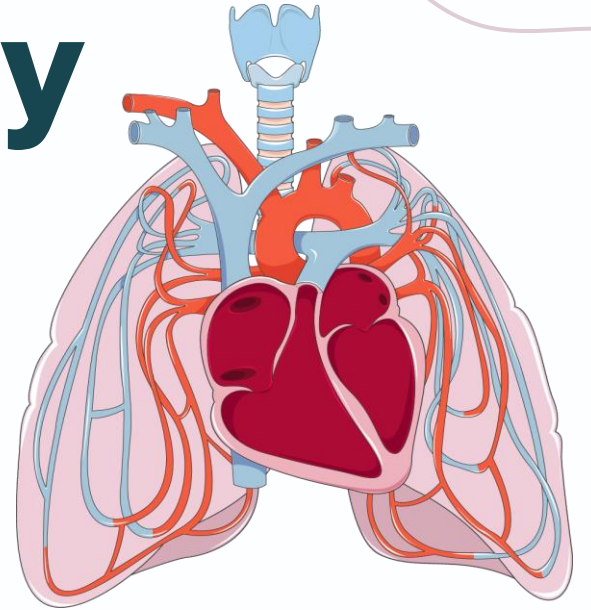


The background features decorative wavy lines in teal and purple colors, framing the central text.

