Management of Shock After Cardiac Surgery

Jean Deschamps, MDCM[a,*], Ren Jie Yao, MD[a], Nakul Kumar, MD[a], Luai Zakaria, MD[b], Chase Donaldson, MD[a], Kamrouz Ghadimi, MD, MHSc[c,d]

KEYWORDS

• Cardiac surgery • Postcardiotomy cardiogenic shock • Temporary mechanical circulatory support • Volume management • Mechanical ventilation

KEY POINTS

- Hemodynamic profiling requires a multimodal approach that considers the nuances of postcardiopulmonary bypass physiology.
- Volume management in postcardiotomy cardiogenic shock requires careful repletion of losses balanced with risks of volume overload for cardiac dysfunction and organ congestion.
- Early use of temporary mechanical circulatory support should be considered before organ dysfunction to promote early recovery in shock states associated to cardiopulmonary bypass.

INTRODUCTION

This article on the management of shock after cardiac surgery is aimed at exploring nuances specific to postcardiotomy shock (PCS). As this article is part of a series on cardiac management, this article will assume basic knowledge that can be found in other article and focus on specific cardiac surgery nuances.

POSTOPERATIVE PATHOPHYSIOLOGY AND IMPACT FROM CARDIOPULMONARY BYPASS

The PCS population is unique due to patients' exposure to cardiopulmonary bypass (CPB) and temporary cardiac arrest, which are associated with cytokine release, vasoplegia syndrome (VPS), coagulopathy, myocardial dysfunction and pulmonary hypertension (PHTN). CPB causes cytokine release from the interaction with the CPB circuit, the removal of blood into a reservoir and reintroduction into the body, and the shear stress from blood passing through the centrifugal impeller.[1] The release of cytokines and inflammatory factors disrupt the normal autoregulation of the vasoconstrictive pathways leading to low vasomotor tone, hypotension and VPS.[1] This inflammatory state affects the integrity of the capillary endothelium and glycocalyx layers resulting in the derangement of hydrostatic and oncotic pressure gradients favoring the shift of fluid out of the intravascular spaces. Inadequate myocardial protection on CPB results in myocardial ischemia and reperfusion injury, which can lead to postcardiotomy cardiogenic shock (PCCS). CPB is associated with the development of PHTN through contributions from reduced pulmonary perfusion resulting in endothelial dysfunction, inflammation with increased oxidative stress, and reduced nitric oxide (iNO) production.[1]

DEFINITION OF POSTCARDIOTOMY SHOCK AND ETIOLOGIES

Conventional cardiogenic shock (CS) definition does not apply to the postcardiotomy (PC)

[a] Division of Surgical Critical Care, Integrated Hospital Institute, Cleveland Clinic, Cleveland, OH, USA; [b] Division of Cardiothoracic Anesthesiology, Integrated Hospital Institute, Cleveland Clinic, Cleveland, OH, USA; [c] Clinical Research Unit, Division of Cardiothoracic Anesthesiology, Duke University School of Medicine, Durham, NC, USA; [d] Clinical Research Unit, Division of Critical Care Medicine, Duke University School of Medicine, Durham, NC, USA
* Corresponding author.
E-mail address: Deschamps.jd@gmail.com

Cardiol Clin 44 (2026) 429–445
https://doi.org/10.1016/j.ccl.2026.05.008

cardiology.theclinics.com

Abbreviations

Ang-II	angiotensin II
CALS	Cardiothoracic Advanced Life Support
CO	cardiac output
CABG	coronary artery bypass graft
CPB	cardiopulmonary bypass
CI	cardiac index
CVP	central venous pressure
CS	cardiogenic shock
dFick	direct Fick
ECG	electrocardiogram
ECMO	extracorporeal membrane oxygenation
eFick	estimated Fick
IABP	intra-aortic balloon pump
iEPO	inhaled epoprostenol
iNO	nitric oxide
ICU	intensive care unit
LCOS	low cardiac output syndrome
LV	left ventricular
LVOT	left ventricular outflow tract
MCS	mechanical circulatory support
NEE	norepinephrine equivalent
PA	pulmonary artery
PAC	pulmonary artery catheter
PCS	postcardiotomy shock
PCCS	postcardiotomy cardiogenic shock
PCVS	postcardiotomy vasoplegic shock
PCWP	pulmonary capillary wedge pressure
PEEP	positive end-expiratory pressure
PHTN	pulmonary hypertension
PPV	positive pressure ventilation
PVR	pulmonary vascular resistance
RCT	randomized controlled trial
RV	right ventricular
RVAD	right ventricular assist device
SAM	systolic anterior mitral
SCAI	Society for Cardiovascular Angiography and Interventions
SvO2	mixed venous oxygen saturation
SVR	systemic vascular resistance
Td	thermodilution
TEE	transesophageal echocardiography
VA	venoarterial
VExUS	venous excess ultrasound
VIS	vasoactive inotropic score
VPS	vasoplegia syndrome
VT	ventricular tachycardia

population due to the high incidence of vasopressor and inotrope requirements that is expected after exposure to CPB and sedation. Although relatively infrequent (occurring in ~0.5% to 1.5% of adult cardiac surgery cases), PCS carries an extremely high mortality approaching 2 in 3 patients.[2] This condition was historically referred to as *low cardiac output syndrome* (LCOS), a term that carries significant definitional heterogeneity. A systematic review of 250 articles by Schoonen and colleagues identified at least 10 distinct LCOS definitions across the cardiac surgery literature, with widely varying incidence estimates depending on the criteria applied—underscoring the challenge of interstudy comparability[3] Literature definitions of PCS are very variable but most imply a degree of tissue hypoperfusion (Table 1). Vasoactive inotropic score (VIS) is best for standardized communication of illness severity across research studies given the variability in vasoactive choices across institutions. The maximum VIS also predicts adverse outcomes and mortality in the PC and mechanical circulatory support (MCS) patient populations.[4] We propose the definition of maximum VIS greater than 20 to 25 required to maintain adequate organ perfusion as a preferred definition for PCS. We prefer to conceptualize PCS as an umbrella term for the coexisting causal pathologies (Table 2). The PCS phenotype classification proposed in this article shares conceptual overlap with the Society for Cardiovascular Angiography and Interventions (SCAI) shock staging system (stages A–E), which includes a CS-specific staging system, and has been validated in postcardiac surgery populations and correlates with mortality outcomes.[5] Application of the SCAI framework alongside the PCCS phenotype classification enables standardized research definitions and facilitates interstudy comparability.

HEMODYNAMIC PROFILING

Postoperative patients can exhibit multiple shock phenotypes which are often a combination of multiple pathologies rather than an isolated mechanism. Hemodynamic profiling entails a systematic assessment of cardiac function, perfusion, and vascular tone in these high-risk patients using standard and advanced monitoring techniques to delineate the predominant shock mechanism and severity (Fig. 1).

Overarching Hemodynamic Profiles

Early identification of structural or arrhythmogenic shock is essential, as these conditions are often rapidly correctable and should be addressed before more nuanced profiling.

Arrhythmogenic shock

Post-CPB patients are vulnerable to arrhythmia-mediated shock, in which loss of atrioventricular synchrony, rapid atrial arrhythmias, ventricular tachyarrhythmias, or pacing failure can acutely reduce effective forward flow.[10] Because hemodynamic collapse can be disproportionate to filling pressures or contractility indices, early rhythm assessment, prompt rhythm correction, and

Table 1
Current definitions of postcardiotomy shock

Author, Year	Definition
Rao et al,[6] 2006	Need for any MCS or inotropic support for >30 min in the ICU.
Sylvin et al,[7] 2010	Inability to separate from CPB, low CI < 2 L/min/m2 with evidence of tissue hypoperfusion.
Fux et al,[8] 2018	Hypoperfusion despite optimal volume loading, vasoactive medical support, and IABP.
Javorski et al,[9] 2024 Caruso et al,[9] 2024	VIS >20–25.

pacing optimization should be the first step in most patients.

Obstructive shock

Obstructive shock is commonly caused by postoperative pericardial or thoracic tamponade, presenting with decreased cardiac output (CO), narrow pulse pressure, elevated filling pressures and signs of impaired tissue perfusion. Postoperative tamponade may be underrecognized due to small and localized collections (92% in one series) that may be missed by transthoracic echocardiography (60% in one series), lacking classic echocardiographic features of tamponade physiology or mistaken for pericardial tissue, and requiring transesophageal echocardiography (TEE) for definitive diagnosis.[11] Rapid volume shifts from bleeding or vasoplegia may blunt expected elevations in filling pressures. Right-chamber tamponade (RA, right ventricular (RV) or RVOT/PA) closely mimics RV failure and is often distinguishable only by echocardiography. Isolated left-atrial tamponade may result in elevated pulmonary capillary wedge pressure (PCWP) despite relatively normal right-sided pressures and preserved left ventricular (LV) function. Thoracic tamponade may present with low filling pressures due to the acute blood loss, particularly in small, restrictive thoracic cavities. Increasing ventilatory pressures may both contribute to and herald evolving cardiac compression.[11] Systolic anterior mitral (SAM) following mitral valve repair is frequently underrecognized and mimics RV failure. TEE is often required to identify SAM, whether primary (due to altered mitral or ventricular geometry) or secondary to RV failure and septal shifting, and to orient toward radically different management strategies (Fig. 2).

Table 2
Postcardiotomy shock phenotypes and definitions

Entity	Definition
PCS	Global circulatory failure after cardiac surgery requiring pharmacologic or mechanical support.
PCCS/LCOS	Depressed myocardial contractility with low CO and elevated filling pressures following surgery.
PCVS	Low SVR with preserved or elevated CO after CPB.
Mixed shock	Combined myocardial dysfunction and vasodilation producing low output with reduced vascular tone.
Obstructive shock	Mechanical impairment of cardiac filling or ejection (eg, tamponade, RV outflow obstruction).
Arrhythmic shock	Hemodynamic compromise from loss of atrioventricular synchrony or malignant arrhythmias.

Cardiogenic and vasoplegic shock

Identifying the relative contributions of postcardiotomy cardiogenic shock (PCCS)/LCOS and postcardiotomy vasoplegic shock (PCVS), which frequently coexist, is the most nuanced part of hemodynamic profiling.[12]

PCCS or LCOS are characterized by impaired end-organ perfusion with a compensatory increase in systematic vascular resistance (SVR). Hypotension, narrow pulse pressure, and elevated filling pressures may be absent early depending on the severity of dysfunction, volume status, and predominant ventricle affected. Echocardiographic assessment of ventricular systolic function may not correlate with CO, and direct cardiac index (CI) measurement is required for accurate interpretation. RV failure may manifest in some cases as preserved CO with predominant systemic venous congestion. This phenotype presents with elevated central venous pressure (CVP), hepatic congestion, rising lactate, and oliguria which may be misinterpreted to another shock states such as tamponade. It is a particularly important mimicker of both distributive and hypovolemic shock, as it frequently presents with a low diastolic BP and wide pulse

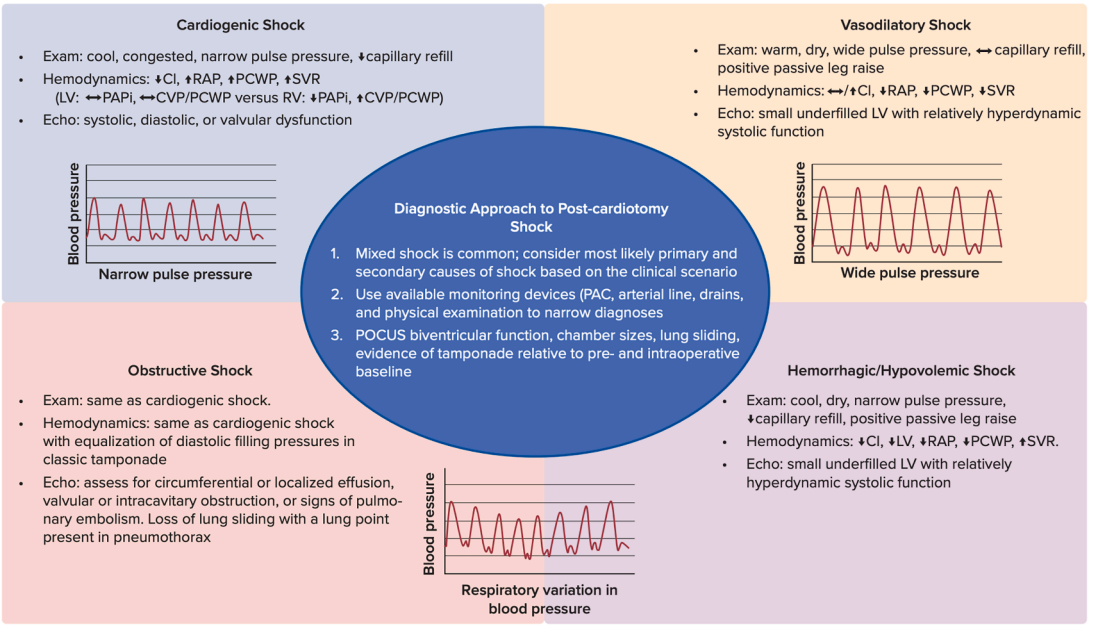

Fig. 1. Initial diagnostic approach to PCS. Algorithmic approach to the differential diagnosis of PCS. PAPi, pulmonary artery pulsatility index; POCUS, point-of-care ultrasound; RAP, right atrial pressure. (Previously published materials: *From* Hall EJ, Papolos AI, Miller PE, et al. Management of Post-cardiotomy Shock. US Cardiol 2024;18:e11. https://doi.org/10.15420/usc.2024.16 with permission (requires permission).)

pressure. PCVS is characterized by severe hypotension with low SVR despite normal or high CO.[13] Vasoplegia is present in a majority of cardiac surgical patients undergoing CPB. PCVS after CPB can resemble early sepsis as both share a systemic inflammatory biology. Post-CPB vasoplegia typically emerges within hours of separation from bypass and chest closure, whereas septic physiology should be suspected greater than 24 to 48 hours with a plausible infectious source, leukocytosis/fever (often elevated in the immediate postoperative period with resolution more than 48–72 hours), and progressive organ dysfunction despite restoration of preload and tone. Procalcitonin typically remains low or declines rapidly in postoperative vasoplegia related to CPB–associated inflammation, whereas rising or elevated procalcitonin levels are suggestive of bacterial sepsis when interpreted in serial measurements.[14] Both PCVS and hypovolemia frequently coexist early after surgery, creating mixed vasodilatory–hypovolemic shock that can obscure diagnosis and delay targeted therapy.[15] Dynamic volume loading to restore a compromised preload until adequate perfusion is achieved is the cornerstone of phenotyping.[15]

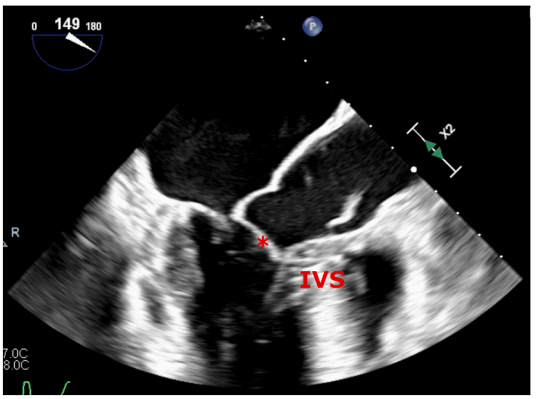

Fig. 2. TEE image of systolic anterior motion of mitral valve. Note the anterior mitral leaflet (*red asterisk*) touching the interventricular septum (IVS) during ventricular systole and causing a dynamic obstruction.

Invasive Measurements

CVP is a frequently used surrogate for preload. In postcardiotomy patients, CVP interpretation is confounded by exacerbated RV compliance abnormalities, pericardial constraint, positive pressure ventilation (PPV), PHTN, and residual RV dysfunction, which may elevate CVP despite relative hypovolemia. It correlates poorly with volume response in many populations including cardiac surgery.[16]

CO measurement is central to inotrope management. Thermodilution (Td) remains the most used method, whereas the direct Fick (dFick) principle is considered the physiologic reference standard but is challenging to use in routine postoperative care. Estimated Fick (eFick) CO rely on assumed oxygen consumption and may be inaccurate in postoperative patients with rapidly changing metabolic demands. Large cohort studies in cardiac surgery demonstrate poor agreement between dFick, eFick, and Td CI with discrepancies greater than 25% and only Td is associated with outcomes.[17] When CO estimates are discordant with the clinical picture, confirmatory assessment using repeat Td, comprehensive pulmonary artery catheter (PAC) profiling, and bedside echocardiography can reduce misclassification of shock phenotype.

Mixed venous oxygen saturation (SvO2) can be useful for trending global oxygen consumption balance, but it is not a direct surrogate for CO. SvO2 may be normal or elevated despite shock when oxygen extraction is impaired or when sedation, hypothermia, or MCS alters VO2; it correlates poorly with CI in the early postcardiac surgery intensive care unit (ICU) phase.[18]

Ultrasonography and Noninvasive Measurements

Postoperative ventricular function may vary due to myocardial stunning, ischemia, or changes following valvular intervention, such as improved systolic performance after aortic valve replacement. New regional wall motion abnormalities or unexpected hemodynamic deterioration should prompt evaluation for ischemia or valve-related complications, particularly in settings where coronary anatomy may be compromised (eg, mitral valve repair or coronary button reimplantation). In such cases, transthoracic imaging may be limited, and TEE is often essential for accurate diagnosis.

Echocardiography may help reconcile discordant Fick and Td CO measurements using LV outflow tract (LVOT) velocity-time integral (VTI)-based CO.[19] Accuracy may be limited, however, after aortic valve replacement, or from transaortic MCS support interference. Dynamic trends with interventions rather than absolute PAC measurements are particularly useful in this setting.

Ventricular chamber size is often used as a surrogate for preload, but dynamic changes in vascular tone (particularly CPB-related vasoplegia) can alter ventricular filling independent of true intravascular volume, limiting its reliability as a marker of volume status.[20] LV ejection fraction remains a useful marker of systolic function but is sensitive to changes in loading conditions, particularly in recent ischemia, stunning and resulting diastolic dysfunction. Commonly used echocardiographic surrogates of RV systolic function such as tricuspid annular plane systolic excursion (TAPSE), tissue Doppler S′, FAC, and RV free wall strain can be intrinsically reduced after pericardiotomy or CPB despite preserved RV ejection fraction and CO.[21] Inferior Vena Cava size and collapsibility are confounded by PPV, elevated intrathoracic pressures, RV dysfunction, and pericardial constraint. Volume decisions should rely on a multimodal assessment integrating dynamic response, ventricular filling/interaction, lung ultrasound for congestion, and end-organ perfusion trends rather than any isolated preload surrogate.[22]

Pulse contour and other noninvasive CO technologies can be less reliable immediately after CPB due to vasoplegia, arrhythmias, open/closed chest transitions, altered arterial compliance, and rapid changes in vascular tone; their greatest value is often in directional trending within a stable calibration window rather than single-point diagnosis.[23]

Integrated Assessment

Consideration of the patient's pre-existing disease for expected metabolic demands postoperatively is essential, and normalized targets should be avoided. Patients with chronic low flow states from long-standing cardiomyopathies, multivalve disease, radiation heart disease or constrictive pericarditis may tolerate lower CI; conversely, patients with infective endocarditis or with concomitant cirrhosis may require higher CI. When available, pre-existing hemodynamic data should be reviewed to inform appropriate targets postoperatively. SvO2, lactate pharmacokinetics, hourly urine output, and clinical peripheral extremity perfusion help assess patient tolerance of lower CI. Early after CPB, the nonpulsatile, CPB circulation may delay metabolic manifestations of hypoperfusion through usual markers.[18] Td CI often detects low flow states before clinical signs such as lactate elevation or oliguria emerge.

Static filling pressures, including CVP, fail to predict preload responsiveness after cardiac

surgery, particularly with open or recently closed chest conditions, abnormal ventricular interaction, decrease RV compliance or pericardial blood accumulation.[16] Pulmonary Capillary Wedge Pressure, Pulmonary Artery Wedge Pressure (PCWP/PAWP) does not uniformly equal LV end-diastolic pressure, particularly in the presence of mitral valve disease, altered LV compliance, high positive end-expiratory pressure (PEEP), or tachyarrhythmias.[24] Contemporary guidance emphasizes that CVP and PCWP should be integrated with dynamic indices, echocardiographic assessment of chamber filling, ventricular interdependence and lung congestion, as well as trends in CO and end-organ perfusion.[25] Transpulmonary and diastolic pressure gradient are used to determine RV-PA coupling, but are sensitive to changes in flow and loading in postcardiotomy physiology or on MCS.[26] Dynamic reassessment of response to targeted interventions using a multimodal approach is the cornerstone of hemodynamic profiling, using a hypothesis testing approach.[27]

Mechanical Circulatory Support

Td CI may be inaccurate on temporary MCS with extracorporeal blood circulation because extracorporeal flow alters pulmonary blood flow and indicator transit; therefore, Td CI should be primarily trended rather than treated as an absolute value.[28] eFick CI estimates are unreliable due to abnormal pulmonary blood flow, recirculation of oxygenated blood into the PA, and unstable oxygen extraction.[28] Garan and colleagues found that complete hemodynamic profiling by PAC before initiating MCS was associated with significantly lower in-hospital mortality (**Table 3**).[12]

FLUID MANAGEMENT

The main goal of fluid management is to maximize CO and tissue perfusion while minimizing complications and volume overload. Guidelines support goal-directed fluid protocols in cardiac surgery that integrate both dynamic fluid volume indices with flow-directed goals as they outperform single-target protocols.[29]

Ongoing fluid resuscitation requires frequent reassessment of static filling pressure and perfusion indices after dynamic interventions to avoid exceeding fluid tolerance. Fluid tolerance considers the negative congestion impact of fluid administration on microcirculatory dysfunction, either as a result or contributing to decreased RV performance.[30] Venous excess ultrasound (VExUS) is a protocol that can identify signs of venous congestion. Limited studies in the cardiac surgery population have shown an association between increased VExUS grades and renal injury after cardiac surgery.[31] Development of RV failure at any point postoperatively warrants consideration for timely volume removal, given association with poor outcomes. Renal dysfunction may require higher than expected doses of loop diuretics or combination diuretic therapy. Urgent continuous renal replacement may be required to improve RV hemodynamics despite adequate solute clearance.[32]

Balanced crystalloids remain the primary choice of fluid for resuscitation after cardiac surgery except for hemorrhagic shock. The use of both 5% and 20% human albumin solutions is controversial. The 2021 Joint Consensus Guidelines indicate that it is *reasonable* to use human albumin due to a reduction in overall fluids required in the perioperative period (Class IIA Level B recommendation).[33] Subsequent randomized controlled trials (RCTs), systematic reviews and meta-analyses have suggested a lack of benefit, resulting in the 2024 Chest Guidelines not recommending albumin for volume replacement. Among RCTs, HAS FLAIR II and ALBICS 2 have shown either neutral or increased risk of acute kidney injury (AKI) and increased use of blood products with the use of 20% albumin solutions in normal and high risk cardiac surgery patients.[34–36] The increasing evidence of lack of benefit of albumin coupled with its significant cost make the place of human albumin after cardiac surgery uncertain[37(p20)].

Resuscitation with blood products is preferred to replace the ongoing loss of red blood cells and coagulation factors in hemorrhagic shock. Guidelines recommend a 1:1 fresh frozen plasma and packed red blood cell ratio; low-quality evidence suggests improved outcomes with increased used of fresh frozen plasma (FFP).[33] Unique risk factors for transfusion-related adverse events specific to cardiac surgery includes emergent operations, milieu of inflammation, and presence of human leukocyte antigens (HLA) or human neutrophil antigens (HNA) antibodies in the donor blood products.[38] Blood salvage interventions can be used during ongoing resuscitation although this can be technically challenging in the ICU. Multiple joint guidelines recommend a restrictive perioperative allogeneic packed red blood cell transfusion strategy, with a target hemoglobin of 7 to 8 g/dL based on the TRACS, TRICS III, and TITRe2 RCTs; transfusion targets more than 10 g/dL are specifically discouraged in stable patients.[39–41] In patients with active hemorrhage after cardiac surgery, our institution targets a hemoglobin of 10 g/dL to maintain oxygen-carrying capacity during ongoing resuscitation. In PCCS, no specific hemoglobin transfusion

Table 3
Summary of common hemodynamic profile scenarios and pitfalls

Mimicker/Scenario	Typical Hemodynamic Pattern	Common Pitfall	Best Discriminating Tools
Vasoplegia vs Sepsis	Low SVR Preserved/high CO, Hypotension	Ignoring surgical timing/context	Time course Preload response End-organ perfusion Infection workup
RV Failure/ Tamponade	Low forward flow Elevated CVP Hypotension	Assuming high CVP = adequate preload	RV size/interaction PA pressures, PAPi TTE/TEE integration
Hypovolemia	Low filling pressures Narrow pulse pressure	CVP used as sole preload marker	Dynamic indices SV response TTE/TEE integration
VA ECMO	Altered pulsatility Discordant CO meas.	Td CO used despite nonreliable	Trend-based PAC data TTE/TEE integration End-organ perfusion

Abbreviations: meas, measurements; PA, pulmonary artery; PAPi, pulmonary artery pulsatility index; SV, stroke volume; TEE, transesophageal echocardiography; TTE, transthoracic echocardiogram.

threshold is established in the literature. Transfusion decisions should be individualized and guided by oxygen delivery targets and serial assessment of tissue perfusion markers, lactate clearance, and ends-organ function. Patients with severely compromised cardiac function or persistent hypoperfusion despite maximal support may warrant a higher hemoglobin target to optimize oxygen delivery. This principle applies across the spectrum of PCCS severity, including patients on MCS: patients awaiting heart and lung transplantation on ventricular assist device (VAD) or ECMO demonstrate that adequate perfusion can be maintained with very restrictive transfusion strategies.

The recently published FARES II trial demonstrated the safety of using prothrombin complex concentrates without increased thrombotic events with the benefits of lower volume resuscitation and improved hemostasis.[42] This study did not specifically distinguish patients that were stable versus in PCCS and excluded patient with VAD. The use of viscoelastic tests to guide coagulation factor specific treatment is supported by guidelines and reduces blood product utilization and reoperations.[43]

LACTATE-BASED MANAGEMENT

Lactate metabolism is a sensitive marker of tissue hypoperfusion and anaerobic metabolism in PCS. Serial lactate measurements and lactate clearance serve as diagnostic and prognostic indicators. Serial trends and clearance are more clinically useful than absolute values in guiding resuscitation decisions. However, lactate interpretation requires careful clinical contextualization in this population: (1) epinephrine administration causes type B lactic acidosis through beta-mediated accelerated glycolysis and increased pyruvate-to-lactate conversion, independent of tissue hypoperfusion[44]; (2) CPB produces lactate through inflammatory mechanisms, circuit interactions, and altered hepatosplanchnic metabolism[45]; (3) hepatic dysfunction impairs lactate clearance, as approximately 60% of lactate is cleared hepatically[46]; (4) right heart disease, PHTN, mitral stenosis, and tricuspid valve pathology are more prone to baseline lactate elevation through venous congestion and reduced hepatic perfusion; and (5) in significant venous congestion, lactate clearance may take multiple days even with adequate systemic perfusion.

PHARMACOLOGIC MANAGEMENT OF SHOCK AFTER CARDIAC SURGERY

Inotropes

No data identify a superior inotrope in cardiac surgery, and each agent has associated lethal arrhythmias and mortality.[47] Comparative inotrope data in PCCS are lacking; however, in CS and acute decompensated heart failure populations, milrinone and dobutamine demonstrate comparable composite outcomes in the DOREMI RCT[48] and a meta-analysis confirms no significant mortality difference between the 2 agents.[49] Inotrope choice is highly variable—differing by up to 30% across institutions—and is often reflects institutional and clinician preference rather than evidence-based selection (**Table 4**).[50] An organized, physiology-guided approach to inotrope

Table 4
Vasopressor and inotropic medications used in postoperative cardiac surgical care

Drug	Typical ICU Dosing (Adult)	CO	SVR	PVR	HR Arrhythmia	Postop Cardiac Surgery Nuances
Norepinephrine	0.01–0.3 μg/kg/min	↔/↑	↑↑	↔/↑	±	First-line vasopressor. May worsen RV afterload in severe PHTN/RV failure.
Vasopressin	0.01–0.04 U/min	↔	↑↑	↔/↓	↔	Second line catecholamine-sparing vasopressor. Less tachycardia than adrenergic agonist agents. Possible peripheral and mesenteric ischemia at higher dose.
Phenylephrine	0.25–2 μg/kg/min (or 25–200 μg/min)	↓/↔	↑↑	↔/↑	↓	Third line vasopressor Helpful if tachyarrhythmias as minimal myocardial activity. May decrease SV and worsen RV failure.
Angiotensin II	10–40 ng/kg/min	↔	↑↑	↔	↔	RAAS-deficient state post-CPB; rapid MAP response; thrombotic risk; DVT prophylaxis recommended; no inotropic effect
Epinephrine	0.01–0.2 μg/kg/min	↑↑	↑	↔/↑	↑↑	Best for mixed cardiogenic and vasoplegic shock. More arrhythmias. Dose-dependent vasoconstriction. Can raise lactate independent of hypoperfusion.
Dopamine	2–10 μg/kg/min	↑	↑	↔/↑	↑↑	Avoided due to arrhythmias compared to norepinephrine Dose-dependent variable effects. Renal dosing unreliable.
Dobutamine	2–10 μg/kg/min	↑↑	↓/↔	↓/↔	↑	Best for low CO with high SVR. Can worsen hypotension in vasoplegia. Caution with SAM/LVOT obstruction.
Milrinone	0.125–0.5 μg/kg/min	↑↑	↓	↓↓	±/↑	Useful for RV failure/high PVR. Hypotension risk from powerful vasodilation. Renally cleared and requires dose adjustment.
Levosimendan	0.05–0.2 μg/kg/min	↑↑	↓	↓	↔	Possible mortality benefit in the preoperative low ejection fraction (<40%)
Isoproterenol	1–10 μg/min (rare)	↑	↓	↓/↔	↑↑↑	Niche chronotrope if pacing limited. High arrhythmia risk.

(*continued on next page*)

Table 4 (*continued*)

Drug	Typical ICU Dosing (Adult)	CO	SVR	PVR	HR Arrhythmia	Postop Cardiac Surgery Nuances
Inhaled iNO	5–40 ppm	↔/↑	↔	↓↓↓	↔	RV unloading in PHTN/RV failure. Minimal systemic hypotension. Wean to avoid rebound PH.
iEPO	10–50 ng/kg/min	↔/↑	↔	↓↓↓	↔	Alternative to iNO for RV failure/PHTN. Delivery/technical considerations.

Abbreviations: DVT, deep vein thrombosis; HR, heart rate; MAP, mean arterial pressure; PH, pulmonary hypertension; RAAS, renin-angiotensin-aldosterone aystem; SAM, systolic anterior motion; SV, stroke volume.
Legend: ↑ increase, ↓ decrease, ↔ no major change, ± variable/dose-dependent.

selection is optimal: agents should be chosen based on the patient's dominant hemodynamic phenotype.[51]

Milrinone (PDE3 inhibitor) is unique by its long context-sensitive half-life of 2 hours which makes it unwieldy in rapidly changing hemodynamic profiles with associated vasoplegia. The frequent renal impairment that coexists with PCS may lead to accumulation that can complicate hemodynamic profiling and management. Milrinone administration may prevent and treat internal mammary artery vasospasm after coronary artery bypass grafting, with in vitro studies showing reversal of vasospasm induced by thromboxane after milrinone administration. It may be useful either early on at low-dose ($\leq$0.25 mcg/kg/hour) as an adjunct to an adrenergic agonist inotropes for its reduction in pulmonary vascular resistance (PVR), or when major hemodynamic shifts have passed.[52]

Low-dose epinephrine acts as an inoconstrictor (<0.1 mcg/kg/minute) and can replace an inodilator in the setting of excessive hypotension in mixed CS and vasodilatory shock. The associated type B lactic acidosis may lead to unnecessary interventions including excessive fluid administration, abdominal computed tomography (CT) scans, or even initiation of MCS.[46]

Dobutamine produces a dose-dependent rise in CI in cardiac surgical patients. Dobutamine may be used when concerns regarding renal failure and monitoring progression of lactic acidosis steer clinicians away from using milrinone and epinephrine respectively. As a rapidly acting inodilator, it can also serve a diagnostic tool to identify the predominant cause of shock between cardiogenic and vasoplegic.

Levosimendan, a calcium sensitizer with inodilatory properties, is available in Europe and Asia and offers theoretic advantages including improved contractility without increasing myocardial oxygen demand. However, 2 RCTs in cardiac surgery[53,54] failed to demonstrate mortality or morbidity benefit with prophylactic or early therapeutic use. These negative trials temper enthusiasm for routine perioperative use, though levosimendan may retain a role in rescue therapy for PCCS unresponsive to standard agents where commercially available.

Vasopressors

Societal recommendations for first-line vasopressor in cardiac surgery are inexistent, with norepinephrine frequently first-line. RCT data in cardiac surgery have demonstrated the safety of vasopressin as a first-line agent compared to norepinephrine, with an improvement in composite outcome driven by decreased renal failure and atrial fibrillation.[55] In vivo data have not demonstrated the in-vitro beneficial PVR sparing effects of vasopressin.[56] A vasopressor strategy limiting myocardial stimulation (vasopressin and phenylephrine) may be beneficial in significant LVOT obstruction or arrhythmic burden. Combination therapy has been shown to reduce mortality and atrial fibrillation in septic shock and is increasingly used in cardiac surgical populations without definite evidence.[57] Reducing total catecholamine after cardiac surgery may reduce comorbid conditions such as atrial fibrillation, mesenteric and digital ischemia, and coronary vasospasm after coronary artery bypass graft (CABG). When combination therapy is inadequate to support vascular tone and mean arterial pressure, refractory shock therapies are considered.

Refractory Shock Therapies

Refractory shock refers to a reduced mean arterial pressure and end-organ dysfunction despite the

use of conventional vasopressors. Several studies define refractory shock as greater than 0.2 mcg/kg/min norepinephrine equivalents (NEEs). A recent report updated the definition of NEE (**Box 1**)[58]:

Several reviews recommend alternative treatments including angiotensin II (Ang-II) (10 ng/kg/minute), methylene blue (1-2 mg/kg bolus followed by 0.5 mg/kg/hour), hydroxocobalamin (5 g bolus more than 15 minutes), and/or stress dose steroids (hydrocortisone 100 mg every 8 hours).[59] While limited data show efficacy in increasing higher MAP, patient outcome data is lacking.[60] Ang-II warrants consideration as a second- or third-line vasopressor in refractory PCVS. The ATHOS-3 RCT demonstrated that Ang-II significantly improved MAP response (69.9% vs 23.4%, $P<.001$) and reduced catecholamine requirements in catecholamine-resistant vasodilatory shock.[61] A post-hoc analysis of ATHOS-3 focusing on 16 postcardiac surgery vasoplegia patients found that 89% of Ang-II-treated patients achieved target MAP response versus 0% in placebo, with rapid catecholamine dose reduction in the majority of responders.[62] Despite improvements in macrocirculation hemodynamics, systemic NO inhibition may block beneficial effects of microcirculatory NO production and lead to significant harm and circulatory failure; those at risk include patients with LCOS and/or PHTN. Careful hemodynamic profiling to confirm vasoplegia as the predominant phenotype is essential before initiating these agents, as these therapies do not provide any inotropic support. MCS should be initiated when inadequate forward flow persists despite vasopressor optimization.

Management of refractory PCVS should start with stress dose steroids (hydrocortisone 100 mg every 8 hours), with rapid escalation if inadequate response within 1 to 2 hours. Next therapies should be dictated by drug availability and contraindications: Ang-II for confirmed low-SVR vasoplegic physiology; methylene blue (G6PD deficiency, concomitant serotonergic medications); hydroxocobalamin (oxalate nephropathy and renal dysfunction).

Box 1
Updated definition of norepinephrine equivalent

NEE = Norepinephrine dose (μg/kg/min) + epinephrine dose (μg/kg/min) + 1/100 × dopamine dose (μg/kg/min) + 0.06 × phenylephrine dose (μg/kg/min) + 2.5 × vasopressin dose (U/min) + 0.0025 × angiotensin II dose (ng/kg/min) + 10 × terlipressin dose (μg/kg/min) + 0.2 × methylene blue dose (mg/kg/h) + 1/8 × metaraminol dose (μg/kg/min) + 0.02 × hydroxocobalamin dose (g) + 0.4 × midodrine dose (μg/kg/min)

Pulmonary Vasodilators

All pulmonary vasodilators may be beneficial, and no data suggest a superior medication. Inhaled nitric oxide (iNO) theoretically offers organ protection via its vasodilatory, anti-inflammatory and antioxidant properties in the setting of ischemic-reperfusion injury after CPB. The INSPIRE-FLO RCT demonstrated equivalent efficacy of both agents in preventing postoperative RV failure in VAD and heart transplant, with inhaled epoprostenol (iEPO) offering cost-effectiveness advantages without compromise in clinical outcomes.[63] In lung transplant recipients, iEPO had similar primary graft dysfunction development to iNO, with no patient outcome differences.[64] Inhaled milrinone may be synergistic with iNO and iEPO and carries the advantage of less systemic hypotension when compared to intravenous milrinone.[65]

MECHANICAL VENTILATION

Cardiopulmonary interactions can exacerbate hemodynamic failure in PCS even in the absence of a primary pulmonary pathology.[66] Postoperative LV and RV failure initially benefit from PPV by decreasing respiratory muscle use and excessive intrathoracic pressure changes, as well as controlling gas exchange and acid–base status.[67] The PPV benefits for LV and RV may be negated by the cardiodepressant effects of sedation such as propofol.[68] Early discontinuation of PPV is beneficial once metabolic and respiratory insults have resolved, even on significant inotropic support, in comparison to noncardiotomy CS.[69]

Evidence in PCCS is limited to retrospective data. In all-comer CS, the use of PPV has been repeatedly shown to improve P/F ratio, respiratory compliance, LV indices, and in some studies, RV indices.[67] Recommendations include targeting lung protective ventilation with tidal volumes between 4 to 8 mL/kg and driving pressure less than 15 cmH2O.[70–72] RCT data in uninjured lungs suggest that this can be extended up to 10 mL/kg.[70] PEEP should be increased as needed to correct hypoxemia, as undertreated atelectasis may be more detrimental than high PEEP to RV function.[73] RCT data in acute respiratory distress syndrome, uninjured lungs, and cardiac surgery without CS have shown that high PEEP is protective against development of acute respiratory distress syndrome (ARDS) and hypoxemia without

detrimental effects on cardiac function.[70,74] Physiologic data on PEEP titration in nonsurgical CS suggest that the cardiovascular impact of PEEP may be dependent on etiology of shock.[73]

LV assist devices are highly susceptible to high ventilatory pressure that reduce transpulmonary flow. RV assist devices (RVADs) are less affected by PPV-induced afterload, but may have decreased flow from PPV-induced reduced preload.[66] An ELSO registry study of 2226 CS venoarterial (VA) extracorporeal membrane oxygenation (ECMO) patients found that survival to discharge was higher in the low intrapulmonary pressure ventilation group (peak inspiratory pressure <30 cm H20).[75] Retrospective outcome data in nonsurgical CS intraaortic balloon pump (IABP) + PPV versus IABP-only group showed no definite survival advantage but some hemodynamic benefits.[76] Early addition of an oxygenator or conversion to ECMO should be considered if metabolic targets cannot be attained with nondetrimental ventilation.

In summary, the practical approach to mechanical ventilation in PCS should incorporate the following principles: (1) target lung-protective tidal volumes of 4 to 8 mL/kg ideal body weight and driving pressure less than 15 cmH2O and minimize respiratory acidosis; (2) titrate PEEP to correct atelectasis and hypoxemia—undertreated atelectasis is more hemodynamically harmful than judicious PEEP application in most PCCS patients; (3) minimize ventilatory pressures to minimize RV afterload including in left ventricular assist device (LVAD)/RVAD and (5) prioritize early liberation from mechanical ventilation once metabolic and hemodynamic stability are achieved, even in the presence of ongoing vasoactive support. **Table 5** summarizes specific mechanical ventilation targets in PCS.

MECHANICAL CIRCULATORY SUPPORT INDICATIONS

Planning of MCS for patients at-risk for PCCS starts with preoperatively. For patients at high risk for PC CS, an axillary CPB cannulation strategy can be considered to facilitate either axillary VA ECMO or axillary microaxial flow pump device as an exit strategy in the perioperative setting. For patients failing initial medical interventions, early escalation to temporary MCS limits time in shock state and prevents multiorgan dysfunction.[77–80]

MCS choice depends on the urgency of initiation, local expertise, the need for isolated univentricular support versus biventricular support, and the need for oxygenator support. The most rapid form of MCS to initiate is VA ECMO. Central VA ECMO can be rapidly deployed in the operating room using the pre-existing CPB cannulas or during chest reopening in cardiac arrest and is often associated with higher flows than peripheral cannulation. The drawbacks of central VA ECMO include higher risk of bleeding and infection, and delayed chest closure. Femoral cannulation can be performed percutaneously in a rapid fashion at the bedside, with drawbacks of more limited flow, risk of north-south syndrome, and limb ischemia. Axillary cannulation is increasingly used as it provides similar benefits to central VA ECMO but can be removed without chest reopening. Upper extremity ischemia and hyperperfusion remain potential complications.

Central and peripheral VA ECMO may require upfront or early LV unloading. Methods of LV unloading include the use of a percutaneous microaxial flow pump (Impella 5.5 or CP) device, IABP, atrial septostomy, left atrial (LA) cannula (left atrial veno-arterial [LAVA] ECMO) and surgical LA or LV drain. A recent meta-analysis that included 2 RCTs showed no difference between upfront LV unloading compared to rescue unloading, though both RCTs used LA drainage cannula as the unloading strategy, despite some prior reported benefit.[81]

In our institution, the goal is to transition off VA ECMO support into a more durable configuration once the patient is out of the acute shock stage and within a 48-hour period. An emerging institutional practice involves concurrent LV unloading with the Impella 5.5 microaxial flow pump at the time of initial ECMO cannulation, particularly when prolonged support is anticipated. This approach is applied selectively to patients with uncertain or expected prolonged LV recovery trajectory and presumed early RV recovery. Combined VA ECMO and Impella support (ECpella) is associated with substantially improved outcomes compared to VA ECMO alone, including lower in-hospital mortality (47% vs 80%) and higher rates of successful bridging to recovery or advanced therapy (68% vs 28%).[82] Impella 5.5 has demonstrated superior outcomes compared to earlier-generation devices, with 76.1% of patients successfully weaned or bridged to transplant or durable support in PCCS (vs 55.7% with Impella 5.0).[83] For patients in whom a 3 to 5 day ECMO course is anticipated due to expected rapid recovery, Impella 5.5 implantation is deferred 48 hours to assess LV recovery trajectory and reassess its benefit for early MCS de-escalation. Surgical or percutaneous RVADs are used as needed for RV support during de-escalation. This combination of devices allows selective weaning based on predominant ventricular recovery and enable physiotherapy in appropriately selected patients. While extubation on VA ECMO is

Table 5
Mechanical ventilation targets in postcardiotomy shock

Parameter	Target	Notes/Evidence
Tidal volume	*4–8 mL/kg IBW (up to 10 mL/kg in uninjured lungs)*	Lung-protective ventilation; titrate based on lung injury status. *PReVENT trial; ARDSNet*
Driving pressure	*< 15 cmH_2O*	Independent predictor of mortality; associated with improved outcomes across ventilated ICU populations.
PEEP	*5–10 cmH_2O*	Titrate to correct hypoxemia. Higher PEEP protective against ARDS; balance against RV afterload and individualize based on RV tolerance.
Extubation timing	*Early when stable*	Pursue early extubation when metabolically and hemodynamically stable; beneficial even with significant inotropic support.

Abbreviation: IBW, ideal body weight.

not routine, concurrent Impella 5.5 insertion has facilitated extubation within the first 24 to 48 hours in selected patients, with reintubation reserved for device reconfiguration or decannulation. In patients considered for durable MCS or transplantation, transitioning to univentricular LV support allows RV function and PVR assessment critical for optimal decision-making.

Providing univentricular MCS can unmask underlying dysfunction in the other apparently functional ventricle. Some clinicians advocate for upfront full biventricular ECMO support with rapid de-escalation to optimize timely organ perfusion and minimize time in the shock state in settings of limited hemodynamic profiling.[84,85] For isolated LV support, Impella 5.5 is more commonly used perioperatively through an axillary graft or tunneled direct aortic cannulation.[86] For isolated RV support, surgical RVAD through a tunneled pulmonary artery graft and peripheral drainage site have been increasingly used as they can be removed without reopening the chest.[87] With the wide variety of MCS options, institutional protocols may be helpful to guide the team's decision-making process, and we propose the following algorithm (**Fig. 3**).

CARDIOTHORACIC ADVANCED LIFE SUPPORT

Cardiothoracic Advanced Life Support (CALS) was developed to optimize survival in this high-risk population by tailoring resuscitation strategies to minimize harm from traditional cardiopulmonary compressions while maximizing perfusion and rapid return of circulation, including early defibrillation, pacing, and, when necessary, emergency resternotomy.[88]

CALS requires a coordinated multidisciplinary team, specialized carts (open chest, ECMO), and trained personnel who have completed CALS-specific competency training. When arrest occurs, the CALS protocol begins with rhythm assessment and immediate intervention:

- Ventricular fibrillation/pulseless ventricular tachycardia (VT): Defibrillate within 1 minute; if unsuccessful, initiate external cardiac massage and administer amiodarone. For ventricular fibrillation (VF) or pulseless VT, immediate defibrillation with up to 3 stacked biphasic shocks (200–360 J) is emphasized, as success declines rapidly over time.
- Asystole/bradycardia: Attempt pacing first; if ineffective, begin compressions. Extreme bradycardia or asystole mandates immediate pacing via epicardial wires or external pads before initiating compressions.
- Pulseless electrical activity: Exclude fine VF, then proceed with compressions.
- If initial measures fail, rapid reopening of the chest allows internal cardiac massage and direct correction of surgical complications such as tamponade or graft occlusion.

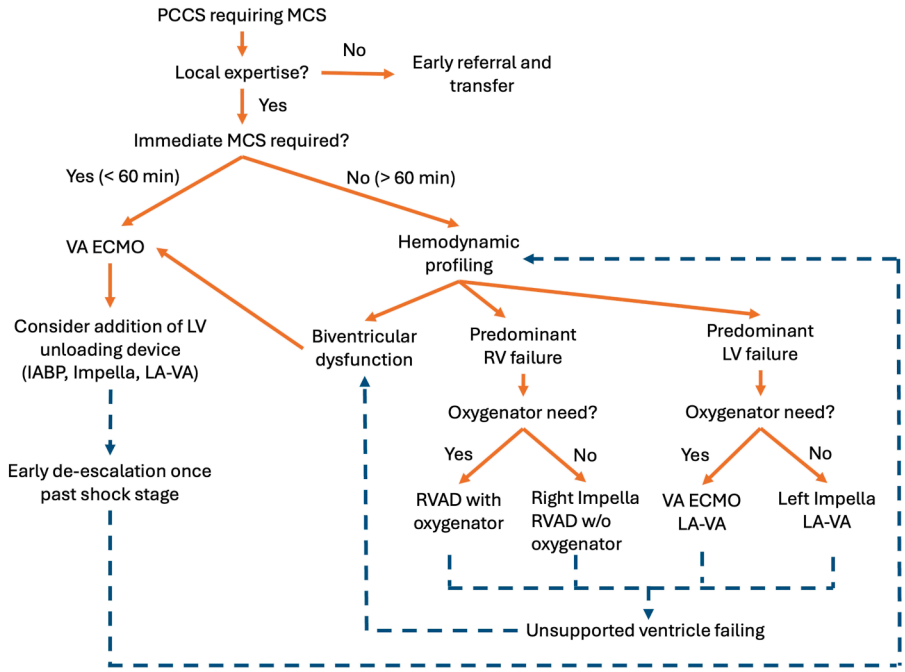

Fig. 3. Algorithmic approach to temporary MCS decision in PCS. Orange full lines represent initial approach to device selection; Blue interrupted lines represent secondary steps after initial attempt at stabilization and require expert reassessment. LA-VA, left atrium VA.

CALS applies to adult patients within 10 postoperative days of a median sternotomy or clamshell incision for procedures including CABG, valve surgery, heart or lung transplantation, ascending aortic repair, and certain VAD placements. Patients with nonsternotomy procedures, durable VADs, or thoracoabdominal repairs are excluded.

The Society of Thoracic Surgeons consensus[89] and other studies reports significant mortality reductions with CALS implementation with improvement in neurologically intact survival rates, attributing success to rapid defibrillation, pacing, and timely surgical intervention; strategies that are absent in conventional ACLS.[88]

SUMMARY

PCS is a complex physiologic phenomenon requiring a multimodal assessment and nuanced management strategies.

CLINICS CARE POINTS

- Conventional cardiogenic shock criteria do not apply after cardiopulmonary bypass; a maximum vasoactive-inotropic score >20–25 with evidence of hypoperfusion offers a practical, standardizable definition of post-cardiotomy shock.
- Static filling pressures (CVP, PCWP) poorly predict preload after cardiac surgery; thermodilution is the only cardiac-output method linked to outcomes, and on extracorporeal mechanical support it should be trended rather than read as an absolute value.
- No inotrope is proven superior in post-cardiotomy shock, so select agents by the dominant hemodynamic phenotype; dobutamine, a rapidly acting inodilator, doubles as a bedside diagnostic to separate cardiogenic from vasoplegic physiology, whereas low-dose epinephrine suits mixed shock but causes type B lactic acidosis independent of perfusion that can prompt unnecessary fluid boluses or imaging.
- Milrinone's long (~2-hour) context-sensitive half-life and renal clearance make it unwieldy amid rapidly shifting hemodynamics and coexisting renal dysfunction; reserve it for established RV failure or high pulmonary vascular resistance, or for use once major hemodynamic shifts have settled, often at low dose alongside an adrenergic inotrope.
- Apply lung-protective ventilation (tidal volume 4–8 mL/kg IBW, driving pressure <15 cmH_2O), titrate PEEP to correct atelectasis, minimize ventilatory pressures with LVAD/RVAD support, and pursue early extubation once the patient is stable, even on significant inotropic support.

- When forward flow remains inadequate despite optimized vasopressors and inotropes, escalate to temporary mechanical circulatory support early, before multiorgan dysfunction develops, to limit time in the shock state; completing pulmonary artery catheter profiling before cannulation is associated with lower in-hospital mortality.

DISCLOSURE

K. Ghadimi receives funding through his institution for research grants from Octapharma for coagulation research. L. Zakaria has research funding through a Society of Cardiac Anesthesia Early Investigator grant. All other authors declare no source of funding or conflict of interest.

REFERENCES

1. Ferreira LO, Vasconcelos VW, Lima JS, et al. Biochemical changes in cardiopulmonary bypass in cardiac surgery: new insights. J Personalized Med 2023;13(10). https://doi.org/10.3390/jpm13101506.
2. Hall EJ, Papolos AI, Miller PE, et al. Management of post-cardiotomy shock. US Cardiol 2024;18:e11.
3. Schoonen A, van Klei WA, van Wolfswinkel L, et al. Definitions of low cardiac output syndrome after cardiac surgery and their effect on the incidence of intraoperative LCOS: a literature review and cohort study. Front Cardiovasc Med 2022;9:926957. https://doi.org/10.3389/fcvm.2022.926957.
4. Pölkki A, Pekkarinen PT, Lahtinen P, et al. Vasoactive inotropic score compared to the sequential organ failure assessment cardiovascular score in intensive care. Acta Anaesthesiol Scand 2023;67(9):1219–28.
5. Rajan R, Jarallah MA, Daoulah A, et al. The utility and validation of SCAI-CSWG stages in patients with acute myocardial infarction-related cardiogenic shock. J Soc Cardiovasc Angiogr Interv 2025;4(1). https://doi.org/10.1016/j.jscai.2024.102461.
6. Rao V. Condition critical: can mechanical support prevent death due to postcardiotomy shock? J Card Surg 2006;21(3):238–9.
7. Sylvin EA, Stern DR, Goldstein DJ. Mechanical support for postcardiotomy cardiogenic shock: has progress been made? J Card Surg 2010;25(4):442–54.
8. Fux T, Holm M, Corbascio M, et al. Venoarterial extracorporeal membrane oxygenation for postcardiotomy shock: risk factors for mortality. J Thorac Cardiovasc Surg 2018;156(5):1894–902.e3.
9. Caruso V, Berthoud V, Bouchot O, et al, ECMOVIS Study Group. Should the vasoactive inotropic score be a determinant for early initiation of VA ECMO in postcardiotomy cardiogenic shock? J Cardiothorac Vasc Anesth 2024;38(3):724–30.
10. Heidenreich PA, Solis P, Estes NAM, et al. 2016 ACC/AHA clinical performance and quality measures for adults with atrial fibrillation or atrial flutter. JACC 2016;68(5):525–68.
11. Price S, Prout J, Jaggar SI, et al. "Tamponade" following cardiac surgery: terminology and echocardiography may both mislead. Eur J Cardio Thorac Surg 2004;26(6):1156–60.
12. Garan AR, Kanwar M, Thayer KL, et al. Complete hemodynamic profiling with pulmonary artery catheters in cardiogenic shock is associated with lower In-Hospital mortality. JACC: Heart Fail 2020;8(11):903–13.
13. Suero OR, Park Y, Wieruszewski PM, et al. Management of vasoplegic shock in the cardiovascular intensive care unit after cardiac surgery. Crit Care Clin 2024;40(1):73–88.
14. Xie M, Chen YT, Zhang H, et al. Diagnostic value of procalcitonin and Interleukin-6 on early postoperative pneumonia after adult cardiac surgery: a prospective observational study: a prospective observational study. HSF 2022;25(1):E020–9.
15. Polyak P, Kwak J, Kertai MD, et al. Vasoplegic syndrome in cardiac surgery: a narrative review of etiologic mechanisms and therapeutic options. J Cardiothorac Vasc Anesth 2025;39(7):1815–29.
16. Rex S, Brose S, Metzelder S, et al. Prediction of fluid responsiveness in patients during cardiac surgery. Br J Anaesth 2004;93(6):782–8.
17. Opotowsky AR, Hess E, Maron BA, et al. Thermodilution vs estimated fick cardiac output measurement in clinical practice: an analysis of mortality from the veterans affairs clinical assessment, reporting, and tracking (VA CART) program and vanderbilt university. JAMA Cardiol 2017;2(10):1090–9.
18. Holm P, Karhu JM, Erkinaro TM, et al. Mixed venous oxygen saturation has a poor association with cardiac index during early intensive care unit stay after cardiac surgery. J Cardiothorac Vasc Anesth 2025;39(8):2088–94.
19. Zhang Y, Wang Y, Shi J, et al. Cardiac output measurements via echocardiography versus thermodilution: a systematic review and meta-analysis. PLoS One 2019;14(10):e0222105. https://doi.org/10.1371/journal.pone.0222105.
20. Preisman S, Kogan S, Berkenstadt H, et al. Predicting fluid responsiveness in patients undergoing cardiac surgery: functional haemodynamic parameters including the respiratory systolic variation test and static preload indicators. Br J Anaesth 2005;95(6):746–55.
21. Singh A, Huang X, Dai L, et al. Right ventricular function is reduced during cardiac surgery independent of procedural characteristics, reoperative status, or pericardiotomy. J Thorac Cardiovasc Surg 2020;159(4):1430–8.e4.
22. Sobczyk D, Nycz K, Andruszkiewicz P. Bedside ultrasonographic measurement of the inferior vena

cava fails to predict fluid responsiveness in the first 6 hours after cardiac surgery: a prospective case series observational study. J Cardiothorac Vasc Anesth 2015;29(3):663–9.
23. Broch O, Bein B, Gruenewald M, et al. Accuracy of cardiac output by nine different pulse contour algorithms in cardiac surgery patients: a comparison with transpulmonary thermodilution. Biomed Res Int 2016;2016: 3468015. https://doi.org/10.1155/2016/3468015.
24. Reddy YNV, El-Sabbagh A, Nishimura RA. Comparing pulmonary arterial wedge pressure and left ventricular end diastolic pressure for assessment of left-sided filling pressures. JAMA Cardiol 2018;3(6):453–4.
25. Chioncel O, Parissis J, Mebazaa A, et al. Epidemiology, pathophysiology and contemporary management of cardiogenic shock – a position statement from the Heart Failure Association of the European Society of Cardiology. Eur J Heart Fail 2020;22(8): 1315–41.
26. Naeije R, Vachiery JL, Yerly P, et al. The transpulmonary pressure gradient for the diagnosis of pulmonary vascular disease. Eur Respir J 2013;41(1):217–23.
27. Osawa EA, Rhodes A, Landoni G, et al. Effect of perioperative goal-directed hemodynamic resuscitation therapy on outcomes following cardiac surgery: a randomized clinical trial and systematic review. Crit Care Med 2016;44(4):724–33.
28. Saxena A, Garan AR, Kapur NK, et al. Value of hemodynamic monitoring in patients with cardiogenic shock undergoing mechanical circulatory support. Circulation 2020;141(14):1184–97.
29. Grant MC, Crisafi C, Alvarez A, et al. Perioperative care in cardiac surgery: a joint consensus statement by the Enhanced Recovery After Surgery (ERAS) cardiac Society, ERAS International Society, and the Society of thoracic surgeons (STS). Ann Thorac Surg 2024;117(4):669–89.
30. Muñoz F, Born P, Bruna M, et al. Coexistence of a fluid responsive state and venous congestion signals in critically ill patients: a multicenter observational proof-of-concept study. Crit Care 2024;28(1):52.
31. Utrilla-Alvarez JD, Gopar-Nieto R, García-Cruz E, et al. Assessing the venous system: correlation of mean systemic filling pressure with the venous excess ultrasound grading system in cardiac surgery. Echocardiography 2023;40(11):1216–26.
32. Zou H, Hong Q, Xu G. Early versus late initiation of renal replacement therapy impacts mortality in patients with acute kidney injury post cardiac surgery: a meta-analysis. Crit Care 2017;21(1):150.
33. Tibi P, McClure RS, Huang J, et al. STS/SCA/AmSECT/SABM update to the Clinical Practice Guidelines on patient blood management. Ann Thorac Surg 2021;112(3):981–1004.
34. Wigmore GJ, Deane AM, Presneill JJ, et al. Twenty percent human albumin solution fluid bolus administration therapy in patients after cardiac surgery-II: a multicentre randomised controlled trial. Intensive Care Med 2024;50(7):1075–85.
35. Shehabi Y, Balachandran M, Al-Bassam W, et al. Postoperative 20% albumin infusion and acute kidney injury in high-risk cardiac surgery patients: the ALBICS AKI randomized clinical trial. JAMA Surg 2025;160(8):835–44.
36. Wigmore GJ, Deane AM, Presneill JJ, et al. The renal effect of 20% human albumin solution fluid bolus therapy in patients after cardiac surgery. A secondary analysis of the HAS FLAIR II randomized clinical trial. J Cardiothorac Vasc Anesth 2025;39(4):967–74.
37. Skubas NJ, Callum J, Bathla A, et al. Intravenous albumin in cardiac and vascular surgery: a systematic review and meta-analysis. Br J Anaesth 2024; 132(2):237–50.
38. Vlaar APJ, Hofstra JJ, Determann RM, et al. Transfusion-related acute lung injury in cardiac surgery patients is characterized by pulmonary inflammation and coagulopathy: a prospective nested case-control study. Crit Care Med 2012;40(10):2813–20.
39. Hajjar LA, Vincent JL, Galas FRBG, et al. Transfusion requirements after cardiac surgery: the TRACS randomized controlled trial. JAMA 2010;304(14): 1559–67.
40. Mazer CD, Whitlock RP, Fergusson DA, et al. Restrictive or liberal red-cell transfusion for cardiac surgery. N Engl J Med 2017;377(22):2133–44.
41. Murphy GJ, Pike K, Rogers CA, et al. Liberal or restrictive transfusion after cardiac surgery. N Engl J Med 2015;372(11):997–1008.
42. Karkouti K, Callum JL, Bartoszko J, et al. Prothrombin complex concentrate vs frozen plasma for coagulopathic bleeding in cardiac surgery: the FARES-II multicenter randomized clinical trial. JAMA 2025;333(20):1781–92.
43. Meco M, Montisci A, Giustiniano E, et al. Viscoelastic blood tests use in adult cardiac surgery: meta-analysis, meta-regression, and trial sequential analysis. J Cardiothorac Vasc Anesth 2020;34(1): 119–27.
44. Totaro RJ, Raper RF. Epinephrine-induced lactic acidosis following cardiopulmonary bypass. Crit Care Med 1997;25(10):1693–9.
45. Maillet JM, Le Besnerais P, Cantoni M, et al. Frequency, risk factors, and outcome of hyperlactatemia after cardiac surgery. Chest 2003;123(5):1361–6.
46. Minton J, Sidebotham DA. Hyperlactatemia and cardiac surgery. J Extra Corpor Technol 2017;49(1): 7–15.
47. Bloom JE, Chan W, Kaye DM, et al. State of shock: contemporary vasopressor and inotrope use in cardiogenic shock. J Am Heart Assoc 2023;12(15): e029787. https://doi.org/10.1161/JAHA.123.029787.
48. Mathew R, Di Santo P, Jung RG, et al. Milrinone as compared with dobutamine in the treatment of

cardiogenic shock. N Engl J Med 2021;385(6):516–25.
49. Biswas S, Malik AH, Bandyopadhyay D, et al. Meta-analysis comparing the efficacy of dobutamine versus milrinone in acute decompensated heart failure and cardiogenic shock. Curr Probl Cardiol 2023;48(8):101245. https://doi.org/10.1016/j.cpcardiol.2022.101245.
50. Mathis MR, Janda AM, Kheterpal S, et al. Patient-Clinician-and institution-level variation in inotrope use for cardiac surgery: a multicenter observational analysis. Anesthesiology 2023;139(2):122–41.
51. Miles TJ, Blackburn KW, Moon MR, et al. Optimal inotropic support strategy in low cardiac output syndrome. Semin Thorac Cardiovasc Surg 2025;S1043-0679(25):00054-1.
52. Huraux C, Makita T, Montes F, et al. A comparative evaluation of the effects of multiple vasodilators on human internal mammary artery. Anesthesiology 1998;88(6). Available at: https://journals.lww.com/anesthesiology/fulltext/1998/06000/a_comparative_evaluation_of_the_effects_of.29.aspx.
53. Landoni G, Lomivorotov VV, Alvaro G, et al. Levosimendan for hemodynamic support after cardiac surgery. N Engl J Med 2017;376(21):2021–31.
54. Mehta RH, Leimberger JD, van Diepen S, et al. Levosimendan in patients with left ventricular dysfunction undergoing cardiac surgery. N Engl J Med 2017;376(21):2032–42.
55. Hajjar LA, Vincent JL, Barbosa Gomes Galas FR, et al. Vasopressin versus norepinephrine in patients with vasoplegic shock after cardiac surgery: the VANCS randomized controlled trial. Anesthesiology 2017;126(1):85–93.
56. Geube M, Abraham A, Kelava M, et al. Cardiopulmonary effects of vasopressin versus norepinephrine in patients undergoing cardiac surgery: a single-center, cluster-randomized crossover trial. J Thorac Cardiovasc Surg 2025;S0022-5223(25):00978-X.
57. McIntyre WF, Um KJ, Alhazzani W, et al. Association of vasopressin plus catecholamine vasopressors vs catecholamines alone with atrial fibrillation in patients with distributive shock: a systematic review and meta-analysis. JAMA 2018;319(18):1889–900.
58. Kotani Y, Di Gioia A, Landoni G, et al. An updated "norepinephrine equivalent" score in intensive care as a marker of shock severity. Crit Care 2023;27:29.
59. Ensor CR, Sabo RT, Voils SA. Impact of early postoperative hydrocortisone administration in cardiac surgical patients after cardiopulmonary bypass. Ann Pharmacother 2011;45(2):189–94.
60. Zakaria L, Shah K, