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Neurological Prognostication after Extracorporeal Cardiopulmonary Resuscitation: From Pathophysiology to Multimodal Integration

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Abstract: Extracorporeal cardiopulmonary resuscitation (ECPR) has emerged as a salvage strategy for refractory cardiac arrest, yet hypoxic-ischemic brain injury (HIBI) remains the predominant determinant of poor long-term outcomes. This review systematically examines the pathophysiological mechanisms of HIBI following ECPR—including primary ischemic injury and secondary reperfusion injury mediated by excitotoxicity, neuroinflammation, oxidative stress, and cerebral edema—and identifies key prognostic factors such as age, arrest location, initial rhythm, low-flow time, intracranial hemorrhage, and hemodynamic targets. We critically evaluate current assessment modalities including bedside ultrasound (TCD, ONSD), cerebral oxygenation monitoring (rSO_2, $SjvO_2$), electrophysiological tools (EEG, aEEG, BIS), biomarkers (NSE, S100B, NfL, GFAP, UCH-L1), and neuroimaging (CT, MRI, low-field POC-MRI). Recognizing the inherent limitations of single-modality approaches, multicenter validation is warranted to establish its clinical utility across diverse settings. Future directions include artificial intelligence-driven dynamic prediction models, validation of emerging biomarkers in ECPR-specific cohorts, and standardized international registries to advance evidence-based neuroprognostication in this challenging population.

1. Introduction

Cardiac arrest (CA) is one of the leading causes of death and severe neurological dysfunction worldwide[1]. Despite continuous advances in conventional CPR(cCPR) techniques, the survival-to-discharge rate for patients with refractory ventricular fibrillation(VF) or pulseless ventricular tachycardia(pVT) remains below 10%, with most survivors suffering from varying degrees of neurological deficits[2]. Extracorporeal cardiopulmonary resuscitation(ECPR) provides extracorporeal circulatory support during cardiac arrest through veno-arterial extracorporeal membrane oxygenation(VA-ECMO), enabling rapid restoration of blood flow and correction of oxygenation, thereby gaining precious time for etiological treatment[3].

In recent years, with the widespread adoption of ECPR technology and the improvement of

emergency care systems, multiple systematic reviews and meta-analyses[4–6] have confirmed that ECPR significantly improves survival rates and rates of favorable neurological outcomes compared with cCPR in patients with refractory cardiac arrest. However, the incidence of acute brain injury(ABI) after ECPR remains as high as 20%–50%[7], representing the leading cause of in-hospital mortality and long-term disability. In 2024, ELSO released the Consensus Guideline[8] on Neuromonitoring and Management of Adult ECMO Patients, which for the first time systematically elaborated five key domains of neurological care during ECMO: neuromonitoring, early post-cannulation physiological goals and ABI, neurotherapeutics, neurological prognostication, and neurological follow-up and outcomes.

Meanwhile, the 2025 joint ERC/ESICM Guidelines[9] for Post-Resuscitation Care further emphasize that neurological prognostication in ECPR patients must adopt a multimodal integrative strategy, avoiding premature decisions to withdraw life-sustaining therapy based on a single diagnostic modality. This article aims to systematically review the latest advances in the pathophysiological mechanisms, key risk factors, bedside assessment modalities, and multimodal integrative prediction models of hypoxic-ischemic brain injury(HIBI) after ECPR, providing reference for clinical practice.

2. Pathological mechanisms

2.1 Primary Ischemic Injury

During cardiac arrest, cerebral blood flow is completely interrupted, and neuronal ATP reserves are rapidly depleted within 2–4 minutes, leading to dysfunction of the sodium-potassium-ATPase pump and neuronal depolarization[10]. This process triggers massive glutamate release, initiating an excitotoxic cascade; simultaneously, anaerobic metabolism results in lactic acidosis, further impairing cellular function. The duration and severity of primary ischemic injury directly determine the extent of immediate neuronal death and the magnitude of subsequent secondary injury.