In the name of GOD
The Compassionate
The Merciful

CardioPulmonary Resuscitation

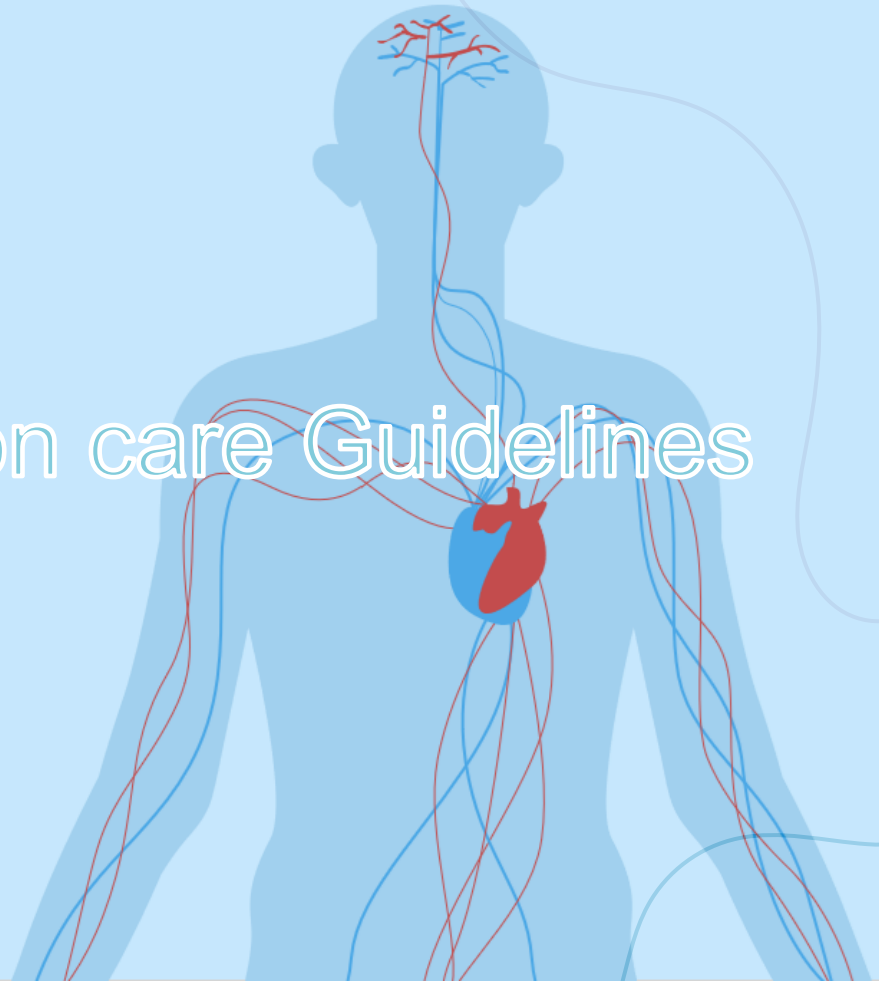




Dr. Navid Shafigh

Assistant Professor at Shahid Beheshti University of Medical Sciences

Post-resuscitation care Guidelines



Post-resuscitation care

1

Normal cerebral
function

2

Stable cardiac rytme

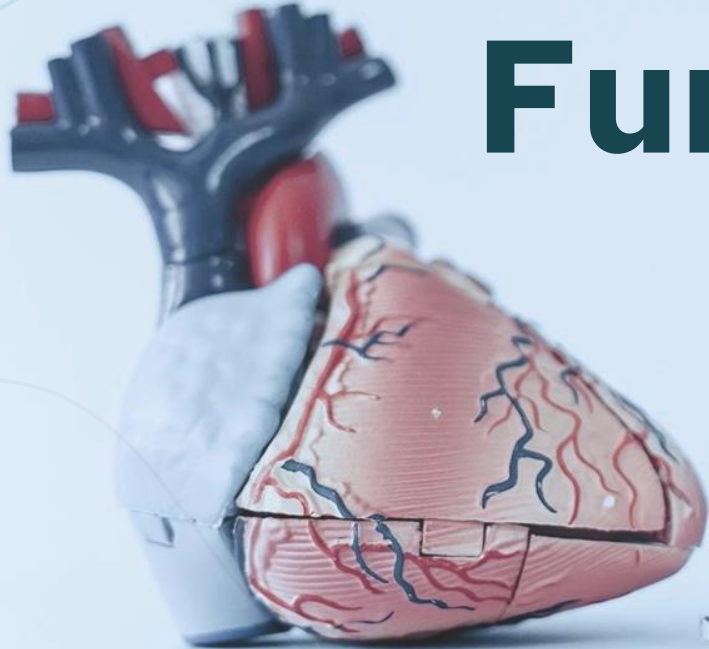
3

Adaquate organ
perfusion

4

Quality of life

Optimising Organ Function



Heart



Ischemia-reperfusion injury

- Reversible myocardial dysfunction for 2-3 days
- Arrhythmias



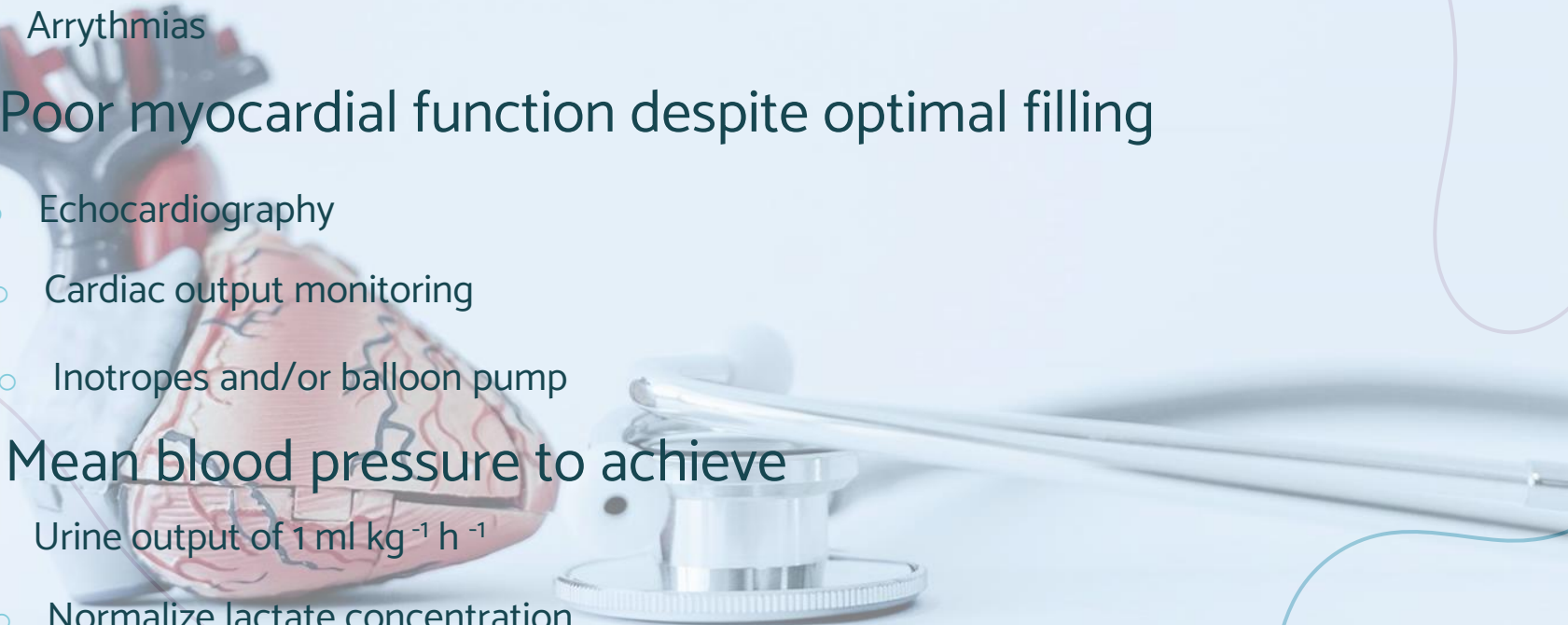
Poor myocardial function despite optimal filling

- Echocardiography
- Cardiac output monitoring
- Inotropes and/or balloon pump



Mean blood pressure to achieve

- Urine output of $1 \text{ ml kg}^{-1} \text{ h}^{-1}$
- Normalize lactate concentration



Disability

Assessment

AVPU

Pupils equal and reacting

Blood glucose 5.9 mmol

No limb movement

No seizures

Treatment

Document arrest accurately

Monitor blood glucose and maintain normal

Consider

Neurological assessment:

- ✓ Glasgow Coma Scale score
- ✓ posture / seizure / limb movement

Targeted Temperature Management (TTM)

Post-cardiac arrest syndrome

- Post-cardiac arrest brain injury
coma, seizures, myoclonus
- Post-cardiac arrest myocardial dysfunction
- Systemic ischemia-reperfusion response
'sepsis-like' syndrome
- Persistence of precipitating pathology



- ✓ Alignment with European Society of Cardiology guidelines for the indications for immediate coronary angiography in post-resuscitation patients without ST-elevation on their 12-lead ECG.
- ✓ Following return of spontaneous circulation (ROSC), aim to maintain a mean arterial blood pressure of > 65 mmHg. Over this threshold optimal blood pressure targets are likely to need to be optimized.
- ✓ Levetiracetam and sodium valproate are preferred instead of phenytoin for the treatment of seizures.

- ✓ Targeted temperature management (TTM) is recommended for adults after either out-of-hospital or in-hospital cardiac arrest (OHCA or IHCA) with any initial rhythm who remain unresponsive after ROSC.
- ✓ Maintain a target temperature at a constant value between 32°C and 36°C for at least 24 h.
- ✓ **Avoid fever** (> 37.7°C) for at least 72 h after ROSC in patients who remain in coma.

Adult Immediate Post-Cardiac Arrest Care Algorithm

VENTILATION/OXYGENATION:

Avoid excessive ventilation. Start at 10 to 12 breaths per minute and titrate to target PETCO₂ of 35 to 40 mm Hg.

DOSES/DETAILS

IV Bolus:

1 to 2 liters normal saline or Lactated Ringer's. If inducing hypothermia, consider 4°C fluid.

Epinephrine IV Infusion:

0.1 to 0.5 mcg/kg per minute

Dopamine IV Infusion:

5 to 10 mcg/kg per minute

Norepinephrine IV Infusion:

0.1 to 0.5 mcg/kg per minute

REVERSIBLE CAUSES:

- Hypovolemia
- Hypoxia
- H⁺ (acidosis)
- Hypothermia
- Hypo-/hyperkalemia
- Tamponade, cardiac
- Toxins
- Tension pneumothorax
- Thrombosis, pulmonary

RETURN OF
SPONTANEOUS
CIRCULATION

Optimize Ventilation and Oxygenation

- Maintain O₂ saturation ≥94%
- Consider advanced airway and waveform capnography
- Do not hyperventilate

Treat hypotension (SBP <90 mm Hg)

- IV/IO fluid bolus
- Vasopressor infusion
- Consider treatable causes
- Assess 12-lead ECG

FOLLOW COMMANDS?

YES

NO

STEMI OR HIGH
SUSPICION OF AMI

CONSIDER
INDUCED
HYPOTHERMIA

CORONARY
REPERFUSION

ADVANCED CRITICAL CARE

NO

YES

The multimodal prognostication guidelines have been updated.

In a comatose patient with a Glasgow Motor Score of $M \leq 3$ at ≥ 72 h from ROSC, in the absence of confounders, poor outcome is likely when two or more of the following predictors are present:

1. No pupillary and corneal reflexes at ≥ 72 h
2. Bilaterally absent N20 SSEP wave at ≥ 24 h
3. Highly malignant EEG (suppressed background or burst suppression) at ≥ 24 h
4. NSE >60 mcg L-1 at 48 h and/or 72 h
5. Status myoclonus ≤ 72 h
6. A diffuse and extensive anoxic injury on brain CT/MRI.
7. Greater emphasis is placed on screening cardiac arrest survivors for physical, cognitive and emotional problems and, where indicated, referring for rehabilitation.

Key Learning Points

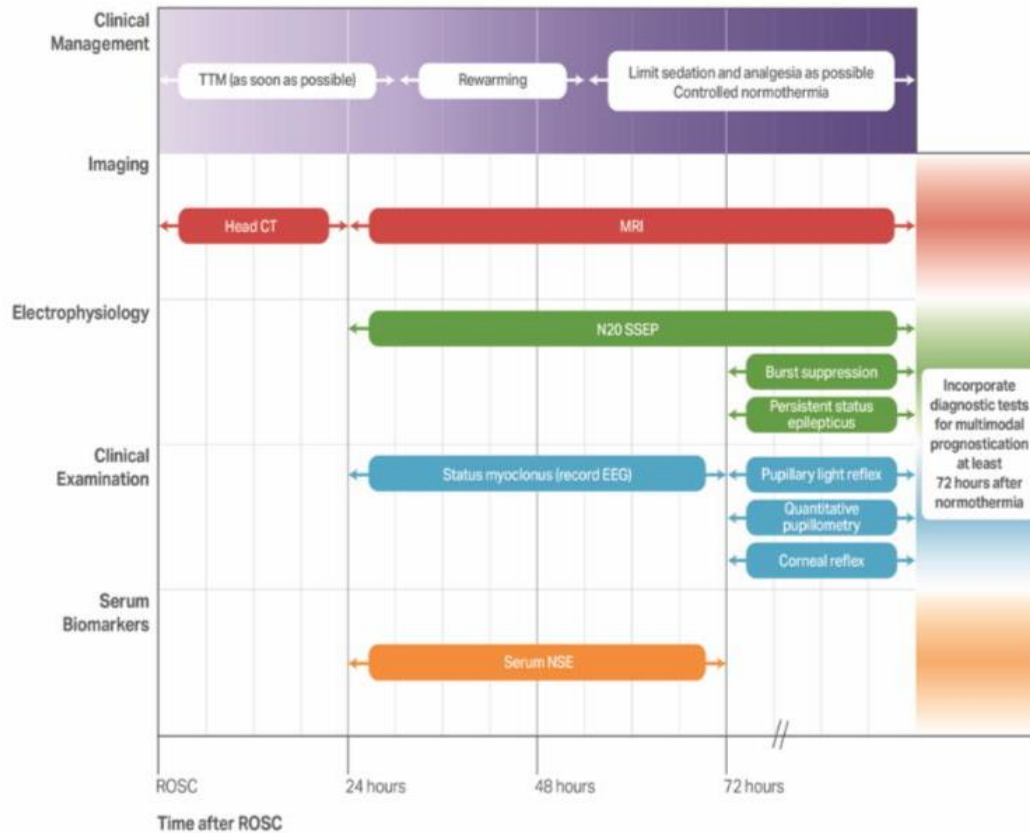
- O₂ sats of 94-98% and normocapnia should be aimed for. (Sackett strength D)A
MAP of 65-100 should be aimed for. (Sackett strength D)
- Tight glycemetic control is not recommended, as the patient is then at higher risk of missed hypoglycemia. (Sackett B)
 - Hyperglycemia must also be avoided. (Sackett B2a)
- Clearance of lactate demonstrates physiological response to therapy and is an independent predictor of mortality. (Sackett B)
 - CTPA or echo will reliably diagnose massive PE. (Sackett B)

