Ventilace, krevní oběh, vnitřní prostředí, kontrola tělesné teploty

František Bednář

Divize akutní kardiologie

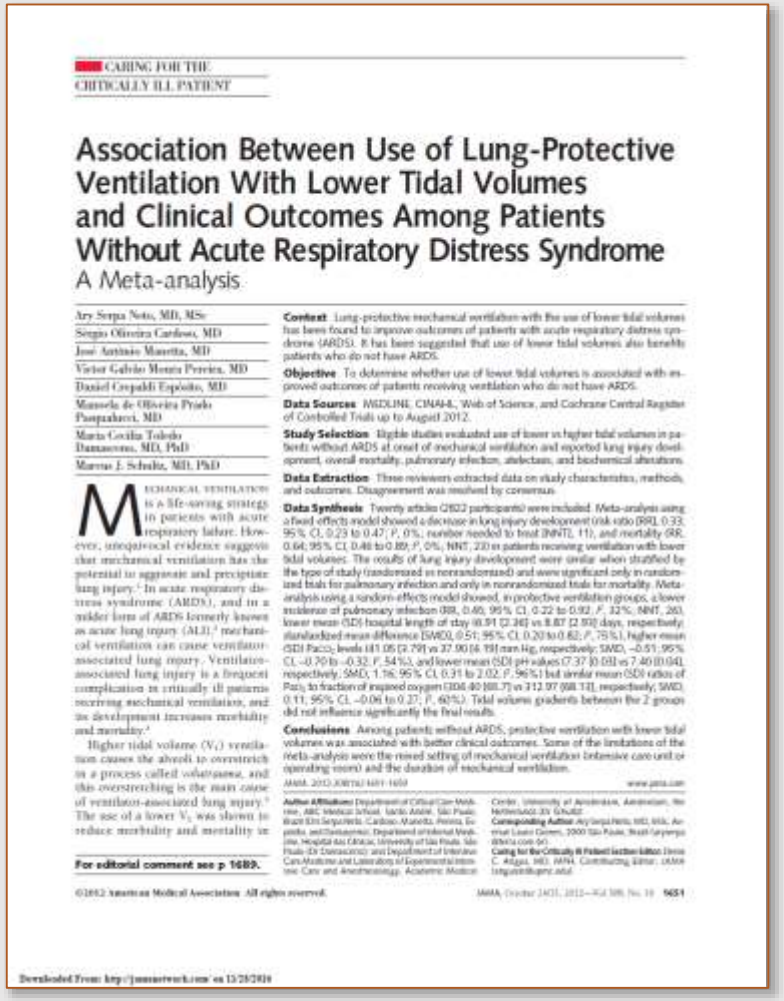
III. Interní-kardiologická klinka FNKV a 3.LF UK Praha

Ventilace po ROSC

- nestudována specificky u pacientů po srdeční zástavě
- racionální je používat doporučení ze studií s ARDS (*protektivní ventilace)*

Protektivní ventilace:
Vt 6-8 ml/kg, PEEP 4-8 cmH2O, Pplat < 30 cmH2O

- ↓incidenci ARDS, plicní infekce, dobu na UPV a mortalitu



CARING FOR THE CRITICALLY ILL PATIENT

Association Between Use of Lung-Protective Ventilation With Lower Tidal Volumes and Clinical Outcomes Among Patients Without Acute Respiratory Distress Syndrome

A Meta-analysis

Ary Serpa Neto, MD, MSc
Sergio Oliveira Cardoso, MD
José Antônio Manetta, MD
Victor Galvão Moura Pereira, MD
Daniel Cespedes Espósito, MD
Manoela de Oliveira Prado Pamplona, MD
Maria Cecília Toledo Damasceno, MD, PhD
Marcus J. Schultz, MD, PhD

Context Lung-protective mechanical ventilation with the use of lower tidal volumes has been found to improve outcomes of patients with acute respiratory distress syndrome (ARDS). It has been suggested that use of lower tidal volumes also benefits patients who do not have ARDS.

Objective To determine whether use of lower tidal volumes is associated with improved outcomes of patients receiving ventilation who do not have ARDS.

Data Sources MEDLINE, CINAHL, Web of Science, and Cochrane Central Register of Controlled Trials up to August 2012.

Study Selection Eligible studies evaluated use of lower vs higher tidal volumes in patients without ARDS at onset of mechanical ventilation and reported lung injury development, overall mortality, pulmonary infection, atelectasis, and biochemical alterations.

Data Extraction Three reviewers extracted data on study characteristics, methods, and outcomes. Disagreement was resolved by consensus.

Data Synthesis Twenty articles (2822 participants) were included. Meta-analysis using a fixed-effects model showed a decrease in lung injury development (risk ratio [RR], 0.33; 95% CI, 0.23 to 0.47; P, 0%; number needed to treat [NNT], 11), and mortality (RR, 0.64; 95% CI, 0.46 to 0.89; P, 0%; NNT, 23) in patients receiving ventilation with lower tidal volumes. The results of lung injury development were similar when stratified by the type of study (randomized vs nonrandomized) and were significant only in randomized trials for pulmonary infection and only in nonrandomized trials for mortality. Meta-analysis using a random-effects model showed, in protective ventilation groups, a lower incidence of pulmonary infection (RR, 0.46; 95% CI, 0.22 to 0.92; P, 32%; NNT, 26), lower mean (SD) hospital length of stay (8.91 [2.36] vs 9.87 [2.93] days, respectively; standardized mean difference [SMD], 0.51; 95% CI, 0.20 to 0.82; P, 75%), higher mean (SD) PaO_2 levels (411.05 [72.79] vs 37.90 [16.19] mm Hg, respectively; SMD, −0.51; 95% CI, −0.70 to −0.32; P, 54%), and lower mean (SD) pH values (7.37 [0.05] vs 7.40 [0.04], respectively; SMD, 1.16; 95% CI, 0.31 to 2.02; P, 96%) but similar mean (SD) ratios of PaO_2 to fraction of inspired oxygen (308.40 [88.77] vs 312.97 [98.13], respectively; SMD, 0.11; 95% CI, −0.06 to 0.27; P, 60%). Tidal volume gradients between the 2 groups did not influence significantly the final results.

Conclusions Among patients without ARDS, protective ventilation with lower tidal volumes was associated with better clinical outcomes. Some of the limitations of the meta-analysis were the mixed setting of mechanical ventilation (intensive care unit or operating room) and the duration of mechanical ventilation.

JAMA. 2012;308(16):1651-1659 www.jama.com

Author Affiliations: Department of Critical Care Medicine, ABC Medical School, Santo André, São Paulo, Brazil (Drs Serpa Neto, Cardoso, Manetta, Pereira, Espósito, and Damasceno); Department of Internal Medicine, Hospital das Clínicas, University of São Paulo, São Paulo (Dr Damasceno); and Department of Intensive Care Medicine and Laboratory of Experimental Intensive Care and Anesthesiology, Academic Medical Center, University of Amsterdam, Amsterdam, The Netherlands (Dr Schultz).

