# PREDICTING A GOOD NEUROLOGICAL OUTCOME IN SURVIVORS OF CARDIAC ARREST

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INTRODUCTION

- About **80% of patients who are successfully resuscitated** from cardiac arrest **are in a coma** upon arrival at the hospital due to post-cardiac arrest brain injury (PCABI) and **their prognosis is uncertain**.

- Much information is available about the predictors of poor neurological outcome after cardiac arrest, while **little is known about the predictors of a good outcome.**
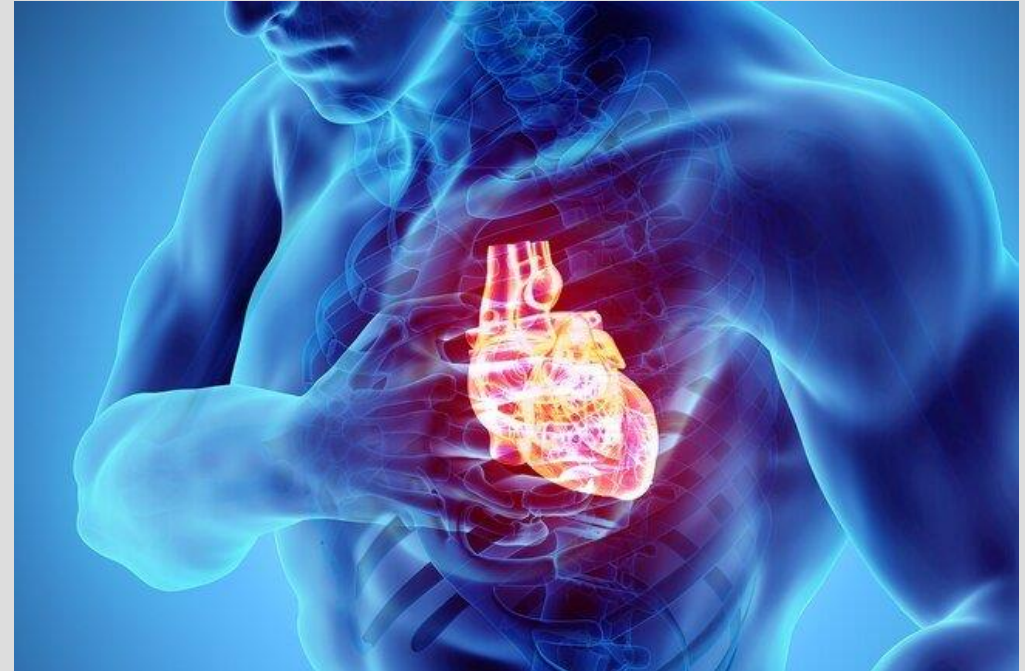
INTRODUCTION

- The outcomes for patients after non-traumatic cardiac arrest are grim. Only <u>6% to 9.6% of all patients with out-of-hospital cardiac arrest</u> (OHCA) survive hospital discharge, and an <u>estimated 22.3% of patients with in-hospital cardiac arrest</u> (IHCA) survive hospital discharge.

- Cardiac arrest-related brain injury is a <u>major determinant of mortality and disability.</u>

NEUROLOGICAL OUTCOME

- Poor neurological outcome is defined as <u>severe neurological disability, persistent vegetative state or death at one month or later after cardiac arrest.</u>

- Is anticipated to be likely when at <u>least two clinical examination unfavorable signs:</u>

  - biomarkers, electroencephalography (EEG),

  - somatosensory evoked potentials (SSEP),

  - brain computed tomography (CT),

  - or brain magnetic resonance imaging (MRI) are present.

- However, when <u>none or only one</u> of these predictors is present, the prognosis

NEUROLOGICAL OUTCOME

Good neurological prognosis (CPC-GP 1 and 2) was significantly associated with:

- the absence of previous illnesses of the patients;

- witnessed cardiorespiratory arrest;

- ventricular fibrillation and pulseless ventricular tachycardia as initial rhythms of cardiorespiratory arrest;

- CPR and defibrillation performed prior to the arrival of the advanced support unit;

- shorter time intervals between collapse and the start of CPR maneuvers.