2.2 Secondary Reperfusion Injury

Paradoxically, restoration of blood flow precipitates a “second hit” that exacerbates cerebral injury through the following mechanisms.

2.2.1 Excitotoxicity

Sustained activation of glutamate receptors drives excessive calcium influx, activating proteases and lipases, promoting mitochondrial dysfunction, and generating reactive oxygen species (ROS)[11].

2.2.2 Inflammatory response

Damage-associated molecular patterns activate microglia and recruit peripheral leukocytes, producing pro-inflammatory cytokines such as tumor necrosis factor-α(TNF-α), interleukin-1β(IL-1β), andIL-6, which disrupt the blood-brain barrier (BBB) and amplify neuronal injury[12].

2.2.3 Oxidative stress

Reperfusion-induced ROS bursts exceed antioxidant defense capacity, leading to lipid peroxidation, protein denaturation, and DNA damage[13].

2.2.4 Cerebral edema

Cytotoxic edema(intracellular swelling) and vasogenic edema(BBB disruption) collectively increase intracranial pressure, potentially reducing cerebral perfusion pressure and precipitating secondary ischemia[14].

3. Key Risk Factors Affecting Neurological Outcomes after ECPR

3.1 Age

Brain aging leads to reduced neuronal numbers, mitochondrial dysfunction, and impaired blood-brain barrier(BBB) integrity, significantly decreasing the tolerance of elderly patients to ischemia-reperfusion injury. Additionally, elderly patients frequently have comorbidities such as hypertension, diabetes mellitus, and chronic kidney disease, which further exacerbate microvascular dysfunction and secondary cerebral edema. A 2025 systematic review further confirmed that age <65years was significantly associated with favorable neurological outcomes after ECPR[15].

3.2 Out-of-Hospital vs. In-Hospital Cardiac Arrest

Compared with in-hospital cardiac arrest (IHCA), out-of-hospital cardiac arrest(OHCA) carries multiple disadvantages: prolonged no-flow time; low-quality bystander CPR, with chest compression fraction(CCF) <60% and cerebral antegrade blood flow only 10%–15% of normal; and uncontrolled low-flow time compared with IHCA[16]. A 2024 single-center retrospective study[17] similarly demonstrated that OHCA was an independent predictor of mortality after ECPR(IHCA accounted for 64% of non-survivors, whereas IHCA accounted for 78% of survivors, $p<0.001$).

3.3 Initial Shockable Rhythm

A shockable rhythm(VF/pVT) suggests a shorter duration of cardiac arrest, allowing rapid restoration of cerebral oxygen supply through early defibrillation. In contrast, pulseless electrical activity(PEA) or asystole typically indicates severe hypoxia and profound metabolic acidosis, with cerebral autoregulatory function already compromised. The 2025 systematic review further confirmed that a shockable rhythm at ECMO initiation was associated with favorable outcomes, and a shockable rhythm at hospital admission also demonstrated predictive value[18].

3.4 Low-Flow Time

Low-flow time is the most critical modifiable factor for neurological prognosis after ECPR and can be further divided into no-flow time and low-perfusion time.

3.4.1 No-flow time

Studies have demonstrated that no-flow time is a critical determinant of neurological prognosis in OHCA patients. Evidence suggests[19] that each additional minute of no-flow time is associated with an approximate 13% reduction in the probability of favorable neurological outcomes. Furthermore, when no-flow time exceeds 12 minutes, the probability of survival approaches less than 1%. These findings underscore the paramount importance of minimizing the interval from collapse to the initiation of bystander CPR, as every minute of delay substantially diminishes the likelihood of meaningful neurological recovery after ECPR.