Key Learning Points

- ✓ There is no role for routine thrombolysis following out of hospital cardiac arrest. (Sackett A1b)
- ✓ Selecting and maintaining a constant, target temperature between 32°C and 36°C for at least 24 hours, in patients with out-of-hospital or in-hospital cardiac arrests who are unresponsive after ROSC, is recommended. Hyperthermia (≥ 37.6 degree centigrade) should be avoided .
- ✓ Absent pupillary or corneal reflexes, absent or extensor motor responses to pain are the best predictors of poor prognosis, but only at 72 hours after cardiac arrest.
- ✓ Myoclonus status epilepticus in the first day after cardiac arrest predicts poor prognosis. (Sackett B2a)

Key Learning Points

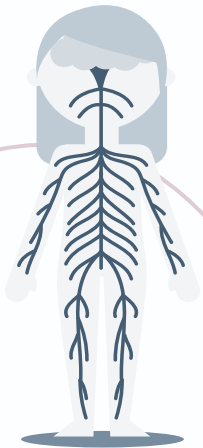
- ✓ Retention of any neurological function (e.g. respiratory effort, coughing/swallowing, pupillary light reflex) after cardiac arrest is predictive of a good functional outcome. (Sackett B2b) An arterial lactate must be more than 16 to be 100% specific for poor neurological outcome (Sackett B2b)
- ✓ Generally, post ROSC comatose patients without significant pre-arrest co-morbidities should be taken to the ICU for supportive care and their individual prognosis decided later by the intensive care team.
- ✓ Where there is no capacity, attorney, or advance decision, the final decision as to withdraw / withhold treatment lies with the most senior clinician caring for the patient, and should be a best interests decision made with the input of those closest to the patient.



NEUROLOGICAL CARE

Neurologic assessment is key, especially when withdrawing care (i.e., brain death) to decrease false positive rates.

Specialty consultation should be obtained to monitor neurologic signs and symptoms throughout the post-resuscitation period.



REVERSIBLE CAUSES OF CARDIAC ARREST

THE H'S	THE T'S
<i>Hypovolemia</i>	<i>Tension pneumothorax</i>
<i>Hypoxia</i>	<i>Tamponade</i>
<i>H⁺ (acidosis)</i>	<i>Toxins</i>
<i>Hypo/Hyperkalemia</i>	<i>Thrombosis (coronary)</i>
<i>Hypoglycemia</i>	<i>Thrombosis (pulmonary)</i>
<i>Hypothermia</i>	<i>Trauma (unrecognized)</i>

ACUTE STROKE

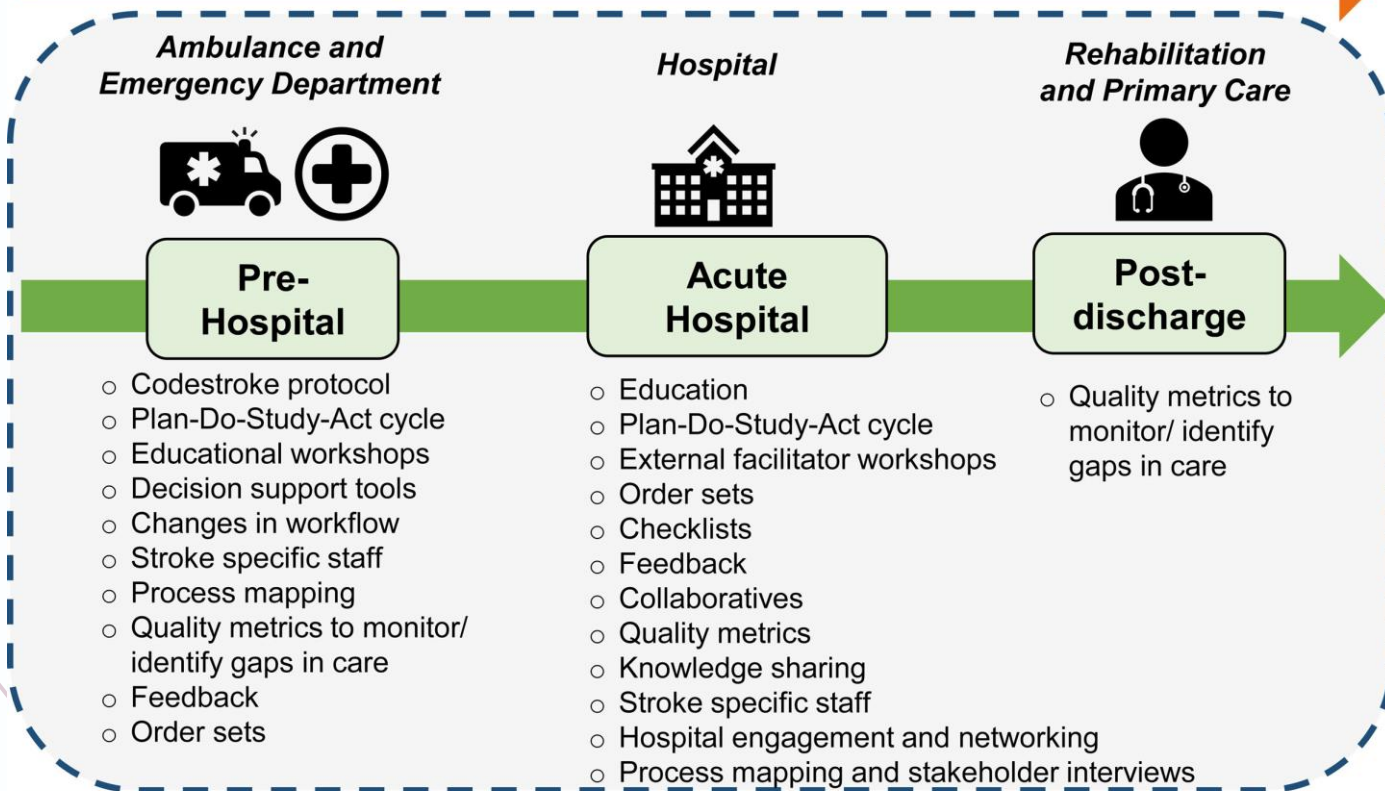
Outcomes for individuals with stroke have improved significantly due to the implementation of Acute Stroke System of Care.

The community is better equipped to recognize stroke as a “brain attack,” and there is greater awareness of the importance of medical care within one hour of symptom onset.

Likewise, EMS systems have been enhanced to transport individuals to regional stroke care centers that are equipped to administer fibrinolytics.

