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Neuroprognostication after Cardiac Arrest

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Abstract

Neuroprognostication is one of the most controversial and sensitive examinations in the field of neurology. Neuroprognostication after cardiac arrest is a particularly important evaluation to complete as it is closely tied to the pathology of cardiac arrest and the time to return of spontaneous circulation (ROSC). With the improvement in healthcare, there are more survivors of cardiac arrest. These survivors have variable outcomes, and often, a difficult prognosis to interpret based on their clinical presentation with hypoxic brain injury. Many patients experience moderate to severe neurological impairment in the form of severe cognitive disability or persistent vegetative state. Withdrawal of life-sustaining measures inevitably leads to death, thus propagating neuroprognostication as a controversial tool used to guide these difficult decisions. Thorough evaluation and tests are required for prognostication, with new guidelines consistently being updated. The non-exhaustive list of tests includes pupillary light response, neurological motor response, computed tomography of the brain, magnetic resonance imaging of the brain, electro-encephalogram, somatosensory evoked potentials, and more. Testing variables and outcomes are also dependent on the patient's clinical picture, including potential hypothermic status. Overall, neuroprognostication after cardiac arrest holds great value in guiding clinical decision-making with the help of physical exam skills, updated algorithmic decision-making guidelines, and technology.

Keywords

Neuroprognostication, Hypoxic Brain Injury, Cardiac Arrest, Clinical Decision-Making, Neurology

1. Introduction

Survival after cardiac arrest is steadily improving, yet most survivors face a second

battle: **hypoxic-ischemic brain injury**. Contemporary U.S. registry data show that roughly **60%** of adults who regain spontaneous circulation develop measurable neurological deficits, and up to **one-third** remain severely disabled at discharge [1]. Many of these survivors experience moderate to severe impairments, such as severe cognitive disabilities or persistent vegetative states [1].

Cognitive disability spans a broad spectrum, from mild executive-function deficits to persistent vegetative state. The U.S. Federal Communications Commission, for instance, classifies intellectual disability, autism-spectrum disorders, severe mental illness, brain injury, stroke, and Alzheimer's disease under the umbrella of cognitive disability [2]. This review focuses on impairments that arise specifically from post-arrest hypoxic-ischemic injury.

Neuroprognostication—the structured, multimodal prediction of long-term neurological outcome—guides decisions on continuing or withdrawing life-sustaining therapy (WLST). 80% of post-cardiac arrest fatalities are due to withdrawal of life-sustaining treatment (WLST). Post-cardiac arrest withdrawal of life-sustaining treatment reflects a complex interplay of regional practices, ethical frameworks, and cultural influences that significantly impact mortality outcomes.

Stark regional variations exist, with North America and Northern Europe showing high WLST rates (30% - 65%). At the same time, Eastern European countries rarely practice WLST [3], revealing how intensely geographic and cultural contexts shape end-of-life decisions. Hospital culture creates further variation, with studies demonstrating that early WLST (before 72 hours) negatively impacts survival rates and differs markedly between institutions within the same country [4]. Religious perspectives profoundly influence these decisions, with most major faiths permitting withdrawal of non-beneficial treatments while others prohibit it entirely. The disconnect between physician and public perspectives—illustrated by a Swedish study showing 82.3% of doctors would withhold treatment in a specific case compared to only 40.2% of the general population—reveals potential ethical misalignments between medical professionals and society [5]. Shared decision-making processes vary widely, with inconsistent family involvement and poorly documented goals-of-care discussions despite their crucial role in determining outcomes. Early WLST, based on perceived poor neurological prognosis, though common, contravenes current guidelines recommending waiting at least 72 hours, potentially resulting in preventable deaths through premature termination of care before accurate prognostication is possible [5] [6]. These factors collectively demonstrate why standardized protocols balancing respect for patient autonomy with evidence-based neurological assessment are essential to ensure ethical decision-making in this vulnerable patient population.

Cardiac arrest abruptly halts effective cardiac output and cerebral perfusion. Etiologies divide broadly into cardiac causes (e.g., hypertension- or hyperlipidemia-driven arrhythmia, myocardial infarction) and non-cardiac causes (respiratory failure, electrolyte derangement, sepsis, trauma). Shared vascular risk factors—hy-

pertension, hyperlipidemia, age, diabetes—link neurological disease with arrest risk. Regardless of the trigger, global hypoperfusion deprives the brain of oxygen and glucose; the extent of injury is governed chiefly by the arrest etiology, the duration of no-flow/low-flow, and the time to return of spontaneous circulation (ROSC) [7] [8].

2. Study Aims

To guide clinicians, patients, and families toward evidence-based decisions, this review pursues three objectives:

1) Summarize the accuracy and limitations of clinical examination, electrophysiology, neuroimaging, and biochemical markers used for neuroprognostication after adult cardiac arrest.

2) Evaluate how current prognostic practices influence WLST timing and functional outcome, emphasizing the risk of self-fulfilling prophecies.

3) Propose a practical, phased algorithm that integrates multimodal data and specifies when prognostic certainty is sufficient to inform WLST versus continued supportive care.

By articulating these aims, we provide a roadmap for clinicians seeking to balance prognostic accuracy with therapeutic optimism.

3. Pathophysiology Cellular Mechanisms (Primary Injury)

The sudden cessation of cerebral perfusion during cardiac arrest halts oxidative phosphorylation, rapidly depleting adenosine triphosphate (ATP) and disabling ATP-dependent ion pumps. Loss of ionic homeostasis permits an uncontrolled influx of calcium, sodium, and chloride, depolarizing neuronal membranes and triggering activation of calcium-dependent proteases, lipases, and endonucleases [9]. Excessive glutamate release and mitochondrial membrane permeabilization generate large quantities of reactive oxygen species (ROS) [9]. The ROS drives both apoptotic and necrotic cell death. Selectively vulnerable neuronal populations—hippocampal CA1 pyramidal neurons, Purkinje cells of the cerebellum, and basal ganglia neurons—are the first to succumb, explaining the characteristic cognitive and motor deficits observed after resuscitation [9]-[11].

4. Reperfusion Injury (Secondary Injury after ROSC)

Restoration of circulation re-oxygenates metabolically primed mitochondria, precipitating an abrupt ROS surge that oxidizes lipids, proteins, and nucleic acids [9]. Endothelial swelling, leukocyte adhesion, and microthrombi create a microvascular "no-reflow" phenomenon, so regional hypoxia persists despite seemingly normal systemic flow. Blood–brain-barrier disruption and failure of cerebral autoregulation promote vasogenic edema and further compromise perfusion [10]. These secondary processes evolve over hours to days, providing a therapeutic window for interventions such as targeted temperature management and antioxidant or anti-inflammatory strategies [10].

5. Neurological Assessment after ROSC

5.1. Clinical Examination

Because many resuscitated patients remain comatose, history must often be gleaned from family, bystanders, or emergency personnel. Initial evaluation centers on establishing the time and quality of ROSC and objectively grading arousal. The Glasgow Coma Scale (GCS) permits serial, standardized assessment of consciousness, distinguishing coma, unresponsive wakefulness (vegetative state), and the minimally conscious state [12]-[21]. Higher admission GCS scores generally portend better neurological recovery [21].

5.2. Advanced Neurodiagnostic Tools

Electroencephalography (EEG) detects electrographic seizures, quantifies background reactivity, and identifies malignant patterns (e.g., burst suppression) associated with unfavorable outcome. Non-contrast computed tomography (CT) rapidly rules out intracranial hemorrhage, mass effect, or severe cerebral edema. At the same time, diffusion-weighted magnetic-resonance imaging (DW-MRI) or functional MRI (fMRI) reveals early ischemic injury and assesses functional connectivity. Finally, carotid duplex ultrasonography evaluates extracranial carotid flow and guides hemodynamic management when cerebral perfusion is in doubt [21]. Together, these complementary modalities integrate structural and functional data to refine prognostication.