3.4.2 Low-perfusion time

Studies have further quantified the relationship between low-flow time and prognosis. A pre-hospital ECPR study[19] showed that for every 10-minute increase in low-flow time, the adjusted odds ratio (aOR) for favorable neurological outcomes was 0.461 (95% CI 0.255–0.834). The 2025 ERC guidelines[9] recommend 45–60 min as the ideal low-flow time window for ECPR. A 2026 risk-stratification study[20] found that favorable outcomes in low-risk patients (shockable rhythm and age <50 years) could accumulate up to 70 min, whereas no additional favorable outcomes were observed in moderate-to-high-risk patients beyond 90 min.

3.5 Incidence of intracranial hemorrhage

The incidence of intracranial hemorrhage (ICH) varies between different modes of extracorporeal membrane oxygenation (ECMO), and regardless of the mode, ICH is associated with significantly increased mortality and poor neurological outcomes. However, the negative impact of ICH on prognosis is unequivocal and severe across ECMO modes. In VA-ECMO patients, in-hospital mortality reaches up to 81%, with a 1-month mortality as high as 86% following ICH[21]. In VV-ECMO patients, overall survival to hospital discharge is only 29.5% for those who develop ICH, compared to 63.7% for those without[22].

3.6 Hemodynamic and Oxygen Delivery Window

3.6.1 Mean arterial pressure

Following CA, the cerebral autoregulatory curve shifts rightward, with its lower limit rising from 60mmHg to 75mmHg. Retrospective studies[23,24] have shown that MAP <60mmHg is associated with decrease in regional cerebral oxygen saturation (rSO_2) and increased risk of poor outcomes.

3.6.2 Arterial partial pressure of oxygen

Available evidence suggests that the relationship between PaO_2 and outcomes in VA-ECMO patients may follow a U-shaped curve – both too low and too high are associated with poor prognosis. A 2025 systematic review[25] and meta-analysis confirmed that hyperoxia (defined as PaO_2 ≥200 mmHg, especially ≥300 mmHg) was significantly associated with increased mortality in ECMO patients(particularly VA-ECMO), with an OR of 1.41 (95% CI 1.22–1.64).

3.6.3 Arterial Partial pressure of carbon dioxide

Available evidence also suggests that $PaCO_2$ levels may exhibit a "U-shaped" relationship with patient outcomes. A multicenter study[26] of OHCA patients receiving VA-ECMO found that, compared with high normocapnia (40 to <50 mmHg), low normocapnia (30 to <40 mmHg) was independently associated with worse 30-day functional outcomes

3.7 Fever and Infection

3.7.1 Body temperature

Elevated body temperatures can increase the brain's metabolic demand, promote the release of excitatory amino acids, and exacerbate calcium overload[27]. The 2025 ERC guideline[9] recommended temperature control for comatose post-CA patients, with a target temperature of ≤37.5℃ maintained for 36-72 hours.

3.7.2 Infection

The incidence of hospital-acquired pneumonia in ECMO patients is 12.7%–55.9%, and bloodstream infection is 5.7%–35.0%[28]. Inflammatory cytokines disrupt the BBB, allowing peripheral immune cells to invade brain parenchyma and induce brain sepsis, aggravating cerebral ischemia-reperfusion injury. Additionally, systemic inflammatory response syndrome(SIRS) can exacerbate cerebral edema and intracranial hypertension[29].

4. Neurological Scoring Systems

4.1 Traditional Scores

The Cerebral Performance Category (CPC), modified Rankin Scale (mRS), and Glasgow Coma Scale (GCS) were widely used tools for assessing resuscitation quality before the ECMO era. However, in the ECPR setting, they exhibit three major limitations: All three rely on subjective physical examination and cannot quantify molecular or physiological damage from ischemia-reperfusion[30]; ECMO cannulation, temperature control, and continuous infusion of analgesic-sedative agents can artificially reduce the GCS to “3T” within 24 hours, resulting in 25%–30% false positives; CPC/mRS can only be determined at discharge or 3-month follow-up, offering no guidance for early decisions regarding continuation of life support[31].