Corresponding Author: Ary Serpa Neto, MD, MSc, Avenue Lauro Gomes, 2000 São Paulo, Brazil (aryserpa@hotmail.com).

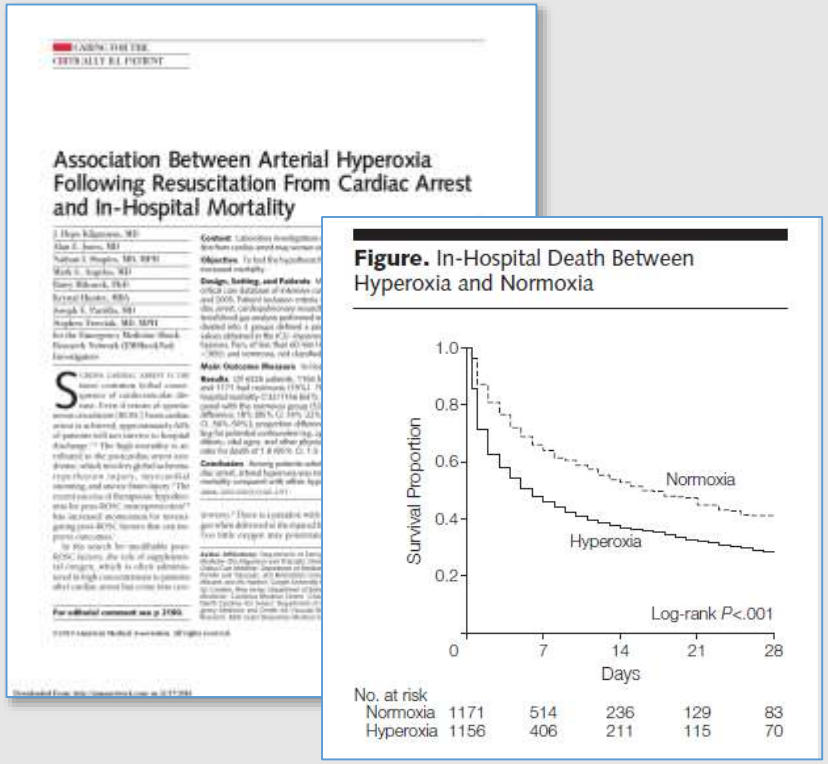
Caring for the Critically Ill Patient Section Editor: Derek C. Angus, MD, MPH. Contributing Editor: JAMA (angusd@upmc.edu).

For editorial comment see p 1689.

JAMA, October 24/31, 2012—Vol 308, No. 16 1651

Vztah PaO_2 a klinických výsledků

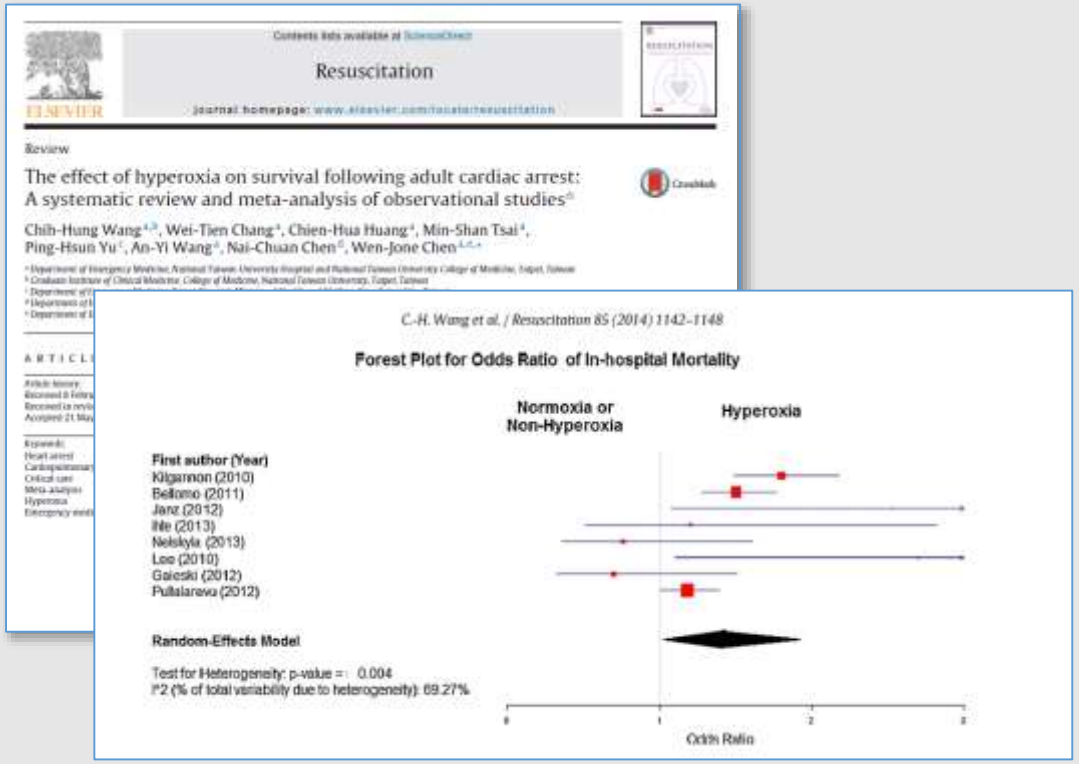
Hyperoxemie (PaO2>300 mmHg) a hypoxemie hod (PaO2< 60 mmHg) v prvních 24 hod zvyšuje mortalitu oproti normoxemii.
Každé zvýšení PaO2 o 100 mmHg zvyšuje mortalitu o 24% !



Association Between Arterial Hyperoxia Following Resuscitation From Cardiac Arrest and In-Hospital Mortality

Figure. In-Hospital Death Between Hyperoxia and Normoxia

Hyperoxemie (PaO2>300 mmHg) koreluje se zvýšenou nemocniční mortalitou, ale pravděpodobně nezhoršuje neurologické výsledky.
Autoři upozoňují na heterogenitu studií a jejich malý počet!



Resuscitation

Review

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

Chih-Hung Wang, Wei-Tien Chang, Chien-Hua Huang, Min-Shan Tsai, Ping-Hsun Yu, An-Yi Wang, Nai-Chuan Chen, Wen-Jone Chen

C.-H. Wang et al. / Resuscitation 85 (2014) 1142–1148

Forest Plot for Odds Ratio of In-hospital Mortality

Vztah $PaCO_2$ a klinických výsledků

Poznámky:

- $PaCO_2$ je určující faktor regulace mozkové perfúze (CBF)
- Hypokapnie ($PaCO_2$ < 35mmHg) vede k cerebrální vazokonstrikci a redukci CBF o 40-50% a ke zhoršení mozková ischemie
- Hyperkapnie ($PaCO_2$ > 45 mmHg) zvyšuje CBF

Závěry studií:

Hypokapnie ($PaCO_2$ < 35) je spojena s vyšší mortalitou a horšími neurologickými výsledky v porovnání s normokapnií ($PaCO_2$ 35-45) a mírnou hyperkapnií ($PaCO_2$ 45-50).