POST-CPR CARE

- Until recently, acute post-resuscitation treatment of cardiac arrest survivors <u>was primarily directed at systemic injuries</u> <u>and acute neurological care focused primarily on prognosis</u>, with <u>supportive care</u> of neurological complications.

- Recently, there has been increased interest in providing acute <u>neuroprotective interventions</u>, with the intention of improving survivors' survival and independence.

Activation of Emergency Response → High-Quality CPR → Defibrillation → Advanced Resuscitation → Post–Cardiac Arrest Care → Recovery
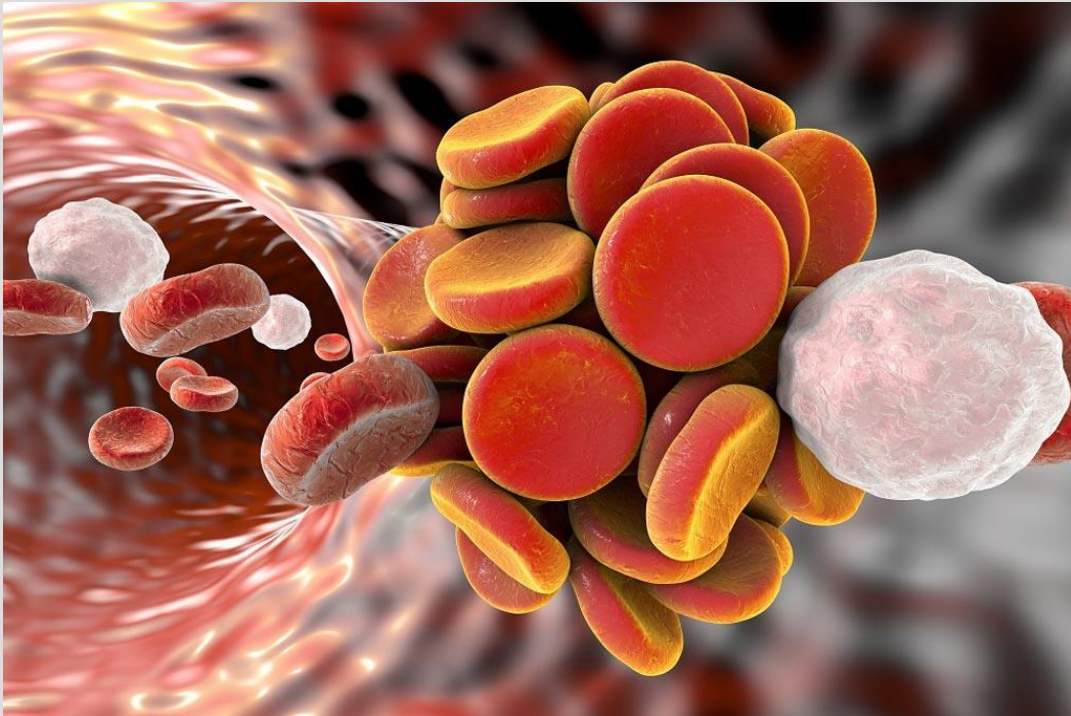
POST-CPR CARE

A recent concept is post-CPR care, which has been considered the <u>fifth link in the chain of survival</u> and has the potential to <u>improve early mortality</u>, caused by hemodynamic instability, <u>failure of multiple organs and systems, and late morbidity and mortality</u>, which result from neurological

POST-CPR CARE

Key measures to be taken include:

- early reperfusion treatment for coronary thrombosis;

- stabilization and maintenance of hemodynamic parameters;

- correction of arterial blood gas disorders;

- maintenance of normal glucose, hemoglobin, and electrolyte values;

POST-CPR CARE

Key measures to be taken include:

- control of the water balance;

- introduction of early enteral nutrition, as well as sedation and analgesia;

- seizure prevention and treatment;

- induction of therapeutic hypothermia;

