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Review Article

Cardiopulmonary Bypass-Related Determinants of Adverse Postoperative Outcomes in Adult Cardiac Surgery: A Comprehensive Narrative Review

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ABSTRACT

Background: Cardiopulmonary bypass (CPB) remains the cornerstone of modern adult cardiac surgery. However, obligate exposure to the extracorporeal circuit precipitates a systemic inflammatory response syndrome (SIRS), hemodilution, and coagulopathy. These non-physiologic states drive significant multi-organ morbidity and perioperative mortality.
Objective: To present qualitative evidence on CPB-related determinants of adverse outcomes focusing on extent of extracorporeal exposure, inflammatory activation and systemic inflammatory response syndrome and coagulopathy. The review further aims to discuss postoperative multiorgan dysfunction and its actionable mitigation strategies in perioperative period.
Methods: A comprehensive literature search was conducted across PubMed, Cochrane Library, and Google Scholar up to 2026. Keywords included cardiopulmonary bypass, systemic inflammatory response, coagulopathy, acute kidney injury, transesophageal echocardiography, and perioperative outcomes.
Results: Prolonged CPB time more than 120 minutes independently increases postoperative mortality by 1.6-fold. The incidence of adverse outcomes including acute kidney injury (AKI), postoperative atrial fibrillation (POAF) and reoperation for bleeding (re-exploration) was 10–30%, 20–40% and 2–7% respectively. Evidence-based mitigation strategies demonstrate substantial efficacy and reduction in post operative adverse outcomes. Lung-protective ventilation reduces pulmonary complications by 25%, routine antifibrinolytic therapy decreases transfusion requirements by up to 30%, and goal-directed viscoelastic monitoring reduces bleeding and transfusion associated adverse outcomes. Furthermore, real-time advanced hemodynamic monitoring facilitates like transesophageal echocardiography

aid in early detection and management of hemodynamic compromise and cardiac dysfunction paving way for early action and mitigation.
Conclusion: The pathophysiological insults of CPB are predictable and highly modifiable. Standardized implementation of evidence-based perioperative bundles, driven by precise pharmacological, mechanical, and intraoperative monitoring strategies, drastically improves clinical outcomes and optimizes resource utilization in adult cardiac surgery.

Keywords: *cardiopulmonary bypass, systemic inflammatory response, extracorporeal circulation, coagulopathy, transesophageal echocardiography, postoperative outcomes, adult cardiac surgery.*

INTRODUCTION

Since its successful clinical introduction by John Gibbon in 1953, cardiopulmonary bypass (CPB) has evolved into an indispensable technology, facilitating over one million adult cardiac surgeries globally each year [1,2]. By providing a bloodless, motionless surgical field while maintaining systemic perfusion and gas exchange, CPB allows for intricate structural heart repairs, complex coronary revascularization, and major aortic reconstructions.

Despite its life-saving nature, the extracorporeal circuit acts as a profound physiological stressor. The obligate contact of human blood with vast synthetic surface areas, combined with non-pulsatile flow, hemodilution, and ischemia-reperfusion injury, reliably triggers a systemic inflammatory response syndrome (SIRS) [3,4]. This systemic derangement contributes heavily to postoperative morbidity, including acute kidney injury (AKI), acute respiratory distress syndrome (ARDS), neurocognitive decline, and severe coagulopathy which subsequently prolongs intensive care unit (ICU) stays and increases healthcare expenditures [5].

The transition from purely operative survival to optimizing long-term morbidity requires an understanding of CPB-induced pathophysiology. This comprehensive narrative review synthesizes the current statistical evidence surrounding CPB-related risk determinants and highlights modifiable, highly effective interventions that multidisciplinary teams comprising cardiac surgeons, anesthesiologists, and perfusionists as a team can seamlessly integrate into clinical practice.

STATISTICAL METHODS AND DATA SYNTHESIS:

The present narrative review, a qualitative literature search and analysis was performed rather than a quantitative systematic review and meta-analysis. For each major cardiopulmonary bypass–related complication (pulmonary complications, acute kidney injury, postoperative atrial fibrillation, stroke/major neurologic events, and transfusion), we extracted the reported incidences, odds ratios, hazard ratios and 95% confidence intervals from recent large cohort studies, registries, and high-quality reviews. Incidence values were synthesized descriptively to define plausible ranges (i.e. acute kidney injury (AKI), the incidence of approximately 20–30% (for all KDIGO staging, subclinical as well as clinical) , postoperative atrial fibrillation (POAF) 20–40%, and stroke or major neurologic events 1–3%), based on simple inspection and averaging of point estimates across contemporary series, without formal weighting or computation of pooled effect sizes. Adjusted effect estimates were quoted directly from the original publications, and no re-estimation of effect sizes was attempted. Heterogeneity between studies was assessed qualitatively by comparing study design, population, era, and outcome definitions, and findings were integrated using standard narrative synthesis principles to link cardiopulmonary bypass exposures, pathophysiology, and clinical outcomes.