4.2 NECPR Score

In 2019, the team led by Ryu derived the “nECPR score” based on V-A ECMO patients over 8 years from a single center[32]. This was the first proposal to incorporate five variables obtainable within 12 hours after cannulation into a logistic model. All components of this scoring system are obtainable within 12 hours of ECMO cannulation. This characteristic provides a timely and objective basis for early clinical decisions, including continuation or withdrawal of life-supporting treatments.

4.3 Modified OHCA Score

In 2021, Song et al. proposed a modification to the existing OHCA score by introducing objective neuroimaging variables to replace the conventional no-flow time component[33]. The study demonstrated that this modified score significantly improved predictive accuracy, with an area under the receiver operating characteristic curve (AUROC) of 0.96 (95% CI 0.90–0.99), a sensitivity of 74.5%, and a specificity of 100% for predicting poor neurological outcomes at 3 months

5. Categories of Assessment Modalities

5.1 Bedside Ultrasound

Transcranial Doppler(TCD) enables early detection of elevated ICP via pulsatility index (PI) and end-diastolic velocity (EDV)[34]. However, interpretation is highly operator-dependent. Optic Nerve Sheath Diameter(ONSD)≥7.2 mm on day 1 after CA is associated with a doubled in-hospital mortality, with an optimal cut-off of 6.5mm[35]. Combining ONSD with PI improves predictive accuracy for HIBI.

5.2 Cerebral Oxygenation Monitoring

In the setting of hypoxic-ischemic brain injury after cardiac arrest, both regional and global cerebral oxygenation monitoring play critical roles in prognostication and pathophysiological characterization. Regional cerebral oxygen saturation (rSO_2) ≤15% has been validated as a biological threshold for “futile ECPR” [36]. $SjvO_2$ reflects the global cerebral oxygen extraction ratio (OER). $SjvO_2$ can identify not only inadequate oxygen delivery but also the "impaired oxygen utilization" pathological phenotype specific to HIBI[37].

5.3 Electrophysiological Assessment

5.3.1 Conventional electroencephalography

The primary value of conventional EEG lies in identifying “highly malignant patterns” to predict poor neurological outcomes. However, its clinical utility is constrained by two major limitations: first, it has high specificity but limited sensitivity, which predisposes to false-negative results; second, it is susceptible to interference from sedative medications, which restricts its early application[38].

5.3.2 Amplitude-integrated electroencephalography

Continuous amplitude-integrated electroencephalography (aEEG) offers a bedside-accessible, real-time tool for early neurological prognostication in comatose patients after cardiac arrest. A prospective ECPR+TTM cohort[39] demonstrated that CNV background within 24 hours predicted good outcome with 100% sensitivity, 93% specificity, 83% positive predictive value, and 100% negative predictive value.

5.3.3 Bispectral index

BIS integrates time-domain, frequency-domain, and higher-order spectral features into a 0–100 dimensionless index. Arbas-Redondo et al. reported that in the first 12 hours of TTM, a mean BIS value >26 predicted good neurological outcomes (CPC 1–2) with a sensitivity of 89.5% and a specificity of 75.8%. The same study also found that a mean suppression ratio(SR) >24 in the first 12 hours predicted poor outcomes with 91.5% sensitivity and 81.8% specificity[40], which suggest that early BIS monitoring offers valuable prognostic information in post-arrest care.

5.4 Biomarkers

5.4.1 Neuron-specific enolase

NSE is the γ-γ enolase isoenzyme, located in the cytoplasm, with a half-life of 24 hours. Serum neuron-specific enolase (NSE) serves as a useful adjunct biomarker for neurological prognostication after cardiac arrest[41]. Hemolysis is the major confounding factor, particularly common in ECMO patients (due to pump head shear stress and circuit thrombosis), requiring cautious interpretation[42].

