International Journal of Emergency Medicine

REVIEW

Open Access

Physiological monitoring and physiology-informed strategies in cardiac arrest: current evidence and clinical translation

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Abstract

Background Despite advances in cardiac arrest management, survival with favorable neurological outcomes remains limited. Physiological monitoring may complement standardized resuscitation by providing real-time information about circulatory effectiveness, reversible causes, and treatment responses. However, whether physiology-guided interventions improve patient-centered outcomes remains uncertain. This narrative review examines the current evidence and clinical applicability of physiological monitoring during resuscitation, physiology-informed rescue strategies, and individualized post-cardiac arrest care.

Main Body During cardiopulmonary resuscitation, invasive arterial pressure monitoring, waveform capnography, and echocardiography provide complementary information about the circulation generated by chest compressions and potential mechanisms of resuscitation failure. Although observational and experimental studies support their physiological relevance, no adult diastolic pressure target or pressure-guided vasopressor strategy has been validated to improve survival or neurological recovery. Recent randomized trials of transesophageal echocardiography have demonstrated physiological or procedural effects without establishing clinical benefit. Among rescue strategies, extracorporeal cardiopulmonary resuscitation has yielded inconsistent results across randomized trials, with potential benefits dependent on patient selection, intervention timing, and system capabilities. Resuscitative endovascular balloon occlusion of the aorta has not improved clinical outcomes in randomized evaluation, while head-up resuscitation remains investigational. Following the return of spontaneous circulation, randomized trials have challenged the benefits of uniformly higher blood pressure and oxygenation targets, mild hypercapnia, and deeper or prolonged temperature control. Individualized approaches incorporating cerebral autoregulation, venous congestion, and cardiopulmonary physiology are physiologically appealing but lack prospective validation.

Conclusion Physiological assessment may complement established resuscitation algorithms by refining clinical decisions during and after cardiac arrest. However, improvements in physiological measurements should not be equated with improved patient outcomes. Future trials should determine whether predefined, physiology-guided treatment strategies improve survival and neurological recovery.

Keywords Cardiac arrest, Cardiopulmonary resuscitation, Neurologic outcome, Extracorporeal cardiopulmonary resuscitation, Hemodynamic monitoring

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Introduction

Despite substantial advances in cardiopulmonary resuscitation (CPR) systems of care, neurological injury remains the leading cause of death and long-term disability after cardiac arrest [1, 2]. Although return of spontaneous circulation (ROSC) rates have improved in many settings, survival with meaningful neurological recovery continues to lag behind [3, 4]. This gap reflects an important reality: successful resuscitation is not synonymous with brain salvage.

Traditional resuscitation algorithms emphasize simplicity, reproducibility, and rapid implementation and remain the foundation of cardiac arrest care [5, 6]. In selected monitored settings, physiological measurements may complement these algorithms by showing whether CPR is generating adequate perfusion and whether a specific intervention produces the expected response. However, evidence that this approach improves survival or neurological outcomes remains limited, and several monitoring methods are not widely available. Patients also differ in arrest etiology, no-flow duration, vascular tone, ventricular function, and response to treatment, which provides the rationale for studying more individualized resuscitation strategies [6, 7].

The approaches discussed in this review differ in how physiological information is used and in their readiness for clinical application. Arterial pressure, end-tidal carbon dioxide, POCUS, and TEE can provide real-time information that may lead to changes in chest compressions, vasopressor administration, or treatment of reversible causes [7–10].

In contrast, head-up CPR, REBOA, and ECPR are discussed here as physiology-informed rescue strategies: their use is supported by physiological rationale, while initiation depends primarily on patient selection, timing, and available resources [5, 10–13]. This distinction concerns the decision to initiate a rescue intervention and does not exclude subsequent adjustment using physiological measurements. Post-arrest management represents a third domain in which individualized hemodynamic, respiratory, and neurological targets are being investigated.

This narrative review examines how physiological measurements obtained during and after cardiac arrest can inform clinical management, with a focus on clinicians involved in adult resuscitation and post-cardiac arrest care. For the purposes of this review, physiology-guided care refers to the use of patient-specific physiological measurements obtained in real time to select or adjust treatment. We also review emerging monitoring methods, their potential influence on treatment, their practical limitations, and the current gaps between physiological rationale and evidence of improved clinical outcomes.

Methods

This narrative review was prepared with reference to the principles of the Scale for the Assessment of Narrative Review Articles (SANRA) [14]. We searched PubMed/MEDLINE, Embase, from database inception through July 2026 using combinations of terms related to cardiac arrest, physiology-guided resuscitation, hemodynamic monitoring, echocardiography, head-up CPR, REBOA, ECPR, and post-arrest physiological targets. Reference lists of relevant guidelines, reviews, and primary studies were also examined. Original randomized trials were prioritized for treatment effects, and original observational or experimental studies were used for monitoring associations and mechanistic findings. Guidelines were used to distinguish current practice from investigational approaches; secondary reviews were retained for background and synthesis. Physiological endpoints, ROSC, survival, and neurological outcomes were considered separately. Selection was narrative rather than exhaustive, and no formal risk-of-bias assessment, evidence grading, or consensus process was performed.

