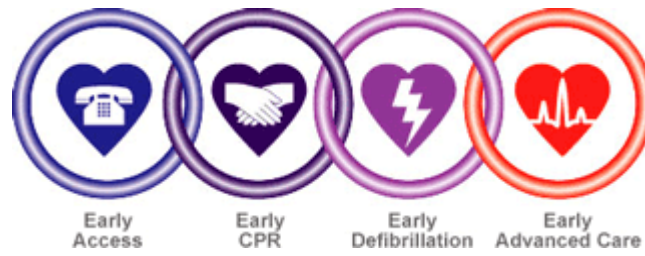


Post Resuscitation Care

Resus 2011

Dr. Fergal H. Cummins FRCSedA&E, MCEM, DMMD, EMDM, FCEM, FACEM
Consultant in Emergency Medicine, Retrieval Specialist
Retrieval, Emergency and Disaster Medicine
Research and Development Unit
Department of Emergency Medicine
Mid Western Regional Hospital - Limerick, Ireland





- 1.** Intervention du témoin
- 2.** Enclenchement rapide des SPU
- 3.** Soins donnés par les premiers répondants
- 4.** Soins préhospitaliers spécialisés
- 5.** Soins hospitaliers
- 6.** Rétablissement



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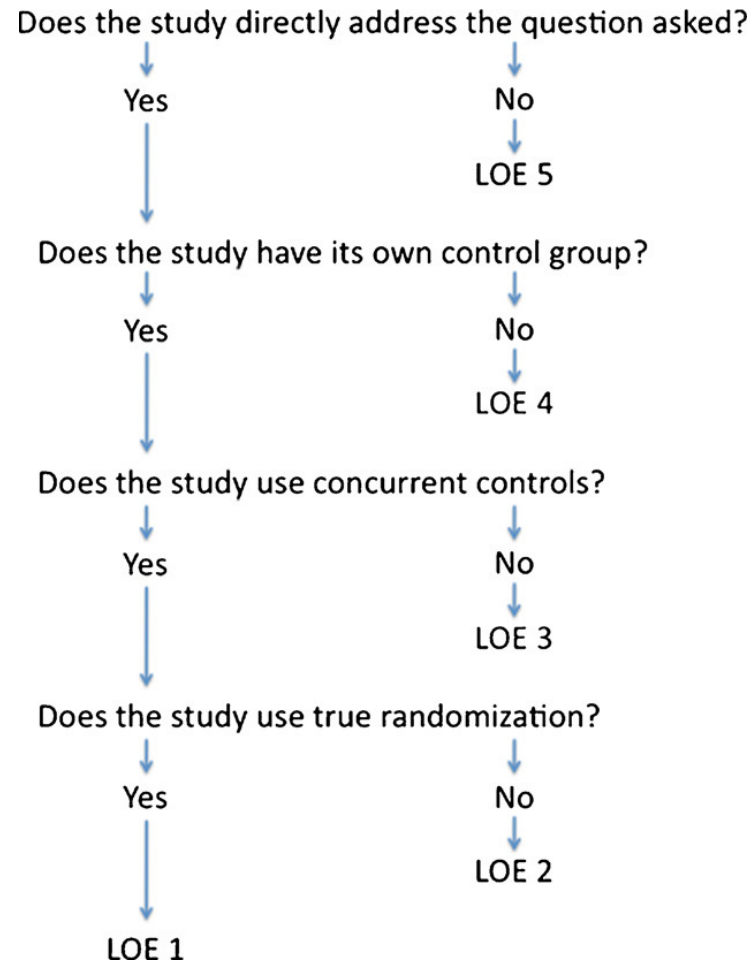


Part 8: Advanced life support

2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations^{☆,☆☆}

Charles D. Deakin (Co-chair)^{*1}, Laurie J. Morrison (Co-chair)¹, Peter T. Morley, Clifton W. Callaway, Richard E. Kerber, Steven L. Kronick, Eric J. Lavonas, Mark S. Link, Robert W. Neumar, Charles W. Otto, Michael Parr, Michael Shuster, Kjetil Sunde, Mary Ann Peberdy, Wanchun Tang, Terry L. Vanden Hoek, Bernd W. Böttiger, Saul Drajer, Swee Han Lim, Jerry P. Nolan, on behalf of the Advanced Life Support Chapter Collaborators

Part 3: Evidence evaluation process
2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations



Allocating levels of evidence

Postresuscitation care

Postresuscitation treatment protocol

ALS-PA-047A, ALS-PA-047B

Implementation of a standardised treatment protocol for post resuscitation care after out-of-hospital cardiac arrest

Kjetil Sunde^{a,b,*}, Morten Pytte^{a,b}, Dag Jacobsen^c, Arild Mangschaud^d, Lars Petter Jensen^a, Christian Smedsruda^e, Tomas Draegnia^f, Petter Andreas Steena^g
Resuscitation (2007) 73, 29–39

Summary

Background:

Introduced and implemented a standardised post resuscitation protocol focusing on vital organ function including therapeutic hypothermia, percutaneous coronary intervention (PCI), control of haemodynamics, blood glucose, ventilation and seizures.

Methods:

All patients with OHCA of cardiac aetiology admitted to the ICU from September 2003 to May 2005 (intervention period) were included in a prospective, observational study and compared to controls from February 1996 to February 1998.

Results:

In the control period 15/58 (26%) survived to hospital discharge with a favourable neurological outcome versus 34 of 61 (56%) in the intervention period (OR 3.61, CI 1.66–7.84, $p = 0.001$).

Inhospital standardised treatment plan after ROSC at Ullevål University Hospital

Goal: to reduce the vital organ injuries (brain, heart), through:

1. Initial optimising haemodynamics and oxygenation

**2. (a) Treat the cause of arrest; reperfusion (PCI) after STEMI and
(b) Therapeutic hypothermia (33 °C in comatose patients for 24 h)**

ice-cold 0.9% NaCl i.v. together with icebags. Endovascular cooling/external cooling

3. A standardised treatment protocol for the following days

3.1. Factor Goal Strategy

Reperfusion, Blood pressure, Central venous pressure, ECG, rate/ischaemia, Ventilator, Blood glucose, Electrolytes Normal values Replacement/specific treatment, Haemoglobin, Diuresis, Buffers, Seizures Prevent/treat seizures

3.2. Sedation Fentanyl and propofol (paralysis when indicated)

3.3. Monitoring

3.4. Vasopressors/inotropic agents First choice: dopamine If pump failure/cardiogenic shock IABP

3.5. Awakening protocol/respirator weaning After 24 h of cooling, patients should be slowly rewarmed

Postresuscitation care

Postresuscitation treatment protocol

ALS-PA-047A, ALS-PA-047B

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of comprehensive treatment protocol, as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

NoRCTs

Before-and-after studies - increase in survival out-of-hospital cardiac arrest with implementation of a comprehensive treatment protocol (LOE 2; LOE 3).

Protocols included multiple elements

The independent effect of each element of the bundle of care could not be established.

Treatment recommendation

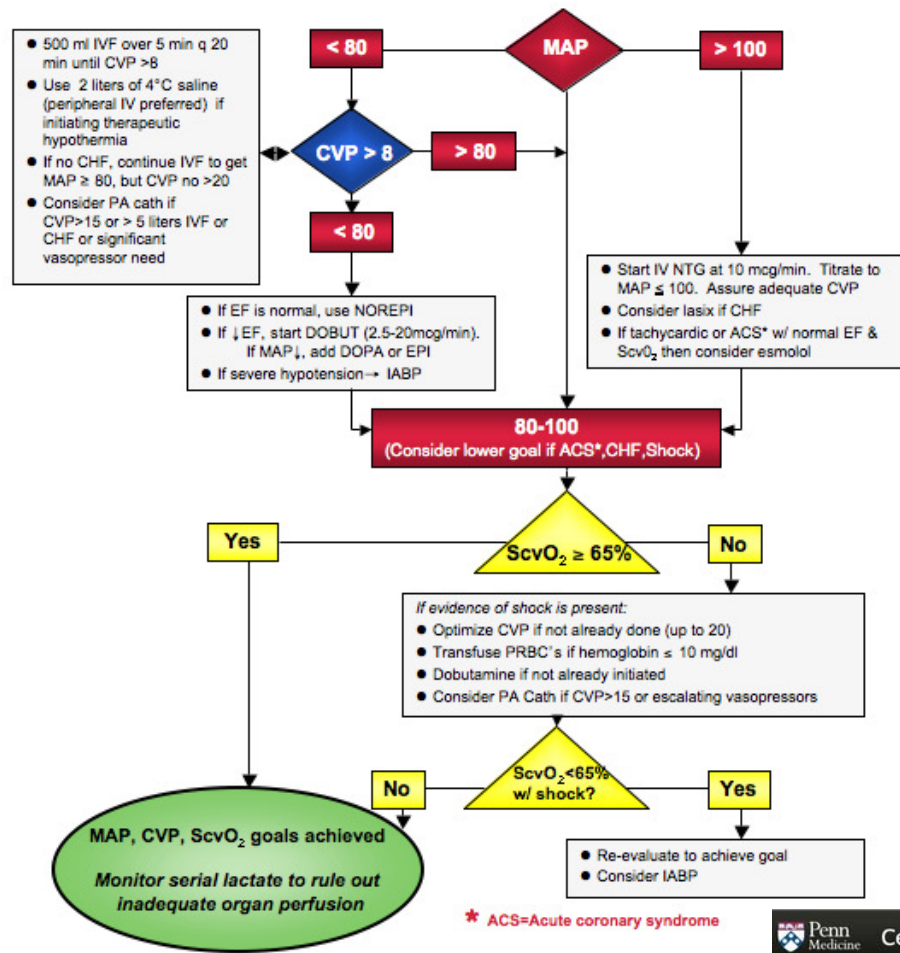
A comprehensive treatment protocol that includes multiple interventions provided in a structured way may improve survival after cardiac arrest.



Post-Cardiac Arrest Early Goal-Directed Therapy

- Who needs this?**
Resuscitated patients with:
- Pulseless < 60 min
 - GCS Motor score < 6
 - No other reason for coma
 - Not DNR or DNI status
 - If pregnant consult Ob/Gyn

- Getting Started**
- Stat ECG, echocardiogram, and cardiology consult
 - Stat head CT
 - Insert arterial pressure monitoring line in radial or femoral artery
 - Initiate therapeutic hypothermia if indicated (after arterial line)
 - Insert Presep[®] CVC in subclavian or internal jugular vein
 - Notify Bed Coordinator for ICU bed and EEG fellow for EEG



* ACS=Acute coronary syndrome



Treatment of precipitating causes of cardiac arrest- Pulmonary embolism

ALS-PA-046A, ALS-PA-046B

Treatment of precipitating causes of cardiac arrest- Pulmonary embolism

ALS-PA-046A, ALS-PA-046B

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital) diagnosed as pulmonary embolism, does the use of early fibrinolytic therapy with or without thrombectomy, as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

Good theoretical reasons why fibrinolysis might be beneficial

No direct evidence to that effect.

No significant increase in survival to hospital discharge. (LOE 5)(LOE 4)

Treatment recommendation

In patients with diagnosed or suspected pulmonary embolism after ROSC following cardiac arrest, there is inadequate evidence to recommend for or against the use of fibrinolytic therapy in addition to heparin.

Part 9: Acute coronary syndromes

Reperfusion strategies

Reperfusion strategies

Out-of-hospital fibrinolytics for STEMI

Prehospital fibrinolytics for STEMI

ACS-018B

In patients with STEMI in the prehospital setting, does the use of prehospital fibrinolytics, compared with in-hospital fibrinolytics, improve outcomes (e.g., chest pain resolution, infarct size, ECG resolution, survival to discharge, 30/60 days mortality)?

Consensus on science

Reduced time to treatment when fibrinolytics were given to patients with STEMI in the **prehospital** setting by either physicians, nurses, or paramedics Nineteen studies (LOE 1, LOE 2, LOE 3).

Prehospital fibrinolysis had **shorter duration and increased frequency of total resolution of chest pain** by the time of admission, **ECG resolution, and decreased mortality** Eleven studies (LOE 1; LOE 2).

Treatment recommendations

In patients with STEMI diagnosed in the prehospital setting, reperfusion may be achieved by administration of fibrinolytics by healthcare providers in the field.

Alternately, fibrinolytic therapy may be administered on arrival at hospital.

Fibrinolysis should be started as soon as possible

Reperfusion strategies

Choice of reperfusion strategy in the hospital

PPCI versus fibrinolytic therapy for STEMI

ACS-025B

In patients with suspected STEMI in the ED setting, does the use of PPCI compared with fibrinolytic therapy, improve outcome (e.g., arrhythmias, infarct size, ECG resolution, survival to discharge, 30/60 days mortality)?

Consensus on science

Hospital with PCI facilities

PPCI conferred clinical benefit compared with fibrinolysis both in terms of mortality and morbidity. 2 studies (LOE 1).

Hospital without PCI facilities

Two studies showed benefit associated with transferring patients for PPCI versus on-site fibrinolysis in terms of reinfarction and stroke and a trend to a lower mortality in the PPCI group (LOE 2).

For patients with cardiogenic shock, early revascularization improves survival at 6 months. One randomised trial NB survival benefit <75 years of age.

(LOE 1).

Reperfusion strategies

Choice of reperfusion strategy in the hospital

PPCI versus fibrinolytic therapy for STEMI

ACS-025B

In patients with suspected STEMI in the ED setting, does the use of PPCI compared with fibrinolytic therapy, improve outcome (e.g., arrhythmias, infarct size, ECG resolution, survival to discharge, 30/60 days mortality)?

Treatment recommendations

Shorter intervals to reperfusion independently of the method chosen.

Programs to reduce the time to PCI.

The benefit of mechanical intervention over fibrinolysis varies considerably depending on the patient's condition and the duration of PPCI-related delays.

For those patients with a contraindication to fibrinolysis, PCI should still be pursued despite the delay, rather than offering no reperfusion therapy.

STEMI patients in shock, PCI (or CABG) is the preferred reperfusion option. Fibrinolysis should only be considered if there is a substantial delay to PCI.

Reperfusion strategies

Fibrinolytics and immediate PCI (facilitated PCI) versus immediate PCI

ACS-028A,ACS-028B

In patients with suspected STEMI in the ED and prehospital settings, does the use of fibrinolytics and immediate PCI, compared with immediate PCI, improve outcome (e.g., chest pain resolution, infarct size, ECG resolution, survival to discharge, 30/60 days mortality)?

Consensus on science

Poorer outcome with routine PCI shortly after fibrinolysis.

12 studies (LOE 1; LOE 2; LOE 5).

Facilitated PCI strategy supported

Eleven studies (LOE 1; LOE 2; LOE 3; LOE 5).

No benefit of PPCI over fibrinolysis

Thirty studies (LOE1; LOE 2; LOE 5).

Treatment recommendations

The routine use of fibrinolysis-facilitated PPCI, compared with PPCI, is not recommended in patients with suspected STEMI.

NB Rescue angio is reasonable

Ventilation

ALS-PA-053B

Ventilation

ALS-PA-053B

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of a specific ventilation strategy (including specific CO₂ goal), as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

Limited studies

Reduced cerebral flow response to hyperventilation (LOE 5). One study (LOE 2), animal studies (LOE 5)

Avoiding hyperventilation, as part of a bundle of care, improved long-term outcome in humans (LOE 3) and in dogs (LOE 5), independent effect of ventilation not determined.

Tidal volumes ≤ 9 mL/kg associated with increased incidence of atelectasis (LOE 3).

Manipulation of tidal volume and PEEP are not associated **independently** with improved (LOE 2; LOE 3).

Treatment recommendation

After restoration of circulation, routine hyperventilation leading to hypocapnia should be avoided in order to prevent additional cerebral ischaemia.

Controlled oxygenation

ALS-PA-061A, ALS-PA-061B

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

[J. Hope Kilgannon](#) Emergency Medicine Shock Research Network (EMShockNet) Investigators *JAMA*. 2010;303(21):2165-2171.

- **Objective** To test the hypothesis that postresuscitation hyperoxia is associated with increased mortality.
- **Design, Setting, and Patients** Multicenter cohort study using the Project IMPACT critical care database of intensive care units (ICUs) at 120 US hospitals between 2001 and 2005.
- Patients were divided into 3 groups defined a priori based on PaO₂ on the first arterial blood gas values obtained in the ICU. Hyperoxia; hypoxia, and normoxia.
- **Main Outcome Measure** In-hospital mortality.
- **Results** Of 6326 patients, 1156 had hyperoxia (18%), 3999 had hypoxia (63%), and 1171 had normoxia (19%).
- **The hyperoxia group had significantly higher in-hospital mortality (732/1156 [63%; 95% confidence interval {CI}, 60%-66%])** compared with the normoxia group (532/1171 [45%; 95% CI, 43%-48%]; proportion difference, 18% [95% CI, 14%-22%]) and the hypoxia group (2297/3999 [57%; 95% CI, 56%-59%]; proportion difference, 6% [95% CI, 3%-9%]).
- **Hyperoxia exposure had an odds ratio for death of 1.8 (95% CI, 1.5-2.2).**
- **Conclusion** Among patients admitted to the ICU following resuscitation from cardiac arrest, arterial hyperoxia was independently associated with increased in-hospital mortality compared with either hypoxia or normoxia.

Controlled oxygenation

ALS-PA-061A, ALS-PA-061B

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of a controlled oxygenation strategy (including specific oxygenation goal), as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

One neutral randomised prospective clinical trial compared ventilation with 30% oxygen or 100% oxygen for the first 60 min after ROSC (LOE 1).

Mean partial pressure of oxygen in arterial blood (PaO₂) at 60min after ROSC was 14.6±3.3 kPa (110±25mmHg) in the 30% oxygen group and 46.5±23.2 kPa (343±174mmHg) in the 100% oxygen group.

No statistical difference was detected in serum biomarkers of acute brain injury, survival to hospital discharge, or the percent of patients with good neurological outcome (cerebral performance category of 1 or 2) at hospital discharge.

Not adequately powered to detect important differences in survival, cerebral performance category, efficacy or harm (n = 14 per group)

Treatment recommendations

There is insufficient clinical evidence to support or refute the use of inspired oxygen concentration titrated to arterial blood oxygen saturation in the early care of cardiac arrest patients following sustained ROSC.

Knowledge gaps

Prospective randomised controlled clinical trials are needed to compare ventilation with 100% oxygen versus ventilation with inspired oxygen titrated to an arterial blood oxygen saturation goal (possibly 94–96%) for the first hour after sustained ROSC. Studies evaluating combined myocardial infarction and cardiac arrest are needed to evaluate the impact of post-cardiac arrest arterial hyperoxaemia on cardiovascular outcomes.

Support of the circulation

Support of the circulation- Fluid therapy

ALS-PA-043A, ALS-PA-043C

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital) who have cardiovascular dysfunction, does the use of IV fluids, as opposed to standard care (or other IV fluids), improve outcome (e.g., survival)?

Consensus on science

No human studies that compare the use of IV fluids versus no IV fluids.

One small human study used IV fluid as part of EGDT in post-cardiac arrest syndrome and found an improvement in survival. Not statistically significant (LOE 5).

IV fluids were administered as part of a package of care (including PCI and therapeutic hypothermia) that improved survival with favorable neurological outcome in adult patients.)(LOE 5).

Rapid infusion of fluids (500–3000mL) to induce therapeutic hypothermia after sustained ROSC produced little evidence of harm. Six human studies (LOE 5).

Treatment recommendation

There is insufficient evidence to support or refute the routine use of IV fluids following sustained ROSC after cardiac arrest.

Rapid infusion of cold 0.9% saline or lactated Ringer's appears to be well tolerated when used to induce therapeutic hypothermia.

Based on the pathophysiology of post-cardiac arrest syndrome, it is reasonable to use IV fluids as part of a package of post-cardiac arrest care.

Support of the circulation- Haemodynamic optimisation

ALS-PA-056B

In adult patients (out-of-hospital and in-hospital) with ROSC after cardiac arrest, does early haemodynamic optimisation, as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

NoRCTs addressing early haemodynamic optimisation after cardiac arrest.

Only **one study** suggested that the introduction of haemodynamic optimisation (fluids, inotropic agents, intra-aortic balloon pump, and reperfusion) **as part of a bundle of interventions improved outcome** in comparison with historical controls (LOE 3). The independent effect of early haemodynamic optimisation was not assessed in this study.

A recent study that included early haemodynamic optimisation as part of a post-cardiac arrest treatment bundle was not powered to measure a survival benefit (LOE 3).

Treatment recommendation

Despite limited clinical data, the known pathophysiology of post-cardiac arrest syndrome provides a rationale for titrating haemodynamics to optimise organ perfusion.

Knowledge gaps

Clinical research is needed to define the optimal targets for haemodynamic optimisation and the best strategies to achieve these targets (fluids, vasopressors, inotropes, circulatory support, etc.)

Support of the circulation- Cardioactive drugs

ALS-PA-057A

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital) who have cardiovascular dysfunction, does the use of any specific cardioactive drugs, as opposed to standard care (or different cardioactive drugs), improve outcome (e.g., survival)?

Consensus on science

No clinical trials that have determined or compared the independent effect of vasopressor and/or inotrope use in the post-cardiac arrest period on cardiovascular dysfunction and/or survival to discharge.

Four clinical trials have suggested improved survival to discharge with vasopressor or inotropes, but have been confounded by multiple simultaneous treatments and/or they are underpowered for survival (LOE 3, LOE 4).

Six experimental studies showed improvement in postresuscitation cardiac dysfunction (left ventricular function) with the administration of cardioactive drugs, such as dobutamine or levosimendan, but none have shown that such improvement in function translates into improved survival (LOE 5).

Treatment recommendation

There is insufficient evidence to support or refute the routine use of vasopressors and/or inotropes for improving survival in adult patients with cardiovascular dysfunction after resuscitation from cardiac arrest.

Support of the circulation- Antiarrhythmic drugs

ALS-PA-058A, ALS-PA-058B

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of prophylactic antiarrhythmic drugs, as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

No controlled studies addressed specifically and directly the use of amiodarone, lidocaine, or β -blockers early or immediately after resuscitation from cardiac arrest.

One uncontrolled retrospective study did not demonstrate an improvement in six-month survival when amiodarone or lidocaine was given to patients resuscitated from VF or tachycardia during early (first 72 h) in-hospital postresuscitation care (LOE 4).

One single prospective nonrandomised study suggested that recurrent VF was reduced and long- and short term survival were improved in patients treated with β -blockers during electrical storm (LOE 5).

One study reported an incidence of approximately 5% for VF or VT in hospitalised post-cardiac arrest patients (LOE 4).

Five RCTs documented consistent improvement in all-cause mortality and sudden death when implantable cardioverter defibrillators were inserted as late, secondary prophylaxis compared with amiodarone or β -blocker administration to patients that survived VF or VT cardiac arrest (LOE 5).

Treatment recommendation

There is no evidence to support or refute continued administration of amiodarone or lidocaine in post-cardiac arrest patients after ROSC.

Support of the circulation-

Mechanical circulatory support

ALS-PA-060

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital) who have cardiovascular dysfunction, does the use of mechanical circulatory support, as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

There are no studies directly addressing the use of mechanical circulatory support in patients with sustained ROSC but who have cardiovascular dysfunction.

One human study showed that patients with severe cardiovascular dysfunction who were nonresponsive to standard care can be supported with mechanical chest compressions during PCI (LOE 4); No patient survived.

Five studies of nonarrested patients in cardiogenic shock or severe heart failure showed that left ventricular assist device or continuous aortic flow augmentation improved haemodynamics but not survival (LOE 5).

Two case series reported the use of the intraaortic balloon pump in patients with severe myocardial dysfunction after sustained ROSC, but the effect was impossible to isolate from other interventions (LOE 4).

Treatment recommendation

There is insufficient evidence to support or refute the use of mechanical circulatory support in post-cardiac arrest patients who have cardiovascular dysfunction.

Temperature Control



Temperature Control – Therapeutic hypothermia

ALS-PA-044

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does therapeutic hypothermia, compared with usual care, improve morbidity or mortality?

Consensus on science

Who to cool?.

All studies of post-cardiac arrest therapeutic hypothermia have included only patients in coma.

Improved neurological outcome at hospital discharge or at 6 months after hospital discharge in comatose patients after out-of-hospital **VF cardiac arrest**. One randomised trial (LOE 1) and a pseudo randomised trial (LOE 2)

Two studies with historical control groups (LOE 3) showed improvement in neurological outcome after therapeutic hypothermia for comatose survivors of **VF cardiac arrest**.

One systematic review demonstrated that conventional cooling methods were more likely to reach a best cerebral performance category score of 1 or 2 with a relative risk of 1.55 (99.5% CI 1.22 to 1.96) and more likely to survive to hospital discharge (relative risk of 1.35 95% CI 1.1 to 1.65) compared with standard postresuscitation care (LOE 1).

One small (n = 30) randomised trial showed **reduced plasma lactate** values and oxygen extraction ratios in a group (n = 16) of comatose survivors after cardiac arrest with asystole or PEA who were cooled with a cooling cap (LOE 1)

Temperature Control – Therapeutic hypothermia

ALS-PA-044

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does therapeutic hypothermia, compared with usual care, improve morbidity or mortality?

Consensus on science

Who to cool?.

One study with historical controls showed better neurological outcome after **VF cardiac arrest but no difference after cardiac arrest from other rhythms** (LOE 3).

Two nonrandomised studies with concurrent controls indicated possible benefit of hypothermia following cardiac arrest from other initial rhythms in- and out-of-hospital (LOE 2).

One registry study, which included almost 1000 cooled comatose patients following cardiac arrest **from all rhythms**, showed that **survival with good** outcome at 6 months was **56% after initial VT/VF, 21% after initial asystole, and 23% after initial PEA** (LOE 4).

Temperature Control – Therapeutic hypothermia

ALS-PA-044

How to cool?

Nineteen studies indicated that cooling could be initiated safely with **IV ice-cold fluids** (30mLkg⁻¹ of saline 0.9% or Ringer's lactate) (LOE 3)

Six studies indicated that cooling with **IV cold saline** can be initiated in the **prehospital** phase (LOE 1; LOE 2; LOE 3).

Thirteen studies documented the use of an **intravascular heat exchanger** to induce and maintain hypothermia (LOE 2; LOE 3; LOE 4).

Twelve studies documented the use of **ice packs and either water- or air-circulating blankets** to induce and maintain hypothermia (LOE 2; LOE 3; LOE 4).

Seven studies documented the use of **ice packs** (sometimes combined with wet towels) alone to induce and maintain hypothermia (LOE 2; LOE 3; LOE 4).

Four studies documented the use of **ice packs** alone to maintain hypothermia (LOE 3; LOE 4).

Seven studies documented the use of **cooling blankets or pads alone** to induce and maintain hypothermia (LOE 2; LOE 3; LOE 4).

Eight studies documented the use of **water-circulating, gel-coated pads** to induce and maintain, or just maintain, hypothermia (LOE 3; LOE 4).

One RCT (LOE 1) used a **cold-air tent** and another used a **cooling helmet** to induce and maintain hypothermia.

In one registry study, cooling was maintained with **ice packs** (17%), **air cooling** (8%), **circulating water blankets** (63%), **an intravascular cooling device** (16%), and **other methods** (8%) (LOE 4).

Temperature Control – Therapeutic hypothermia

ALS-PA-044

When to cool?.

One registry-based case series of 986 comatose post-cardiac arrest patients suggested **that time to initiation of cooling was not associated with improved neurological outcome** post discharge (median 90 min; interquartile range [IQR] 60 to 165 min) (LOE 4).

A case series of 49 consecutive comatose post-cardiac arrest patients who were intravascularly cooled after out-of-hospital cardiac arrest also documented that **time to target temperature was not an independent predictor of neurological outcome**(median 6.8 h; [IQR 4.5 to 9.2 h]) (LOE 4).

Safe with percutaneous coronary intervention?. Five studies indicated that the combination of therapeutic hypothermia and PCI is feasible and safe after cardiac arrest caused by acute myocardial infarction (LOE 3; LOE 4).

Temperature Control – Therapeutic hypothermia

ALS-PA-044

Treatment recommendation

Comatose adult patients with spontaneous circulation after out-of-hospital VF cardiac arrest should be cooled to 32–34 °C for 12–24 h.

Induced hypothermia might also benefit comatose adult patients with spontaneous circulation after out-of-hospital cardiac arrest from a non-shockable rhythm, or cardiac arrest in hospital.

Rapid infusion of ice-cold IV fluid 30mLkg⁻¹ or ice packs are feasible, safe, and simple methods for initially lowering core temperature up to 1.5 °C.

When IV fluids are used to induce hypothermia, additional cooling strategies will be required to maintain hypothermia.

Limited available evidence suggests that PCI during therapeutic hypothermia is feasible and safe and may be associated with improved outcome.

Knowledge gaps

Seizure control

ALS-PA-050A, ALS-PA-050B

Seizure control

ALS-PA-050A, ALS-PA-050B

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of seizure prophylaxis or effective seizure control, as opposed to standard care (no prophylaxis or ineffective seizure control), improve outcome (e.g., survival)?

Consensus on science

No controlled clinical trials directly addressed prophylactic treatment for seizures after cardiac arrest. Five studies documented a 3–44% incidence of seizures after sustained ROSC (LOE 4).

Two studies reported no difference in neurological outcome after use of single-dose diazepam or magnesium or both; or thiopental given after sustained ROSC (LOE 5).

No studies addressing prompt and aggressive treatment after the first seizure occurring after circulation was restored. Seizures in the postarrest period may be refractory to multiple medications (LOE 4).

No reported difference in the occurrence of seizures after sustained ROSC in patients treated with therapeutic hypothermia or with normothermia care (LOE 5).

Treatment recommendation

There are insufficient data to support or refute the use of specific antiseizure medication in the prevention or treatment of seizures after ROSC.

Blood glucose control

ALS-PA-045A, ALS-PA-045B

Blood glucose control

ALS-PA-045A, ALS-PA-045B

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of a specific strategy to manage blood glucose (e.g., target range), as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

Strict glucose compared with moderate glucose control in patients resuscitated from prehospital cardiac arrest with VF found **no survival benefit with strict glucose control** (LOE 1). One human prospective randomised interventional study

Post-cardiac arrest patients suggested an association of **higher glucose levels with increased mortality** and worse neurological outcomes.(LOE 4). Five retrospective studies

A randomised trial of intensive glucose control versus conventional glucose control in the largest number of ICU patients to date reported **increased mortality in patients treated with intensive glucose control** (LOE 5).

Two meta-analyses of studies of tight glucose control versus conventional glucose control in critically ill patients showed **no significant difference in mortality** but found tight glucose control was associated with a significantly **increased risk of hypoglycaemia** (LOE 5).

Treatment recommendation

Strategies to treat hyperglycaemia >10mmolL⁻¹ (>180mgdL⁻¹) should be considered in adult patients with sustained ROSC after cardiac arrest.

Hypoglycaemia should be avoided.

Steroid therapy

ALS-PA-048A

Steroid therapy

ALS-PA-048A

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does treatment with corticosteroids, as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

Two observational studies (LOE 2) and two animal studies (LOE 5) failed to demonstrate any benefit or harm from the use of steroids after successful resuscitation from cardiac arrest.

One small, single-centre randomised placebo-controlled trial showed benefit from the use of a package of care consisting of vasopressin and dexamethasone in addition to adrenaline during resuscitation, combined with the treatment of post-cardiac arrest shock with hydrocortisone in the study group (LOE 1). Complex design

Treatment recommendation

There is insufficient evidence to support or refute the use of corticosteroids for patients with ROSC following cardiac arrest.

Knowledge gaps

It is important to determine the incidence of adrenal insufficiency after sustained ROSC following cardiac arrest. Clinical trials are needed to determine the effect of exogenous steroids administered after cardiac arrest.

Haemofiltration

ALS-PA-054A

Haemofiltration

ALS-PA-054A

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of haemofiltration as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

One RCT demonstrated no difference in survival or neurological outcome between groups treated with high-volume haemofiltration (200mLkg⁻¹ h⁻¹ for 8 h) with or without mild hypothermia, and control group without haemofiltration (LOE 1).

The combined haemofiltration-only and haemofiltrationplus- hypothermia groups had increased survival at 6 months after cardiac arrest when compared to controls. One study suggested improved survival and neurological outcome in patients treated with high-volume haemofiltration after resuscitation from cardiac arrest (LOE 2).

Treatment recommendation

There is insufficient evidence to support or refute the use of haemofiltration in patients with sustained ROSC after cardiac arrest.

Neuroprotective therapy

ALS-PA-055A, ALS-PA-055C

Neuroprotective therapy

ALS-PA-055A, ALS-PA-055C

In adult patients with ROSC after cardiac arrest (out-of-hospital or in-hospital), does the use of neuroprotective drugs, as opposed to standard care, improve outcome (e.g., survival)?

Consensus on science

One small pilot study in witnessed, out-of-hospital cardiac arrests of presumed cardiac aetiology **showed improved survival at 3 months** when therapeutic hypothermia (35 °C) and the oral administration of coenzyme Q10 was compared with therapeutic hypothermia alone; however, there was **no difference in neurologically intact survival** (LOE 1).

Four RCTs (LOE 1) using nimodipine, lidoflazine or diazepam in out-of-hospital cardiac arrest showed **no benefits** from any of the drugs when compared with standard care.

Two RCTs (LOE 1) using thiopental or nimodipine in out-of-hospital cardiac arrest - **No benefits** when compared with standard care.

A retrospective analysis using glucocorticoids in out of- hospital cardiac arrest - **No benefits** when compared with standard care (LOE 2).

Treatment recommendation

The value of routine use of coenzyme Q10 in patients treated with hypothermia is not certain.

There are insufficient data to recommend for or against the use of neuroprotective drugs (thiopental, glucocorticoids, nimodipine, lidoflazine, or diazepam) alone or as an adjunct to therapeutic hypothermia in comatose cardiac arrest after ROSC.

Emerging pharmaceutical therapies in cardiopulmonary resuscitation and post-resuscitation syndrome

A.F. Charalampopoulos, N.I. Nikolaou / Resuscitation 82 (2011) 371–377

Table 1
Effects of drugs tested during cardiac arrest and post-resuscitation syndrome.

Studies	Observed effects	References
Vasopressin Humans, RCT	No overall benefit in survival or mental performance of survivors of in-hospital and OHCA in comparison to adrenaline. Increased survival to hospital discharge in OHCA patients with asystole.	6,8,11
Corticosteroids Humans, NRT	Increased ROSC but not short-term survival.	16
β -Blockers Animals	Evidence for reduced VF inducibility, reduction of number of shocks needed to defibrillate, rendering resuscitation more effective, improving ROSC and short-term survival. Amelioration of myocardial dysfunction post-ROSC.	21–23
Humans, retrospective	Survival benefit if β -blockers administered before CA or post-ROSC.	29
Sodium–hydrogen exchanger inhibitors Animals/rat-heart model	Decrease in peri-arrest arrhythmias, amelioration of ischaemic contracture, improvement of myocardial performance post-ROSC.	37,38
Humans, RCT	Cariporide decreased MI rates but increased cerebrovascular events and short term mortality after CABG.	39,40
Erythropoietin Animals	Increase in coronary and mean aortic perfusion pressure and reduction of adrenaline dose during CPR. Improvement of cardiac performance and short-term survival after CA. Reduced ischaemic damage of brain.	42–45
Humans NRTs	Increased ROSC and survival to hospital discharge. Trend towards better neurological recovery in OHCA patients	46–47
Inotropes Animals	Evidence of increased coronary perfusion pressure, ROSC rate, and brain regional oxygen saturation during CPR with levosimendan. Levosimendan may be more effective than dobutamine in improving myocardial function post ROSC. Levosimendan may also improve post-ROSC survival with less brain damage.	50–54
δ -Opioids agonists Animals/rat-heart model	Reduction of myocardial metabolism during ischaemia, amelioration of myocardial dysfunction and increased survival post-ROSC.	56–58
Thrombolysis Humans	No treatment benefit from routine use in OHCA patients. May be useful in selected patients with STEMI and pulmonary embolism	61–63
Neurotensin Animals	Prolonged decrease of body temperature.	64
ATP-sensitive potassium channel activators. Animals	Cardio- and neuroprotection.	69
Humans RCT	Supplementing blood cardioplegia with diazoxide safely improves myocardial protection during cardiac surgery.	70