Quality Improvement Initiatives to Improve Stroke Care

Stroke systems of care (local, regional, or national)



Economic Evaluations of Quality Improvement Projects

GOALS OF ACUTE ISCHEMIC STROKE CARE

The overall goal of stroke care is to minimize brain injury and optimize the individual's recovery.

Preferential transport to stroke-capable centers has been shown to improve outcomes. Stroke centers are equipped with resources often not available at smaller community hospitals.

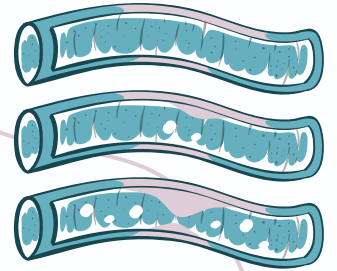
The presence of specialists, including neurologists and stroke care specialists, multidisciplinary teams experienced in stroke care, advanced imaging modalities, and other therapeutic options make transport to stroke centers the most suitable option.

The goal of the stroke team, emergency physician, or other experts should be to assess the individual with suspected stroke within ten minutes.

The 8 D's of Stroke Care

DETECTION	<i>Rapid recognition of stroke systems</i>
DISPATCH	<i>Early activation and dispatch of EMS by 911</i>
DELIVERY	<i>Rapid EMS identification, management, and transport</i>
DOOR	<i>Transport to stroke center</i>
DATA	<i>Rapid triage, evaluation, and management in ED</i>
DECISION	<i>Stroke expertise and therapy selection</i>
DRUG	<i>Fibrinolytic therapy, intra-arterial strategies</i>
DISPOSITION	<i>Rapid admission to the stroke unit or critical care unit</i>

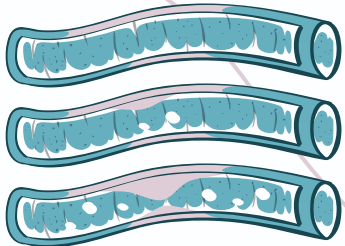
ACUTE CORONARY SYNDROME



For individuals with acute coronary syndrome (ACS), proper care starts during the call to EMS.

First responders must be aware of and look for signs of ACS. Quick diagnosis and treatment yield the best chance to preserve healthy heart tissue.

It is very important that health care providers recognize individuals with potential ACS in order to initiate evaluation, appropriate triage, and timely management.



GOALS OF ACS TREATMENT

Early EMS communication allows for preparation of emergency department personnel and cardiac catheterization ED EVIDENCE BASED CARE REPERFUSION WITH PCI OR FIBRINOLYTICS lab and staff.

Once the ACS patient arrives at the receiving facility, established protocols should direct care. The shorter the time is until reperfusion, the greater the amount of heart tissue that can be saved, and the more optimal the overall outcome.

QUALITY POST-MI CARE REDUCE MYOCARDIAL NECROSIS TO PRESERVE HEART FUNCTION TREAT ACS COMPLICATIONS (VF, VT, SHOCK) Major adverse cardiac events (MACE) includes death and non-fatal myocardial infarction.

Life-threatening complications of ACS include ventricular fibrillation, pulseless ventricular tachycardia

GOALS OF ACS TREATMENT

- ✓ Bradyarrhythmia, cardiogenic shock, and pulmonary edema. EMS should have the capacity to perform ECGs on scene and on the way to the hospital. The receiving hospital should be made aware of possible ACS, especially ST elevation myocardial infarction elevation (STEMI) and non-ST-elevation myocardial infarction (NSTEMI).
- ✓ BLOOD PRESSURE SUPPORT AND VASOPRESSORS
- ✓ Consider blood pressure support in any individual with systolic blood pressure less
- ✓ than 90 mm Hg or mean arterial pressure (MAP) less than 65.
- ✓ Unless contraindicated, 1 to 2 liters of IV saline or Lactated Ringer's is the first intervention.

GOALS OF ACS TREATMENT

- ✓ When blood pressure is very low, consider vasopressors (commonly referred to as “pressors”)
- ✓ If no advanced airway, 30:2 compression to ventilation ratio
- ✓ Epinephrine is the pressor of choice for individuals who are not in cardiac arrest.- Dopamine, phenylephrine, and methoxamine are alternatives to epinephrine.- Norepinephrine is generally reserved for severe hypotension or as a last-line agent.
- ✓ Titrate the infusion rate to maintain the desired blood pressure.

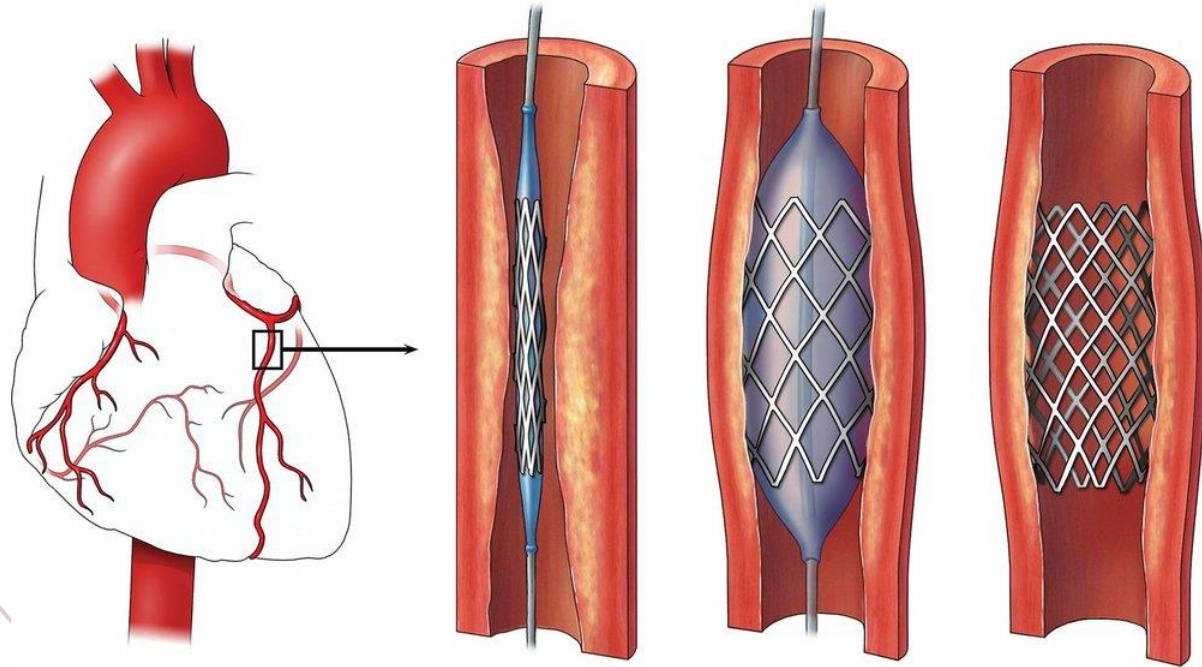
PERCUTANEOUS CORONARY INTERVENTION

Percutaneous coronary intervention (PCI) is preferred over thrombolytics.

Individual should be taken by EMS directly to a hospital that performs PCI.

If the individual is delivered to a center that only delivers thrombolytics, they should be transferred to a center that offers PCI if time permits.