PATHOPHYSIOLOGICAL PERTURBATIONS OF EXTRACORPOREAL CIRCUIT (CPB)

Circuit Mechanics and Hemodynamic Alterations: The standard CPB circuit operates as an integrated, temporary replacement for the human cardiopulmonary axis. Blood is drained via venous cannulae into a reservoir, propelled by a roller or centrifugal pump, oxygenated, temperature-regulated, filtered, and returned to the arterial circulation. Exposure to this circuit disrupts homeostasis across multiple physical and biochemical domains such as switching continuous pulsatile circulation to a Non-Pulsatile Flow, diminishes endothelial shear stress. Prolonged loss of pulsatility alters microvascular autoregulation, increasing the risk of splanchnic and renal ischemia by up to 20% compared to physiologic hemodynamics [6,7]. Obligate use of nearly 1000–1500 mL of crystalloid or colloid as priming solution acutely dilutes circulating blood components leading to hemodilution. A drop in intraoperative hematocrit to less than 22% during CPB is independently associated with a 30% increased risk of postoperative AKI and a 1.5-fold increase in mortality [8,9]. Deliberately induced hypothermia for organ protection, typically in the range of 28°C–32°C is utilized to reduce cerebral and myocardial metabolic demand by approximately 7% per degree Celsius drop. However, it intrinsically paralyzes the enzymatic reactions of the coagulation cascade and exacerbates platelet dysfunction [10].

Systemic Inflammatory Response Syndrome (SIRS): The hallmark of CPB is the ubiquitous activation of systemic inflammation. This is driven by several concurrent mechanisms including contact activation of blood components with the biomaterials of the circuit activates Factor XII, triggering both the intrinsic coagulation cascade and the kallikrein-kinin system, leading to bradykinin-induced vasodilation and capillary leak [11]. Subsequent complement cascade activation, generating C3a and C5a anaphylatoxins promotes massive neutrophil degranulation [12]. This may result in cytokine Storm characterized by Interleukin-6 (IL-6), Interleukin-8 (IL-8), and Tumor Necrosis Factor-alpha (TNF-α) peaking 4 to 6 hours after separating from CPB. Severe vasoplegia secondary to this cytokine surge complicates 10–45% of adult cardiac surgeries, requiring high-dose vasopressor support [13,14]. Increase vasopressor score is an independent predictor of AKI and mortality following cardiac surgery. Ischemia-Reperfusion (I/R) Injury following release of aortic cross-clamping creates global myocardial ischemia. Upon cross-clamp release, the sudden influx of oxygen generates highly reactive oxygen species (ROS), causing lipid peroxidation, cellular membrane damage, and myocardial stunning [15].

THE KEY DETERMINANTS AND THE STRATEGIES TO MITIGATE THE ADVERSE OUTCOME

Extent of Extracorporeal Exposure (Duration of Cardiopulmonary Bypass)

The duration of extracorporeal circulation is the most robust, modifiable, independent predictor of adverse outcomes. CPB times exceeding 120 minutes are associated with a 1.6-fold increase in 30-day mortality, while times exceeding 180 minutes doubles the mortality risk [16,17]. It has been observed that each 30-minute increment in CPB time beyond baseline increases the odds of prolonged mechanical ventilation (>24 hours) by 15%, and the odds of severe AKI by 20% [18]. Aortic cross-clamp (ACC) time exceeding 90 minutes correlate heavily with postoperative low cardiac output syndrome (LCOS) and the need for mechanical circulatory support, such as intra-aortic balloon pump (IABP) insertion [19]. The long CPB time increases the hypothermic time on CPB. Prolonged and deep hypothermia lead to cessation of enzyme activity, tissue hypoperfusion and coagulopathies following CPB.