RCT: randomized controlled trials, CA: cardiac arrest, OHCA: out- of -hospital cardiac arrest, NRT: non randomised trial, ROSC: return of spontaneous circulation, CABG:

Post Resuscitation Care - Summary

Goal-Directed Strategies for Improving Outcome

- Hypothermia
- Coronary Reperfusion
- Normal Oxygenation
- Normal Ventilation
- Hemodynamic Optimization
- Moderate Glycemic Control



*National Institute for
Health and Clinical Excellence*

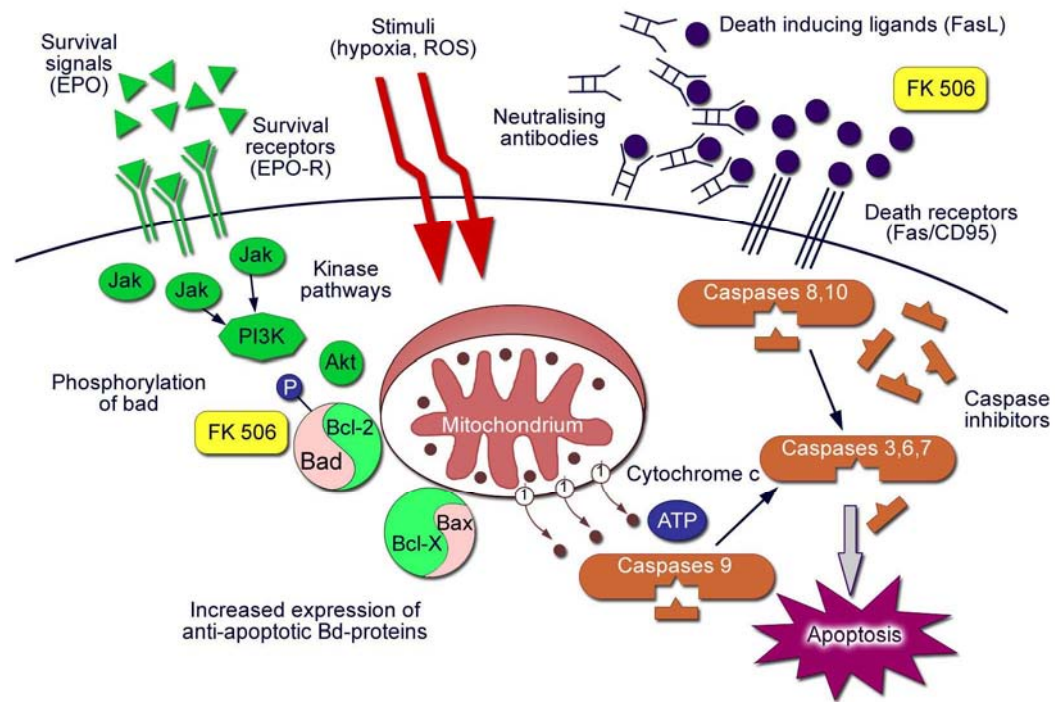
Therapeutic hypothermia following cardiac arrest

- **Description**
- People who have a cardiac arrest can sometimes develop neurological problems because of the lack of oxygen to the brain.
- In this procedure, after resuscitation a cooling device is used to reduce the person's core temperature to 32–34°C to reduce the risk of developing neurological problems.

Guidance 1.1

Current evidence on the safety and efficacy of therapeutic hypothermia following cardiac arrest is adequate to support the use of this procedure

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

V. A. NEGOVSKY

*Laboratory of Experimental Resuscitation, Academy of Medical Sciences of the U.S.S.R.,
9, October 25th Street, Moscow, U.S.S.R.*

In the first stages of the development of the science of resuscitation, ‘reanimatology’, research workers have been limited mainly to the study of the pathology of death, and to the elaboration of a series of techniques of resuscitation. We now have at our disposal some knowledge of the process of disintegration of physiological functions during the dying of an organism, and of their restoration during resuscitation. We also have at our disposal a number of methods available to a large circle of practising doctors. Extensive experimental studies and clinical findings have clearly proved that after the first step in resuscitation when heart function and respiration have been restored, the second step in resuscitation arises—the more complicated problems of treating the after-effects of a general hypoxia. There are characteristic disturbances in the functions of the central nervous system and internal organs, in metabolism and in homeostasis among other systems.

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 changes occur during clinical death and after resuscitation.



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Part 1: Executive summary

2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations[☆]

Jerry P. Nolan (Co-chair)^{*,1}, Mary Fran Hazinski (Co-chair)¹, John E. Billi, Bernd W. Boettiger, Leo Bossaert, Allan R. de Caen, Charles D. Deakin, Saul Drajer, Brian Eigel, Robert W. Hickey, Ian Jacobs, Monica E. Kleinman, Walter Kloeck, Rudolph W. Koster, Swee Han Lim, Mary E. Mancini, William H. Montgomery, Peter T. Morley, Laurie J. Morrison, Vinay M. Nadkarni, Robert E. O'Connor, Kazuo Okada, Jeffrey M. Perlman, Michael R. Sayre, Michael Shuster, Jasmeet Soar, Kjetil Sunde, Andrew H. Travers, Jonathan Wyllie, David Zideman