PERCUTANEOUS CORONARY INTERVENTION



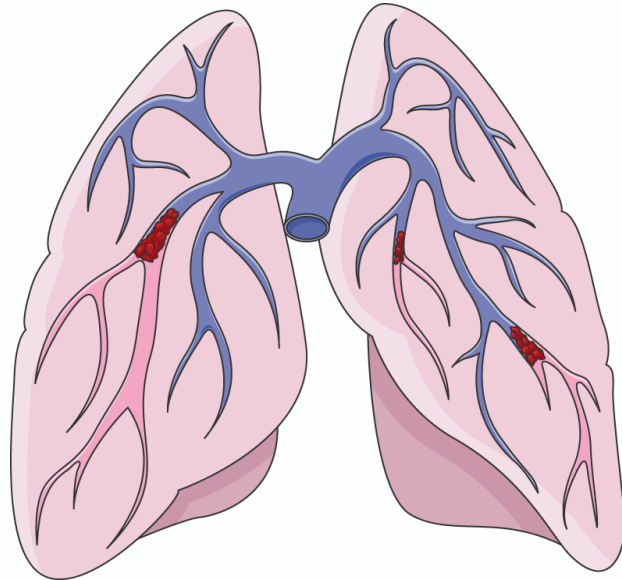
OPTIMIZATION OF HEMODYNAMICS AND VENTILATION

100% oxygen is acceptable for early intervention but not for extended periods of time. Oxygen should be titrated, so that individual's pulse oximetry is greater than 94% to avoid oxygen toxicity.

Do not over ventilate to avoid potential adverse hemodynamic effects.

Ventilation rates of 10 to 12 breaths per minute to achieve $ETCO_2$ at 35 to 40 mmHg. IV fluids and vasoactive medications should be titrated for hemodynamic stability.

New technologies in resuscitation





Your partner in life support

The LUCAS device is an easy-to-use mechanical chest compression device that helps lifesaving teams around the world deliver high-quality, guidelines-consistent chest compressions to sudden cardiac arrest patients; in the field, on the move and in the hospital.

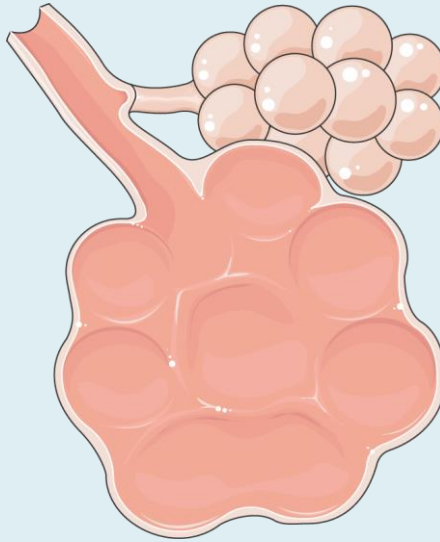
High-Perfusion CPR Technology Dramatically Improves Cardiac Arrest Survival

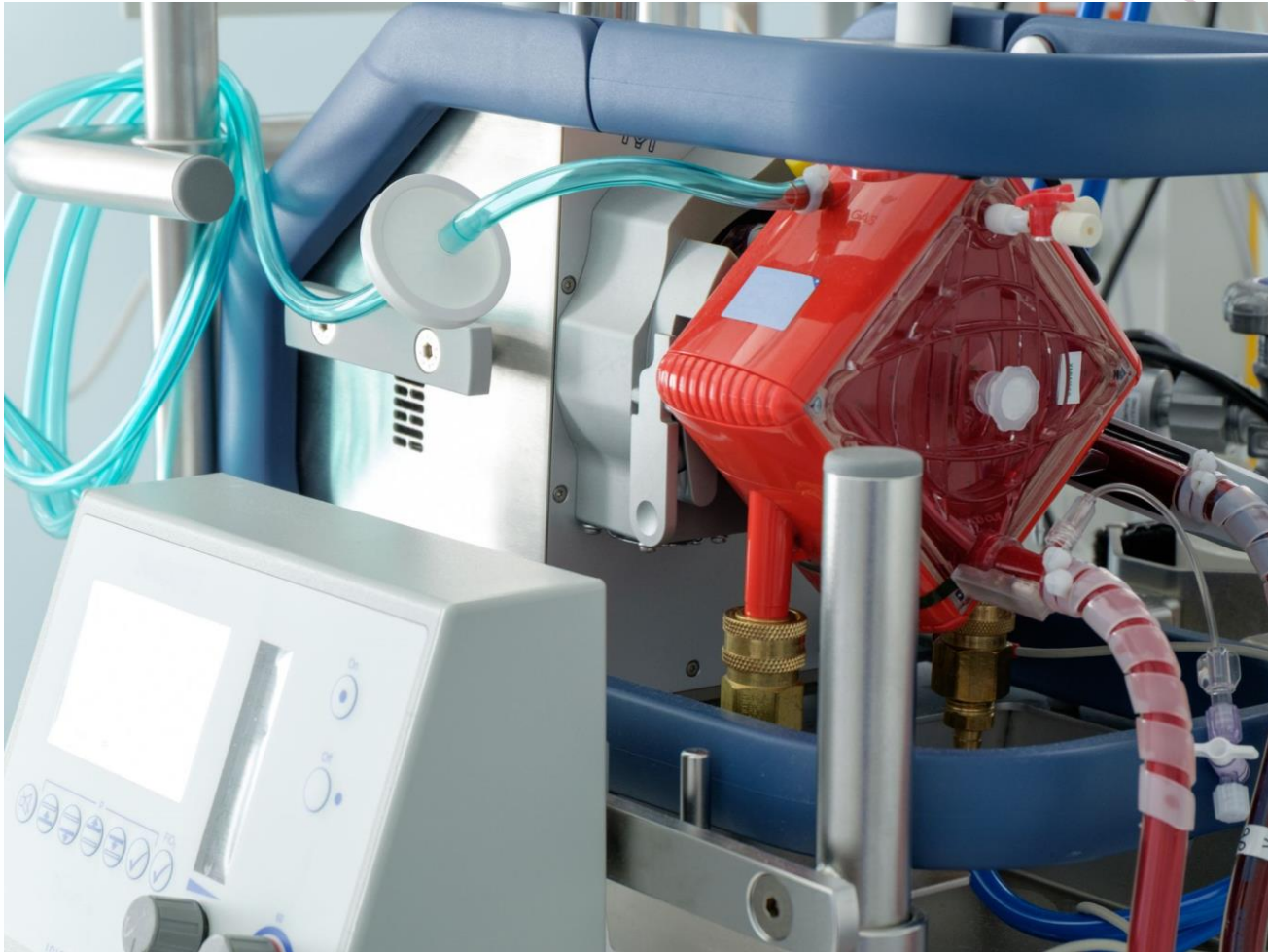




ECMO

Extracorporeal membrane oxygenation in cardiac arrest





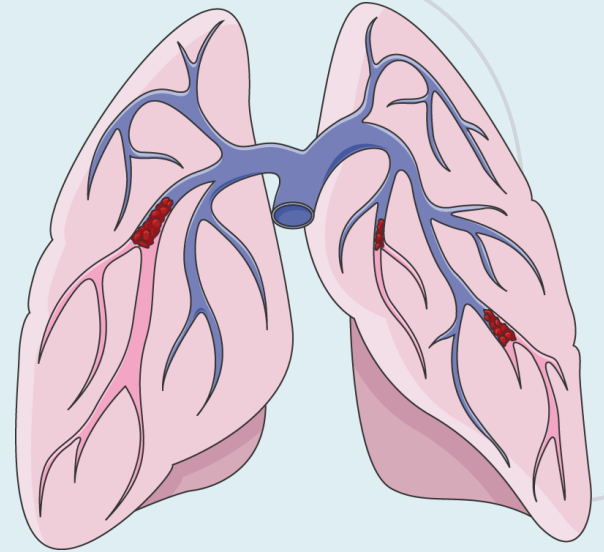
Extracorporeal membrane oxygenation in cardiac arrest