Contents lists available at SciVerse ScienceDirect

Resuscitation

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

Clinical paper

Arterial carbon dioxide tension and outcome in patients admitted to the intensive care unit after cardiac arrest☆

Antoine G. Schneider[a,b,1], Glenn M. Eastwood[a,1], Rinaldo Bellomo[a,b,*], Michael Bailey[b], Miklos Lipcsey[a,c], David Pilcher[a], Paul Young[e,f], Peter Stow[g], John Santamaria[h], Edward Stachowski[i], Satoshi Suzuki[a], Nicholas C. Woinarski[a], Janine Pilcher[g]

ARTICLE INFO

ABSTRACT

1. Introduction

Doporučení I.

1. Během KPR používáme 100% O_2
2. Post ROSC spontánně ventilující: O_2 maska při SpO2< 94%
3. Po dosažení ROSC - *protektivní ventilaci:* Vt 6-8 ml/kg, PEEP 4-8 cmH_2O, Pplat < 30 cmH_2O
4. Udržujeme PaO_2 60 - 300 mmHg nebo SpO_2 94-98%
5. Udržujeme $PaCO_2$ 35 – 50 mmHg
6. Zavedení nosogastrické sondy (dekomprese žaludku, uvolnění bránice, zlepšení ventilace)
7. Doporučujeme protokol sedace a event. myorelaxace
8. ATB prevence pneumonie při TTM – spíše ano (*ANTHARTIC trial ?* …)

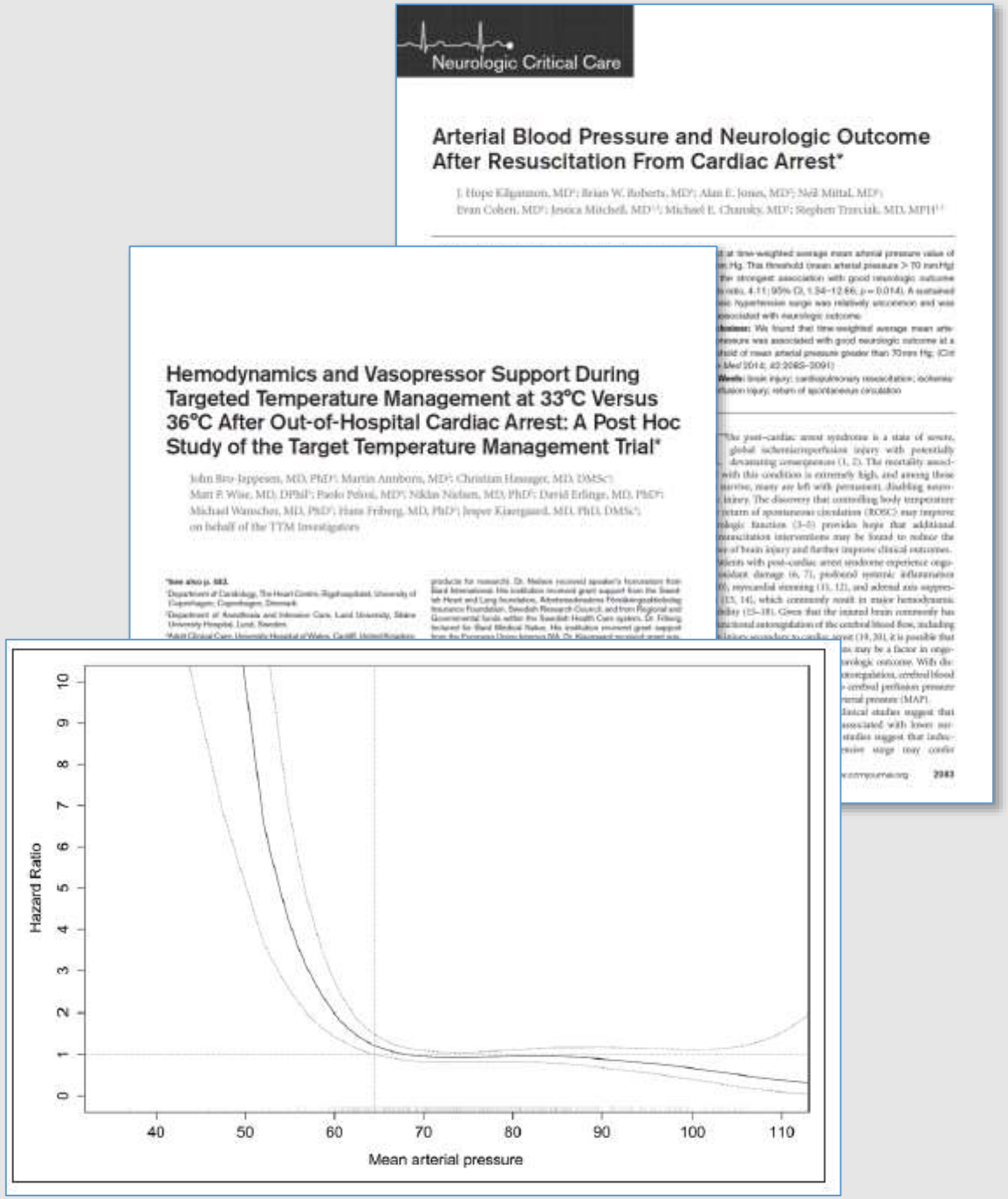
Krevní oběh po ROSC

Poznámky:

- Post cardiac arrest syndrom – myokardiální dysfunkce + vazodilatace (SIRS) (hypotenze, ↓CO, arytmie)
- Zotavení ↓CO cca 24 hod (i déle), zotavení vazodilatace až 3 dny (i déle)
- myokardiální dysfunkce – často inotropní podpora
- SIRS – vazoplegie – noradrenalin, tekutiny
- Kardiovaskulární selhání – nejčastější příčina úmrtí během prvních cca 3 dnů

Optimální hodnota TK

- Optimální TK post-CA sy není znám
- Hodnoty často odvozeny z prací u sepse
- Post CA-syndrom je jistě více komplexnější než sepse
- Mozková autoregulace je porušena u více jak 1/3 pacientů po CA na rozdíl od sepse
- MAP > 65-70 mmHg - lepší neurologický outcome a nižší mortalita (TTM trial)