Focused surgical approach with protocolized and shorter run of CPB can help reducing duration of CPB. Practicing shorter pump runs helps to mitigate hypothermia and its side-effects. [10,18,19] Reducing the cardiopulmonary bypass time and aortic cross clamp and deep hypothermia by the surgeon is a key strategy to minimize the post-operative adverse events following surgery. Proactive attenuation of circuit-induced trauma involves precise mechanical and pharmacological strategies. Use of miniaturized extracorporeal circulation (MiECC) helps to reduce priming volumes to 200–500 mL and significantly limits hemodilution and blunts the inflammatory response, demonstrating a 15–20% reduction in postoperative transfusion requirements [20,21]. Biocompatible coatings like heparin-coated and phosphorylcholine-modified circuits mimic human endothelial surfaces, significantly reducing complement activation and preserving platelet counts, can reduce the post-operative coagulopathy, acute kidney injury and neurocognitive dysfunctions leading to better clinical outcome [22]. Perioperative Pharmacological therapy with corticosteroids (e.g., methylprednisolone 10-20 mg/kg intravenous) reliably suppresses pro-inflammatory cytokine transcription. While large randomized trials (e.g., the SIRS trial) showed no difference in overall 30-day mortality, steroid use significantly reduces the incidence of new-onset atrial fibrillation and respiratory failure [23,24]. Pulmonary dysfunction is the most frequent complication following CPB, with clinical manifestations ranging from mild atelectasis (occurring in 50–70% of patients) to acute respiratory distress syndrome (ARDS), which occurs in 1–2% of patients but carries a devastating mortality rate of up to 50% [25,26]. Cessation of mechanical ventilation during CPB leads to dense alveolar collapse. Concurrently, complement activation traps neutrophils in the pulmonary microvasculature, leading to endothelial damage, surfactant depletion, and interstitial edema is the key mechanism of respiratory distress following CPB [27]. Practicing Lung-Protective Ventilation strategy by maintaining ventilation during CPB remains debated, but immediate postoperative lung-protective ventilation (tidal volumes of 6–8 mL/kg predicted body weight, moderate PEEP of 5–8 cm H_2O, and driving pressures of <15 cm H_2O reduces major pulmonary complications by 25% compared to historical high-tidal-volume strategies [28,29]. Practicing early extubation using fast-track anesthesia and enhanced recovery after cardiac surgery (ERAS) protocols targeting extubation within 6 hours reduce ventilator-associated pneumonias and decrease ICU length of stay by an average of 1.2 days without increasing reintubation rates [30]. Adapting these protocols can improve the post-operative outcome to a greater extent.

Cardiopulmonary Bypass (CPB) induced Coagulopathy

Up to 50% of cardiac surgery patients receive allogeneic blood products. Reoperation for bleeding occurs in 2–7% of cases and is an independent predictor of adverse outcomes, carrying a 3- to 4-fold increase in hospital mortality [31,32]. Furthermore, every single unit of transfused packed red blood cells (PRBCs) increases the risk of serious infection by 10% and AKI by 20% [33]. The coagulopathy of CPB is multifactorial: consumptive loss of fibrinogen, profound platelet exhaustion (receptor shedding), and acquired hyperfibrinolysis triggered by tissue plasminogen activator (tPA) release [34]. The routine prophylactic administration of Tranexamic Acid (TXA) is a Class I recommendation. Standard dosing (loading 10–15 mg/kg followed by an infusion) reduces bleeding volumes by 30% and the need for re-exploration by up to 50%, without increasing the risk of thrombotic events [35,36]. Goal-Directed Viscoelastic Testing has become a standard of care in modern practice of cardiac surgery. Empiric transfusion of plasma is obsolete. The use of point-of-care thromboelastography (TEG) or rotational thermoelectrometry (ROTEM) allows clinicians to precisely identify specific defects (e.g., isolated fibrinogen deficiency vs. delayed clot kinetics) in real-time. Implementation of TEG/ROTEM-guided algorithms reduces total blood product utilization by 30–40% and significantly decreases rates of severe bleeding [37,38]. Blood conservation protocols aiming at autologous blood salvage and practicing retrograde priming, use of cell-saver devices to wash and reinfuse shed mediastinal blood reduces the need for allogeneic PRBCs by 30% [39].