5.3. Limitations of Neurodiagnostic Tools

Interpretation of ancillary tests requires care. Sedatives, neuromuscular blockade, and targeted temperature management can suppress cortical activity on EEG, creating patterns that mimic electrocerebral silence, and inter-observer variability further limits specificity [22] [23]. Non-contrast CT may overlook subtle early hypoxic-ischaemic changes [24]. Although quantitative grey-to-white-matter ratio analysis improves sensitivity, repeated radiation exposure complicates serial imaging [24]. MRI and fMRI are logistically challenging in haemodynamically unstable patients, and ferromagnetic implants can preclude their use; prolonged acquisition times necessitate additional sedation that may itself alter blood-oxygen-level-dependent (BOLD) signals and delay care [25]. Even when feasible, fMRI data are hampered by motion artifact, hemodynamic lag, and the absence of standardized connectivity thresholds [26]. Carotid duplex ultrasonography is operator dependent and provides no insight into the intracranial microcirculation, where the "no-reflow" phenomenon predominates [27]. Consequently, reliable neuroprognostication relies on a multimodal paradigm correlating serial clinical examinations with at least two independent ancillary investigations.

6. Evaluation

Neuroprognostication following cardiac arrest involves a comprehensive, multimodal approach that includes a detailed examination under specific conditions.

The patient should be evaluated without sedation, and temperature regulation should be maintained with a goal temperature below 36.5 ˚C [28]. Essential assessments include arterial blood gas analysis, metabolic and hematological blood tests, and serial serum neuron-specific enolase (NSE) levels. Additional diagnostic modalities may encompass somatosensory evoked potentials, brain computed tomography, continuous electroencephalography, brain magnetic resonance imaging, carotid duplex or cerebral angiography, and, when indicated, advanced functional imaging such as positron-emission tomography or single-photon emission computed tomography [28]. In rare circumstances, an apnea test can evaluate brainstem function when other prognostic markers remain inconclusive. This specialised, stepwise testing strategy equips clinicians with a more definitive prognosis and enhances the overall accuracy of neuroprognostication. The recommended algorithm for evaluating comatose cardiac-arrest survivors illustrates the day-by-day steps and expectations for patient care (Figure 1).

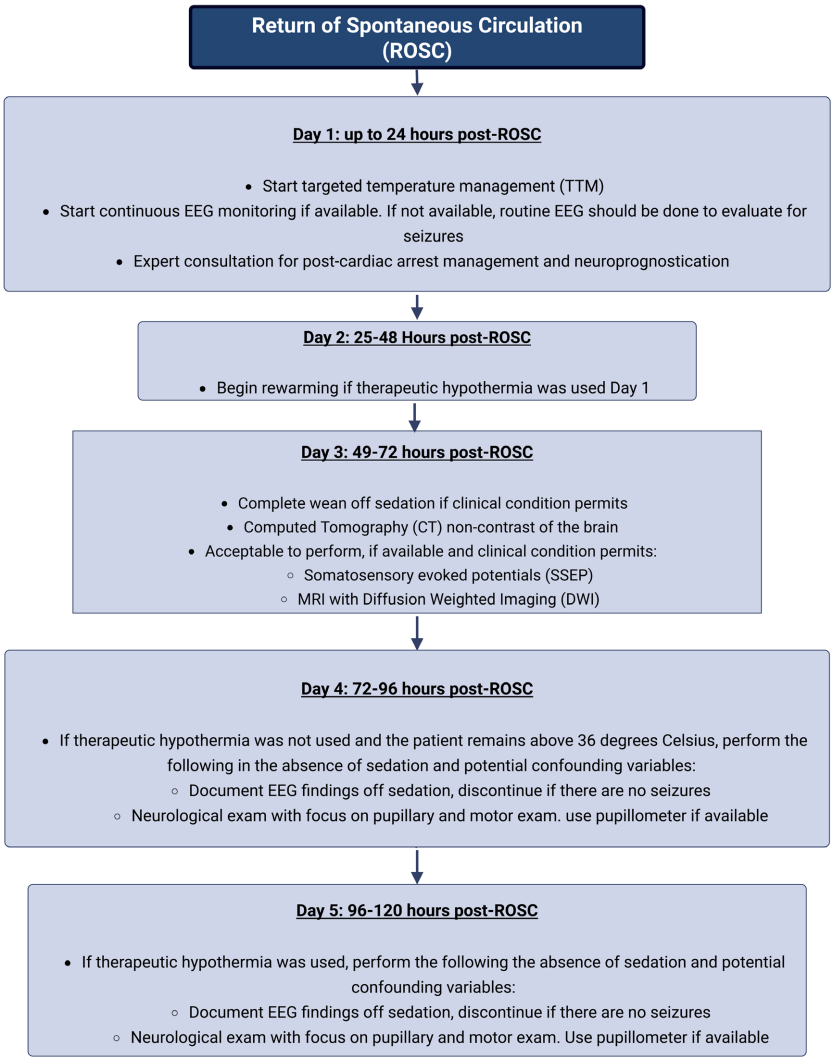

Figure 1. Algorithm for neuroprognostication in adult comatose cardiac arrest survivors. The evidence-based algorithm determines functional outcome after 3 months.

