



L'arrêt cardiaque News in ICU ? And after...

Les MARES contre-attaque

Dr LE SAUX Audrey

Dr BOURENNE Jeremy



- 10 lits de réanimation
- 1 lits de déchocage
- 600 admissions / an
- Dont 100 arrêts cardiaques



Cardiac Arrest network

Consultation de suivi

La RDU aux JDU



Syndrome post arrêt cardiaque

The second step in resuscitation—the treatment of the ‘post-resuscitation disease’

V. A. NEGOVSKY *Resuscitation* (1972)



There is much evidence that the organism experiences a specific pathological condition after resuscitation. We are inclined to call this condition ‘the post-resuscitation disease’, and to examine it as an independent nosological form. Indeed, irreversible

Metabolism

Liver function

Heart and circulation

Kidney function

Respiration

Central nervous system function

Syndrome post arrêt cardiaque



Pathologie causale



Dysfonction myocardique

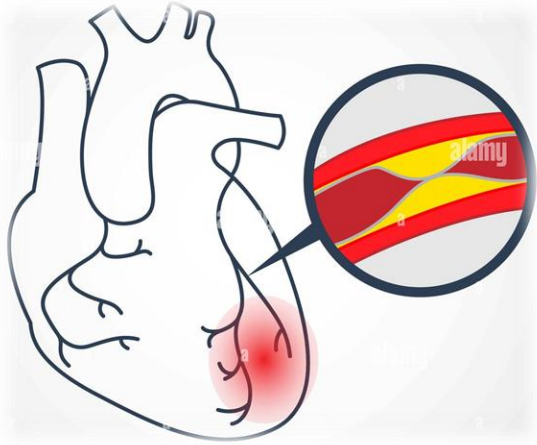


Encéphalopathie post anoxique

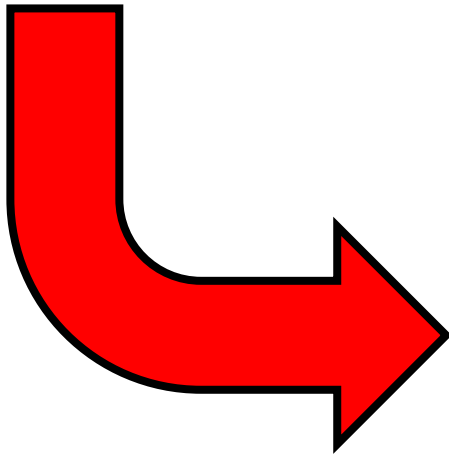


**Syndrome d'Ischémie – Reperfusion
Défaillance multi-viscérale**

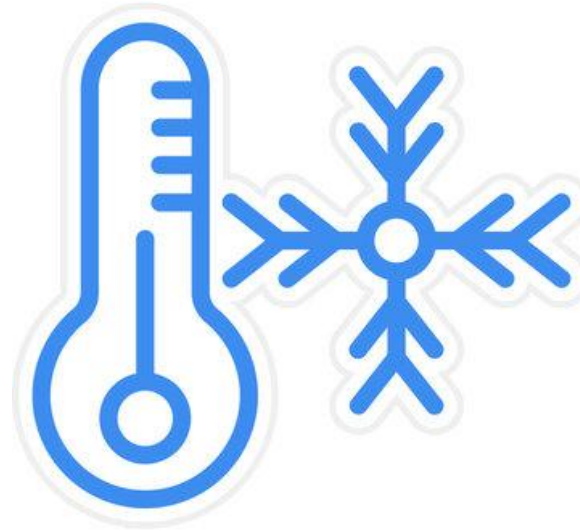
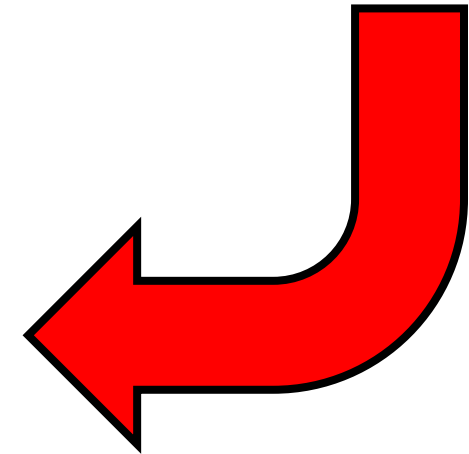
Encéphalopathie post anoxique



Une coronarographie ?



Des paramètres à optimiser ?



Un contrôle ciblé ?

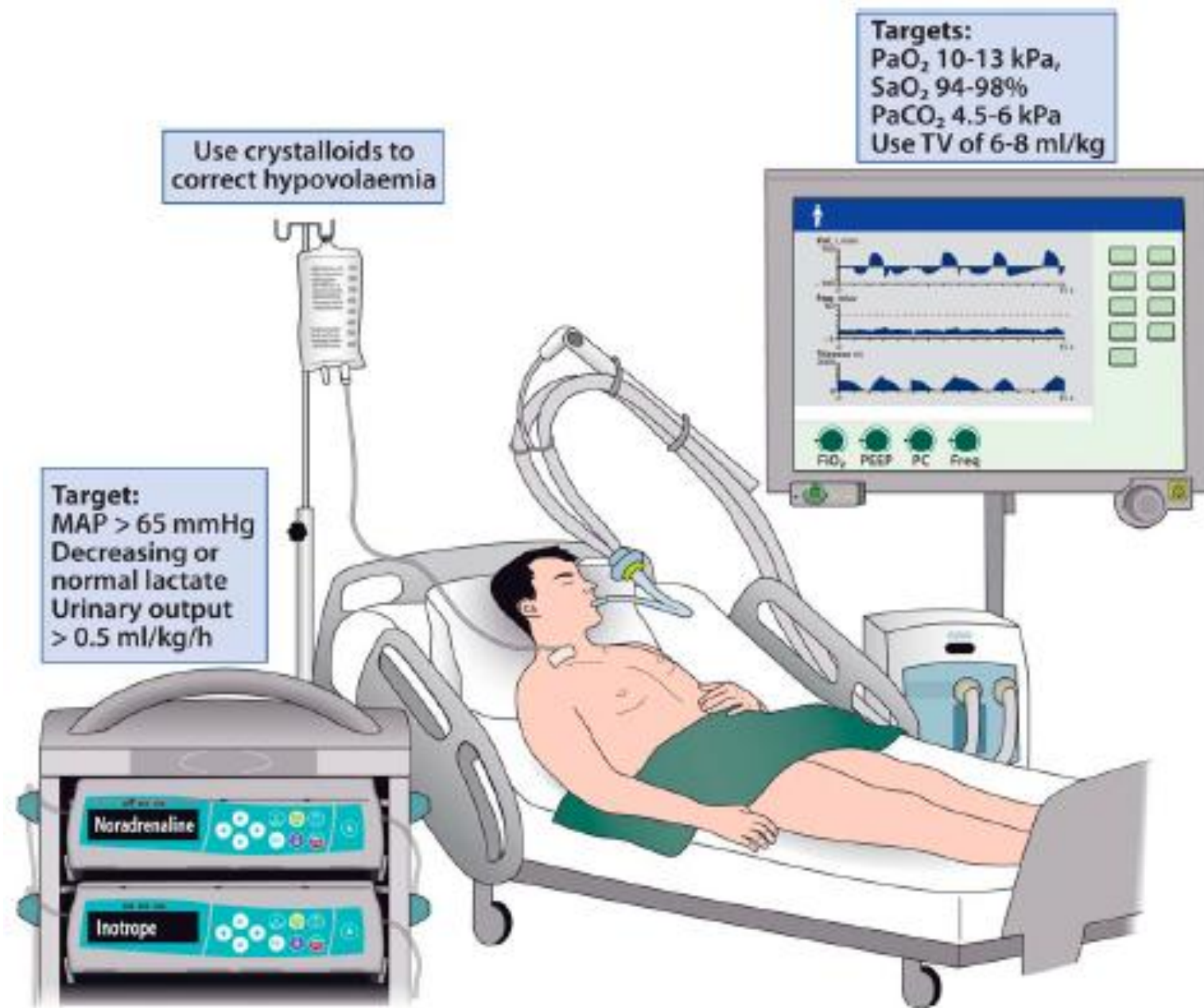
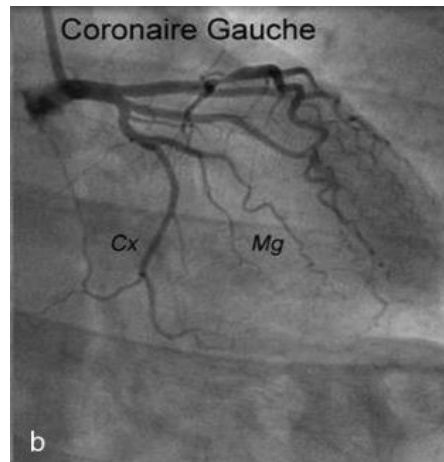
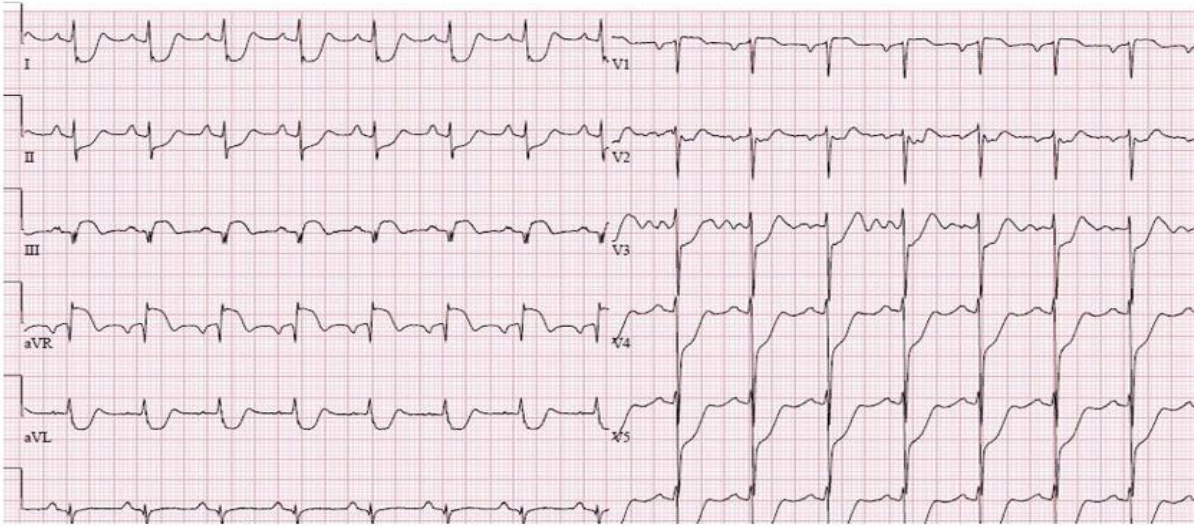


Fig. 3 – Haemodynamic, oxygenation and ventilation targets.

Faut-il faire une coronarographie d'emblé



IMMEDIATE CORONARY ANGIOGRAPHY IN SURVIVORS OF OUT-OF-HOSPITAL
CARDIAC ARREST

CHRISTIAN M. SPAULDING, M.D., LUC-MARIE JOLY, M.D., ALAIN ROSENBERG, M.D., MEHRAN MONCHI, M.D.,
SIMON N. WEBER, M.D., JEAN-FRANÇOIS A. DHAINAUT, M.D., PH.D., AND PIERRE CARLI, M.D.

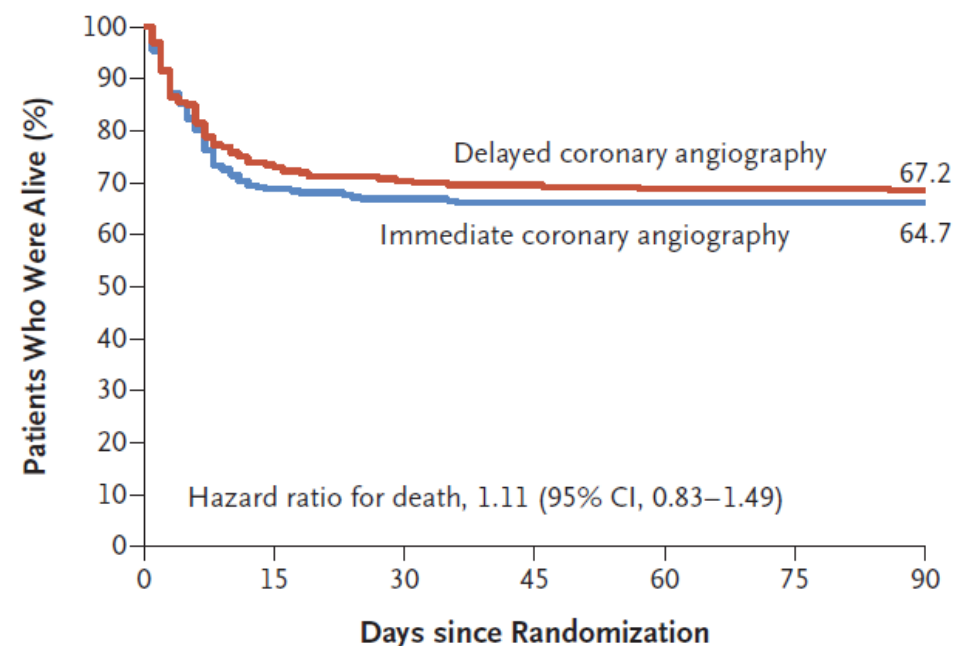
VARIABLE	VALUE	VARIABLE	No. OF PATIENTS	No. WITH RECENT CORONARY-ARTERY OCCLUSION (%)
Normal coronary arteries — no. (%)	17 (20)	ST-segment elevation and chest pain		
Clinically insignificant coronary artery disease (≤ 50 percent stenosis) — no. (%)	7 (8)	Present	15	13 (87)
Clinically significant coronary artery disease — no. (%)	60 (71)	Absent	69	27 (39)
Single-vessel disease	22	ST-segment elevation or chest pain		
Two-vessel disease	13	Present	49	31 (63)
Three-vessel disease	24	Absent	35	9 (26)
Isolated left main coronary artery disease	1			
Left ventricular ejection fraction — %	33.9 ± 10.5			
Left ventricular end-diastolic pressure — mm Hg	25.3 ± 9.5			





Coronary Angiography after Cardiac Arrest without ST-Segment Elevation

Outcome	Immediate Angiography Group (N=273)	Delayed Angiography Group (N=265)	Effect Size (95% CI) [†]
Primary end point			
Survival at 90 days — no. of patients (%) [‡]	176 (64.5)	178 (67.2)	OR, 0.89 (0.62 to 1.27)
Secondary end points			
Survival with good cerebral performance or mild or moderate disability — no. of patients/total no. (%)	171/272 (62.9)	170/264 (64.4)	OR, 0.94 (0.66 to 1.31)
CPC score at 90 days — no./total no. (%) [§]			
1	157/272 (57.7)	159/264 (60.2)	Reference
2	14/272 (5.1)	11/264 (4.2)	OR, 1.29 (0.56 to 2.92)
3	4/272 (1.5)	5/264 (1.9)	OR, 0.81 (0.21 to 3.07)
4	0/272	2/264 (0.8)	NA
5	97/272 (35.7)	87/264 (33.0)	OR, 1.13 (0.78 to 1.63)
Survival until hospital discharge — no. of patients (%)	178 (65.2)	182 (68.7)	OR, 0.85 (0.60 to 1.22)
Neurologic status at ICU discharge			
GCS score			
Median (IQR)	15 (14 to 15)	15 (14 to 15)	
Geometric mean (95% CI)	13.7 (13.2 to 14.2)	13.5 (12.9 to 13.7)	1.02 (0.96 to 1.04)
CPC score — no./total no. (%) [§]			
1	74/258 (28.7)	86/249 (34.5)	Reference
2	59/258 (22.9)	56/249 (22.5)	OR, 1.22 (0.76 to 1.98)
3	36/258 (14.0)	30/249 (12.0)	OR, 1.39 (0.78 to 2.48)
4	4/258 (1.6)	9/249 (3.6)	OR, 0.52 (0.15 to 1.75)
5	85/258 (32.9)	68/249 (27.3)	OR, 1.45 (0.93 to 2.27)

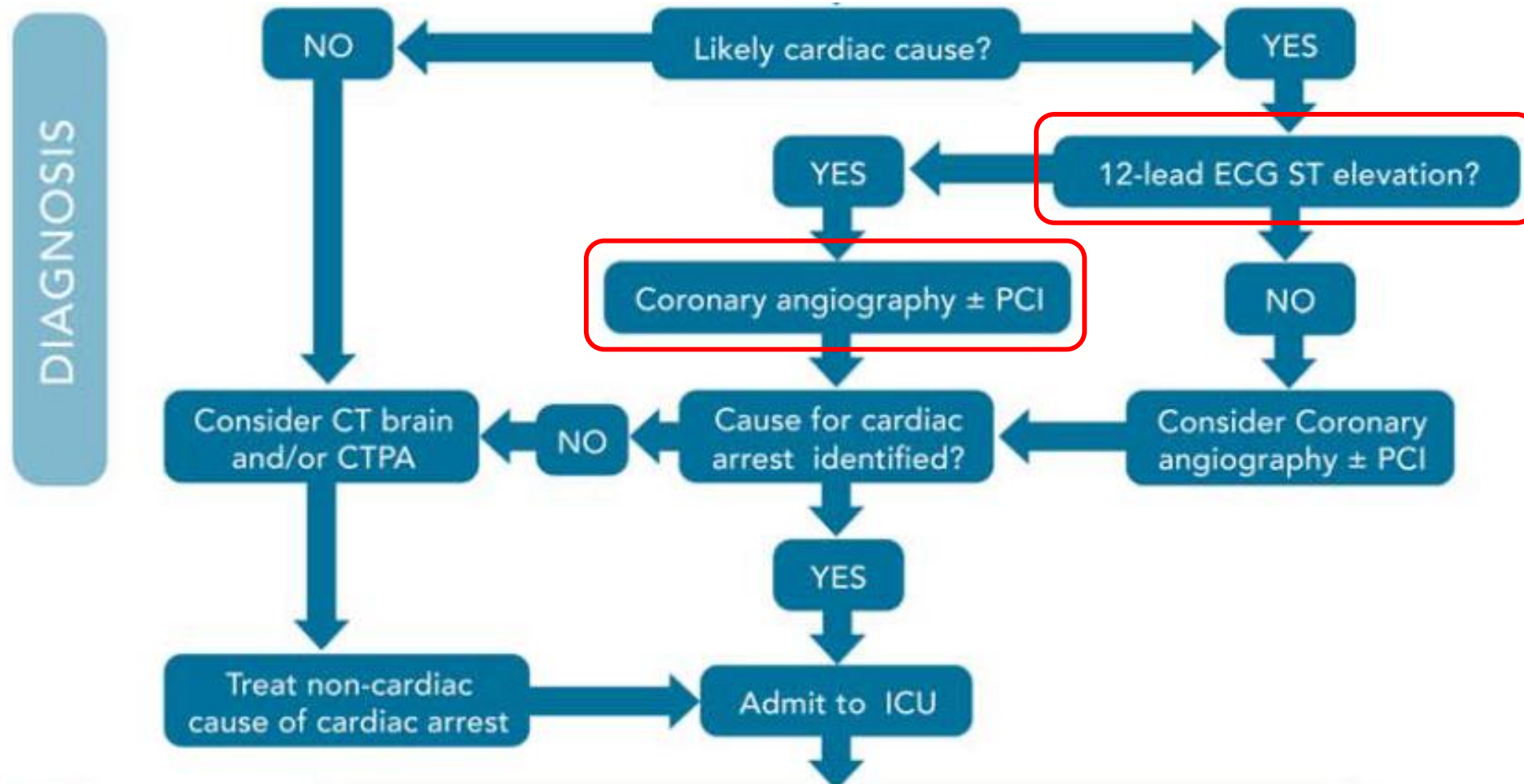


No. at Risk

Delayed	265	191	183	181	179	179	178
Immediate	273	183	178	176	176	176	176

European Resuscitation Council and European Society of Intensive Care Medicine Guidelines 2021: Post-resuscitation care[☆]

POST-RESUSCITATION CARE



Pubmed: Arrêt cardiaque et Contrôle thermique

The screenshot shows the PubMed search results page. At the top, the NIH National Library of Medicine logo is visible. The search bar contains the text 'cardiac arrest and hypothermia'. Below the search bar, the text '9,210 results' is circled in red. To the right of the search bar, there are buttons for 'Save', 'Email', and 'Send to'. Below these buttons, there are links for 'Advanced', 'Create alert', and 'Create RSS'. On the right side of the page, there are buttons for 'Sort by: Best match' and 'Display options'. The page number '1' is shown in a box, followed by 'of 921' and navigation arrows.

NIH National Library of Medicine
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Log in

PubMed®

cardiac arrest and hypothermia

Search

Advanced Create alert Create RSS User Guide

Save Email Send to

Sort by: Best match

Display options

MY CUSTOM FILTERS

9,210 results

Page 1 of 921

Plus de 9000 references.

Induced hypothermia and fever control for prevention and treatment of neurological injuries

Kees H Polderman

Vol 371 June 7, 2008



Prevention de l'apoptose		Time frame after injury
Prevention of apoptosis*	Ischaemia can induce apoptosis and calpain-mediated proteolysis. Hypothermia can prevent or reduce this process	Hours to many days or even weeks
Reduced mitochondrial dysfunction and improved energy homeostasis	Hypothermia reduces metabolic demands and might improve mitochondrial function	Hours to days
Reduction of excessive free radical production†	Production of free radicals such as superoxide, peroxynitrite, hydrogen peroxide, and hydroxyl radicals is typical in ischaemia	Hours to days
Reduction de la production excessive de radicaux libres		
	Suppressed by hypothermia	Hours to days
Reduced permeability of the blood-brain barrier and the vascular wall; reduced oedema formation*	Blood-brain barrier disruptions induced by trauma or ischaemia are moderated by hypothermia. The same effect occurs with hypothermia	Hours to days
Reduced permeability of cellular membranes (including membranes of the blood-brain barrier)	Decreased leakage of cellular membranes, with associated improvements in cell function and cellular homeostasis, including decrease of intracellular acidosis and mitigation of DNA injury	Hours to days
Attenuation de la reperfusion		
Impaired neurotransmission	Excessive release of neurotransmitters such as glutamate and prolonged activation of numerous enzyme systems (kinases) and induces a state of permanent hyperexcitability (excitotoxic cascade), which can be moderated by hypothermia	First minutes to 72 h
Reduction of metabolism*	Cellular oxygen and glucose requirements decrease by an average of 5–8% per degree Celsius decrease in temperature	Hours to days
Reduction de la permeabilite des membranes cellulaires		
Depression of the immune response and various potentially harmful proinflammatory reactions*	Susceptibility to infection during ischaemia can be blocked or mitigated by hypothermia	5 days
Effets anticoagulants		
	Higher temperatures than the surrounding areas and differences in blood flow can increase dramatically during injury, with up to 10°C in some areas of the brain. Hyperthermia can increase the damage to injured brain cells; this is mitigated by hypothermia	Minutes to many days
Anticoagulant effects*	Microthrombus formation might add to brain injury after CPR. Anticoagulant effects of hypothermia might protect against thrombus formation. Thrombolytic therapy has been shown to improve outcome after stroke	Minutes to days
Depression de la reponse immunitaire		
Suppression of epileptic activity and seizures*	Many patients with severe traumatic brain injury develop seizures. Hypothermia has been shown to mitigate epileptic activity	Hours to days
Diminution HTIC		
On the basis of clinical observations (eg, reduction in inflammatory response and pro-inflammatory cytokine levels) and experimental data (eg, reduction in excitatory transmitters measured using microdialysis probes in human beings; decrease in local brain hyperthermia). CPR=cardiopulmonary resuscitation. *Some supporting clinical evidence. †Animal studies only.		

Table 1: Possible mechanisms underlying protective effects of hypothermia

The New England Journal of Medicine

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VOLUME 346

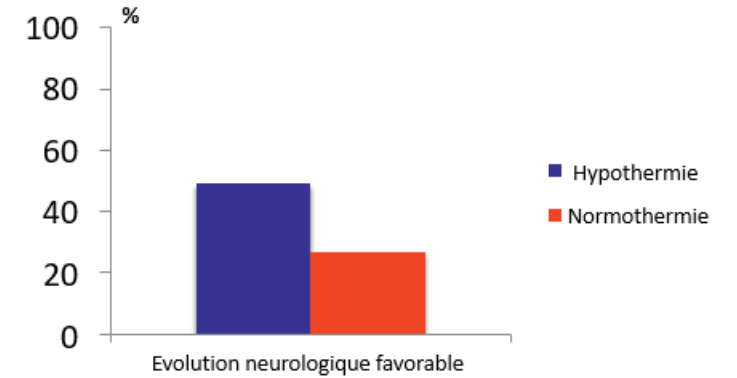
FEBRUARY 21, 2002

NUMBER 8



MILD THERAPEUTIC HYPOTHERMIA TO IMPROVE THE NEUROLOGIC OUTCOME AFTER CARDIAC ARREST

THE HYPOTHERMIA AFTER CARDIAC ARREST STUDY GROUP*



INDUCED HYPOTHERMIA AFTER OUT-OF-HOSPITAL CARDIAC ARREST

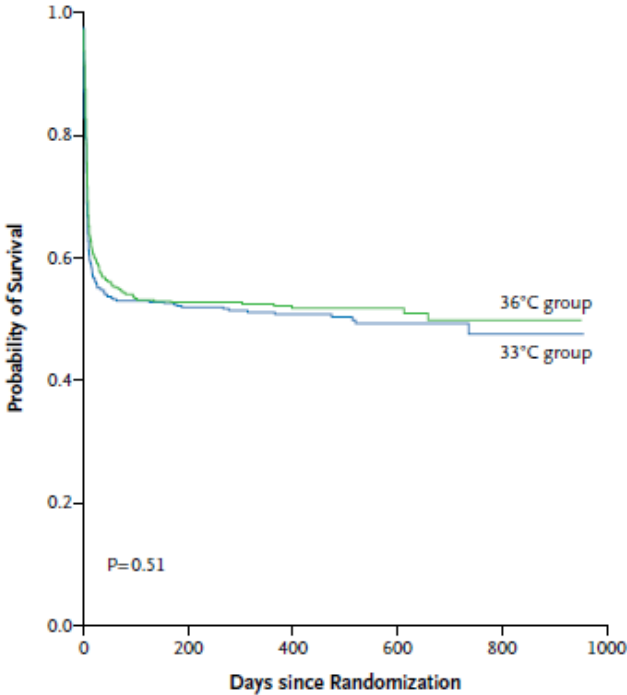
TREATMENT OF COMATOSE SURVIVORS OF OUT-OF-HOSPITAL CARDIAC ARREST WITH INDUCED HYPOTHERMIA

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.

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., Jørund 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*



No. at Risk	473	230	151	64	15
33°C group	473	235	144	68	12
36°C group	466	235	144	68	12

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

ORIGINAL ARTICLE

Hypothermia versus Normothermia after Out-of-Hospital Cardiac Arrest

J. Dankiewicz, T. Cronberg, G. Lilja, J.C. Jakobsen, H. Levin, S. Ullén, C. Rylander, M.P. Wise, M. Oddo, A. Cariou, J. Bělohávek, J. Hovdenes, M. Saxena, H. Kirkegaard, P.J. Young, P. Pelosi, C. Storm, F.S. Taccone, M. Joannidis, C. Callaway, G.M. Eastwood, M.P.G. Morgan, P. Nordberg, D. Erlinge, A.D. Nichol, M.S. Chew, J. Hollenberg, M. Thomas, J. Bewley, K. Sweet, A.M. Grejs, S. Christensen, M. Haenggi, A. Levis, A. Lundin, J. Düring, S. Schmidbauer, T.R. Keeble, G.V. Karamasis, C. Schrag, E. Faessler, O. Smid, M. Otáhal, M. Maggiorini, P.D. Wendel Garcia, P. Jaubert, J.M. Cole, M. Solar, O. Borgquist, C. Leithner, S. Abed-Maillard, L. Navarra, M. Annborn, J. Undén, I. Brunetti, A. Awad, P. McGuigan, R. Bjørkholt Olsen, T. Cassina, P. Vignon, H. Langeland, T. Lange, H. Friberg, and N. Nielsen, for the TTM2 Trial Investigators*

ABSTRACT

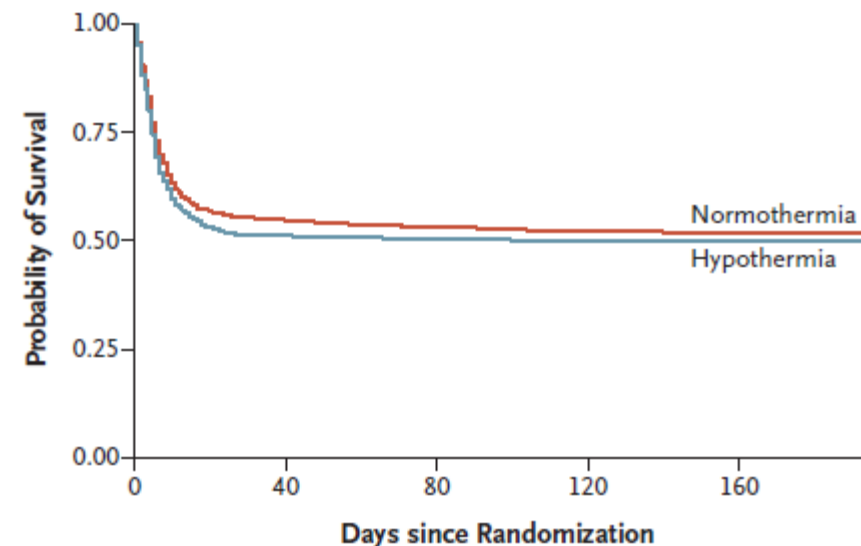


Figure 3. Probability of Survival until 180 Days after Randomization.

Shown are Kaplan–Meier estimates of the probability of survival until 180 days after randomization among patients assigned to undergo hypothermia or normothermia. Data are for the 1850 patients for whom survival status (including time of death) was available. Data were censored according to the last day of follow-up.



ERC-ESICM guidelines on temperature control after cardiac arrest in adults

Table 2 ERC-ESICM Recommendations for temperature control after cardiac arrest in adults



GOOD PRACTICE

We **recommend** continuous monitoring of core temperature in patients who remain comatose after ROSC from cardiac arrest.



LOW

We **recommend** actively preventing fever (defined as a temperature $> 37.7^{\circ}\text{C}$) in post-cardiac arrest patients who remain comatose.



GOOD PRACTICE

We **recommend** actively preventing fever for at least 72 hours in post-cardiac arrest patients who remain comatose.



GOOD PRACTICE

Temperature control can be achieved by exposing the patient, using anti-pyretic drugs, or if this is insufficient, by using a cooling device with a target temperature of 37.5°C .



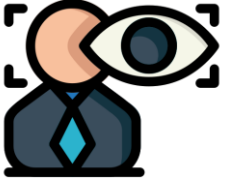
GOOD PRACTICE

There is currently insufficient evidence to recommend for or against temperature control at $32\text{--}36^{\circ}\text{C}$ in sub-populations of cardiac arrest patients or using early cooling, and future research may help elucidate this. We **recommend not** actively rewarming comatose patients with mild hypothermia after ROSC to achieve normothermia.



MODERATE

We **recommend not** using prehospital cooling with rapid infusion of large volumes of cold IV fluid immediately after ROSC.



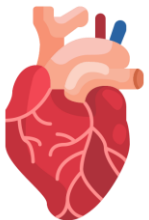
TEMOIN/ PAS TEMOIN, NF



INTRA/EXTRA HOSPITALIER



RYTHME CHOCABLE / NON CHOCABLE



CAUSE CARDIAQUE / NON CARDIAQUE

Quid des sédations ?



Pas de
refroidissement
=
pas de sédations ?



Rôle
neuroprotecteur de
la sédation



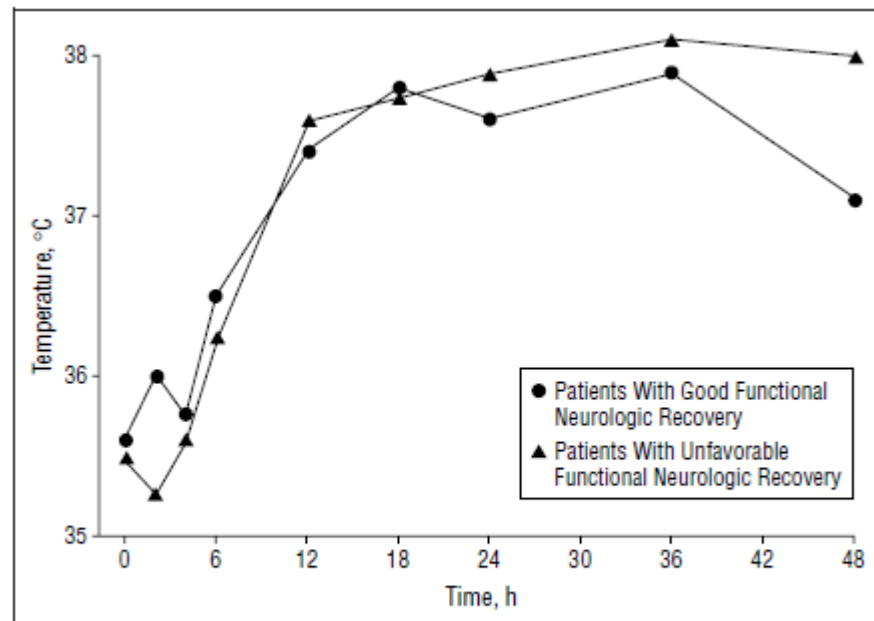
Evaluation
neurologique rapide



Switch sédations
si possible

Hyperthermia After Cardiac Arrest Is Associated With an Unfavorable Neurologic Outcome

Andrea Zeiner, MD; Michael Holzer, MD; Fritz Sterz, MD; Waltraud Schörkhuber, MD; Philip Eisenburger, MD; Christof Havel, MD; Andreas Kliegel, MD; Anton N. Laggner, MD



Temperature curves within 48 hours after successful cardiopulmonary resuscitation. Data are expressed as the median.

Hypothermia versus Normothermia after Out-of-Hospital Cardiac Arrest

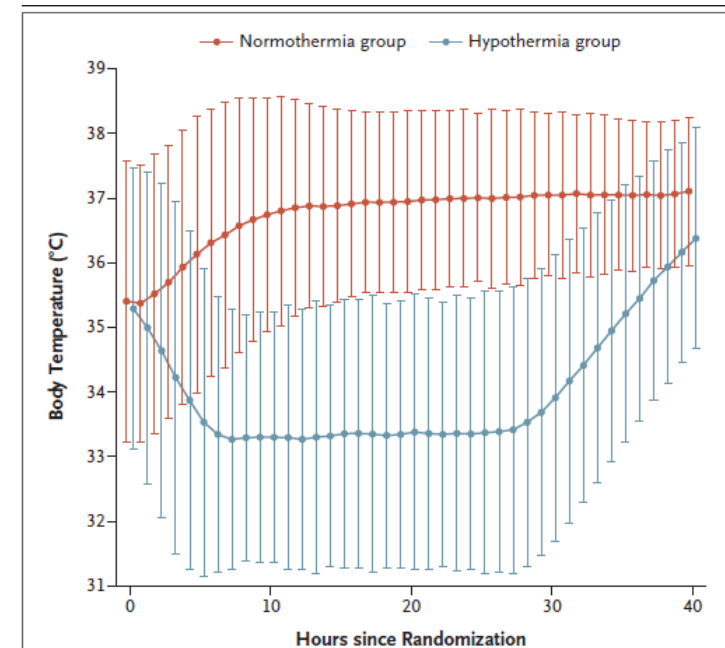


Figure 1. Body Temperature during the Intervention Period.

Shown are body-temperature curves in the hypothermia and normothermia groups for the patients in whom a bladder temperature was recorded. The median number of temperature recordings was 38 in both the hypothermia group and the normothermia group, out of 41 possible recordings. The temperature curves show the means, and the I bars indicate ± 2 SD (95% of the observations are within the error bars). The median time from cardiac arrest to randomization in the trial was 135 minutes.

Comparison of two sedation regimens during targeted temperature management after cardiac arrest

[Marine Paul](#)^{a,b,1} · [Wulfran Bougouin](#)^{a,c,d,1} · [Florence Dumas](#)^{a,c,d,e} · ... · [Jean-Paul Mira](#)^{a,b} · [Claudio Sandroni](#)^{h,2} · [Alain Cariou](#)^{a,b,c,d,2}  ... [Show more](#)



Fig 2b

Proportion of patients

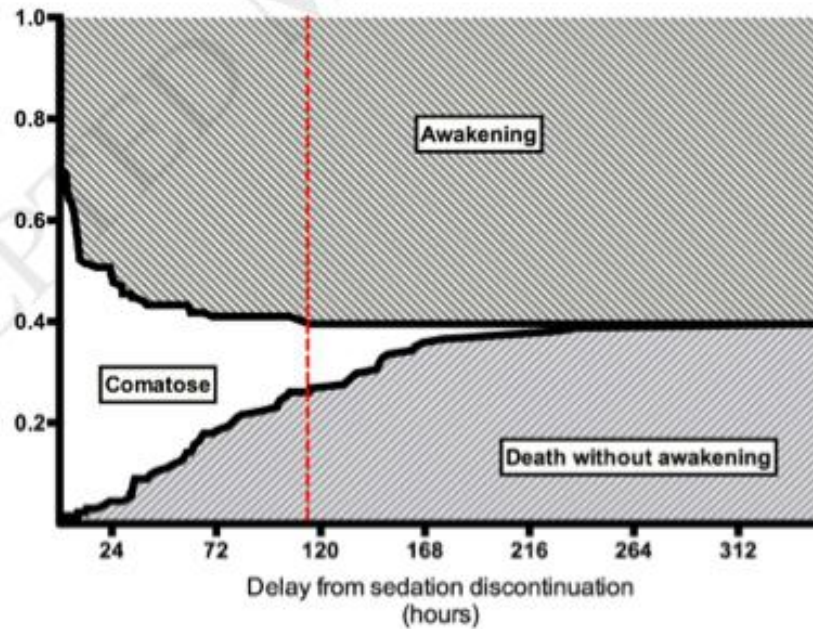
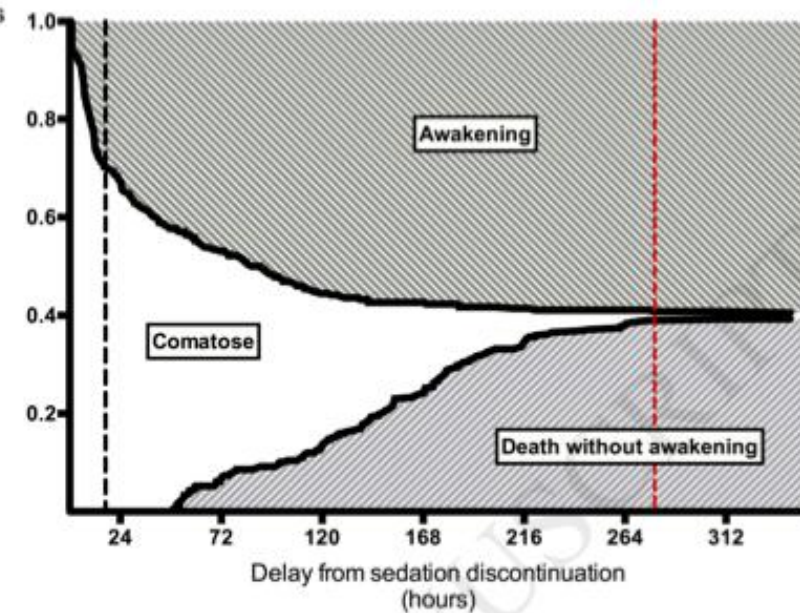


Fig 2a

Proportion of patients





Recherche clinique

metaphore 

Comparaison de deux niveaux de pression artérielle moyenne après arrêt cardiaque extra-hospitalier

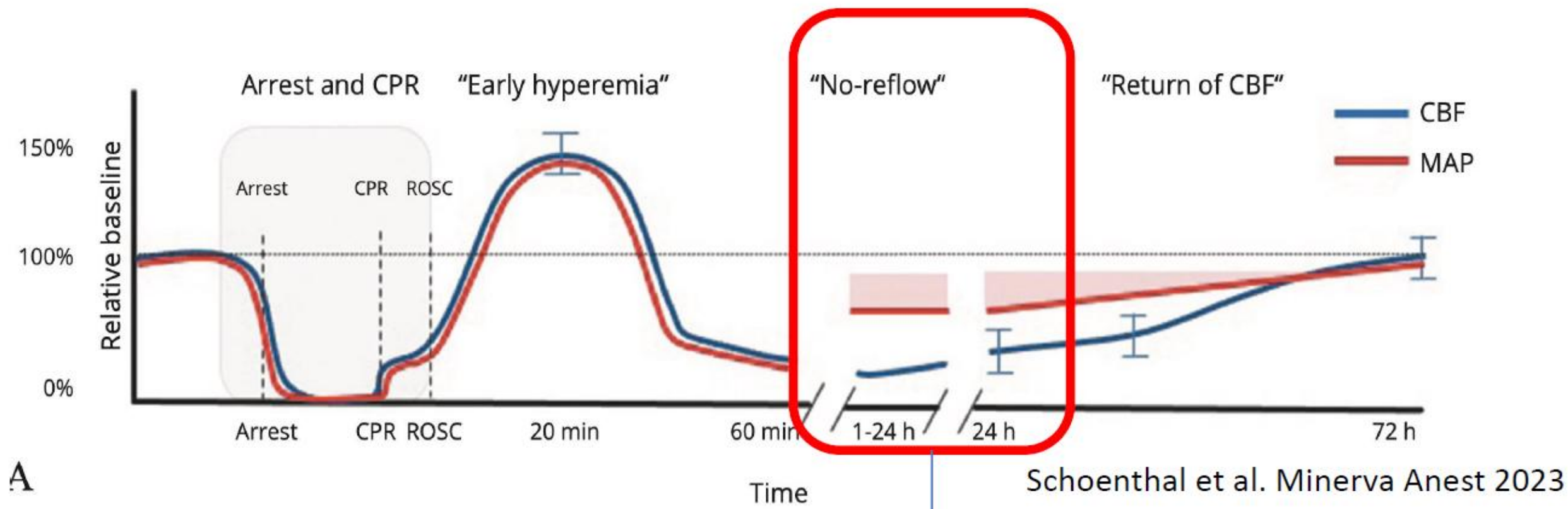
MEan arTeriAl Pressure after out-of-HOspital cardiac arREst

V1 19/09/2024



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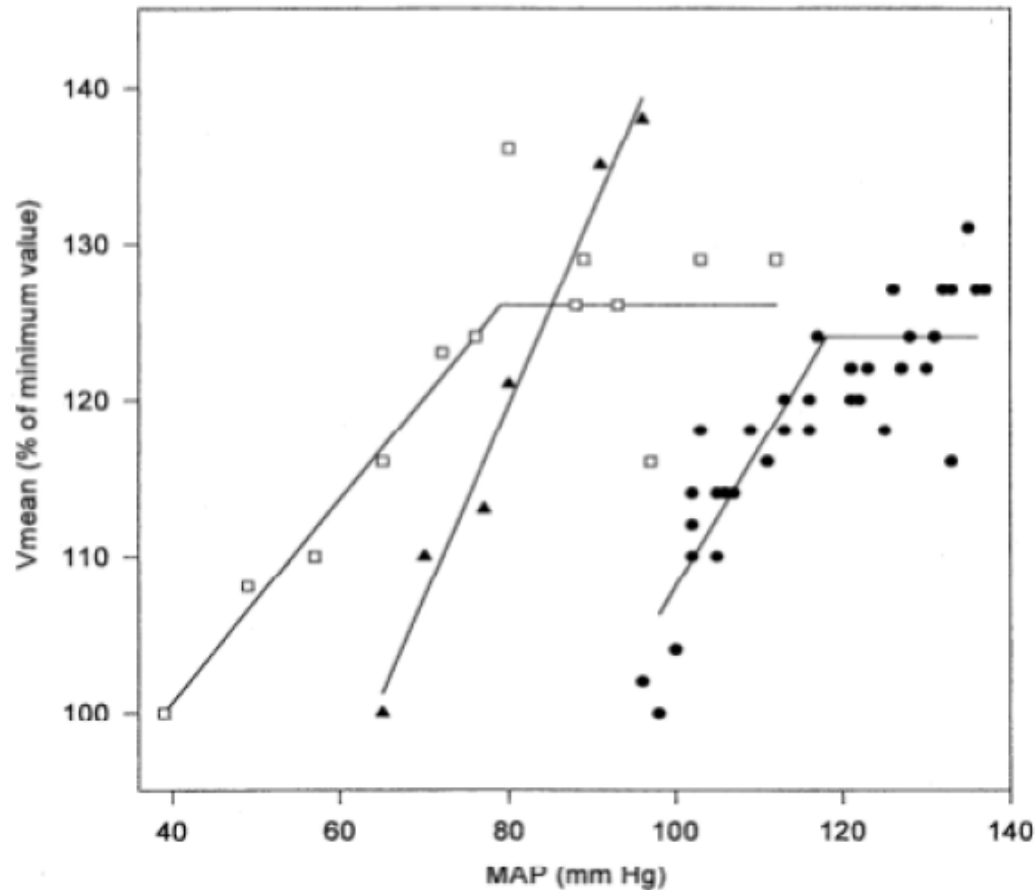


Vasoconstriction
 ↘ NO
 Microthrombi
 ↑ EO2

Safar et al. Ann Emerg Med 1993
Takagi et al. Stroke 1977
Safar et al. Arch Neurol 1976

Autoregulation of Cerebral Blood Flow in Patients Resuscitated From Cardiac Arrest

Claus Sundgreen, MD; Fin Stolze Larsen, MD; Tina Maria Herzog, MD; Gitte Moos Knudsen, MD; Søren Boesgaard, MD; Jan Aldershvile, MD



18 patients (50% choquables)

6 sujets sains

↑ PAM 30 mmHg (norépinéphrine)

Vm (Doppler transcrânien)

Absence d'autorégulation : 8 patients sur 18

Seuil inférieur dévié à droite: 5 patients sur 18

SEUIL INFÉRIEUR D'AUTORÉGULATION

76
mmHg

SUJETS SAINS

114
mmHg
[80-120]

PATIENTS

Sundgreen et al. Stroke 2001

An observational near-infrared spectroscopy study on cerebral autoregulation in post-cardiac arrest patients: Time to drop 'one-size-fits-all' hemodynamic targets?☆

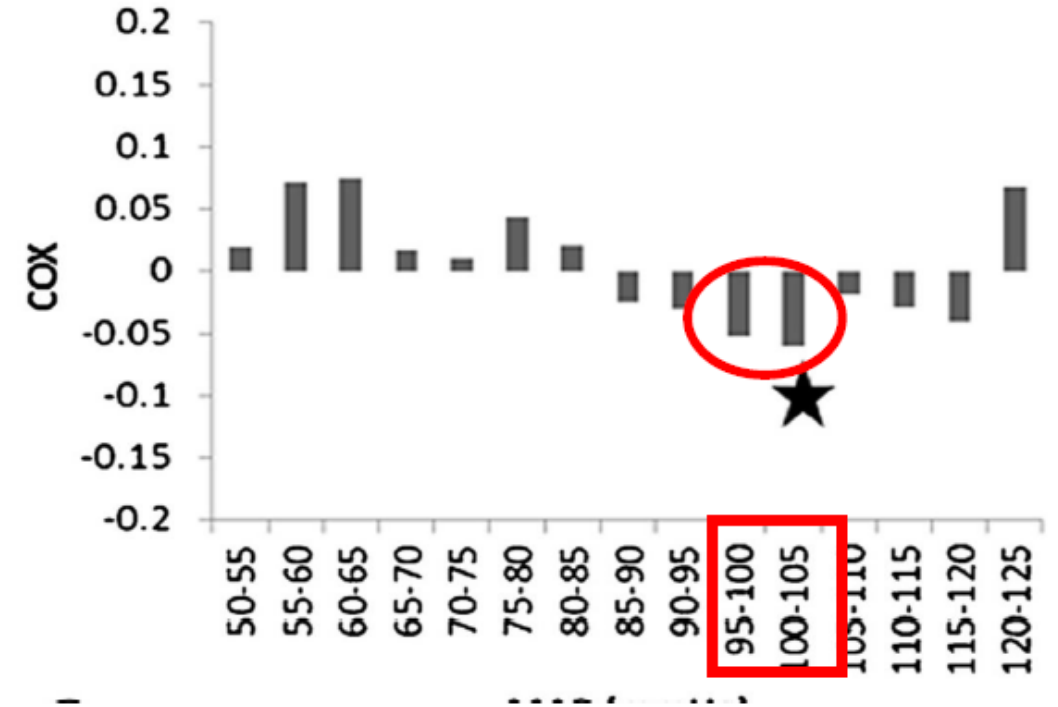
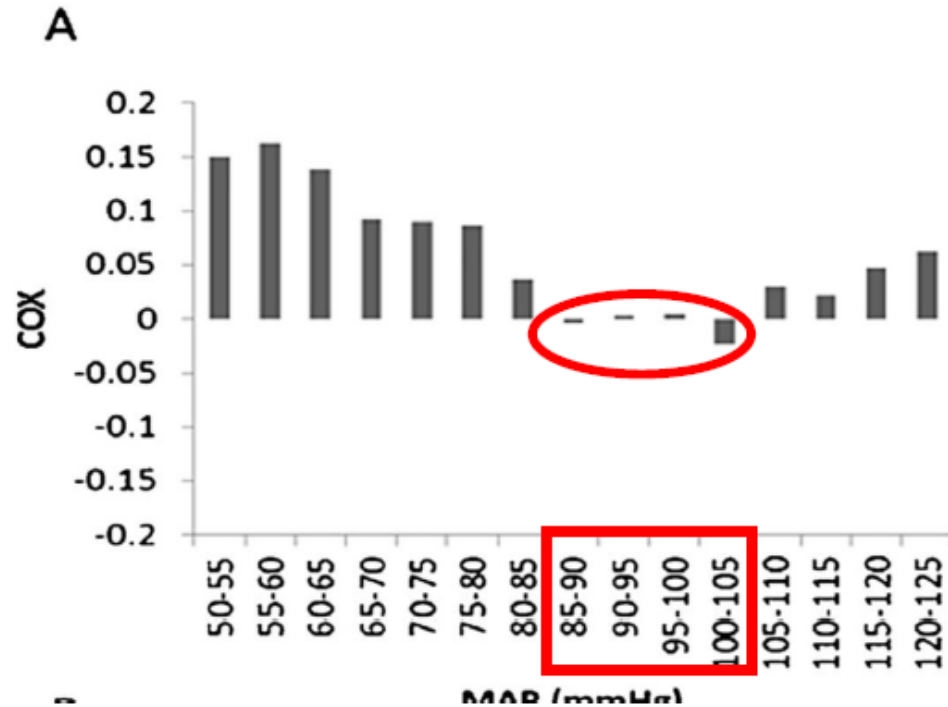


K. Ameloot^{a,*,1}, C. Genbrugge^{b,c,1}, I. Meex^{b,c}, F. Jans^{b,c}, W. Boer^b, M. Vander Laenen^b, B. Ferdinande^a, W. Mullens^{a,c}, M. Dupont^a, J. Dens^{a,c}, C. DeDeyne^{b,c}

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^b Department of Anesthesiology and Critical Care Medicine, Ziekenhuis Oost-Limburg, Genk, Belgium

^c Faculty of Medicine and Life Sciences, University Hasselt, Diepenbeek, Belgium



PAM optimale 85-100 mmHg?

An observational near-infrared spectroscopy study on cerebral autoregulation in post-cardiac arrest patients: Time to drop 'one-size-fits-all' hemodynamic targets?☆

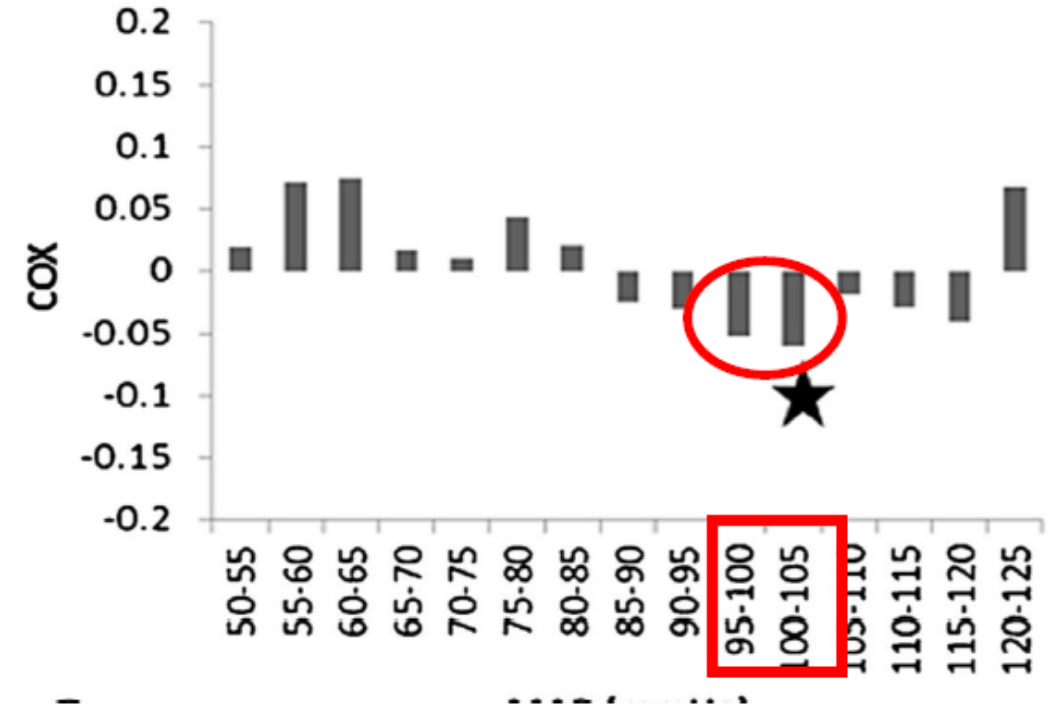
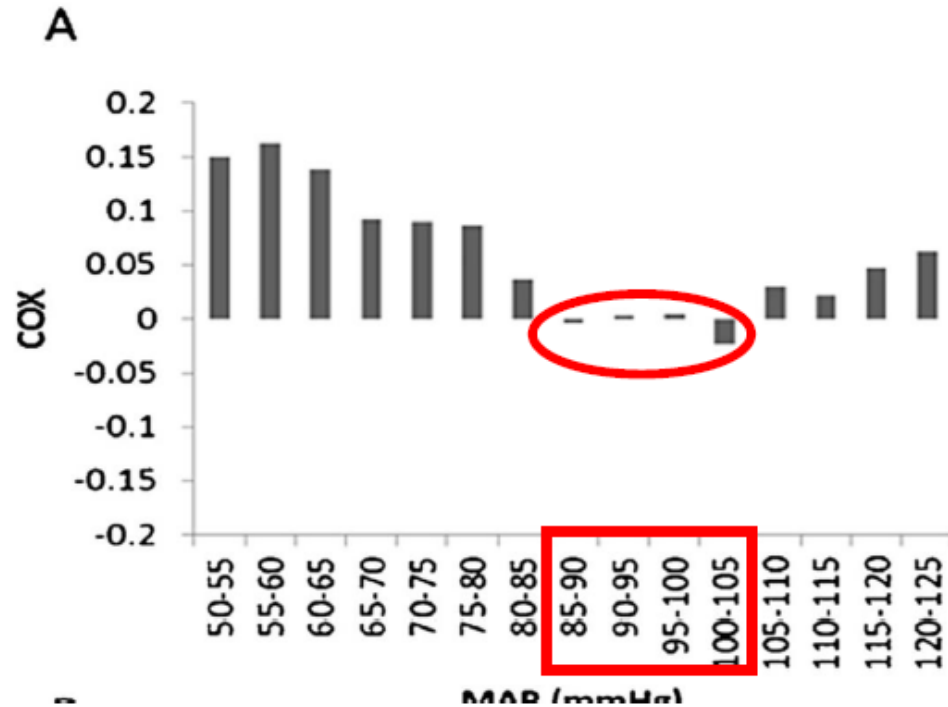


K. Ameloot^{a,*,1}, C. Genbrugge^{b,c,1}, I. Meex^{b,c}, F. Jans^{b,c}, W. Boer^b, M. Vander Laenen^b, B. Ferdinande^a, W. Mullens^{a,c}, M. Dupont^a, J. Dens^{a,c}, C. DeDeyne^{b,c}

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^c Faculty of Medicine and Life Sciences, University Hasselt, Diepenbeek, Belgium



PAM optimale 85-100 mmHg?

ORIGINAL

Targeting low-normal or high-normal mean arterial pressure after cardiac arrest and resuscitation: a randomised pilot trial



Jakkula et al. Intensive Care Medicine 2018

Early goal-directed haemodynamic optimization of cerebral oxygenation in comatose survivors after cardiac arrest: the Neuroprotect post-cardiac arrest trial

Ameloot et al. Eur Heart J 2020

PAM 65-75 vs 80-100 mmHg

Taux de NSE ($\mu\text{g/L}$) à 48h:

- 21.2 [15.1-34.0] vs 22.0 [13.6-30.9] $p=0.392$

PAM 65 mmHg vs 85-100 mmHg + SvO₂ 65-75%

% de voxels avec un score ADC $< 650.10^6$ mm²/s:

- 12% [9-16] vs 16% [13-21], $p=0.09$

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Blood-Pressure Targets in Comatose Survivors of Cardiac Arrest

DESIGN

Etude randomisée
2 centres au Danemark
Mars 2017-Décembre
2021

INTERVENTION

**PAM ≥ 63 mmHg vs PAM
 ≥ 77 mmHg**

TTM 36°C

Remplissage vasculaire,
noradrénaline, dopamine



RÉSULTATS



799 patients:

- Age 63 ans
- 45.9% hypertension artérielle



Caractéristiques de l'arrêt cardiaque:

- Extra-hospitalier
- Cause cardiaque présumée ou non connue
- 84.8% Rythme choquable
- 88% RCP par un témoin
- Durée No flow+ Low Flow 21 minutes
- 45.3% STEMI

Blood-Pressure Targets in Comatose Survivors of Cardiac Arrest

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Etude randomisée
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Remplissage vasculaire,
noradrénaline, dopamine

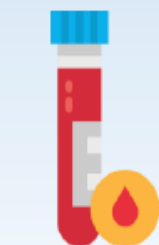


RÉSULTATS



Décès ou CPC 3 ou 4 à J90:

- 127 (32%) vs 133 (34%), 1.08 [0.84-1.37], $p=0.56$



NSE à 48h:

- 18 $\mu\text{g/L}$ [11-34] vs 18 $\mu\text{g/L}$ [11-37]

Score MoCA à J90:

- 26 [24-29] vs 27 [24-29]

✓ Admission en réanimation après un arrêt cardiaque extra-hospitalier

✓ Reprise d'une activité cardiaque spontanée efficace persistante > 20 minutes

✓ Patient sous ventilation mécanique invasive pour coma défini par un score de Glasgow $\leq 8/15$

✓ Consentement d'un membre de la famille ou d'un proche ou procédure d'inclusion en urgence

Critère de jugement principal = proportion de patients mRS 0-3 dans chaque bras

65
mmHg

30 %

90
mmHg

38 %

Puissance 80%

Risque alpha 5% (2 analyses intermédiaires)

20% de chevauchement entre les 2 bras

1380 patients
(690 / bras)

Période d'inclusion : 3 ans
1.4 inclusion / mois / centre

2 analyses intermédiaires (n = 400 et 800)

✓ mortalité toute cause à J28

✓ complications globales J0 à J7

HYPOTHÈSE

Cibler une PAM haute (≥ 90 mmHg) pendant les 24 premières heures de prise en charge après un arrêt cardiaque extra-hospitalier améliore la survie et le devenir neuro-fonctionnel à J180.

Comparer les effets d'une cible haute de PAM (≥ 90 mmHg) et d'une cible standard de PAM (≥ 65 mmHg) sur le devenir neurologique 6 mois après un arrêt cardiaque extra-hospitalier

Proportion de patients avec évolution neurologique favorable (**score modifié de Rankin entre 0 et 3**) à J180

RANDOMISATION

Algorithme de minimisation (HTA, rythme, centre)

Bras interventionnel
PAM ≥ 90 mmHg pendant 24h

*Dose minimale de
NORADRÉNALINE*

*↑ 0.05 $\mu\text{g/kg/min}$ toutes les 10 min
↓ 0.05 $\mu\text{g/kg/min}$ toutes les heures*

Pas de valeur maximale de PAM
**Anti-hypertenseurs non
recommandés** pendant 24h sauf
complications*

Puis PAM ≥ 65 mmHg

**En cas de
complications,
modification de la cible
et anti-hypertenseurs au
libre choix du clinicien*

Bras « standard of care »
PAM ≥ 65 mmHg pendant 24h

*Dose minimale de
NORADRÉNALINE*

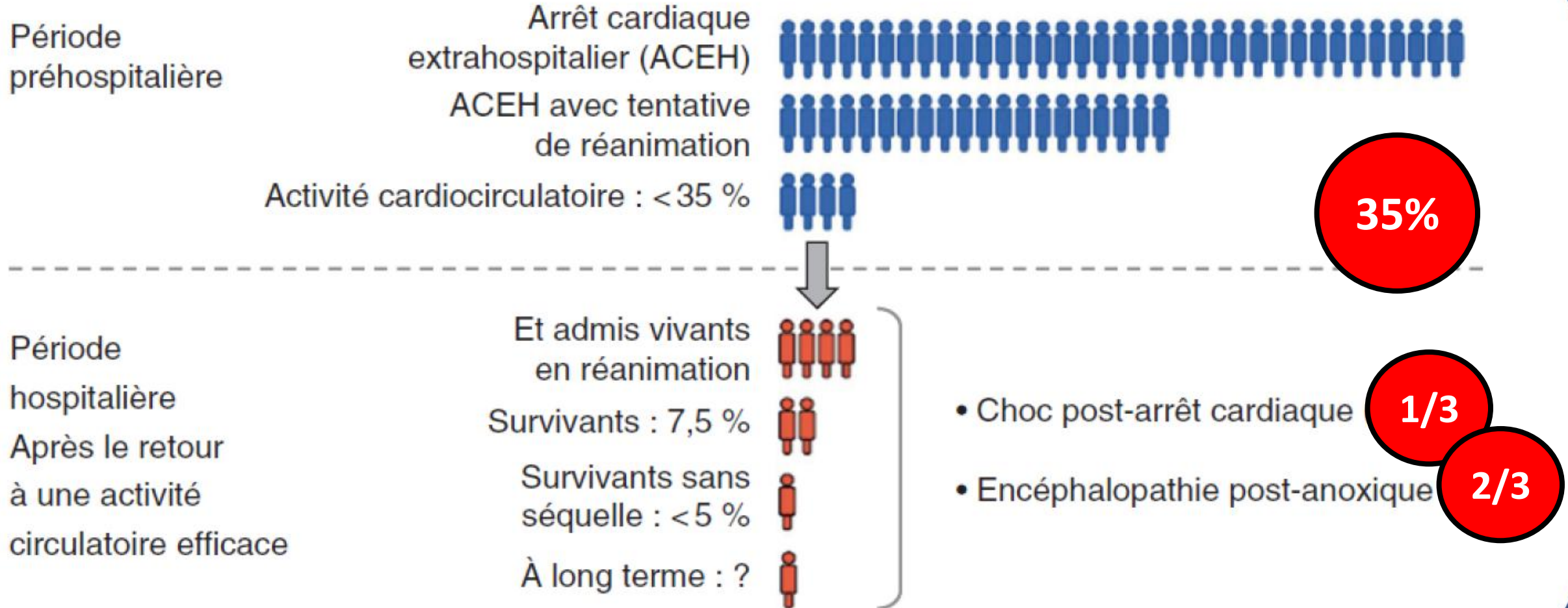
*↑ 0.05 $\mu\text{g/kg/min}$ toutes les 10 min
↓ 0.05 $\mu\text{g/kg/min}$ toutes les heures*

Pas de valeur maximale de PAM
**Anti-hypertenseurs non
recommandés** pendant 24h sauf
complications*

Puis PAM ≥ 65 mmHg

La stratégie doit être appliquée dès la randomisation et tout doit être mis en œuvre pour atteindre la cible dans les 30 minutes qui suivent la randomisation!

Survie après arrêt cardiaque

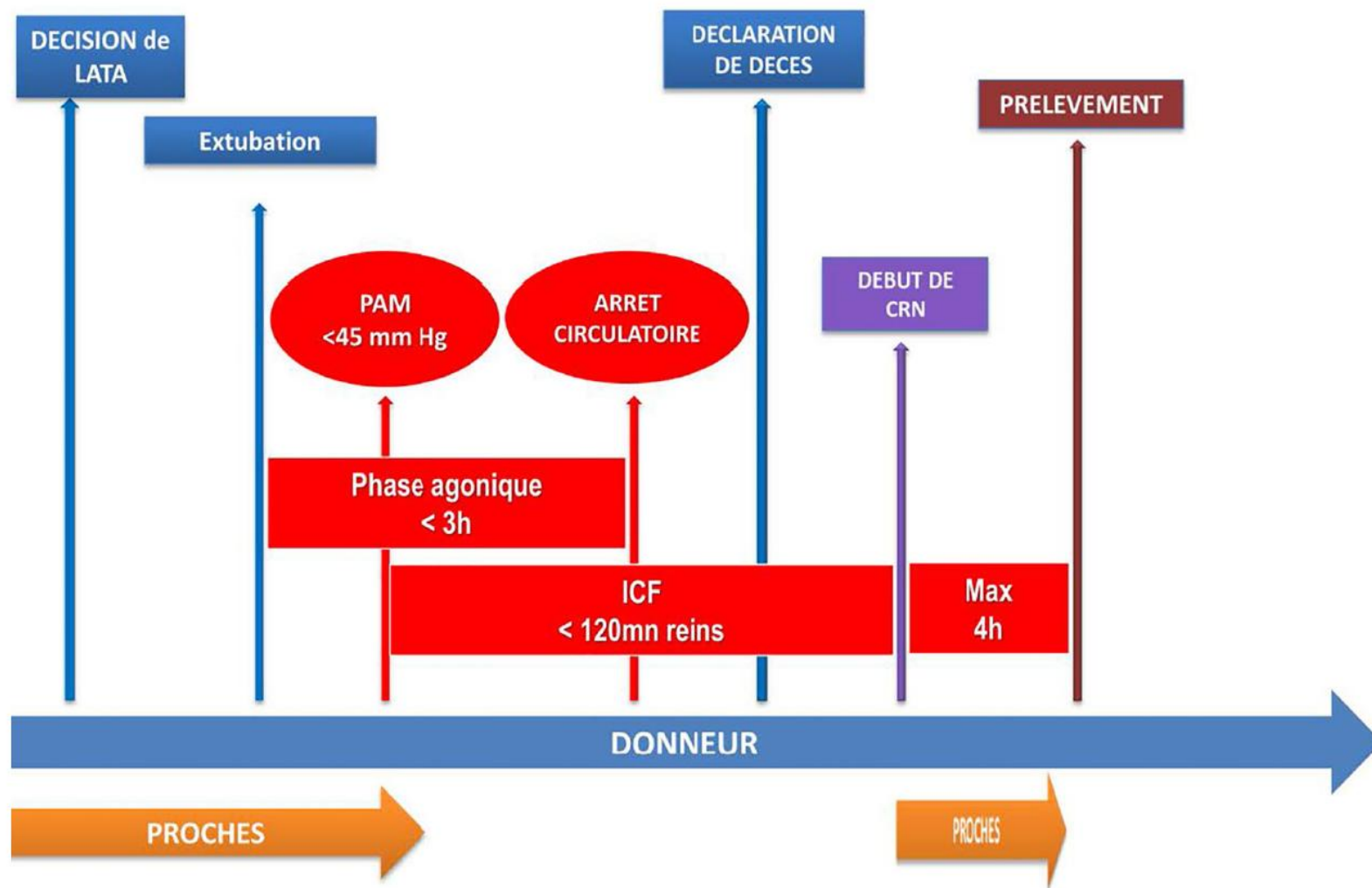


Survie après arrêt cardiaque

Classification internationale dite de Maastricht 1995, révisée en 2013

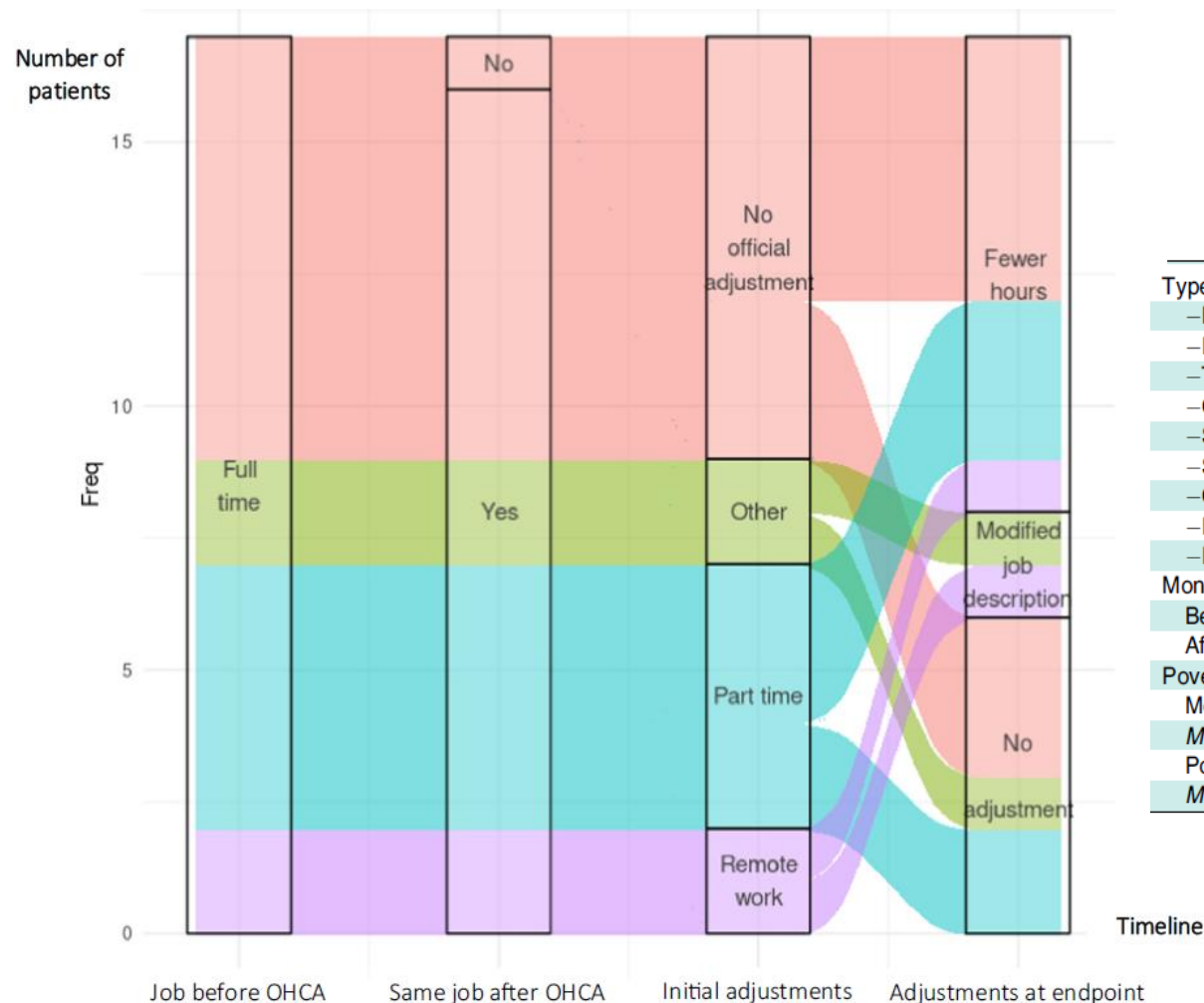
- | | |
|---------------|---|
| Catégorie I | Les personnes qui font un arrêt circulatoire en dehors de tout contexte de prise en charge médicalisée, déclarées décédées à la prise en charge |
| Catégorie II | Les personnes qui font un arrêt circulatoire avec mise en œuvre d'un massage cardiaque et d'une ventilation mécanique efficaces, mais sans récupération d'une activité circulatoire |
| Catégorie III | Les personnes pour lesquelles une décision de limitation ou d'arrêt programmé des thérapeutiques est prise en raison du pronostic des pathologies ayant amené la prise en charge en réanimation |
| Catégorie IV | Les personnes décédées en mort encéphalique qui font un arrêt circulatoire irréversible au cours de la prise en charge en réanimation |

**Questionnement
Ethique ?**



Return to work after out of hospital cardiac arrest, insights from a prospective multicentric French cohort

Nolwen Flajoliet^{a,*}, Jeremy Bourenne^{b,h}, Nathalie Marin^a, Jonathan Chelly^{c,h}, Jean Baptiste Lascarrou^{d,h}, Cédric Daubin^{e,h}, Wulfran Bougouin^{f,h}, Alain Cariou^{a,h}, Guillaume Geri^{g,h}



	No return to work at endpoint <i>n</i> = 14	Return to work at endpoint <i>n</i> = 17	<i>p</i> -value
Type of job – ISCO 08. N			
–Managers	1	5	
–Professionals	0	7	
–Technicians and Associate Professionals	3	0	0.005
–Clerical Support Workers	2	0	
–Services and Salesworkers	2	1	
–Skilled Agricultural, Forestry and Fishery Workers	0	1	
–Craft and Related Trades Workers	2	1	
–Plant and Machine Operators and Assemblers	3	1	
–Elementary Occupation	1	1	
Monthly income. median (Q1Q3)			
Before OHCA. median (Q1Q3)	1800 (1500–2500)	3500 (1950–6000)	0.024
After OHCA. median (Q1Q3)	900 (800–1200)	3400 (2200–6000)	<0.001
Poverty rate and modified poverty rate. median (Q1Q3)			
Modified poverty rate. median (Q1Q3)	20.00 (14.57–27.95)	15.20 (12.50–19.65)	0.001
Missing data	6	5	
Poverty rate. median (Q1Q3)	17.70 (11.97–20.27)	10.80 (9.20–12.45)	<0.001
Missing data	8	6	

Conclusion

Après la RACS , c'est le début des problèmes