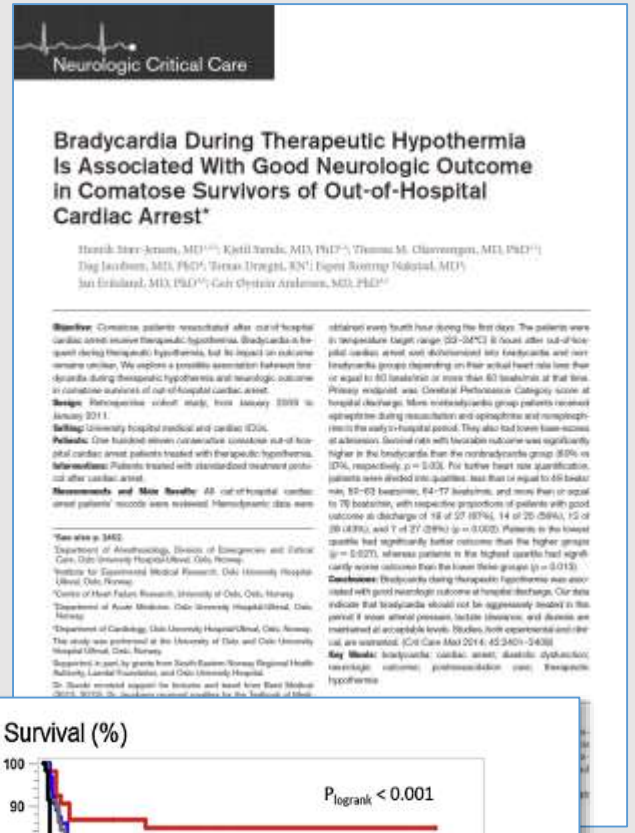
Optimální hodnota TF

- Bradykardie – fyziologická reakce při hypotermii
- Bradykardie během TTM může představovat prognostický marker
- Bradykardie ≥ 40/min lze tolerovat , pokud je TK, laktát, diureza.., nutno zvážit EF …)


Resuscitation

Clinical Paper

Sinus bradycardia during hypothermia in comatose survivors of out-of-hospital cardiac arrest – A new early marker of favorable outcome?


Neurologic Critical Care

Bradycardia During Therapeutic Hypothermia Is Associated With Good Neurologic Outcome in Comatose Survivors of Out-of-Hospital Cardiac Arrest*

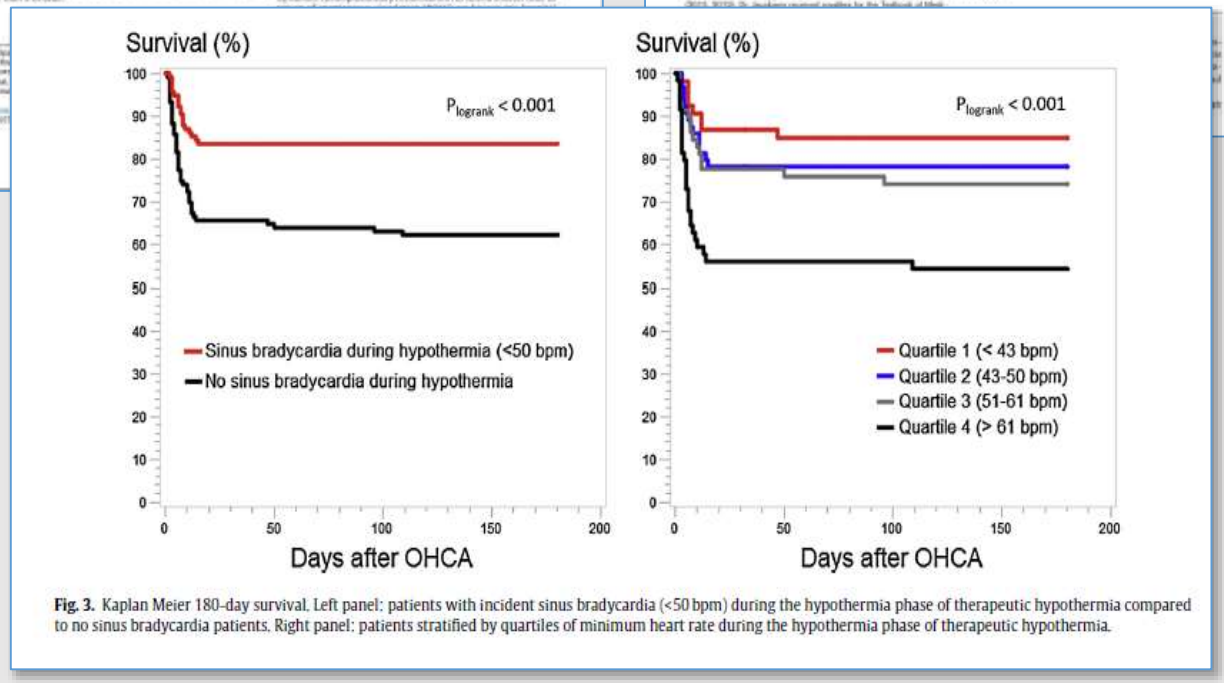


Fig. 3. Kaplan Meier 180-day survival. Left panel: patients with incident sinus bradycardia (<50 bpm) during the hypothermia phase of therapeutic hypothermia compared to no sinus bradycardia patients. Right panel: patients stratified by quartiles of minimum heart rate during the hypothermia phase of therapeutic hypothermia.

Doporučení II.

1. Tekutiny k optimalizaci plnících tlaků (F1/1)
2. MAP ≥ 65 mmHg a TF ≥ 40/min – použij NA ± DOBU
3. Cíl: → diuréza 1 ml/kg/hod
   normální/klesající laktát
   smíšená žilní saturace > 70%

   (cave: hypotermie ↑ diurézu a ↓clearance laktátu)
4. vždy zvážit pacientův normální TK, myokardiální dysfunkci, komorbidity…
5. Vhodná invazivní monitorace TK a event. CŽK, dle potřeby PAC
6. Použij info z ECHO, RTG S+P
7. SKG – viz dále

Koronární reperfuze

- Prevalence akutních koronárních lézí (AKL) u OHCA 59-71%
- STE nebo BBB 80% má AKL
- nonSTE 25-58% má AKL

Resuscitation

Clinical paper

Acute coronary angiography in patients resuscitated from out-of-hospital cardiac arrest—A systematic review and meta-analysis

Jacob Moesgaard Larsen, Jan Ravkilde

Resuscitation

Clinical paper

Post-resuscitation electrocardiograms, acute coronary findings and in-hospital prognosis of survivors of out-of-hospital cardiac arrest

Julio García-Tejada, Alfonso Jurado-Román, Jesús Rodríguez, Maite Velázquez, Felipe Hernández, Agustín Albarrán, Roberto Martín-Asenjo, Carolina Granda-Nistal, Raúl Coma, Juan Tascón

Koronární reperfuze
Indikace urgentní koronarografie

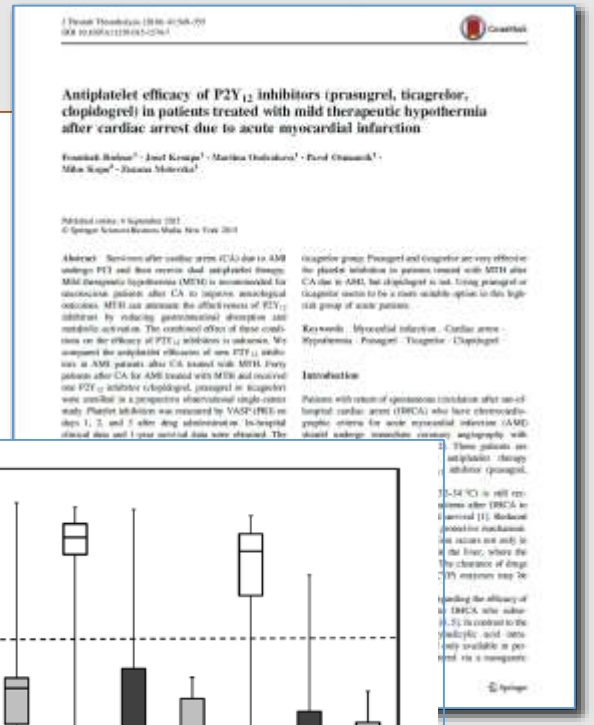
Periprocedurální antitrombotická léčba

1) ASA 250 mg (Kardegic ½ amp) i.v.