7. Treatment/Management

The latest Neurocritical Care Society (NCS) guidelines have identified eleven clinical variables to support clinician decision-making and family discussions. The American Heart Association guidelines recommend delaying neurological prognostication until at least 72 hours after return to normothermia in post-cardiac arrest patients based on substantial evidence demonstrating the risks of premature assessment. This crucial waiting period accounts for the delayed clearance of sedatives and neuromuscular blockers in hypothermia-treated patients, which can confound clinical examinations [29]. Multiple studies reveal that false-positive rates for poor outcome prediction decrease significantly when assessments occur after this timeframe, with evidence that 15% - 30% of patients with good outcomes may not awaken until 48 hours to 12 days after sedation cessation [6]. Prospective research reveals that approximately one-third of patients had life-sustaining therapy withdrawn within 72 hours of admission, potentially contributing to 2300 preventable deaths annually in the U.S. [30]. This recommendation reflects the understanding that approximately 80% of patients destined for good outcomes recover consciousness within 3.5 days post-ROSC, while delayed awakening commonly occurs until 7 days, with documented cases extending beyond this timeframe [30]. Current guidelines emphasize a multimodal prognostication approach conducted no earlier than 72 hours after normothermia to maximize accuracy and allow sufficient neurological recovery time [31].

Brain MRI performed 2 - 7 days post-cardiac arrest provides optimal prognostic information as supported by multiple lines of evidence. This timing window coincides with the evolution of apparent diffusion coefficient (ADC) changes, which reach nadir between 3 - 5 days after arrest [32], allowing for maximal detection of hypoxic-ischemic injury. Current guidelines formally recommend brain MRI at 2 - 5 days after return of spontaneous circulation based on established studies, though recent evidence validates the utility of imaging through day 7 [33]. This extended window is particularly valuable as quantitative thresholds have been validated, showing that > 10% of brain tissue with an ADC < 650×10^{-6} mm^2/s identifies poor outcomes with high specificity [34]. The 2 - 7 day period allows for clearance of sedative medications and completion of therapeutic temperature management, reducing confounding factors and improving prognostic accuracy. Multimodal prognostication guidelines from neurological societies support this timing, emphasizing that diffusion restriction patterns across bilateral anterior and posterior circulation, involving both cortex and deep gray matter, are most predictive when assessed during this interval [28]. This timing also aligns with the broader recommendation to delay definitive prognostication until at least 72 hours after normothermia, creating a comprehensive approach to neurological assessment following cardiac arrest.

Neuromuscular blocking agents (NMBAs) present a double-edged sword in

post-cardiac arrest neuroprognostication. They mask critical clinical examination findings like motor responses and brainstem reflexes, while significantly enhancing the reliability of somatosensory evoked potentials (SSEPs). When administered during SSEP testing, NMBAs eliminate muscular artifacts that could otherwise contaminate recordings and compromise signal quality. Current guidelines specifically recommend their use to improve signal-to-noise ratios below the critical threshold of 0.25 μV [35]. Unlike sedatives and analgesics that can alter cortical waveforms, NMBAs act exclusively at the neuromuscular junction without affecting central signal transmission, preserving the integrity of cortical N20 responses critical for prognostication [28]. This selective peripheral action makes SSEPs particularly valuable within a multimodal assessment framework, as they remain relatively resistant to the confounding effects of both hypothermia and sedation compared to EEG patterns or clinical examination findings. The Canadian Cardiovascular Society's position statement emphasizes this advantage, recommending NMBAs during SSEP recording to minimize artifacts and false positives, while cautioning that their use necessitates delaying clinical examination until complete clearance [3]. Prudent practice involves scheduling SSEP assessments when NMBAs are clinically indicated for shivering control during targeted temperature management, followed by adequate time for drug clearance before clinical examination, supporting the standard 72-hour post-normothermia timeline for comprehensive neuroprognostication [36].