Physiological foundations of modern resuscitation

Blood flow during closed-chest CPR is generated by a variable combination of direct cardiac compression and changes in intrathoracic pressure. External force displaces blood toward the arterial circulation, while chest recoil lowers intrathoracic and right atrial pressures, promoting venous return and ventricular filling. The relative contribution of these mechanisms varies among patients and may be influenced by chest anatomy, compression location, airway pressure, circulating volume, and ventricular compliance. Incomplete recoil or excessive ventilation may therefore reduce forward flow even when compression rate and depth remain within recommended ranges [15–17].

Coronary perfusion pressure (CPP) is defined as the difference between aortic diastolic pressure and right atrial pressure during decompression. Paradis and colleagues demonstrated a close association between this gradient and the probability of ROSC [18](Fig. 1). Although the aortic component often receives greater attention, right atrial pressure (RAP) is equally relevant. Elevated RAP during decompression may result from incomplete chest recoil, increased intrathoracic pressure, impaired venous drainage, right-sided cardiac distension, or mechanical obstruction. In these situations, coronary perfusion and venous return may remain inadequate despite an apparently acceptable arterial diastolic pressure.

The mechanical response of the chest can also change during prolonged CPR. Repeated compressions may alter chest-wall compliance or produce thoracic injuries, changing the transmission of external force to the heart and great vessels. The same compression depth may

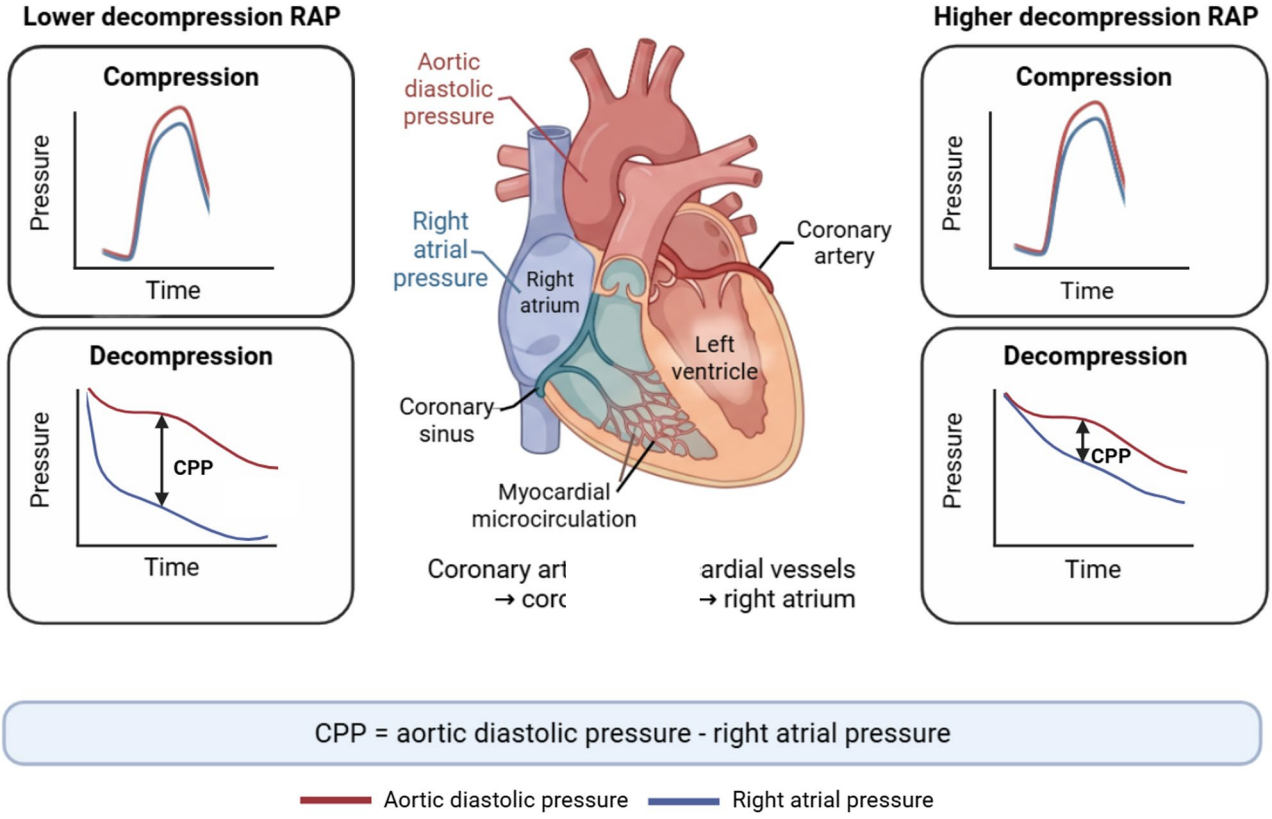


Fig. 1 Aortic and right atrial pressures during cardiopulmonary resuscitation. The central illustration depicts the coronary circulation from the aorta through the myocardial microvasculature and coronary sinus to the right atrium. During decompression, coronary perfusion pressure is approximated by the difference between aortic diastolic pressure and right atrial pressure. For the same aortic pressure, a higher right atrial pressure reduces this gradient. Red and blue curves represent aortic and right atrial pressures, respectively; double-headed arrows indicate coronary perfusion pressure. The lower panels provide expanded schematic views of decompression rather than continuous extensions of the upper panels. Waveforms are illustrative and do not represent patient recordings or treatment targets. CPR, cardiopulmonary resuscitation; CPP, coronary perfusion pressure; RAP, right atrial pressure. Created in BioRender. Melo, R. (2026) https://BioRender.com/ea1uz9c

therefore have different hemodynamic effects as resuscitation progresses. Conventional feedback devices may not detect this deterioration because they measure how compressions are delivered rather than the blood flow they generate [16, 19].