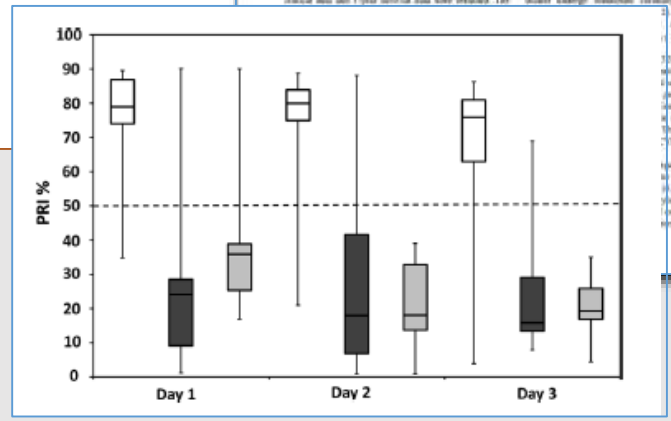
2) heparin 70-100 IU/kg i.v.

3) P2Y12 blokátory rozdrtit do NGS

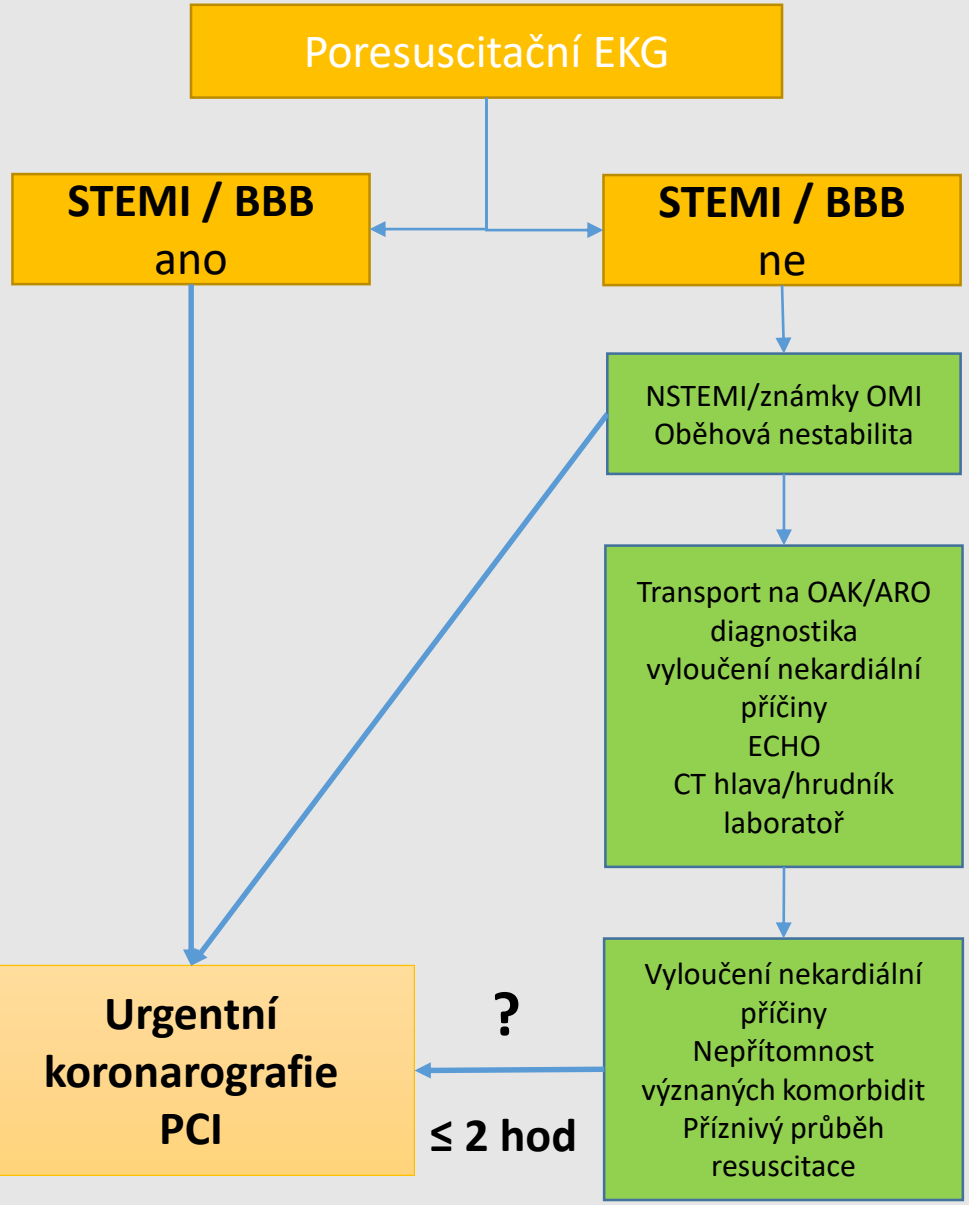
prasugrel 60mg
ticagrelor 180 mg
clopidogrel neúčinný



Antiplatelet efficacy of $P2Y_{12}$ inhibitors (prasugrel, ticagrelor, clopidogrel) in patients treated with mild therapeutic hypothermia after cardiac arrest due to acute myocardial infarction



Neúčinnost (PRI > 50%) clopidogrelu (*bílé obdélníky*) podávaného NGS během prvních tří dnů po OHCA pro AIM.



Vnitřní prostředí
acido-bazická rovnováha

Poznámky:

- CA – ischemie –tkáňová hypoxie - produkce laktátu - ↓pH
- ↓pH – hyperventilace - ↓pCO2 – vazokonstrikce- zh**oršen**í mozkové ischemie
- Těžká acidóza (pH< 7.20) - ↓CO, ↓TK, arytmie, ↓účinnosti KA
- ABR při hypotermii:
  a) hodnoty krevních plynů závislé na teplotě
  b) ↓ T zvyšuje rozpustnost O2 a CO2 a tím snižuje pO2 a pCO2 (O2 / pCO2snižuje o 5/2 mmHg/1°C)
  c) ↓ T snižuje spotřebu O2 a produkci CO2
  d) Měření krevních plynů:
  alpha-stat (teplota nekorigována, 37oC) nebo pH –stat (korigováno na aktuální teplotu pacienta)
  v klinické praxi nemá větší význam

Vnitřní prostředí
hladina draslíku

- Hypokalemie (≤ 3.5 mmol/l) nacházíme u 30-50% pacientů po OHCA
- **Příčiny:**
  - uvolnění katecholaminů
  - korekce metabolické a respirační acidózy
  - hypotermie
- Hypokalemie zvyšuje riziko arytmií (≤2.5 ↑QT a VT)
- Udržovat v rozmezí **4-4.5mmol/l**

Best Practice & Research Clinical Anaesthesiology

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

6

Metabolic and electrolyte disturbance after cardiac arrest: How to deal with it

Rinaldo Bellomo, MBBS, MD (Hons), FRACP, FCICM, PG Dip Echo, Professor, Director *,
Johan Mårtensson, MD, PhD, Consultant Anaethestist,
Glenn Matthew Eastwood, PhD, ICU Research Manager, RN, BN, BN(Hons), GradDipNurs(Crit Care)

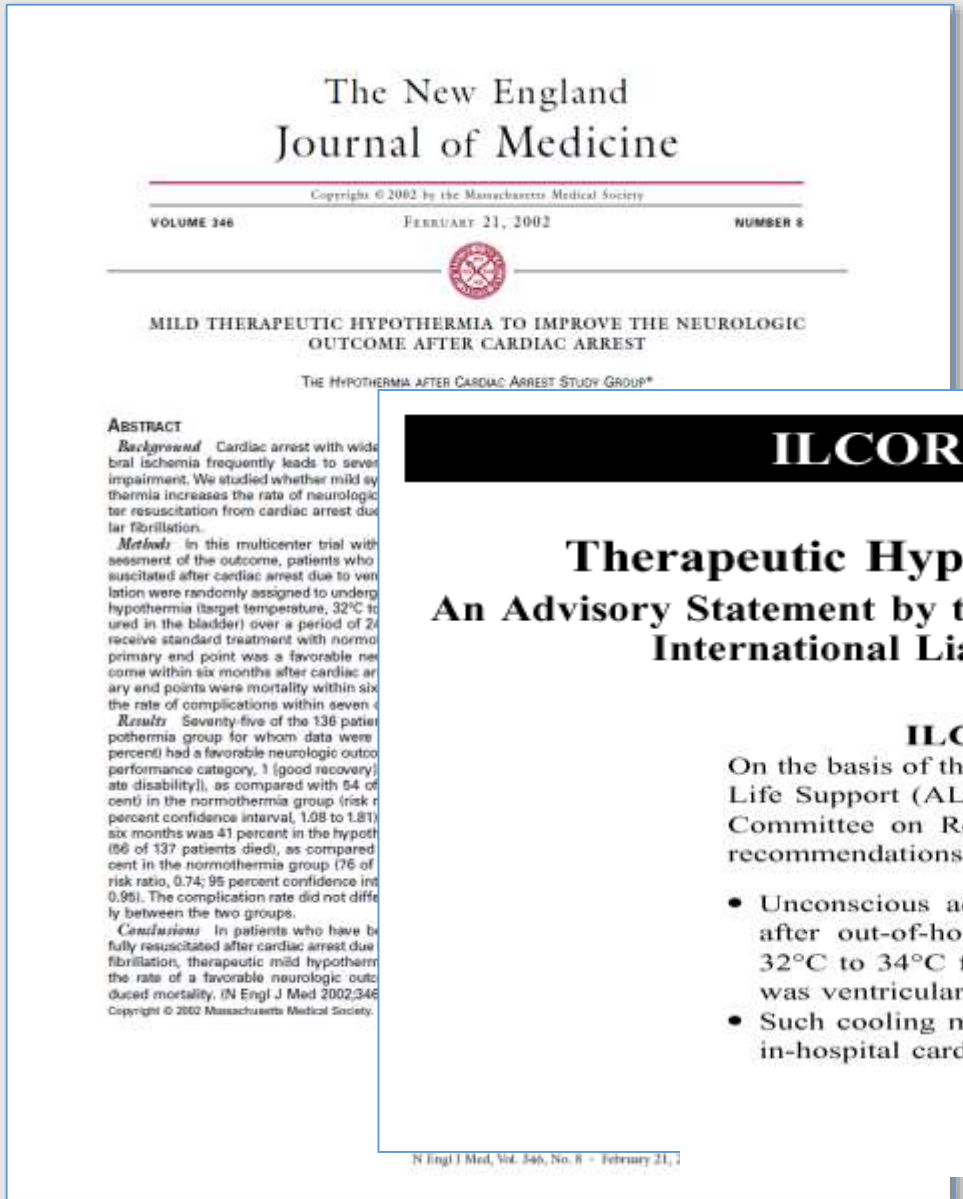
Kontrola glykemie

- Přísná kontrola glykemie (4-6 mmol/l) zvyšuje riziko hypoglykemie a mortalitu oproti méně striktní kontrole (≤ 10 mmol/l) u ICU pacientů
- závislost mezi hodnotami glykemie a mortalitou má tvar U-křivky
- Závislost platí především pro nediabetiky, pro pacienty s DM je méně vyjádřena
- Hypotermie: ↑glykemie (↓inzulinová sekrece a senzitivita), ↑potřeba inzulinu

Doporučení III.

1. Těžká acidóza (pH< 7.20)- korekce oběhu, oxygenace a ventilace a až následně bikarbonát (2mmol/kg)
2. Udržovat glykemii 6-10 mmol/l
3. Udržovat kalemii v rozmezí 4.0-4.5mmol/l

Kontrola tělesné teploty po OHCA



The New England
Journal of Medicine

VOLUME 346 FEBRUARY 21, 2002 NUMBER 8

MILD THERAPEUTIC HYPOTHERMIA TO IMPROVE THE NEUROLOGIC OUTCOME AFTER CARDIAC ARREST

THE HYPOTHERMIA AFTER CARDIAC ARREST STUDY GROUP*

ABSTRACT

Background Cardiac arrest with wide
bral ischemia frequently leads to sever
impairment. We studied whether mild sy
thermia increases the rate of neurologic
ter resuscitation from cardiac arrest due
lar fibrillation.

Methods In this multicenter trial with
sessment of the outcome, patients who
suscitated after cardiac arrest due to ven
lation were randomly assigned to underg
hypothermia (target temperature, 32°C to
ured in the bladder) over a period of 24
receive standard treatment with normo
primary end point was a favorable ne
come within six months after cardiac ar
ary end points were mortality within six
the rate of complications within seven

Results Seventy-five of the 136 patie
pothermia group for whom data were
percent) had a favorable neurologic outco
performance category, 1 [good recovery]
ate disability]), as compared with 54 of
cent) in the normothermia group (risk r
percent confidence interval, 1.08 to 1.81)
six months was 41 percent in the hypoth
(56 of 137 patients died), as compared
cent in the normothermia group (76 of
risk ratio, 0.74; 95 percent confidence int
0.95). The complication rate did not diffe
ly between the two groups.