When prognosis remains indeterminate, clinicians should communicate the possibility of a prolonged and uncertain recovery trajectory. Shared decision-making demands that the wishes of the patient and their surrogate be incorporated; in many cases, an extended observation period may be appropriate and should be discussed transparently.

Predictive markers in the NCS framework are stratified as **reliable**, **moderately reliable,** or **not reliable** based on three-month functional outcomes. These markers, summarized in **Table 1**, feed into an evidence-driven algorithm (**Figure 2**) that guides day-to-day prognostic expectations and therapeutic choices.

Alongside the collected patient history, physical examination findings, imaging results, and neuroprognostic indicators, the provided algorithms also support clinicians in navigating the neuroprognostication process for suitable patients [18] [19].

8. Complications and Conclusions

These guidelines are essential for navigating neurologically complex situations that frequently intertwine medical, ethical, and legal considerations [28]. Every case is unique and warrants comprehensive, ongoing dialogue with the family. Such communication ensures that care aligns with patient values and clarifies what constitutes a meaningful quality of life. Most hospitals provide dedicated ethics and legal consult services when additional complexities emerge.

Table 1. Reliable and moderate predictors of functional outcomes in patients with cardiac arrest at 3 months or later.

Category of predictors	Interpretation
Reliable predictors of poor functional outcome at 3 months or later	
Pupillary light response (PLR) ≥ 72 h from ROSC	• Bilateral absence of the PLR. • Use quantitative pupillometry where available. • Where a pupillometer is unavailable and the PLR is thought to be absent, consult ophthalmology or use a magnifying glass. • Consider potential confounders such as medications (mydriatic ophthalmic drops, nebulized bronchodilators) and prior ophthalmic surgery.
Somatosensory Evoked Potentials (SSEP) ≥ 48 h from ROSC	• Target-measured N20 amplitude is < 4 μV at 48 - 72 h from ROSC; it indicates a poor outcome. • Responses must be present at Erb's point and the cervical spine, as a prerequisite to prognostication. • Consider routine use of neuromuscular blockade during testing to minimize artifact. • Studies should be interpreted as indeterminate in the presence of significant background noise, which may obscure the N20 response. • Severe hypothermia may abolish the N20 response.
Moderately reliable predictors of poor functional outcome at 3 months or later	
CT Head, non-contrast ≥ 48 h following ROSC	• Diffuse pattern—loss of gray–white differentiation and sulcal effacement should be present across vascular distributions in the bilateral anterior and posterior circulation, with involvement of cerebral cortex and deep gray matter. • Do not use in the presence of artifact from sources such as EEG electrodes, patient movement, or beam hardening from bone.
MRI brain with DWI sequence, 2 - 7 days following ROSC	• Diffuse pattern- restricted diffusion should be present across vascular distributions in the bilateral anterior and posterior circulation, with cerebral cortex involvement and deep gray matter. • Do not use in the presence of artifact from sources such as patient movement. • Seizures and their potential etiologies of restricted diffusion must be ruled out.
Electroencephalography (EEG)	• Presence of suppression or burst suppression. • Suppression is defined as a background voltage < 10 μV for > 99% of the record. • Burst suppression is defined as a suppressed (< 10 μV) pattern present for 50% - 99% of the record. • Exclude confounders such as sedation, toxic-metabolic encephalopathy, and hypothermia.
Not reliable predictors of poor functional outcome at 3 months or later	
Neurological exam	• An absent or extensor motor response or absent withdrawal, localization, or command-following at any time.