These limitations have encouraged the use of physiological signals as complementary measures of CPR effectiveness. Arterial pressure, end-tidal carbon dioxide, echocardiography, and selected measures of cerebral or systemic blood flow can provide information about the circulation generated during resuscitation. Transthoracic impedance and intrathoracic pressure are less established but may offer additional information in the future [20, 21].The PERSEUS protocol provided an early framework for integrating hemodynamic, ventilatory, and metabolic variables in highly monitored cardiac arrests, although it has not been prospectively validated as a treatment algorithm [22].

The following sections apply this framework to physiological monitoring during CPR, rescue interventions for refractory arrest, and individualized management after ROSC (Fig. 2).

Physiology-guided monitoring and CPR

Diastolic blood pressure monitoring during CPR

Among the physiological variables available during monitored cardiac arrest, DBP has emerged as one of the most clinically useful because it can be measured continuously and interpreted in real time throughout resuscitation. Adult observational studies have reported an association between higher intra-arrest DBP and an increased likelihood of ROSC, supporting its role as a physiological marker of CPR effectiveness [7–9, 23, 24]. Consequently, recent American Heart Association (AHA) guidelines acknowledge physiology-guided resuscitation as a reasonable adjunct when invasive arterial monitoring is already available [6].

DBP should be interpreted as a marker of the hemodynamic effect of CPR rather than as an isolated treatment target. Persistently low DBP should first prompt confirmation of waveform quality and reassessment of

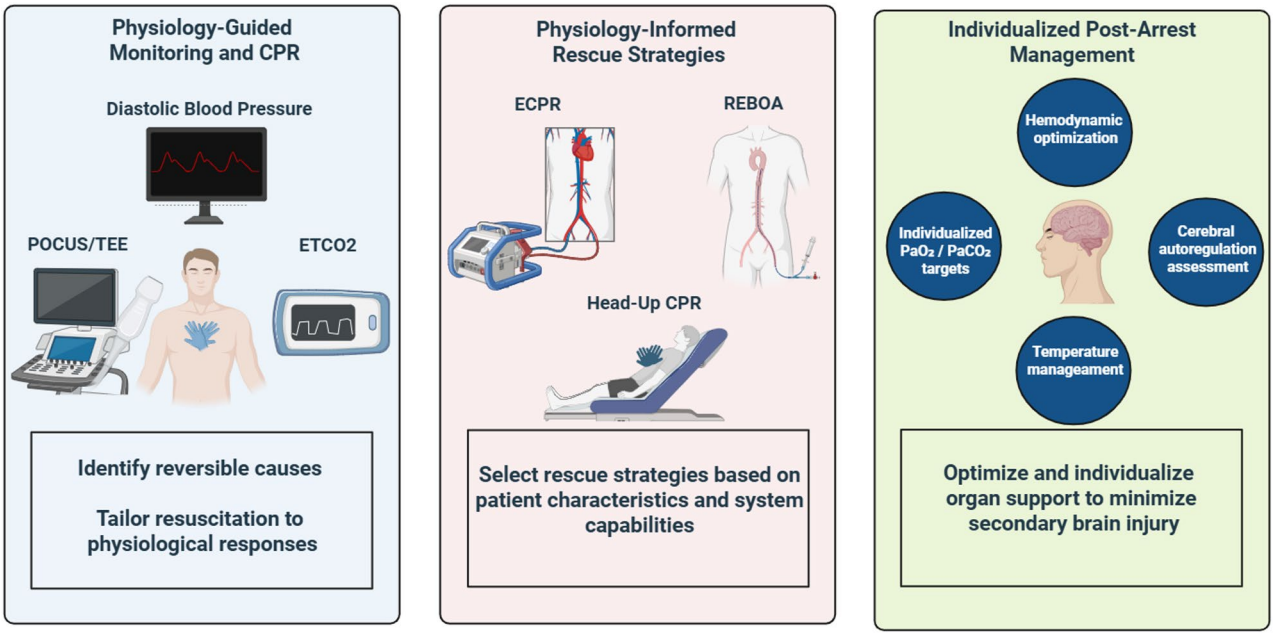


Fig. 2 Physiological monitoring and physiology-informed strategies across cardiac arrest care. Schematic representation of three complementary domains of cardiac arrest care: (Left) physiological monitoring to assess CPR effectiveness and identify reversible causes; (Center) rescue strategies selected according to patient characteristics and system capabilities; and (Right) individualized post-arrest management based on hemodynamic, respiratory, and cerebral physiology. These approaches do not represent a sequential treatment algorithm, and their clinical benefits vary according to the available evidence.CPR, cardiopulmonary resuscitation; DBP, diastolic blood pressure; ECPR, extracorporeal cardiopulmonary resuscitation; $ETCO_2$, end-tidal carbon dioxide; $PaCO_2$, arterial partial pressure of carbon dioxide; PaO_2, arterial partial pressure of oxygen; POCUS, point-of-care ultrasound; REBOA, resuscitative endovascular balloon occlusion of the aorta; TEE, transesophageal echocardiography. Created in BioRender. Melo, R. (2026). https://BioRender.com/03399tl

compression depth, hand position, chest recoil, ventilation, and potentially reversible causes. The arterial pressure response to epinephrine may provide additional physiological information, but no validated strategy supports modifying vasopressor dose or timing according to DBP. Experimental studies suggest that hemodynamic-directed CPR can improve perfusion and short-term survival compared with a uniform resuscitation strategy [25, 26].

