approach. We undertook this study to evaluate the effect of the administration of tissue plasminogen activator (t-PA) during CPR in adults with undifferentiated pulseless electrical activity (i.e., with an unknown or presumed cardiovascular cause) that was not responsive to initial therapy.

METHODS

Location of the Study

This randomized, double-blind, placebo-controlled trial was conducted between February 12, 1998, and September 30, 1999, in the greater Vancouver region in Canada at seven advanced-life-support paramedic base stations and in the emergency departments of three tertiary teaching hospitals. This region has a population of over 2 million persons served by the British Columbia Ambulance Service. In this region, victims of out-of-hospital cardiac arrest are treated by paramedics using protocols based on American Heart Association guidelines,[26] and they are not transported to a hospital unless a perfusing rhythm develops, a shockable rhythm persists, or extenuating circumstances exist.

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Case Report

Successful use of intra-arrest thrombolysis for electrical storm due to acute myocardial infarction☆

PULMONER EMBOLİ

Retrospektif karşılaştırmalı bir çalışmada PE'ye bağlı kardiyak arrest sırasında rt-PA ile fibrinolitik tedavi alan grupta, ROSC (% 67'ye% 43), 24 saat sağkalım (% 53'e% 23) ve hastaneden taburculuk (% 19'a karşı% 7) açısından anlamlı olarak daha iyi sonuçlar elde edildi.

Major bleeding complications in cardiopulmonary resuscitation: the place of thrombolytic therapy in cardiac arrest due to massive pulmonary embolism. Resuscitation. 2003;57(1):49-55.

Kılavuz:

- Şüpheli PE'de ampirik trombolizin etkinliğini destekleyen veya reddeden herhangi bir kanıt yoktur.

- Bu hastalarda tromboliz düşünülebilir.(SınıfIIb-KD C-LD)

- Dahil etme kriterleri, trombolitik zamanlama ilaç ve doz konusunda fikir birliği yok.

- Tromboliz, cerrahi veya mekanik embolektemi acil tedavi seçenekleri arasındadır.(SınıfIIa-KD C-LD)

- Tromboliz göğüs kompresyonları yapıldığı sırada bile faydalı olabilir.(SınıfIIa-KD C-LD)

Hangi durumda şüphelenelim?

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

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Review

Evidence-based diagnosis and thrombolytic treatment of cardiac arrest or periarrest due to suspected pulmonary embolism☆,☆☆

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ABSTRACT

When a previously healthy adult experiences atraumatic cardiac arrest, providers must quickly identify the etiology and implement potentially lifesaving interventions such as advanced cardiac life support. A subset of these patients develop cardiac arrest or periarrest due to pulmonary embolism (PE). For these patients, an early, presumptive diagnosis of PE is critical in this patient population because administration of thrombolytic therapy may significantly improve outcomes. This article reviews thrombolysis as a potential treatment option for patients in cardiac arrest or periarrest due to presumed PE, identifies features associated with a high incidence of PE, evaluates thrombolytic agents, and systemically reviews trials evaluating thrombolytics in cardiac arrest or periarrest. Despite potentially improved outcomes with thrombolytic therapy, this intervention is not without risks. Patients exposed to thrombolytics may experience major bleeding events, with the most devastating complication usually being intracranial hemorrhage. To optimize the risk-benefit ratio of thrombolytics for treatment of cardiac arrest due to PE, the clinician must correctly identify patients with a high likelihood of PE and must also select an appropriate thrombolytic agent and dosing protocol.

1-Ritim:

NEA. Diğer ritimlere kıyasla daha fazla PE insidansına sahiptir.

2-Arrest sırasında ve prearrest dönemde bulunan faktörler:

- PE'ye bağlı kardiyak arrest hastalarında şu triad ilişkili bulunmuştur:

-Şahitli kardiyak arrest
-NEA
-Yas<65-70

Resuscitation 2001;49:265-72.
Resuscitation 2005;65:57-64.