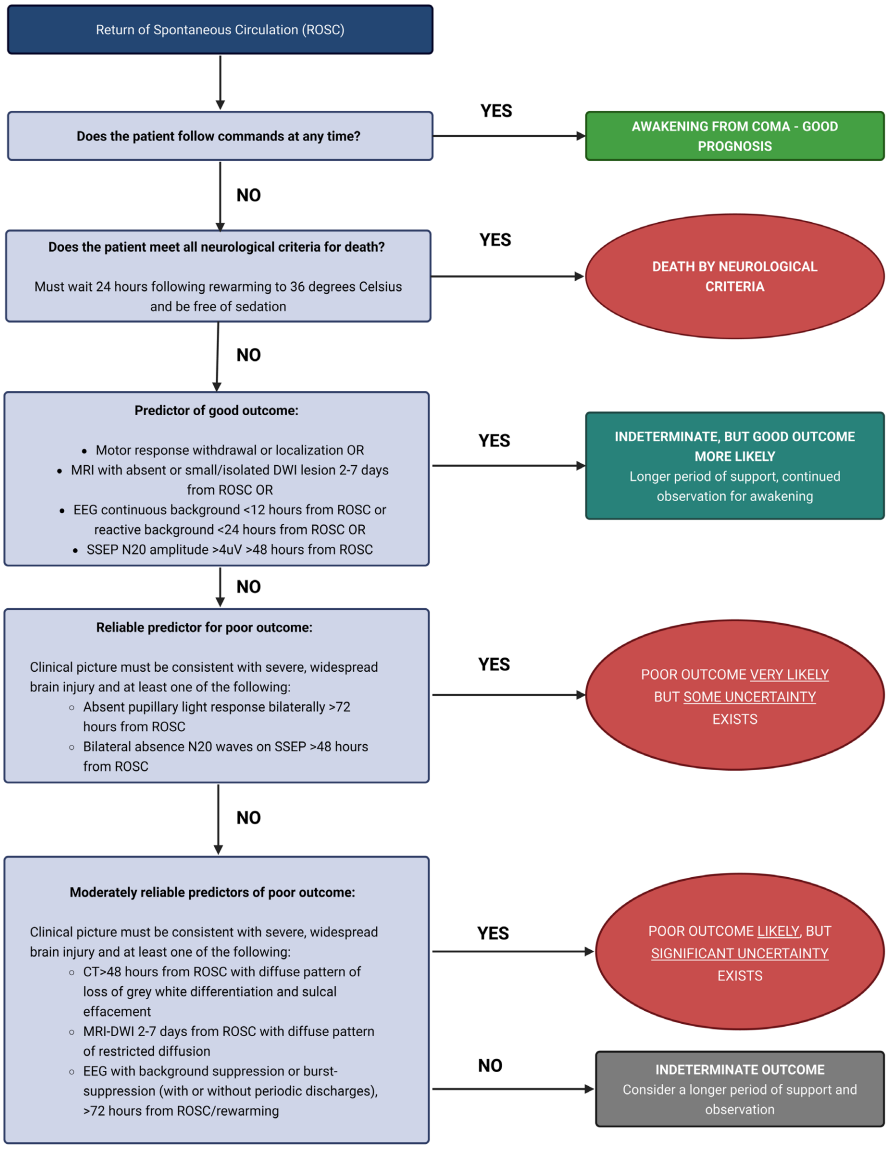

Figure 2. Algorithm for neuroprognostication in adult comatose cardiac arrest survivors: predictors & prognosis.

As clinician expertise in neuroprognostication grows—and as algorithms and ancillary tools become more widespread—favourable outcomes for cardiac-arrest survivors should continue to rise. Nonetheless, the effectiveness of these strategies remains contingent on each institution's resources and interdisciplinary collaboration [28]. As clinician expertise in neuro prognostication grows and algorithms and ancillary tools become more widespread, favorable outcomes for cardiac-arrest survivors should continue to rise. Nonetheless, the effectiveness of these strategies remains contingent on each institution's resources and interdisciplinary collaboration [28]. In Rajajee's study, resource utilization varied amongst models, thus representing the contrast in financial resources between low- and high-resource healthcare settings. Given the importance of thoughtful conversation regarding a patient's goals of care, resource-intensive testing such as MRI

and NSE might not be necessary. Instead, utilizing low-cost methods of prognostication, including clinical exam in concordance with a patient's discussed wishes, offers significant predictive value. However, without proper discussion and investigation according to predictive algorithms, there is a possibility for long-term expenditure on equipment, facilities, and resource teams, including transport. That being said, limiting the scope of practice to testing only in ways that will alter or enhance clinical treatment is encouraged, particularly in low-resource settings.

Neuroprognostication sits at the intersection of medicine, ethics, and law. Transparent, ongoing dialogue with families aligns care with patient values and clarifies what constitutes an acceptable quality of life. Where disputes arise, institutional ethics or legal services may be required [28].

As clinicians become familiar with multimodal prognostication and protocols, tools, and algorithms mature, cardiac-arrest survivors' outcomes should continue to improve. Nevertheless, success depends on local resources and interdisciplinary collaboration [28].

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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