Cardiopulmonary Bypass (CPB) induced Neurological and Cognitive Dysfunction

Type I neurological deficits (stroke, TIA, coma) occur in 1.5–5% of adult cardiac surgeries. Type II deficits, notably Postoperative Cognitive Dysfunction (POCD), are remarkably common, affecting 30–50% of patients at discharge and persisting in 10–20% at 3 months postoperatively [40,41]. Embolic burden (solid atheroemboli from a calcified aorta or gaseous micro emboli from the circuit) accounts for the majority of strokes. Hypoperfusion relative to the patient's cerebral autoregulatory threshold drives ischemic watershed injuries [42]. Meticulous surgical technique, epiaortic ultrasound to guide cannulation away from severe atheroma, and the use of 40-micron arterial line filters [43]. Near-infrared spectroscopy (NIRS) allows continuous, non-invasive monitoring of regional cerebral oxygen saturation. Interventions targeting a reversal of NIRS desaturation (e.g., increasing MAP, optimizing CO_2, or transfusing for severe anemia) reduce the incidence of major cognitive decline [44].

Cardiopulmonary Bypass and Cardiac Surgery Associated Acute Kidney Injury (CSA-AKI)

CSA-AKI complicates 10–30% of procedures, depending on the diagnostic criteria (e.g., KDIGO). Alarmingly, 1–2% of patients will require renal replacement therapy (RRT). The requirement for postoperative RRT is an ominous prognosticator, associated with an in-hospital mortality rate approaching 40–50% [45,46]. The kidneys are exquisitely sensitive to CPB-induced non-pulsatile flow, micro emboli, free hemoglobin toxicity (due to circuit hemolysis), and systemic hypoperfusion [47]. Goal-directed oxygen delivery is paramount. Maintaining a targeted oxygen delivery index (DO_2) >270 mL/min/m^2 during CPB, primarily by avoiding extreme hemodilution and maintaining adequate pump flow, significantly reduces the incidence of CSA-AKI [48,49]. Strict avoidance of perioperative nephrotoxins and protocolized volume management are standard of care [50].

Cardiovascular Dysfunction Following CPB

Postoperative Atrial Fibrillation (POAF) is the most common arrhythmia, occurring in 20–40% of patients, peaking on postoperative day 2 or 3, and extending hospital length of stay by 2–3 days [51]. Low Cardiac Output Syndrome (LCOS) occurs in 5–15% of cases [52]. Continuation of preoperative beta-blockers is the cornerstone of POAF prevention, reducing incidence by 30% [53]. Effective mitigation of cardiovascular complications heavily relies on advanced, real-time hemodynamic monitoring. In accordance with updated perioperative guidelines from the ASA and ACC for cardiac patients, comprehensive intraoperative TEE is considered indispensable [54]. TEE allows the cardiac anesthesiologist to perform continuous, dynamic assessments of left and right ventricular systolic and diastolic function, accurately guide goal-directed fluid therapy, and detect critical, sudden-onset regional wall motion abnormalities or paravalvular leaks immediately upon weaning from CPB [55,56]. The ability to rapidly diagnose the etiology of hemodynamic instability (e.g., distinguishing acute right ventricular failure from profound hypovolemia or vasoplegia) drastically reduces the time to implement targeted inotropic, vasopressor, or surgical interventions, directly curbing postoperative mortality [57].

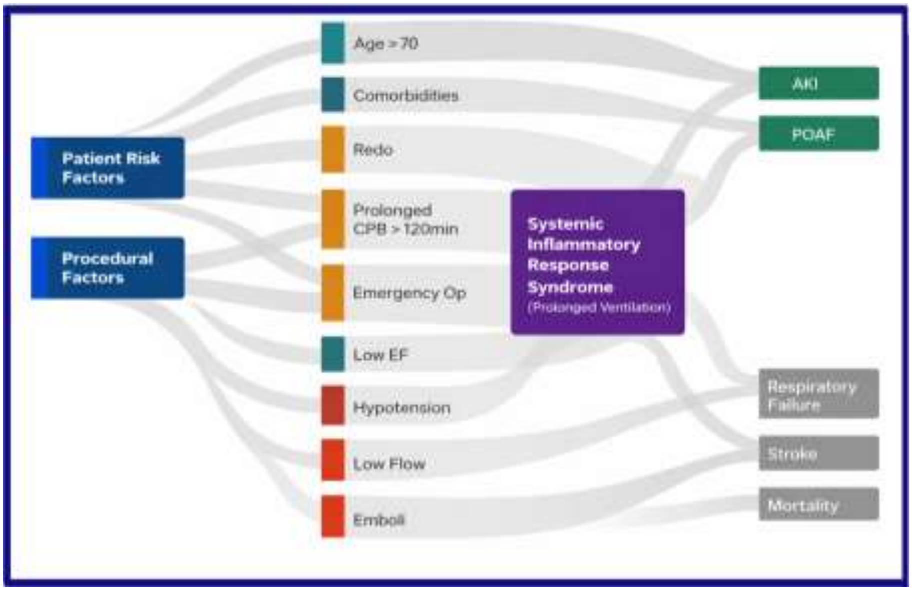

Figure 1 : Patient Related (Non modifiable) and Procedural (Modifiable) Risk Factors And Outcomes Following CPB in Cardiac Surgery

Figure 1 :- Flow diagram illustrating the relationship between patient and procedural risk factors and their downstream outcomes following cardiopulmonary bypass (CPB) in cardiac surgery. Patient-related factors (age >70, comorbidities, low ejection fraction, hypotension) and procedural factors (redo surgery, prolonged cardiopulmonary bypass >120 min, emergency operation, low flow, emboli) converge to trigger systemic inflammatory response syndrome (SIRS), represented centrally. SIRS is associated with prolonged ventilation and leads to adverse outcomes including acute kidney injury (AKI), postoperative atrial fibrillation (POAF), respiratory failure, stroke, and mortality.[58,59,60]

A Comprehensive, Evidence-Based Bundle Approach for Minimizing CPB related organ dysfunction and Improving post CPB Outcomes Following Cardiac Surgery

Quality improvement registries, such as the Society of Thoracic Surgeons (STS) database, repeatedly demonstrate that isolated, singular interventions yield marginal improvements. Conversely, structured, algorithmic "care bundles" yield synergistic benefits, accelerating recovery and reducing major complication rates by 30–50% [61,62]. Table 1 outlines a rigorously validated, scalable optimization bundles applicable across diverse resource settings.

Implementation science suggests that even partial adherence to such bundles in resource-constrained environments provides outsized benefits in reducing morbidity and overall healthcare costs [64].

Table 1: Comprehensive CPB Optimization and Enhanced Recovery Bundle

Care Domain	Key Evidence-Based Interventions	Expected Clinical Impact
Preoperative	Risk stratification (Euro SCORE II); optimization of baseline anemia; continuation of beta-blockers.	Decreased incidence of POAF; lower baseline transfusion risk [63].
Circuit & Perfusion	Miniaturized circuits (MECC); heparin-coated surfaces; target hematocrit >22%; DO_2 >270 mL/min/m².	20% reduction in CSA-AKI; dampened SIRS response [48,21].

Hemostasis	Standardized TXA protocols; strict use of cell-salvage; TEG/ROTEM-guided factor replacement algorithms.	30% reduction in bleeding volumes; significant drop in allogeneic PRBC use [35,37].
Anesthesia & Monitoring	Comprehensive TEE for ventricular function/volume; NIRS monitoring; lung-protective ventilation.	Rapid identification of LCOS; prevention of prolonged cerebral desaturation [55,44].
Postoperative	Fast-track extubation (<6 hrs); early, protocolized mobilization (<24 hrs); multimodal, opioid-sparing analgesia.	25% reduction in pulmonary complications; decreased ICU length of stay [28,30].

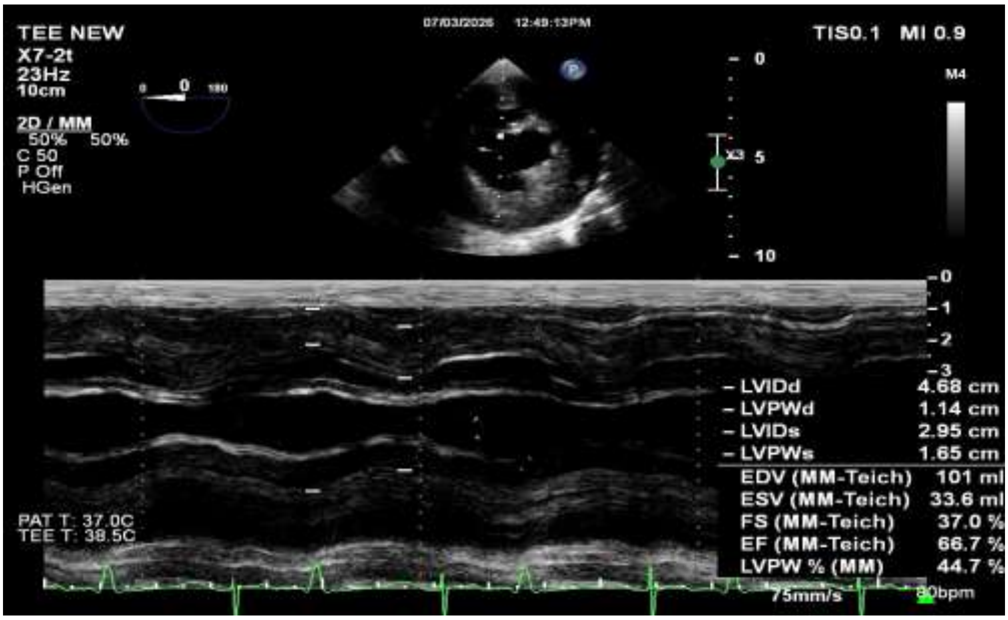

Figure 2 : Intra-operative Transesophageal Echocardiography for Monitoring of Post CPB Cardiovascular Function as a Part of Enhanced Recovery Bundles for Improving Outcomes Following Cardiac Surgery

Figure 2. M-mode interrogation of the trans-gastric mid-short axis view during real-time transesophageal echocardiography applied for cardiovascular monitoring. Assessment of left ventricular function following separation from cardiopulmonary bypass (CPB) demonstrates preserved systolic performance with a normal ejection fraction (EF = 66.7%) prior to decannulation. This evaluation is critical in guiding the decision to terminate CPB support. Parameters measured include left ventricular internal diameters in diastole (LVIDd) and systole (LVIDs), left ventricular posterior wall thickness in diastole (LVPWd) and systole (LVPWs), end-diastolic volume (EDV), end-systolic volume (ESV), fractional shortening (FS), and ejection fraction (EF) derived using the Teichholz method. The measured EF of 66.7% confirms preserved systolic function.

Future Directions

The horizon of extracorporeal circulation focuses heavily on application of multimodal techniques in mitigating the blood-biomaterial interface and moving toward precision, and individualized, patient specific perfusion strategy in choosing the circuitry, extent of hypothermia, choice of cardioplegia, evidence-based pharmacotherapy and blood conservation strategies. Targeted Cytokine Adsorption, technique utilizing the integration of hemadsorption devices (e.g., CytoSorb) directly into the CPB circuit to actively scavenge peak intraoperative cytokines (IL-6, TNF-α) shows promise in preventing severe postoperative vasoplegia in high-risk subjects undergoing complex cardiovascular surgeries [65,66]. Researches into seeding extracorporeal circuits with functional endothelial cells (Endothelialized circuits) aims to create a truly physiologic, non-thrombogenic surface, potentially eliminating the need for massive systemic heparinization [67]. Introduction of artificial intelligence (AI) and machine learning algorithms utilizing continuous high-fidelity data streams (MAP, NIRS, DO_2) are being developed to predict real-time hypotensive and ischemic events before they occur, allowing for automated, closed-loop perfusion adjustments for improved patient outcome [68].

CONCLUSION

Cardiopulmonary bypass remains one of the most remarkable physiological feats in modern medicine, granting surgeons the critical time needed to correct complex structural heart disease. However, it exacts a predictable systemic toll. The extracorporeal circuit instigates a violent inflammatory storm, profound coagulopathy, and risks end-organ ischemia.

Favorable postoperative outcomes are no longer merely a function of surgical technical success, but rather the result of meticulous, multidisciplinary perioperative care. By strictly minimizing bypass duration, leveraging biocompatible circuitry, utilizing goal-directed coagulation and advanced echocardiographic monitoring, and adhering to evidence-based recovery bundles, clinical teams can significantly blunt the physiological trauma of CPB. As technology evolves, the continuous refinement of these protocols will further reduce morbidity and ensure the highest standard of care for adult cardiac surgery patients worldwide.

Declaration by Authors

Ethical Approval: Not Applicable

Authors' Contribution:

For this narrative review, NPJ and ABS contributed to the concept and overall design of the review. NPJ contributed to literature collection, drafting, and manuscript preparation. ABS contributed to critical interpretation, revision, and final approval of the manuscript. SSS and APP were involved in review of literature and revision. All authors approved the final version for submission.

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Informed Consent: Obtained from subject who underwent routine intraoperative transesophageal echo for the image incorporation in manuscript (figure 2).

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