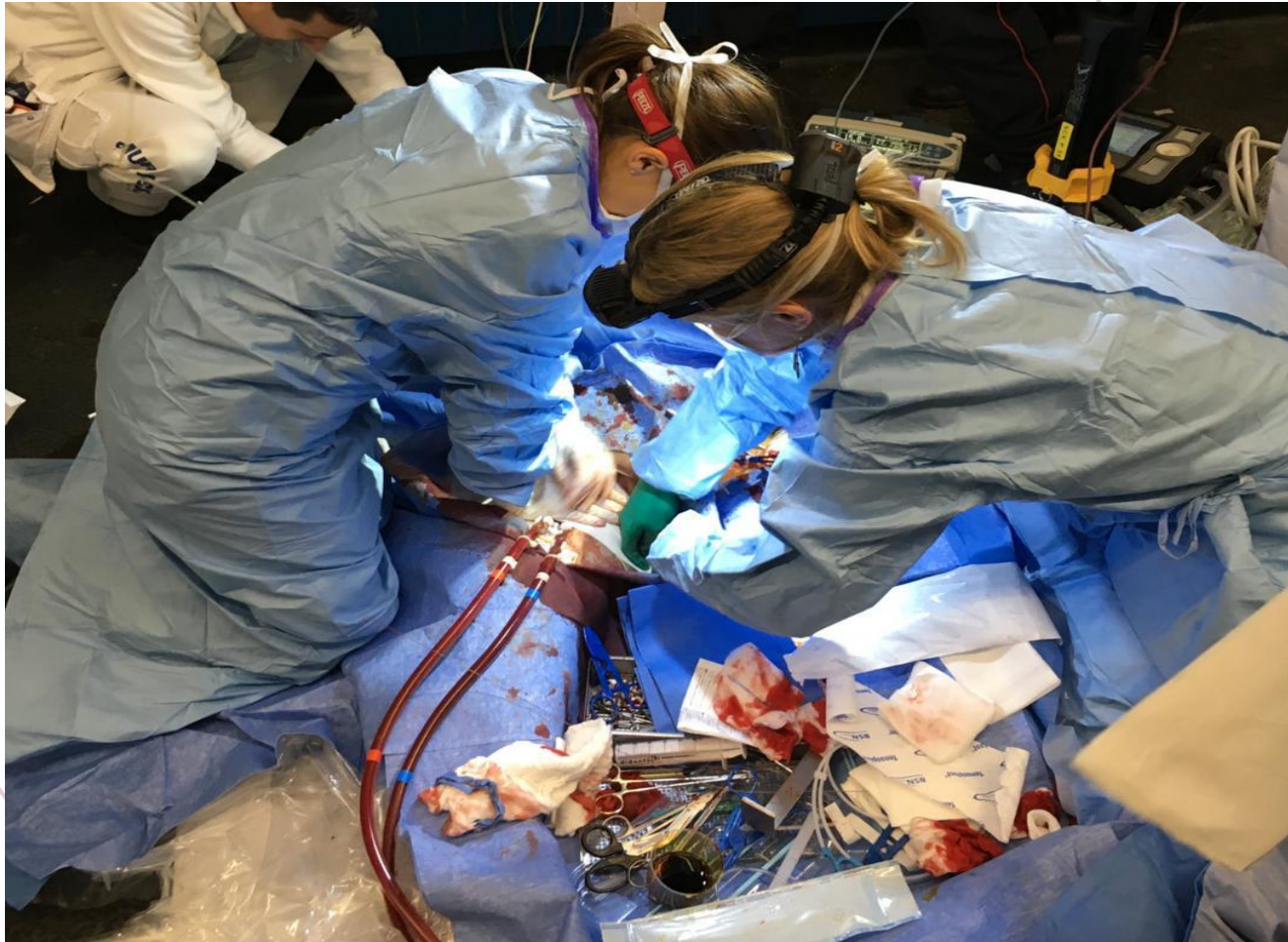
Boon Kiat Kenneth Tan, MBBS, MCEM

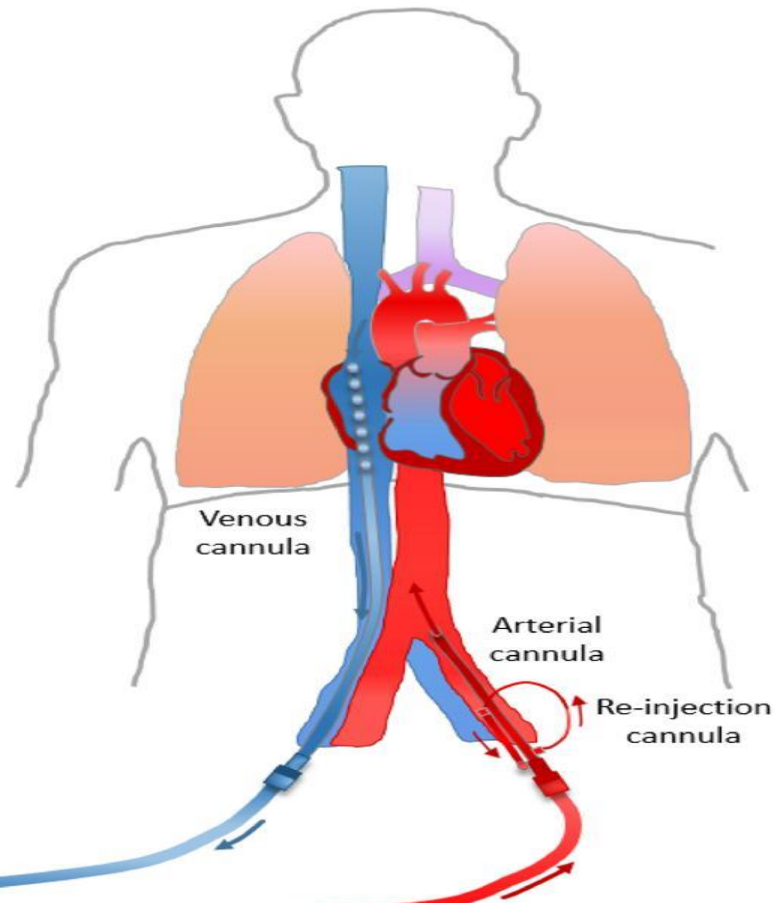
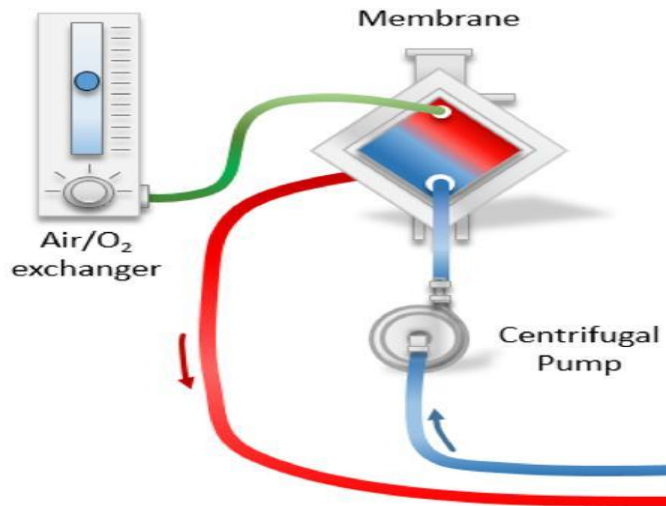
[Additional article information](#)

CONCLUSION

- ✓ ECPR is only one component of the post-cardiac arrest regimen (which includes therapeutic hypothermia, early reperfusion, treatment of root cause, tight glycemic control and low tidal volume ventilation) and functions as a bridge to definitive therapy. As the CHEER trial and studies on therapeutic hypothermia have aptly demonstrated, therapeutic hypothermia and early reperfusion can give excellent patient outcomes. It is thus important to complement ECPR with good post-cardiac arrest care. ECPR is still an evolving field; as more studies are conducted and clinical experience improves, there is hope that patients who would otherwise die will be given a second chance at life.