- Ortak prearrest bulgular ise;

- Solunum güçlüğü
- Şİŕ 0.8
- Mental durum değişikliği

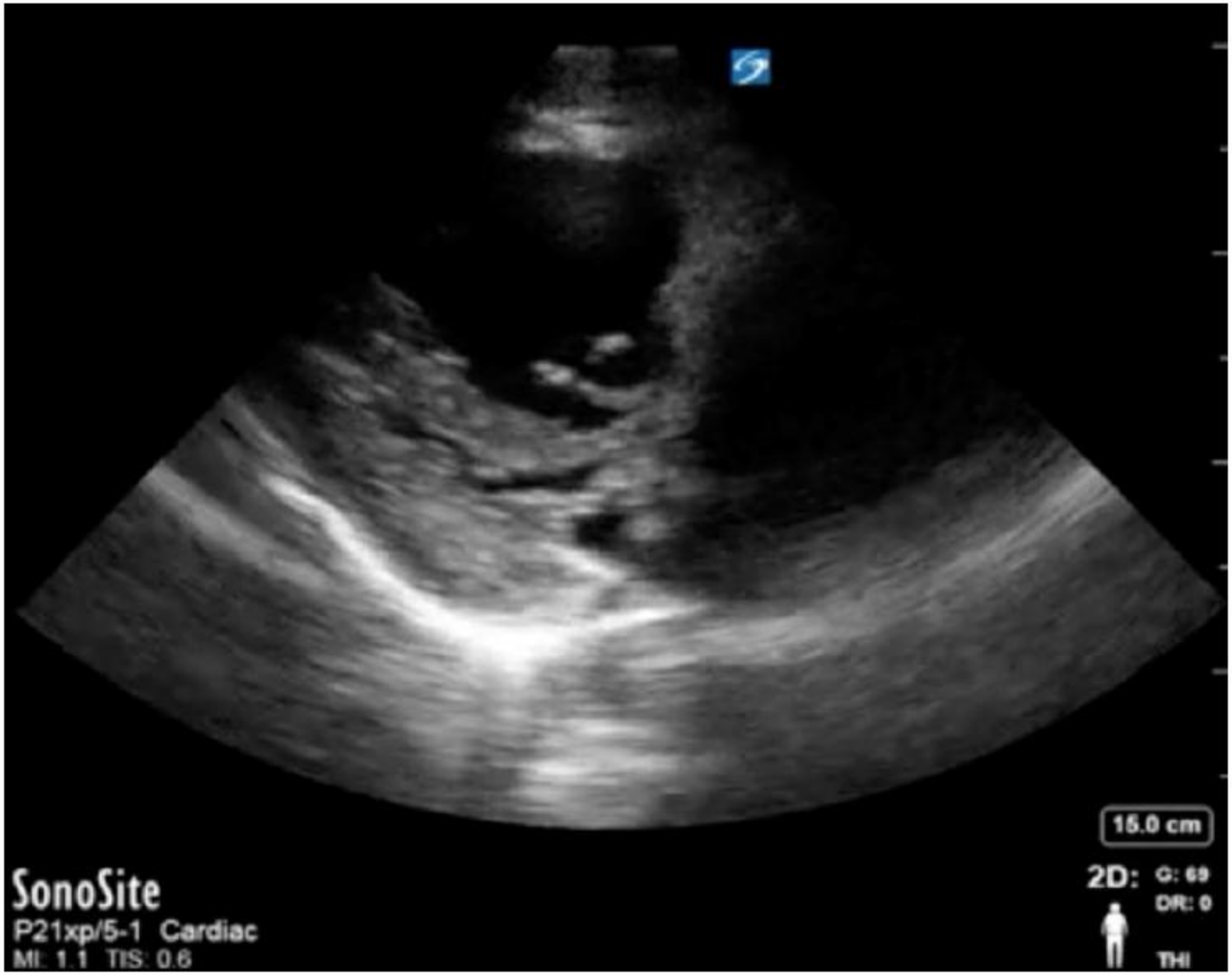
Resuscitation 2001;49:265-72.
Resuscitation 2005;65:57-64.

3-Geleneksel PE ile ilişkili faktörler:

- Tek taraflı bacak şişliği
- Saturasyon<%95
- Aktif kanser
- İmmobilizasyon
- Ortapedik cerrahi
- Havayolu ile seyahat

4-USG

Arrest sırasında kardiyak hareket ve kontraktil aktiviteyi tanımlamak ve tedavi edilebilir durumları (tamponat, miyokard enfarktüsü, pulmoner emboli, pnömotoraks ve hipovolemi) teşhis etmek için kullanılabilir.