- Establishing the patient's prognosis.
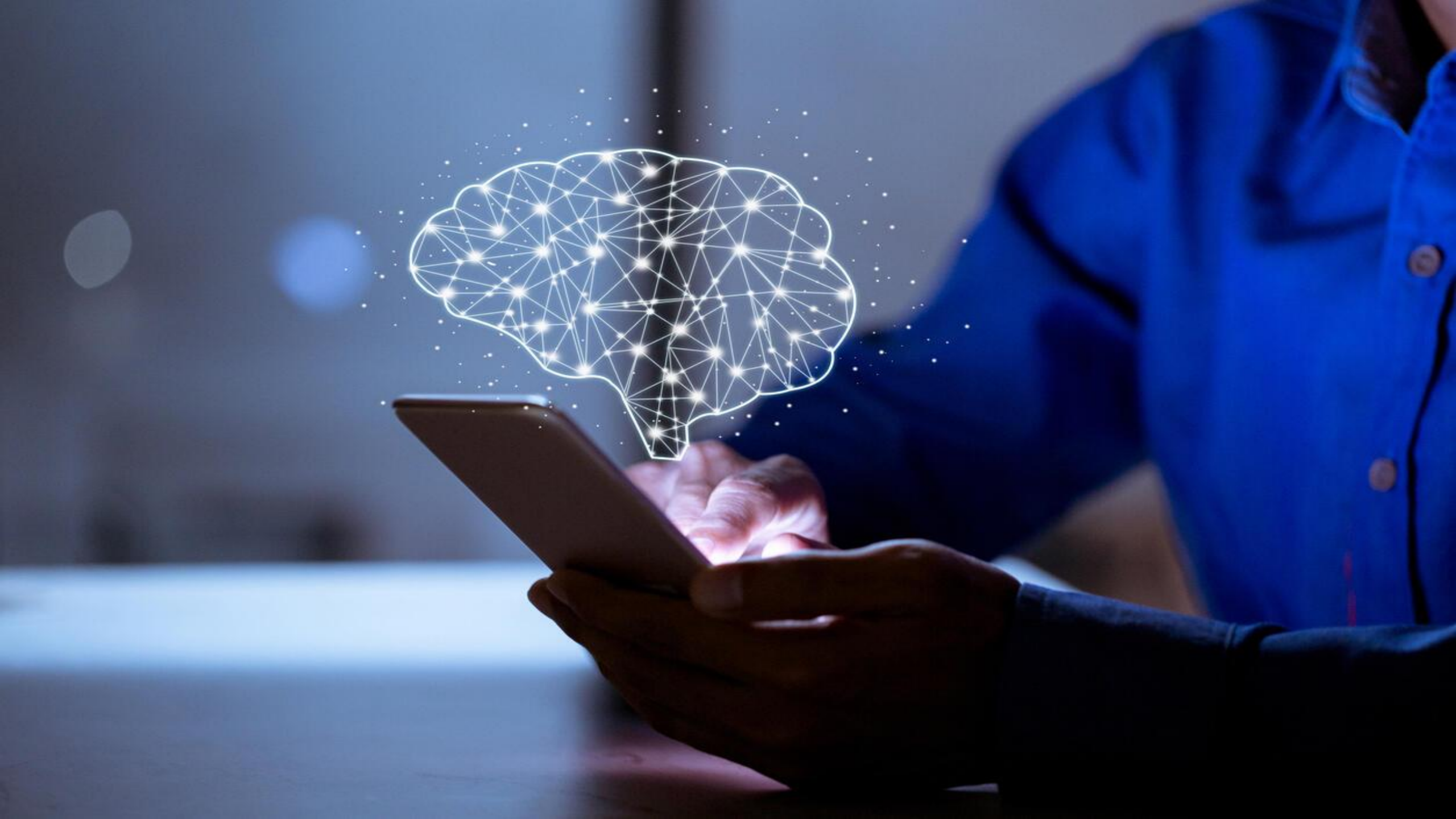
PREDICTION OF GOOD NEUROLOGICAL OUTCOME

Predicting a good neurological outcome after cardiac arrest has several advantages:

- It can <u>reduce uncertainty</u> in forecasting.

- Detecting a chance of <u>good neurological recovery can reassure patients' family</u> members and inform the conversations they have with doctors.

- It can help inform <u>decisions about organ support escalation</u>.