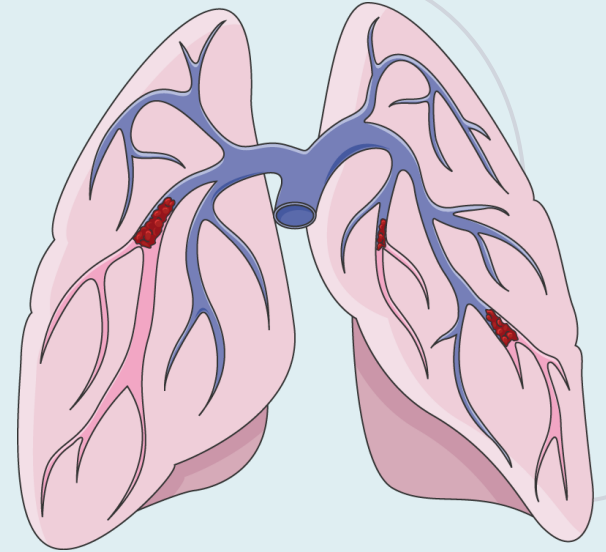
Rambam Maimonides Medical Journal

Rambam Health Care Campus

CPR and ECMO: The Next Frontier

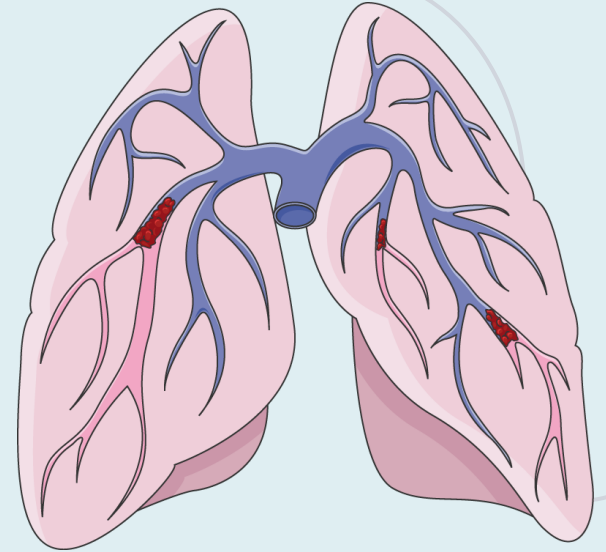
Daniel I. Ambinder, M.D., Matt T. Oberdier, Ph.D.,
[...], and Henry R. Halperin, M.D., M.A.

- ✓ There are two common configurations of ECMO which enable customization of support for each patient. In general, for respiratory support during which oxygenation is affected, such as with severe refractory acute respiratory distress syndrome, veno-venous ECMO (VV-ECMO) can be used to remove deoxygenated blood from the venous system, pass it through an oxygenator, then return the oxygenated blood into the circulation via the venous system. However, in cases of cardiac failure during which oxygenated blood is not adequately circulated throughout the body, additional support can be obtained through veno-arterial ECMO (VA-ECMO)



CONCLUSION

- ✓ There are abundant opportunities to improve future sudden cardiac arrest outcomes via progress in research and technology of ECMO, and through the synergy of CPR, ECMO, and therapeutic hypothermia. These advances will ultimately result in ECMO being more commonly utilized for sudden cardiac arrest at a larger distribution of clinical sites by a more broadly trained pool of providers. Organ transplant recipients may also benefit from expanded ECMO application that includes sudden cardiac arrest.



THERAPEUTIC HYPOTHERMIA

- Recommended for comatose individuals with return of spontaneous circulation after a cardiac arrest event.
- Individuals should be cooled to 89.6 to 93.2 degrees F (32 to 36 degrees C) for at least 24 hours.



B Leak Proof Technology

A Patented 3-layer Construction

C
Simple, Safe &
Effective

- Therapeutic hypothermia is a type of treatment. It's sometimes used for people who have a cardiac arrest. Cardiac arrest happens when the heart suddenly stops beating. Once the heart starts beating again, healthcare providers use cooling devices to lower your body temperature for a short time. It's lowered to around 89°F to 93°F (32°C to 34°C). The treatment usually lasts about 24 hours.

- The mechanisms for TTM is controversial, these are non-mutually exclusive possibilities: avoidance of hyperthermia (decreased metabolic demand and fever-related tissue injury)reduction in metabolic demand (through prevention of fever, seizure control, cooling, sedation and neuromuscular blockade)improved overall care (focusing the coordinated efforts of an expert team with close monitoring and prioritization of therapies on a critically ill patient)reduction in ischemic-reperfusion injury (including effects on excitotoxicity, neuroinflammation, apoptosis, free radical production, seizure activity, blood-brain barrier disruption, blood vessel leakage and cerebral thermopooling)

- The medical team may start the hypothermia within 4 to 6 hours after the cardiac arrest. A healthcare provider will give you medicine to help you relax (sedative). It makes you sleep and keeps you from shivering. You won't remember anything about the procedure afterward. You may also get another medicine to keep you from moving. Your heart rate, blood pressure, and other vital signs will be closely watched. Providers use special thermometers to check your internal temperature. The provider may use cooling blankets, ice packs, or cooling pads to bring the body temperature down.

INDICATIONS AND CONTRA-INDICATIONS

- ✓ Inclusion and exclusion criteria vary between institutions The following are suggested
- ✓ inclusion criteria based on the T33C targeted protocol at my institution: Post cardiac arrest (any cause)ROSC < 30 mins from team arrival Time < 6 hours from ROSCPatient is comatose MAP \geq 65mmHg
- ✓ Exclusions may include:
Advanced directive stipulating DNR (absolute)Traumatic arrest
Active bleeding (including intracranial)Pregnancy, recent major surgery, severe sepsis

Inclusion Criteria for TH

- ✓ Witnessed arrest Post-cardiac arrest patient
- ✓ regardless of initial rhythm with ROSC within 60 min of initiation of ACLS
- ✓ Within 6 hours following cardiac arrest
- ✓ Maximum down time = 15 minutes
- ✓ Comatose state GCS < 5 Intubated

Exclusion Criteria for TH

- Intracranial hemorrhage
- Major surgery within 14 days
- Severe systemic infection/sepsis
- Preexisting coagulopathies
- Pregnancy
- DNR status/terminal illness
- Drug induced coma
- MAP <60mmHg for > 30 min after ROSC
- Temperature < 30 ° C (86F)
- GCS > 5

PHASES

There are 3 phases to TTM :

- Induction

Aim to reduce the core body temperature to T_{33C} (other protocols aim for between $32-34^{\circ}C$ ($90-93^{\circ}F$))– within 6 hours

- Maintenance

Maintain core body temperature for 24 hours (other protocols 12-36 hours)

- Rewarming

Either controlled or passive rewarming to normothermia $37^{\circ}C$ ($98.6^{\circ}F$)– $0.25^{\circ}C$ per hour (others target $0.5^{\circ}C$ per hour)– over 8-12 hours– avoid hyperthermia– can adjust rate if necessitated by hemodynamic instability

METHODS

- My institution uses IV cold saline 2-3 mL/kg stat cooling vest and cooling machinesedation and paralysisPros and cons of different methods.
- After TTM if a T36C is targeted then boluses of cold saline are unlikely to be required or lower volumes will be used

EFFECTS OF THERAPEUTIC HYPOTHERMIA

- ✓ CVS
- ✓ Bradycardia
- ✓ Hypotension
- ✓ decreased cardiac output (matched by reduced metabolic demand)
- ✓ AF is common
- ✓ severe dysrhythmias are more common below 30°C (86°F)
- ✓ Other ECG changes in hypothermia include prolongation of the PR, QRS and QT intervals, as well as Osborn waves (or J-waves)

EFFECTS OF THERAPEUTIC HYPOTHERMIA

- ✓ Therapeutic hypothermia is very helpful for some people.
- ✓ But it has some rare risks. Some of these risks include:
- ✓ Another abnormal heart rhythm,
- ✓ especially slow heart rates
- ✓ Severe blood infection (sepsis)
- ✓ Blood is less able to clot.
- ✓ This can cause bleeding.
- ✓ Electrolyte and metabolic problems
- ✓ Raised blood sugar levels Imbalance of the pH (acid-base) levels in the body
- ✓ These risks may vary based on your age and other health problems.
- ✓ Ask your healthcare provider about the risks specific to you.