SonoSite
P21xp/5-1 Cardiac
MI: 1.1 TIS: 0.6
15.0 cm
2D:
G: 69
DR: 0
THI

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Review

Echocardiography in cardiac arrest: An emergency medicine review☆

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ABSTRACT

Introduction: Cardiac arrest management primarily focuses on optimal chest compressions and early defibrillation for shockable cardiac rhythms. Non-shockable rhythms such as pulseless electrical activity (PEA) and asystole present challenges in management. Point-of-care ultrasound (POCUS) in cardiac arrest is promising.
Objectives: This review provides a focused assessment of POCUS in cardiac arrest, with an overview of transthoracic (TTE) and transesophageal echocardiogram (TEE), uses in arrest, and literature support.
Discussion: Cardiac arrest can be distinguished between shockable and non-shockable rhythms, with management varying based on the rhythm. POCUS provides a diagnostic and prognostic tool in the emergency department (ED), which may improve accuracy in clinical decision-making. Several protocols incorporate POCUS based on different cardiac views. TTE includes parasternal long axis, parasternal short axis, apical 4-chamber, and subxiphoid views, which may be used in cardiac arrest for diagnosis of underlying cause and potential prognostication. TEE is conducted by inserting the probe into the esophagus of intubated patients, with several studies evaluating its use in cardiac arrest. It is associated with few adverse effects, while allowing continued compressions (and evaluation of those compressions) and not interrupting resuscitation efforts.
Conclusions: POCUS is a valuable diagnostic and prognostic tool in cardiac arrest, with recent literature supporting its diagnostic ability. TTE can guide resuscitation efforts dependent on the rhythm, though TTE should not interrupt other resuscitation measures. TEE can be useful during arrest, but further studies based in the ED are needed.
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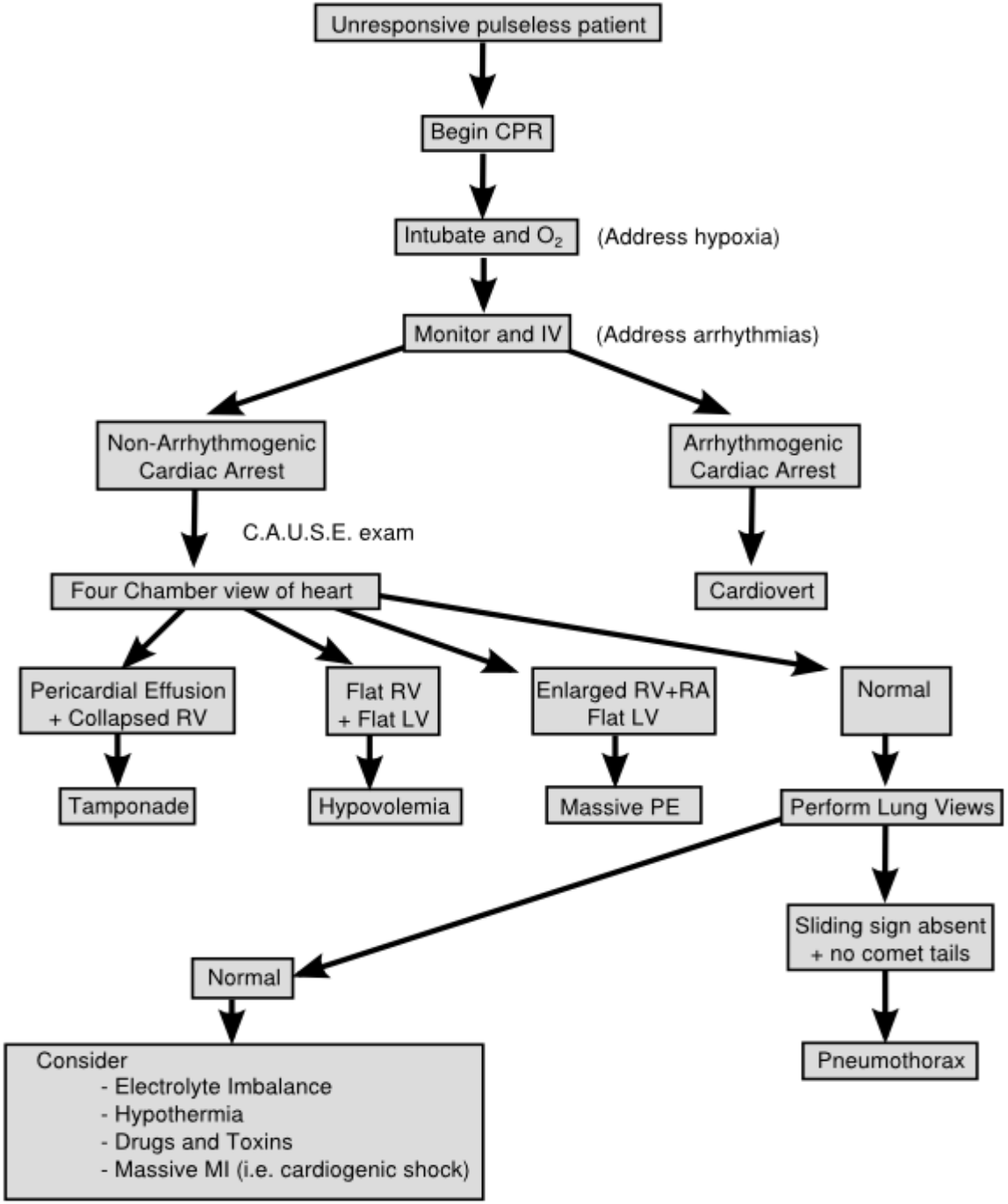
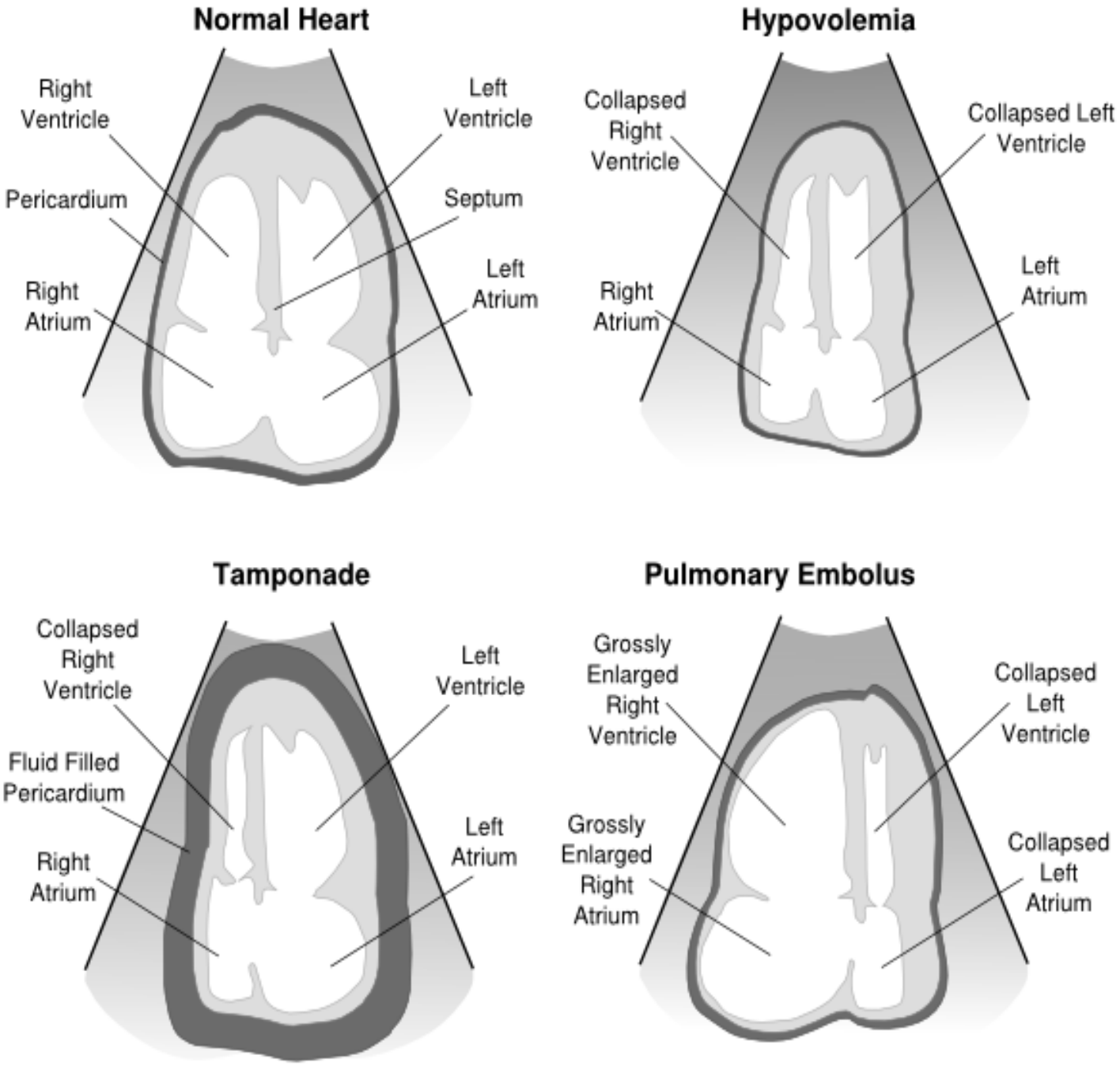
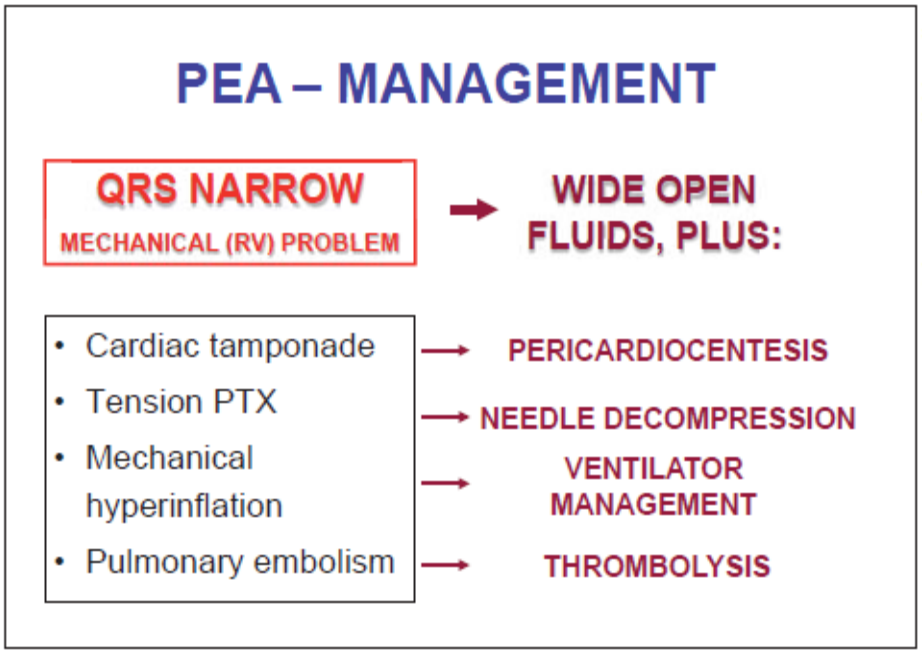
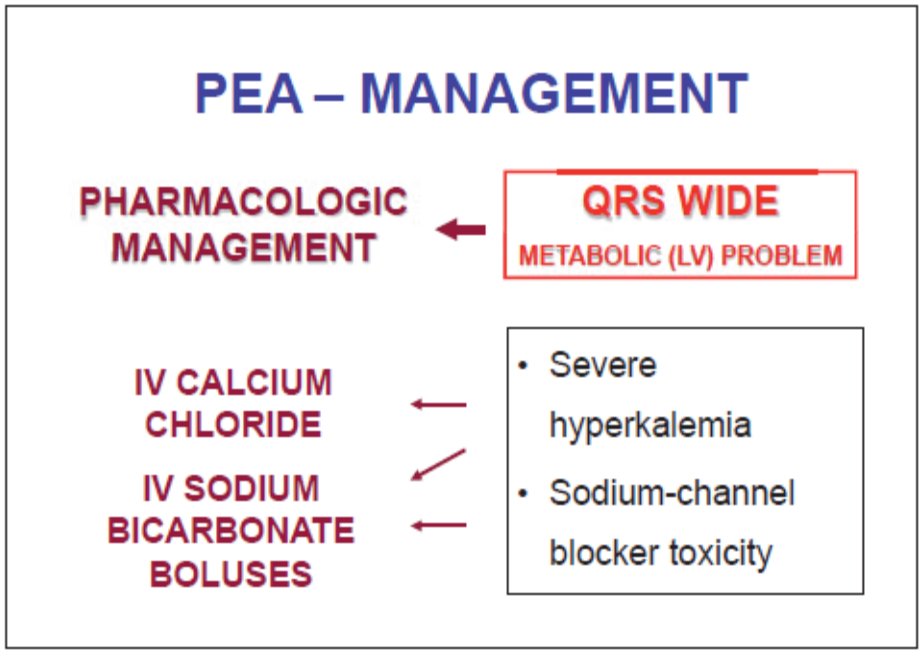

Unresponsive pulseless patient
Begin CPR
Intubate and O_2
(Address hypoxia)
Monitor and IV
(Address arrhythmias)
Non-Arrhythmogenic Cardiac Arrest
Arrhythmogenic Cardiac Arrest
C.A.U.S.E. exam
Four Chamber view of heart
Cardiovert
Pericardial Effusion + Collapsed RV
Flat RV + Flat LV
Enlarged RV+RA Flat LV
Normal
Tamponade
Hypovolemia
Massive PE
Perform Lung Views
Sliding sign absent + no comet tails
Normal
Pneumothorax
Consider
- Electrolyte Imbalance
- Hypothermia
- Drugs and Toxins
- Massive MI (i.e. cardiogenic shock)


Normal Heart
Right Ventricle
Left Ventricle
Pericardium
Septum
Right Atrium
Left Atrium
Hypovolemia
Collapsed Right Ventricle
Collapsed Left Ventricle
Right Atrium
Left Atrium
Tamponade
Collapsed Right Ventricle
Left Ventricle
Fluid Filled Pericardium
Right Atrium
Left Atrium
Pulmonary Embolus
Grossly Enlarged Right Ventricle
Collapsed Left Ventricle
Grossly Enlarged Right Atrium
Collapsed Left Atrium



Color version available online



USG

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REVIEW

Point-of-care ultrasound in cardiopulmonary resuscitation: a concise review

Pablo Blanco[1] · **Carmen Martínez Buendía**[2]

Table 1 POCUS protocols to be used in CPR

	CAUSE[10]	FEEL[3] FEER[11]	SESAME[13, 14]	PEA[12] *
Cardiac	1	1	4	2
Lung ultrasound: pneumothorax	2	–	1	1
Proximal DVT (lower extremities)	–	–	2–3 (first in non-traumatic arrest)	3
Abdomen: e.g., free fluid, ruptured abdominal aorta	–	–	2–3 (first in traumatic arrest)	3

Numbers refer to the order in which scans are performed

CAUSE cardiac arrest ultrasound exam, *FEEL* focused echocardiographic evaluation in life support, *FEER* focused echocardiographic evaluation in resuscitation, *PEA* parasternal-Epigastric-Abdomen and other scans (*the order of scans can vary according to clinical setting), *SESAME* sequential emergency scanning assessing mechanism or origin of shock of indistinct cause, *DVT* deep venous thrombosis

Guidelines for the Use of Transesophageal Echocardiography (TEE) in the ED for Cardiac Arrest

[Ann Emerg Med. 2017;70:442-445.]

1. INTRODUCTION

The American College of Emergency Physicians (ACEP) has developed these criteria to assist practitioners performing emergency ultrasound studies (EUS) of the heart using transesophageal echocardiography (TEE) during cardiac arrest.

Ultrasound has been shown in cardiac arrest to accurately identify the presence or lack of intrinsic cardiac activity and in some cases the cause of arrest, including left ventricular failure, right ventricular failure, pulmonary embolism, pericardial tamponade, and hypovolemia. These findings can lead to lifesaving changes in management such as administration of intravenous fluids, blood products, vasopressors, or thrombolytics, or

STROKE