Despite its physiological rationale, important limitations remain. Evidence supporting DBP monitoring during adult CPR is predominantly observational, and randomized trials demonstrating improved clinical outcomes with DBP-guided interventions are lacking. In addition, invasive arterial monitoring is unavailable in most cardiac arrests, particularly outside monitored hospital environments. Although arterial catheter placement during ongoing CPR has been described in highly organized in-hospital and prehospital systems [27, 28], its implementation depends on expertise and resources and must not interfere with high-quality CPR. Accordingly, DBP monitoring may complement conventional resuscitation in selected settings, but specific DBP-guided treatment algorithms remain unvalidated.

Hemodynamic assessment of vasopressor response during cardiac arrest

Epinephrine remains a cornerstone of advanced life support because its α-adrenergic effects increase vascular tone, augment aortic diastolic pressure, and improve the likelihood of ROSC. In the PARAMEDIC2 trial, epinephrine increased 30-day survival compared with placebo (3.2% vs. 2.4%), without a statistically significant improvement in favorable neurological outcomes [29]. Observational studies have associated higher cumulative epinephrine doses with poorer neurological outcomes [30, 31]. However, these associations are difficult to interpret because patients requiring more doses generally experience longer and more refractory arrests. Moreover, analyses restricted to survivors are susceptible to selection bias. These findings do not establish a causal relationship between cumulative epinephrine exposure and neurological injury.

These observations have stimulated interest in a more individualized approach to vasopressor administration during CPR. When invasive arterial monitoring is available, the hemodynamic response to epinephrine can be assessed in real time. A limited pressure response may reflect severe vasoplegia, inadequate circulating volume, impaired drug delivery during low-flow CPR, or a persistent mechanical cause of circulatory failure. Conversely,

an increase in arterial pressure indicates a measurable hemodynamic response but does not necessarily imply adequate coronary or cerebral perfusion [24, 32, 33].

This physiological approach remains investigational. No randomized trial has demonstrated that tailoring vasopressor therapy according to arterial pressure improves survival or neurological outcomes compared with current guideline-based practice. Similarly, although combination regimens including vasopressin and corticosteroids have shown encouraging results in selected studies of in-hospital cardiac arrest, evidence remains insufficient to support routine adoption [33–35]. More experimental strategies, including intra-aortic epinephrine administration, have also been proposed to improve drug delivery during low-flow CPR, with preliminary physiological findings but no established clinical benefit [36, 37]. At present, physiological monitoring may complement conventional vasopressor administration in selected monitored environments but should not replace established resuscitation algorithms.

Waveform capnography during CPR

Waveform capnography has two established uses during resuscitation: confirming and monitoring tracheal tube placement and detecting changes compatible with ROSC [38–40]. During CPR, ETCO2 also reflects delivery of carbon dioxide to the lungs and can provide an indirect signal of pulmonary blood flow when ventilation is reasonably stable. In an observational study, ETCO2 was associated with chest compression depth and ventilation rate, highlighting its sensitivity to both circulatory and ventilatory factors [41]. These findings support monitoring, not proof that treatment titrated to an ETCO2 target improves survival.

Low or falling ETCO2 should prompt assessment of compressions, ventilation, airway position, and circuit integrity. A sustained abrupt increase may indicate ROSC and should be checked against the rhythm, arterial waveform, and clinical findings without unnecessary interruption of compressions. Values below 10 mmHg have been associated with poor outcomes, while values above 10 mmHg, and preferably above 20 mmHg, may support mechanically adequate CPR [6, 41]. They are not standalone treatment thresholds or guarantee of adequate cerebral or coronary perfusion.

Interpretation is affected by ventilation, dead space, arrest etiology, airway technique, and drug administration. Sodium bicarbonate can transiently increase exhaled carbon dioxide, and vasopressors may change pulmonary perfusion independently of compression quality. Trends are therefore more useful when compared under similar ventilatory conditions. The historic association between low ETCO2 after prolonged CPR and death arose from selected intubated patients with pulseless electrical activity [42]; it should not be generalized to every arrest. ETCO2 must not be used alone to decide termination of resuscitation.

POCUS and transesophageal echocardiography during arrest

POCUS can provide physiological and diagnostic information during cardiac arrest, particularly by identifying potentially reversible causes such as cardiac tamponade, severe hypovolemia, tension pneumothorax, and ventricular dysfunction. However, findings must be interpreted in context: right ventricular dilation alone does not establish pulmonary embolism, and the presence of cardiac motion does not necessarily indicate effective perfusion. When integrated into structured rhythm checks [43], ultrasound may help distinguish true pulseless electrical activity from pseudo-pulseless electrical activity and inform targeted interventions, provided that image acquisition does not prolong interruptions in chest compressions [6, 10, 44].

Transesophageal echocardiography (TEE) allows continuous cardiac imaging during chest compressions, providing information about ventricular filling, cardiac compression, and potential mechanical causes of inadequate forward flow. It may identify ineffective ventricular compression or left ventricular outflow tract obstruction, allowing adjustment of hand position or mechanical compression devices. A published case report of relief of LVOT obstruction illustrates this physiological mechanism but does not establish improved clinical outcomes [45–47].