- It can <u>counteract a falsely pessimistic signal from predictors of a poor neurological outcome.</u>
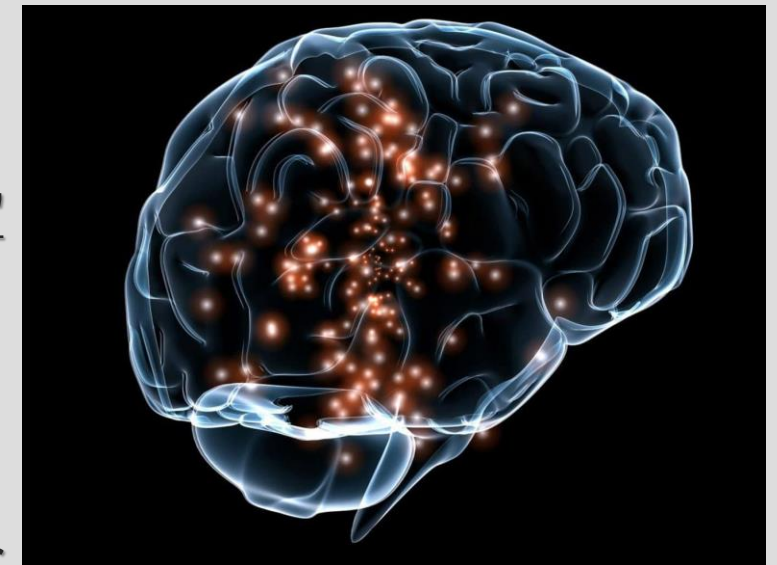
PREDICTORS OF GOOD NEUROLOGICAL OUTCOME

- 1) GCS motor score 4 or 5 immediately or at 72-96 h after return of spontaneous circulation (ROSC);

- 2) Normal blood values of neuron-specific enolase (SES) at 24-72 h after ROSC;

- 3) Absence of diffusion restriction in the cortex or deep gray matter on MRI in the 2-7 days after

- 4) Favorable EEG background (continuous, normal voltage, or both) without abundant discharges at 12-72 h after ROSC;

- 5) SSEP N20 wave amplitude above 4 μV on at least one side predicted a good outcome with a specificity >85% at 12-96 h after ROSC.

Predictors of mortality and neurological function in ICU patients recovering from cardiac arrest: a spanish nationwide prospective cohort study

A.Loza, F. del Nogal, D. Macias, C. leon, L.Socias, L. Herrera, L.J.Yuste, J.Ferrero, B.Vidal, J. Sanchez, A. Zabalegui, P.Saavedra, A. Lesmes, Om behalf of the spanish PCRR-HT Study Group

The most favorable neurological outcome observed in:

Desfibrilation Rhythm

ROSC <20min

Cardiac Cause of Heart Failure

Neurological Outcome in patients after successful ressuscitation in out-of-hospital settings

Martin Marinsek, Andreja Sinkovic, David Suran

A good neurological outcome is more likely in:

- Early EEG reactivity

- Appropriate EEG Responses to Auditory Stimuli

- No CT/MRI abnormalities within 1 week of ROSC

- Early treatment of ACS with PCI and dual antiplatelet therapy

BIOMARKERS

- **NSE is the most studied biomarker of post-cardiac arrest brain injury;**

- **In current ERC-ESICM guidelines for post-resuscitation care, NSE values greater than 60 µg/L at 48 h and 72 h ROSC are recommended as predictors of poor outcomes after cardiac arrest;**
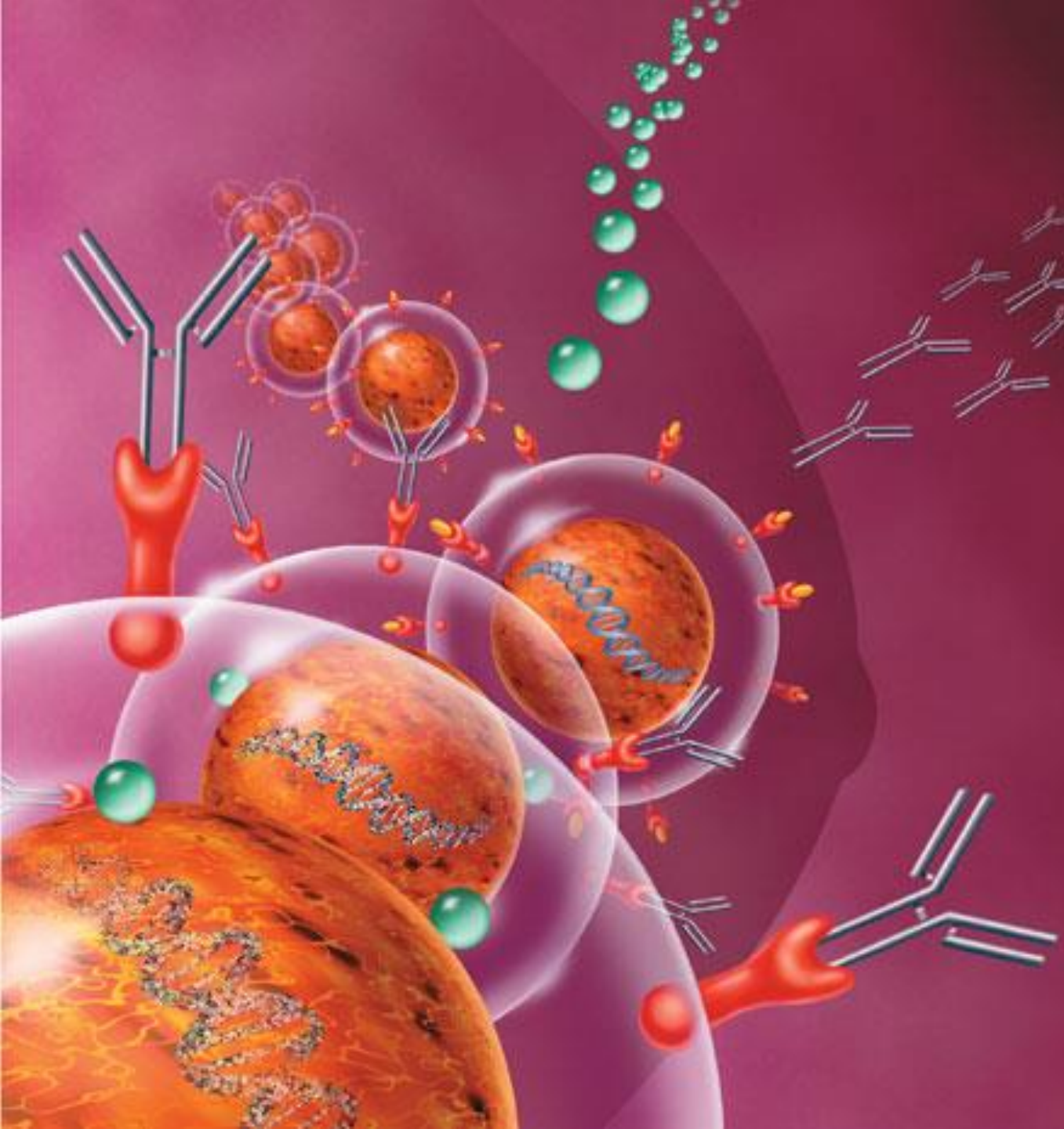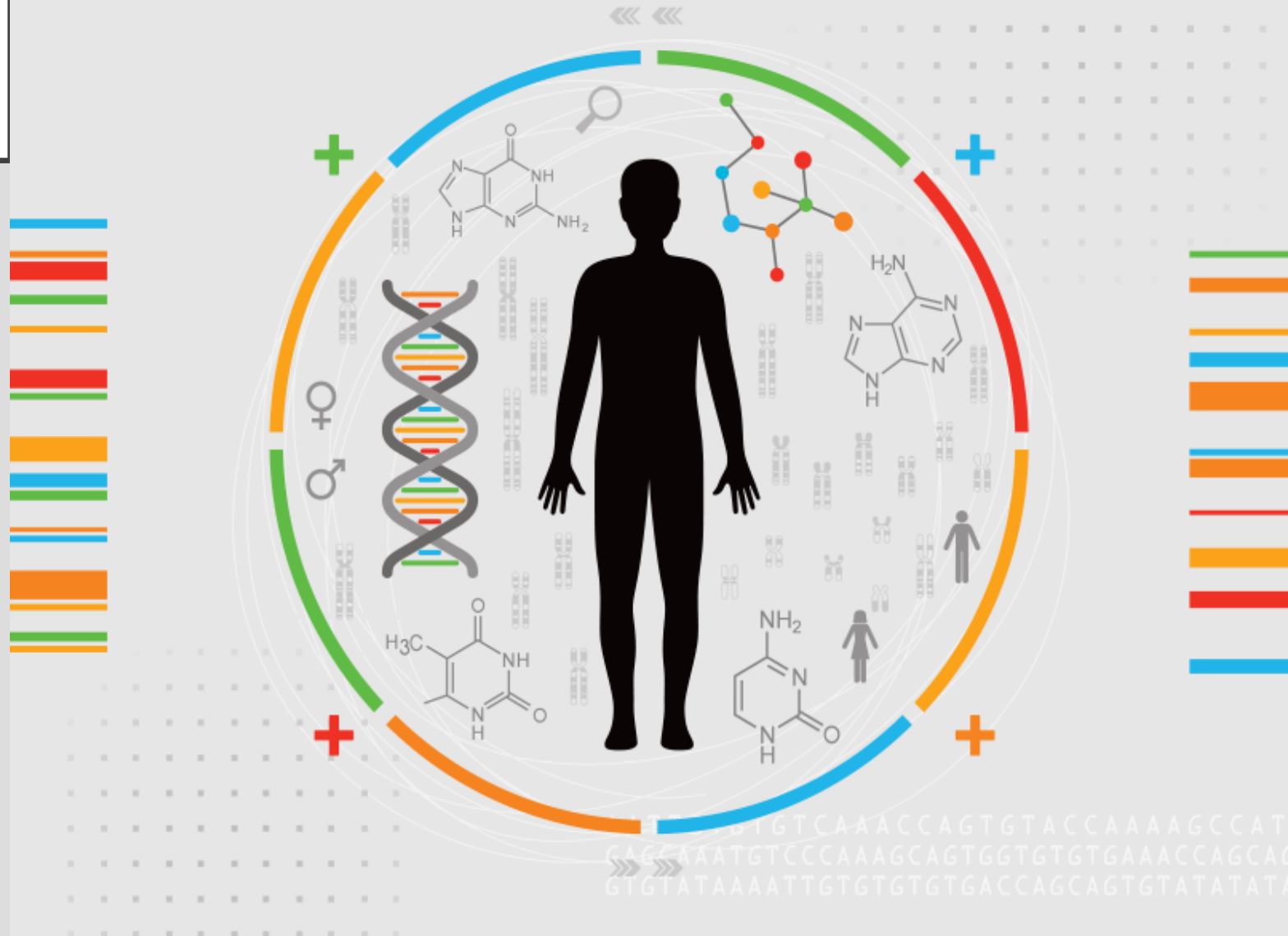
BIOMARKERS

- NSE predicted a good outcome with a specificity consistently greater than 80%, even when measured just 24 hours after ROSC.

- This trend is different from that observed for the prediction of poor outcomes, where the best accuracy is achieved at 48-72 h of ROSC, which coincides with peak blood NSE values.
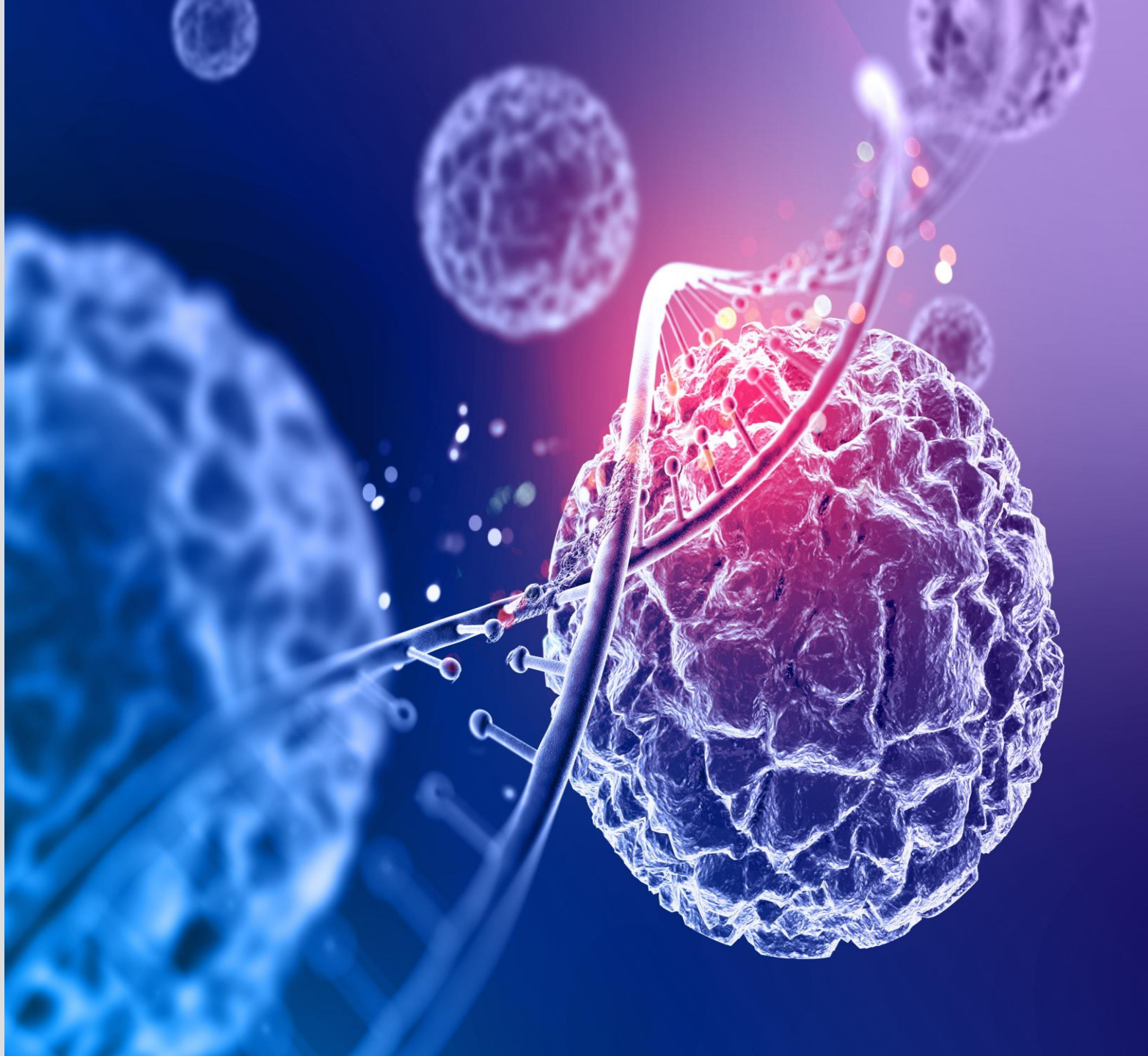
BIOMARKERS

- S-100B is a __biomarker released from astrocytes and is less documented than NSE__ as a predictor of outcomes after cardiac arrest;

- __S-100B increases more rapidly, reaching its maximum blood values within 24 h__ of ROSC, and __has a shorter half-life compared to NSE__. This could make S-100B a potential __test for good prediction of early results after arrest__. However, these results need to be confounded by other studies.

BIOMARKERS

Other biomarkers such as:

- NFL

- GFAP,

- Tau protein,

- UCH-L1 has recently been investigated as an alternative to NSE or S100-B.

- Of these, NFL and GFAP had the highest specificities.

Identification of novel biomarkers for prediction of neurological prognosis following cardiac arrest

Jung Woo Eun, Hee Doo Yang, Soo Hyun Kim, Sungyoup Hong, Kyu Nam Park, Suk Woo Nam and Siyoug Jeong

- <u>MAPK3</u>
- <u>BCL2</u>
- <u>AKT1</u>
- <u>Independent predictors of poor neurological outcomes ($P < 0.05$)</u>

ELECTROPHYSIOLOGY SOMATOSENSORY EVOKED POTENTIALS

- Studies showed that a <u>very low-amplitude N20 wave excludes good output with high probability</u>, which reduces the area of uncertainty.

- Indirect confirmation of the reliability of the <u>N20 SSEP waveamplitude</u> for outcome prediction came from a recent <u>study showing that this amplitude was inversely proportional to the severity of PCABI detected at autopsy.</u>
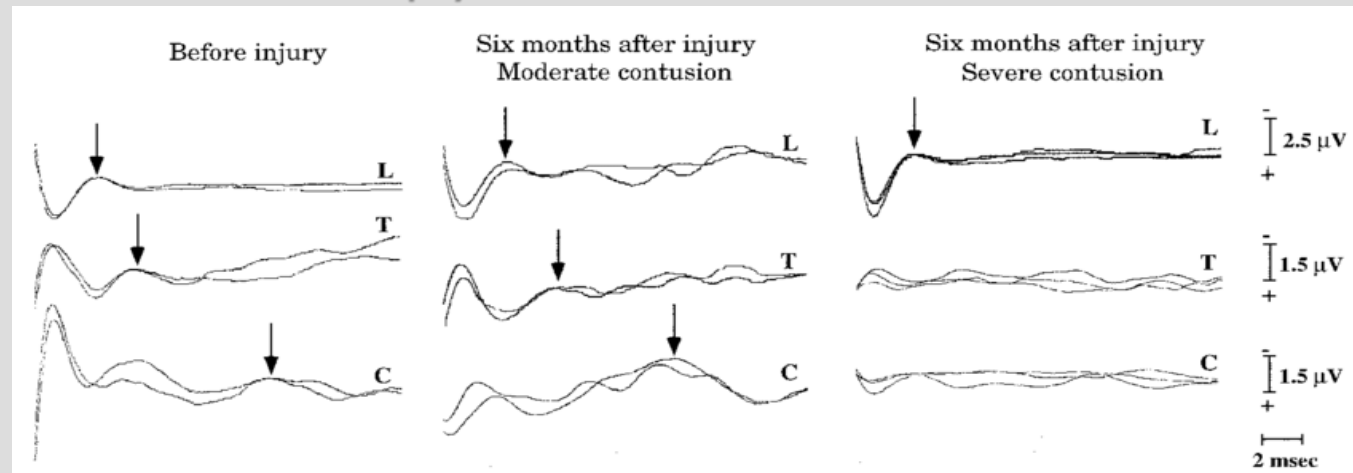
EEG

- EEG measures the <u>"electrophysiological functional recovery" of the cortex and its connections to subcortical structures after cardiac arrest.</u>

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- Immediately after ROSC, the <u>EEG background is suppressed and then gradually recovers to a continuous normal voltage in most patients. This process is not specific to a good neurological</u> outcome, but its timing is, since in patients with good neurological outcome, recovery usually occurs sooner.

EEG

- In one study, the EEG background was recovered to a continuous normal voltage within 24 hours in 95% of patients with good outcome versus 11% of patients with poor outcome.

- This occurred in 75% of patients with good results in another study.

IMAGING

PCABI leads to <u>cytotoxic edema that manifests on brain CT</u> as a decrease in brain parenchymal density that mainly affects gray matter symmetrically, with consequent reduction in the density ratio of gray matter to white matter (GWR).

Other signs of cerebral edema of PCABI <u>include an efacement of the cerebrospinal fluid spaces, and pseudo-subarachnoid hemorrhage and white cerebellar signs</u>.

All of these signs suggest <u>a poor neurological outcome</u>.

REDUCE BRAIN INJURY AFTER CPR

For patients who are in a coma in whom the baseline heart rhythm is VT/VF or asystole/pulseless electrical activity (PEA) after OHCA, target <u>temperature management (36°C for 24 hours, followed by 8 hours of rewarming at 37°C, and keeping temperature below 37.5C for up to 72 hours) is probably as effective as HT and is an acceptable alternative (Level B).</u>

REDUCE BRAIN INJURY
AFTER CPR

The absence of a clear understanding of the mechanisms by which hypothermia exerts its neuroprotective effects limits the ability to identify the most opportune time to initiate intervention.

When added to HT, coenzyme Q10 showed a survival benefit but no improvement in neurological status at 3 months. More data are needed to define the role of coenzyme Q10.

REDUCE BRAIN INJURY
AFTER CPR

Evidence refutes the use of 100% oxygen in post-resuscitation.

REDUCE BRAIN INJURY AFTER CPR

- The studies investigated xenon gas, lidoflazine nimodipine, selenium, thiopental, magnesium, diazepam, and corticosteroids as putative neuroprotective agents.

- To date, no neuroprotective drug has been shown to be effective in improving survival or neurological outcome in patients who are in a coma after cardiac arrest.

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