5.4.2 S100B

S100B is a calcium-binding protein primarily found in astrocytes, with a half-life of approximately 30–60 minutes, making it suitable for dynamic monitoring. In a retrospective cohort study[43] of 426 patients undergoing V-V ECMO demonstrated that serum S100B levels were

significantly higher in patients with newly detected intracranial pathology. A cut-off value of 1.52 μg/L for S100B was associated with detrimental outcomes, characterized by significantly reduced median survival.

5.4.3 Emerging Biomarkers

Glial fibrillary acidic protein(GFAP), ubiquitin carboxy-terminal hydrolase L1(UCH-L1), and neurofilament light chain(NfL) are significantly elevated after ROSC[44]. NfL has shown tremendous potential in prognostic assessment after cardiac arrest. An international multicenter prospective study[45] confirmed that serum NfL levels at 1–3 days after ROSC had superior predictive performance for poor neurological outcomes at 6 months compared with other biomarkers and standardized neurological monitoring modalities.

5.5 Imaging Assessment

5.5.1 CT

CT evaluation of HIBI is often negative in the early phase. In CA patients, diffuse cerebral edema, narrowed ventricles, and loss of gray-white matter differentiation appearing at 24 hours suggest HIBI[46].

5.5.2 MRI

Compared with CT, Diffusion-weighted imaging–apparent diffusion coefficient (DWI-ADC) can identify ischemic lesions within <1 hour. A whole-brain ADCmean $<650\times10^{-6}$ mm²/s predicts poor outcomes (PPV 94%)[47]. The optimal scanning window is 48–72 hours after ROSC; scanning earlier than 96 hours can reduce false positives.

5.5.3 Bedside point-of-care low-field MRI

Conventional 3.0T MRI is contraindicated in ECMO patients due to ferromagnetic components. A 0.064T low-field system can perform scans at the bedside, with detection rates for acute ischemic stroke consistent with 1.5T MRI (κ=0.92), magnetic induction heating <0.5°C, and no adverse events, providing real-time neuroimaging for ECMO patients[48].

5.6 Multimodal Integration

Given the limitations of single assessment modalities, multimodal integration has become the core concept of neurological prognostication after ECPR. Integrating five dimensions of information—temporal parameters, structural imaging, biochemical markers, electrophysiology, and quantitative EEG—may overcome the limitations of any single indicator and holds promise for early and comprehensive assessment of the extent of neurological injury. Multicentre validation will be essential to determine the applicability of this framework across different healthcare settings and patient populations, thereby providing more reliable decision support for neurological prognostication after ECPR.

6. Conclusions and Future Perspectives

ECPR offers hope for survival in patients with refractory cardiac arrest, yet HIBI remains the primary bottleneck limiting long-term neurological functional recovery. Neurological prognostication is a complex clinical decision-making process requiring comprehensive

consideration of patient baseline characteristics, cardiac arrest type, ECMO operational parameters, and multimodal monitoring information.

Current evidence indicates that: Low-flow time is the strongest modifiable prognostic factor, with an ideal window of 45–60 minutes, and requires risk stratification incorporating age and initial rhythm; Traditional neurological scores have significant limitations in the ECPR setting, while the nECPR score and modified OHCA score provide early predictive tools; Bedside ultrasound, cerebral oxygenation monitoring, electrophysiological assessment, biomarkers, and imaging each have advantages and disadvantages, with no single "gold standard"; Multimodal integration models and the 2025 ERC/ESICM time-adaptive framework represent the direction of development in this field.

Future research should focus on: Validating specific thresholds for emerging biomarkers such as NfL and GFAP in large ECPR cohorts; Promoting the application of 0.064T bedside low-field MRI in ECMO patients; Constructing artificial intelligence-based multi-parameter dynamic prediction models; Conducting ECPR-specific neuroprotection randomized controlled trials; Establishing standardized international ECPR neurological prognostication registries to promote the accumulation of evidence-based medical evidence.

Through multidisciplinary collaboration, standardized monitoring pathways, and prudent prognostic judgment, we can hope to maximize long-term neurological functional recovery and quality of life for patients while improving ECPR survival rates.

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