EFFECTS OF THERAPEUTIC HYPOTHERMIA

- ✓ The goal is to cool as quickly as possible.
- ✓ Another cooling choice is internal cooling. That's when chilled fluids are given through an IV (intravenous) line into your bloodstream.
- ✓ The therapeutic hypothermia will likely last around 24 hours.
- ✓ The medical team will slowly rewarm you over several hours.
- ✓ They may set cooling blankets at gradually warmer temperatures.
- ✓ In some cases, they may also use rewarming devices.

Physiologic Monitoring of CPR Quality During Adult Cardiac Arrest: A Propensity-Matched Cohort Study

Robert M. Sutton, MD MSCE, Benjamin French,
PhD, [...], and for the American Heart
Association's Get With The Guidelines –
Resuscitation Investigators

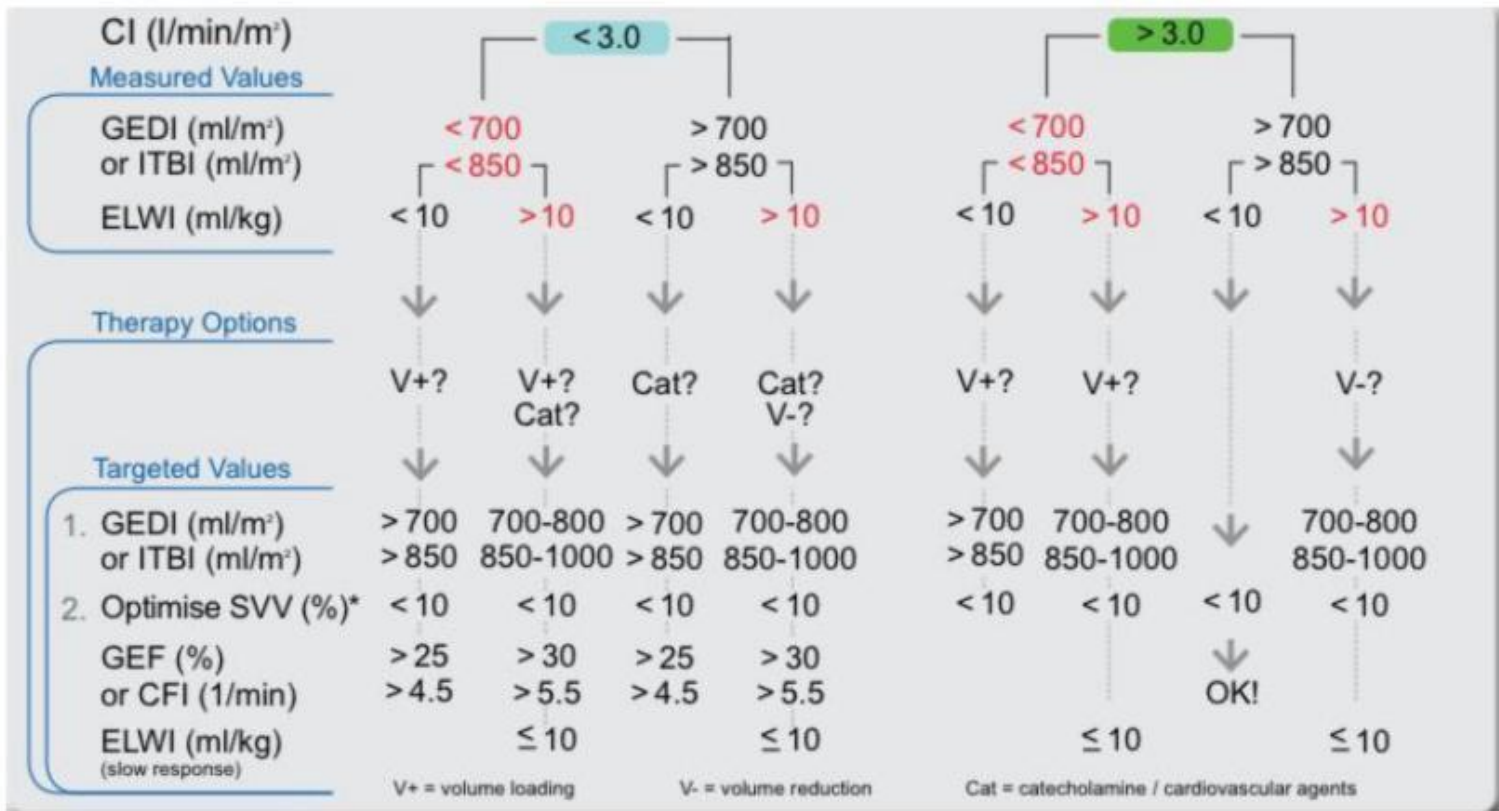
Conclusion

- Clinician-reported use of either ETCO₂ or DBP to monitor CPR quality was associated with improved ROSC. An ETCO₂ >10mmHg during CPR was associated with a higher rate of survival compared to events with ETCO₂ ≤10mmHg





		Central Venous Oxygenation - Oxygenation Balance (Oxygen load of the venous blood after passing through the organs)		ScvO ₂ ^{mm}	70-80 %	
		O ₂ Consumption (Consumption of O ₂ by organs)		VO ₂ l	125-175 ml/min/m ²	
Oxygen Delivery	O ₂ Delivery (Delivery of O ₂ via blood to organs)		DO ₂ l	400-650 ml/min/m ²		
	Haemoglobin (Oxygen transporter in blood)		Hb ^{mm}	8.7-11.2 mmol/l (Male) 7.5-9.9 mmol/l (Female)		
	Arterial / capillary oxygen saturation (Oxygen load of arterial blood)		SaO ₂ / SpO ₂	96-100 %		
	Flow		Cardiac Index	CI	3-5 l/min/m ²	
Blood Flow	Pulse Contour Cardiac Index (Cardiac Index related to body surface)		PCCI	3-5 l/min/m ²		
	Chronotropy Heart Rate		HR	60-80 bpm		
Stroke Volume	Stroke Volume Index (Output per heart beat)		SVI	40-60 ml/m ²		
	Preload	Global Enddiastolic Volume Index (Volume of blood in the heart)		GEDI	680-800 ml/m ²	
		Intrathoracic Blood Volume Index (Volume of blood in heart and lungs)		ITBI	850-1000 ml/m ²	
	Stroke Volume Variation (Dynamic fluid responsiveness)		SVV [°]	0-10 %		
	Pulse Pressure Variation (Dynamic fluid responsiveness)		PPV [°]	0-13 %		
	Afterload	Systemic Vascular Resistance Index (Resistance of vascular system)		SVRI	1700-2400 ^{dynes/cm⁵m²}	
		Mean Arterial Pressure		MAP	70-90 mmHg	
	Contractility	Global Ejection Fraction (Ratio of stroke volume and preload)		GEF	25-35%	
		Left Ventricular Contractility (Increase of arterial pressure over time)		dPmax	Trend information	
		Cardiac Function Index (Ratio of CI and preload)		CFI	4.5-6.5 l/min	
Cardiac Power Index (Global cardiac performance)		CPI	0.5-0.7 W/m ²			
Lung	Extravascular Lung Water Index (Lung oedema)		ELWI	3-7 ml/kg		
	Pulmonary Vascular Permeability Index (Permeability of lung tissue)		PVPI	1.0-3.0		
Liver	Plasma Disappearance Rate ICG (Performance of the liver)		PDR	16-25 %/min		
	Retention rate of ICG after 15 minutes (Performance of the liver)		R15	0-10 %		



THANKS!

Do you have any questions?

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