Recent randomized trials have evaluated whether these physiological and procedural advantages translate into measurable benefits. In EXECT-CPR, 132 patients with ongoing out-of-hospital cardiac arrest were randomized to TEE-guided compression-site adjustment or conventional CPR. Sustained ROSC occurred in 44% and 39%, respectively (cluster-adjusted odds ratio, 1.21; 95% CI, 0.64–2.29), without significant differences in survival or neurological outcomes. ETCO2 was higher in the TEE group during the 11th to 20th minutes after emergency department arrival [48]. The trial was underpowered to detect the anticipated treatment effect, and its findings do not establish clinical superiority.

In the PHTEE-OHCA pilot trial, 35 patients were randomized and 32 analyzed. TEE was associated with a higher chest compression fraction (96.2% versus 91.6%), while median hands-off time was 4 s in both groups. Sustained ROSC occurred in 40% of the TEE group versus 71% of controls (p = 0.083) [49]. This small pilot study was not powered to assess clinical outcomes, and between-group differences limit interpretation of the ROSC findings. The results support the feasibility of

prehospital TEE in physician-staffed systems but do not establish a survival benefit.

Together, these findings indicate that TEE can provide physiological information and improve selected procedural measures, but its impact on patient-centered outcomes remains uncertain. Both transthoracic and transesophageal approaches require trained operators and integration into resuscitation protocols that minimize interruptions. TEE also requires appropriate equipment and consideration of probe-related complications. Its clinical value ultimately depends on whether the information obtained leads to a timely and effective intervention.

Physiology-informed rescue strategies

The strategies discussed in this section are supported by physiological principles but differ from conventional physiology-guided monitoring in their indications and implementation. Their initiation depends primarily on patient selection, timing, available expertise, and system organization, although subsequent adjustments may be guided by physiological measurements. Their inclusion in this review does not imply equivalent evidence or readiness for clinical adoption.

REBOA: physiological rationale and clinical evidence

REBOA has emerged as a potential adjunctive strategy for refractory cardiac arrest by temporarily redistributing the limited blood flow generated during CPR toward the heart and brain. Occlusion of the descending thoracic aorta increases proximal aortic pressure while reducing distal blood flow, potentially augmenting coronary and cerebral perfusion during the low-flow state of resuscitation [11, 50, 51].Experimental studies have demonstrated improvements in proximal arterial pressure, CPP, and cerebral perfusion following aortic occlusion, although these findings do not establish improved clinical outcomes.

Partial REBOA has been proposed to preserve some distal perfusion while maintaining proximal pressure augmentation, potentially reducing ischemic injury associated with complete aortic occlusion. Experimental studies have demonstrated the feasibility of adjusting the degree of aortic occlusion using proximal and distal arterial pressure measurements, although its applicability to non-traumatic cardiac arrest remains uncertain [52, 53].

The REBOARREST trial randomized 200 patients with non-traumatic out-of-hospital cardiac arrest, of whom 179 were included in the reported analysis. Sustained ROSC occurred in 28% of patients receiving REBOA plus advanced life support and 26% receiving advanced life support alone (adjusted risk difference, 1.8%; 95% CI, − 11 to 15; $P = 0.78$) [50]. Although ETCO2 increased significantly after aortic occlusion, no significant improvement in survival or favorable neurological outcomes was demonstrated. These findings illustrate that physiological changes do not necessarily translate into clinical benefit. Interpretation of this trial is limited by deployment timing, its predominantly rural setting, and exclusions following randomization. These considerations may affect generalizability but do not alter the neutral primary result. Furthermore, REBOA requires rapid femoral arterial access, specialized equipment, and trained operators. Current evidence does not support its routine use in non-traumatic cardiac arrest, and its role remains investigational.

Extracorporeal CPR: shortening the low-flow state

Among the advanced rescue strategies discussed in this review, ECPR has undergone the most extensive clinical evaluation. However, evidence of improved survival with favorable neurological outcome is inconsistent and strongly dependent on patient selection and system performance. Unlike interventions that optimize the limited circulation generated by conventional CPR, ECPR restores extracorporeal blood flow while the underlying cause of arrest is identified and treated [54–56].

The potential neurological benefit of ECPR is intrinsically time dependent. Restoration of extracorporeal circulation shortens the duration of low-flow cerebral ischemia, providing an opportunity to preserve viable brain tissue while definitive therapies, including coronary reperfusion, pulmonary thrombectomy, or treatment of other reversible causes, are undertaken. Consequently, ECPR should be viewed not simply as a rescue device, but as part of an integrated reperfusion strategy in which patient selection, rapid transport, timely cannulation, and coordinated post-resuscitation care are equally important determinants of outcome.

Randomized trials have produced discordant results. The single-center ARREST trial demonstrated a benefit within a highly organized pathway for patients with refractory ventricular fibrillation [5], whereas the Prague OHCA [57] and INCEPTION [13] trials did not demonstrate a statistically significant improvement in their primary intention-to-treat outcomes. These trials evaluated different patient populations and treatment pathways, with Prague OHCA specifically assessing a bundled invasive strategy rather than ECPR alone. Collectively, their findings do not establish broad efficacy for ECPR, although selected patients may benefit within highly organized systems capable of rapid cannulation and definitive treatment of the arrest cause.

The impact of head-up positioning on cardiopulmonary resuscitation

Head-up cardiopulmonary resuscitation (HUP-CPR) has emerged as a novel strategy aimed at improving cerebral

perfusion during cardiac arrest. The physiological rationale is based on reducing intracranial pressure while maintaining arterial inflow, thereby increasing cerebral perfusion pressure during the low-flow state of CPR. Experimental studies have consistently demonstrated favorable effects on cerebral hemodynamics, including lower intracranial pressure and improved cerebral and coronary perfusion pressures compared with conventional supine CPR [58–60].

However, these physiological benefits should be interpreted within the context in which HUP-CPR has been studied. Most experimental models combine gradual head and thorax elevation with active compression-decompression CPR and impedance threshold devices following an initial priming period to establish forward blood flow. Consequently, the available evidence reflects a bundled resuscitation strategy rather than body positioning alone, and these components should not be considered interchangeable.

Whether these physiological improvements translate into better neurological outcomes in humans remains uncertain. Current evidence is derived predominantly from experimental studies and surrogate physiological endpoints, whereas clinical outcome data remain limited. Additionally, clinical translation remains challenging because head-up CPR has generally been evaluated as part of a bundle that includes automated elevation, mechanical chest compressions, and an impedance threshold device. Observational data suggest that outcomes may depend on the timing of device application and on establishing adequate circulation before elevation [61]. Accordingly, current AHA guidelines discourage head-up CPR outside clinical trials.

Individualized post-arrest management

Hemodynamic and cerebral perfusion targets

Following ROSC, systemic arterial pressure may not reliably reflect cerebral perfusion because post-arrest myocardial dysfunction, vasoplegia, and impaired cerebral autoregulation can coexist. In the BOX trial, targeting a MAP of 77 rather than 63 mmHg did not improve clinical outcomes [62]. Smaller randomized studies similarly failed to demonstrate consistent benefits from higher fixed MAP targets, although cerebral oxygenation may increase with higher arterial pressure [63].

Vasoactive drugs are frequently required after ROSC to maintain arterial pressure and organ perfusion. Norepinephrine is commonly used when vasodilation predominates, while inotropes such as dobutamine may be considered when myocardial dysfunction and low cardiac output persist despite adequate preload and arterial pressure. However, cerebral perfusion may also be compromised by venous congestion. In an observational study of 48 post-cardiac arrest patients, Ameloot et al. found that central venous pressure was more strongly associated with cerebral oxygen saturation than MAP, even after accounting for cardiac output [64]. These findings suggest that a fixed MAP target may not ensure adequate cerebral oxygenation in patients with elevated venous pressure. Thus, individualized hemodynamic management after ROSC may require consideration of both arterial pressure and venous congestion, although whether interventions targeting these variables improve neurological outcomes remains unknown.

Beyond the influence of venous congestion, impaired or shifted cerebral autoregulation after hypoxic-ischemic injury represents another limitation of fixed MAP targets. Near-infrared spectroscopy has been used to estimate an individual MAP associated with preserved cerebral autoregulation [65], while transcranial Doppler can provide complementary information about cerebral blood flow [66]. However, these methods remain investigational. No prospective trial has demonstrated that targeting an autoregulation-derived MAP improves neurological outcomes. These findings further highlight the limitations of uniform MAP targets after cardiac arrest and the need to evaluate whether individualized targets based on cerebral physiology can improve clinical outcomes.

Oxygenation and ventilation

Arterial oxygenation is only one determinant of cerebral oxygen delivery, which also depends on hemoglobin concentration, cardiac output, and cerebral blood flow. Following ROSC, impaired cerebrovascular function may further dissociate arterial oxygenation from tissue oxygen availability. Randomized trials comparing different PaO2 targets have not demonstrated significant improvements in neurological outcomes with higher oxygenation targets [67]. In a randomized pilot trial, moderate hyperoxia increased cerebral oxygen saturation measured by NIRS without reducing biomarkers of neurological injury [68].

PaCO2 is a major determinant of cerebral vascular tone and blood flow, but its effects must also be considered in the context of systemic and cardiopulmonary physiology. Targeting mild hypercapnia (50–55 mmHg) rather than normocapnia (35–45 mmHg) did not improve neurological outcomes at six months in a large randomized trial [69]. However, uniform PaCO2 targets may not be appropriate for every patient. In patients with chronic hypercapnia, reducing PaCO2 substantially below baseline may be undesirable. Conversely, hypercapnia and respiratory acidosis may increase pulmonary vascular resistance and worsen right ventricular loading conditions in patients with pulmonary hypertension or right ventricular dysfunction. Hypercapnia-induced cerebral vasodilation may also increase intracranial pressure in patients with impaired intracranial compliance. These considerations may justify individualized adjustments,

although no post-arrest trial has validated a phenotype-guided PaCO2 strategy. Normocapnia remains the recommended initial target, with arterial blood gases used to guide adjustments.

Temperature management

Temperature control after cardiac arrest aims to limit secondary hypoxic-ischemic brain injury, although the optimal temperature and duration of treatment remain uncertain. Randomized comparisons of 33 °C versus 36 °C and of 33 °C versus targeted normothermia with early treatment of fever did not demonstrate improved survival or functional outcomes with the lower temperature target [70, 71]. A separate trial in patients with nonshockable rhythms found a modest increase in favorable neurological outcome with a target of 33 °C rather than 37 °C, without a reduction in mortality [72]. More recently, a randomized trial involving 1,158 patients found no improvement in neurological outcomes with longer durations of cooling at 33 °C [73]. These findings suggest that neither a lower temperature target nor prolonged cooling provides consistent benefit across the populations studied. However, differences in the severity and mechanisms of hypoxic-ischemic injury may influence treatment response, and no physiological or clinical marker has been validated to identify patients who might benefit from deeper or longer cooling. Protocolized temperature control and fever prevention remain important components of post-arrest care.

Clinical applicability, implementation challenges, and current limitations

The approaches discussed in this review differ in physiological rationale, evidence, feasibility, and resource requirements (Table 1). High-quality CPR and structured post-arrest care remain universal foundations, whereas invasive monitoring, TEE, REBOA, and ECPR are largely restricted to organized systems with appropriate

Table 1 Physiological rationale and evidence base of the approaches discussed in this review

Approach	Physiological target	Proposed mechanism	Evidence base	Clinical interpretation
DBP monitoring	Aortic DBP and coronary perfusion pressure	Improve myocardial and cerebral perfusion	Human observational and experimental studies; no adult RCT of DBP-guided CPR [7, 8, 24–26]	Useful monitoring adjunct when available; no validated adult DBP target or treatment algorithm.
Vasopressor response assessment	Arterial pressure response during CPR	Assess pressure response to vasopressors	RCT evidence for epinephrine; observational evidence for response-guided assessment [29, 30, 32]	Investigational; should not replace guideline-based dosing
POCUS and TEE	Reversible causes and compression effectiveness	Identify treatable pathology and guide compression position	Diagnostic studies and randomized TEE trials showing procedural or physiological effects without established clinical benefit [43, 46, 48, 49]	Adjunct for experienced operators
Head-up CPR	Cerebral perfusion pressure and intracranial pressure	Improve cerebral venous drainage and arterial inflow	Predominantly animal and observational bundled-protocol studies [59–61]	Investigational and protocol dependent
REBOA	Proximal aortic, coronary, and cerebral perfusion	Redistribute blood flow toward the heart and brain	Experimental and observational studies; neutral REBOARREST randomized trial [50–52]	Investigational; limited to specialized systems
ECPR	Systemic perfusion and low-flow duration	Restore circulation while treating the arrest cause	RCTs with discordant results; observational studies and meta-analyses [5, 13, 57]	Selected patients in protocolized ECPR systems
Individualized post-arrest hemodynamics	MAP and cerebral perfusion	Preserve cerebral blood flow	RCTs of fixed MAP targets were neutral; individualized targets remain observational [62–65]	Support systemic and cerebral perfusion while accounting for cardiac output, venous congestion, and cerebral autoregulation.
Post-arrest oxygenation and ventilation	PaO_2 and $PaCO_2$	Limit secondary cerebral injury	RCTs have not identified superior PaO_2 or $PaCO_2$ targets [67–69]	Avoid hypoxemia, marked hyperoxemia, and hypocapnia
Post-arrest temperature control	Temperature and fever prevention	Limit secondary cerebral injury	RCTs show no consistent benefit from routine cooling to 33 °C [70–73]	Protocolized temperature control; individualized temperature targets and cooling duration remain unvalidated.

Summary of the physiological targets, proposed mechanisms, predominant evidence base, and current clinical interpretation of the monitoring methods, rescue strategies, and post-arrest interventions discussed in this review. Evidence-base descriptions reflect the predominant study designs and do not represent a formal certainty-of-evidence assessment

CerPP, cerebral perfusion pressure; CPP, coronary perfusion pressure; CPR, cardiopulmonary resuscitation; DBP, diastolic blood pressure; ECPR, extracorporeal cardiopulmonary resuscitation; ICP, intracranial pressure; MAP, mean arterial pressure; NIRS, near-infrared spectroscopy; $PaCO_2$, arterial partial pressure of carbon dioxide; PaO_2, arterial partial pressure of oxygen; POCUS, point-of-care ultrasound; REBOA, resuscitative endovascular balloon occlusion of the aorta; ROSC, return of spontaneous circulation; TEE, transesophageal echocardiography

Table 2 Physiological findings, potential clinical responses, and current applicability of monitoring and rescue strategies during cardiac arrest

Approach or signal	Physiological finding	Potential clinical response	Validation and limitations	Current setting
CPR quality metrics	Inadequate compression depth, rate, recoil, or chest compression fraction	Correct compression technique and minimize interruptions	Mechanical metrics do not directly measure blood flow or organ perfusion [6, 16]	All cardiac arrest settings
Arterial DBP	Persistently low DBP during CPR	Confirm waveform quality; reassess compression depth, hand position, recoil, ventilation, and reversible causes; evaluate the pressure response to vasopressors	No validated adult DBP threshold or pressure-guided vasopressor dosing strategy [6– [8, 24]	Cardiac arrest with an arterial line
$ETCO_2$	Low or decreasing $ETCO_2$	Reassess compression quality, ventilation, airway position, and pulmonary blood flow; consider ROSC when $ETCO_2$ rises abruptly	Influenced by ventilation, airway obstruction, vasopressors, and arrest etiology [6, 38, 41, 42]	Cardiac arrest with capnography
POCUS	Findings suggestive of tamponade, pulmonary embolism, hypovolemia, or tension pneumothorax; cardiac activity without a palpable pulse.	Direct treatment toward the suspected reversible cause	Operator-dependent and must not prolong interruptions in chest compressions [6, 43, 44]	Settings with trained operators
TEE	Poor ventricular compression or compression-induced LVOT obstruction	Adjust hand position or mechanical compression device placement	Randomized trials have not established clinical benefit; requires trained operators and specialized equipment [46, 48, 49]	Specialized centers
Vasopressor response	Absent, limited, or brief arterial pressure increase after epinephrine	Reassess drug delivery, vascular tone, CPR-generated flow, and mechanical causes of circulatory failure	No validated strategy for changing vasopressor dose or interval [29, 32]	Cardiac arrest with an arterial line
Head-up CPR	No established bedside physiological trigger	No patient-specific adjustment can currently be recommended	Bundle-dependent; optimal timing and elevation strategy remain uncertain [6, 59–61]	Research protocols or selected expert systems
REBOA	Refractory arrest with inadequate central perfusion	Consider only within a predefined endovascular resuscitation pathway	No validated physiological threshold for initiation; REBOARREST did not improve sustained ROSC [50, 51]	Investigational
ECPR	Refractory arrest with a potentially reversible cause	Activate an established ECPR pathway according to predefined eligibility criteria	Outcomes depend on patient selection, low-flow time, and system performance [5, 13, 57]	Specialized ECPR systems

Potential clinical responses represent actions that may be considered after interpretation of the physiological finding. They should not be interpreted as validated treatment algorithms unless otherwise stated. DBP, diastolic blood pressure; ECPR, extracorporeal cardiopulmonary resuscitation; $ETCO_2$, end-tidal carbon dioxide; LVOT, left ventricular outflow tract; POCUS, point-of-care ultrasound; REBOA, resuscitative endovascular balloon occlusion of the aorta; ROSC, return of spontaneous circulation; TEE, transesophageal echocardiography

expertise and infrastructure. Table 2 links physiological findings to potential clinical responses and summarizes their validation, limitations, and current setting.

Physiological plausibility should not be interpreted as proof of clinical benefit. DBP-guided CPR, REBOA, head-up CPR, and individualized cerebral perfusion targets remain incompletely validated. For diagnostic tools such as POCUS and TEE, direct effects on survival are difficult to establish because benefit depends on whether findings lead to timely and effective treatment.

This review has several limitations. The evidence is heterogeneous and, for many interventions, is based on experimental studies, observational data, or physiological surrogate outcomes rather than patient-centered outcomes. The narrative design also means that study and topic selection may be influenced by author judgment. In addition, the feasibility and effects of these approaches may vary across resuscitation systems. The potential clinical responses discussed in the text and Table 2 have not been established as formal recommendations and should not replace current resuscitation guidelines.

Rather than replacing standardized resuscitation algorithms, physiology-guided strategies should be viewed as complementary tools that may refine decision-making in selected patients and specialized environments. Future progress in cardiac arrest care will likely depend not on a single intervention, but on integrating high-quality CPR with individualized physiological assessment, advanced reperfusion strategies when appropriate, and structured neuroprotective post-arrest care. The central message is not that algorithms should be abandoned, but that future cardiac arrest care may increasingly combine standardized fundamentals with individualized physiological

targets aimed at maximizing meaningful neurological recovery [21, 74, 75].

Conclusion

Physiological monitoring during CPR can provide clinically relevant information about circulatory effectiveness, treatment response, and potentially reversible causes. Physiology-informed rescue strategies may benefit selected patients, although their effectiveness depends on patient selection, timing, and system capabilities. After ROSC, differences in hemodynamic, respiratory, and cerebral physiology may justify individualized assessment beyond uniform treatment targets, but the clinical benefit of physiology-guided interventions remains unproven. Future trials should evaluate whether treatment adjustments based on specific physiological measurements improve survival and neurological recovery.

Abbreviations

ADP	Aortic diastolic pressure
CPP	Coronary perfusion pressure
CPR	Cardiopulmonary resuscitation
DBP	Diastolic blood pressure
ECPR	Extracorporeal cardiopulmonary resuscitation
EEG	Electroencephalography
$ETCO_2$	End-tidal carbon dioxide
HUP-CPR	Head-up cardiopulmonary resuscitation
ICP	Intracranial pressure
ITD	Impedance threshold device
MAP	Mean arterial pressure
NIRS	Near-infrared spectroscopy
POCUS	Point-of-care ultrasound
RAP	Right atrial pressure
REBOA	Resuscitative endovascular balloon occlusion of the aorta
ROSC	Return of spontaneous circulation
TEE	Transesophageal echocardiography

Author contributions

RHM conceived the review, performed the literature review, interpreted the evidence, designed the figures, drafted the manuscript, and coordinated manuscript revisions. DJ contributed to the interpretation of the literature, critically revised the manuscript for important intellectual content, and approved the final version. HPG contributed to the interpretation of the evidence, critically revised the manuscript for important intellectual content, and approved the final version. RHP conceived the overall scientific framework of the review, supervised manuscript development, critically revised the manuscript for important intellectual content, and approved the final version. All authors read and approved the final manuscript.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

This article is a narrative review of previously published literature and did not involve human participants, human data, human tissues, or animal experimentation performed by the authors. Therefore, ethics committee approval and informed consent were not required.

Consent for publication

Not applicable.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this manuscript, the authors used ChatGPT (OpenAI) and other generative artificial intelligence tools to assist with language editing and manuscript organization. BioRender was used to create and assemble elements of the figures. All AI-assisted content was critically reviewed and revised by the authors, who take full responsibility for the final content of the article.

Competing interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Received: 24 July 2026 / Accepted: 22 October 2026

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