Conclusions In patients who have b
fully resuscitated after cardiac arrest due
fibrillation, therapeutic mild hypotherm
the rate of a favorable neurologic outc
duced mortality. (N Engl J Med 2002;346

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. BURST, 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 moder-

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,[2] the use of induced hypothermia to decrease cerebral oxygen demand has been proposed as a treatment option.[3] Although this suggestion has been supported by studies

ILCOR Advisory Statement

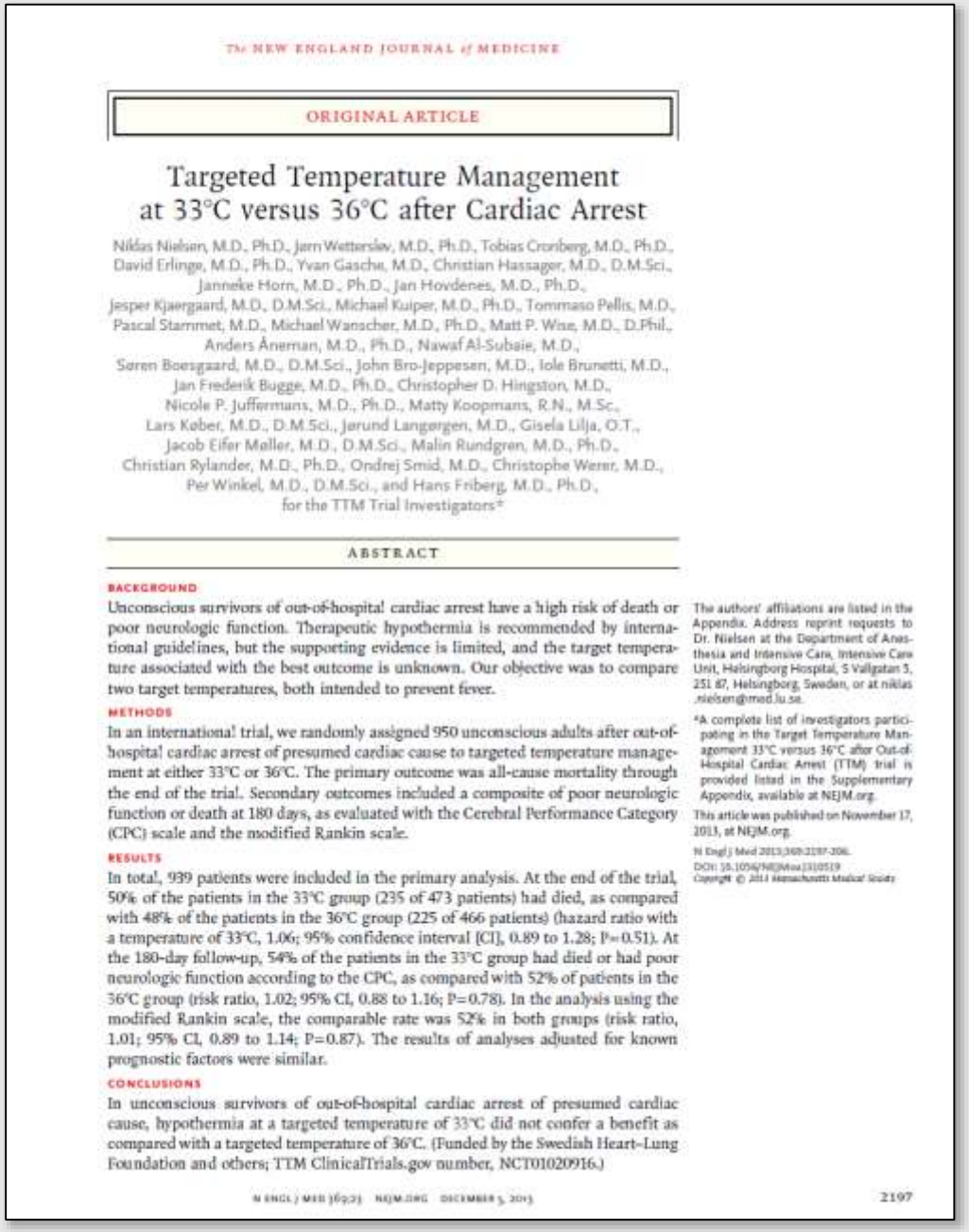
Therapeutic Hypothermia After Cardiac Arrest

An Advisory Statement by the Advanced Life Support Task Force of the International Liaison Committee on Resuscitation

ILCOR Recommendations

On the basis of the published evidence to date, the Advanced Life Support (ALS) Task Force of the International Liaison Committee on Resuscitation (ILCOR) made the following recommendations in October 2002:

- Unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest should be cooled to 32°C to 34°C for 12 to 24 hours when the initial rhythm was ventricular fibrillation (VF).
- Such cooling may also be beneficial for other rhythms or in-hospital cardiac arrest.



The NEW ENGLAND JOURNAL of MEDICINE

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., Tommaso Pellis, M.D., Pascal Stammet, M.D., Michael Wanscher, M.D., Ph.D., Matt P. Wise, M.D., D.Phil., Anders Åneman, M.D., Ph.D., Nawaf Al-Subaie, M.D., Søren Boesgaard, M.D., D.M.Sci., John Bro-Jeppesen, M.D., Iole Brunetti, M.D., Jan Frederik Bugge, M.D., Ph.D., Christopher D. Hingston, M.D., Nicole P. Juffermans, M.D., Ph.D., Matty Koopmans, R.N., M.Sc., Lars Køber, M.D., D.M.Sci., Jarund Langørgen, M.D., Gisela Lilja, O.T., Jacob Eifer Møller, M.D., D.M.Sci., Malin Rundgren, M.D., Ph.D., Christian Rylander, M.D., Ph.D., Ondrej Smid, M.D., Christophe Werer, M.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, 939 patients were included in the primary analysis. At the end of the trial, 50% of the patients in the 33°C group (235 of 473 patients) had died, as compared with 48% of the patients in the 36°C group (225 of 466 patients) (hazard ratio with a temperature of 33°C, 1.06; 95% confidence interval [CI], 0.89 to 1.28; P=0.51). At the 180-day follow-up, 54% of the patients in the 33°C group had died or had poor neurologic function according to the CPC, as compared with 52% of patients in the 36°C group (risk ratio, 1.02; 95% CI, 0.88 to 1.16; P=0.78). In the analysis using the modified Rankin scale, the comparable rate was 52% in both groups (risk ratio, 1.01; 95% CI, 0.89 to 1.14; P=0.87). The results of analyses adjusted for known prognostic factors were similar.

CONCLUSIONS

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. (Funded by the Swedish Heart–Lung Foundation and others; TTM ClinicalTrials.gov number, NCT01020916.)

The authors' affiliations are listed in the Appendix. Address reprint requests to Dr. Nielsen at the Department of Anesthesia and Intensive Care, Intensive Care Unit, Helsingborg Hospital, S Vallgatan 5, 251 87, Helsingborg, Sweden, or at niklas.nielsen@med.lu.se.

*A complete list of investigators participating in the Target Temperature Management 33°C versus 36°C after Out-of-Hospital Cardiac Arrest (TTM) trial is provided listed in the Supplementary Appendix, available at NEJM.org.

This article was published on November 17, 2013, at NEJM.org.

N Engl J Med 2013;369:2197-206.
DOI: 10.1056/NEJMoa1310519

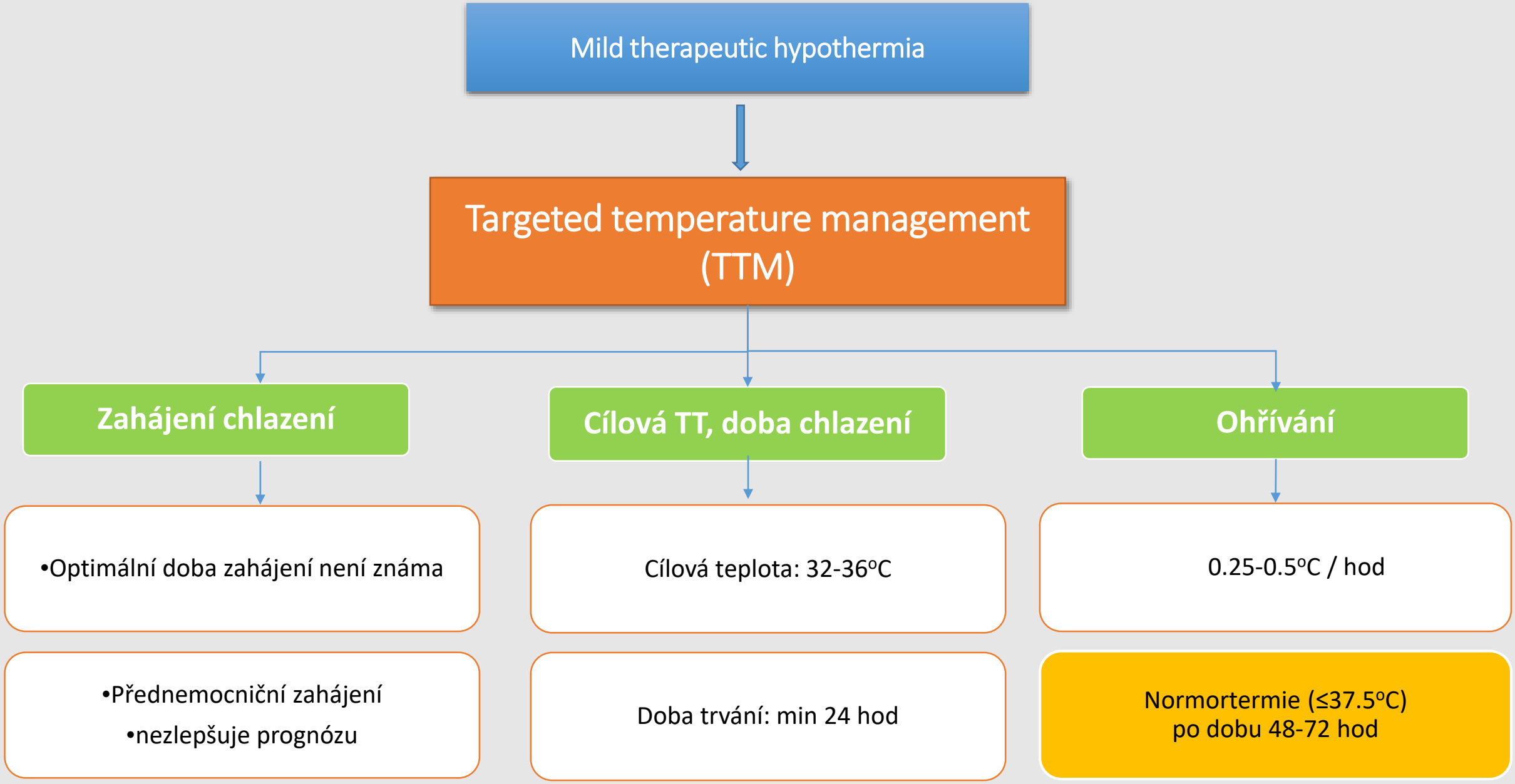
Targeted temperature management (TTM)

Table 2. Outcomes.

Outcome	33°C Group	36°C Group	Hazard Ratio or Risk Ratio (95% CI)*	P Value
	no./total no. (%)			
Primary outcome: deaths at end of trial	235/473 (50)	225/466 (48)	1.06 (0.89–1.28)	0.51
Secondary outcomes				
Neurologic function at follow-up†				
CPC of 3–5	251/469 (54)	242/464 (52)	1.02 (0.88–1.16)	0.78
Modified Rankin scale score of 4–6	245/469 (52)	239/464 (52)	1.01 (0.89–1.14)	0.87
Deaths at 180 days	226/473 (48)	220/466 (47)	1.01 (0.87–1.15)	0.92

CONCLUSIONS

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. (Funded by the Swedish Heart–Lung Foundation and others; TTM ClinicalTrials.gov number, NCT01020916.)


Mild therapeutic hypothermia
Targeted temperature management (TTM)
Zahájení chlazení
Cílová TT, doba chlazení
Ohřívání
•Optimální doba zahájení není známa
Cílová teplota: 32-36°C
0.25-0.5°C / hod
•Přednemocniční zahájení
•nezlepšuje prognózu
Doba trvání: min 24 hod
Normortermie (≤37.5°C)
po dobu 48-72 hod

Doporučení IV.

1. Udržovat teplotu na 32°C - 36°C
2. Doba TTM 24 hod
3. Zabránit hypertermii (≥37.5°C) po dobu 48-72 hod po CA

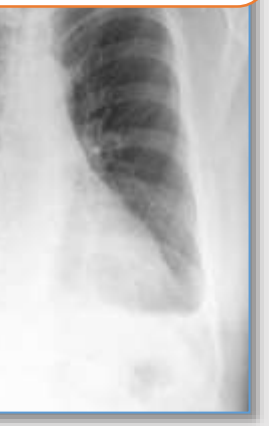
Probíhající studie o TH:

- *TTH48* 33°C 24h vs. 48 hod
- *CAPITAL CHILL* 31°C vs. 34°C

Doporučení pro poresuscitační péči

Oxygenace
paO_2 60-300 mmHg

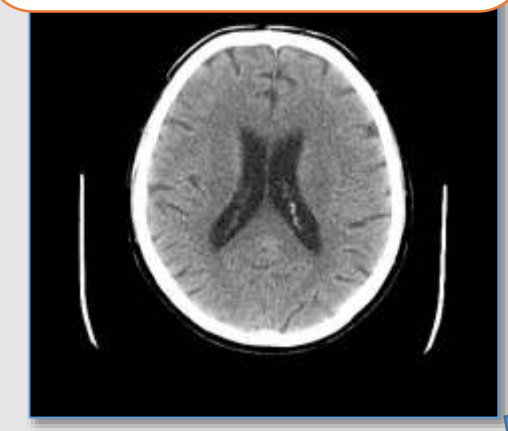
Protektivní ventilace
Vt 6-8 ml/kg
PEEP 4-8 cmH_2O, Pplat < 30 cmH_2O



MAP ≥ 65 mmHg,
± vazopresory / inotropie

Ventilace
$paCO_2$ 35-50 mmHg

TTM (36°C)
Vnitřní prostředí
Glykemie 6-10 mmol/l
Sedace/křečová aktivita



Upraveno podle Sutherasan Y., 2015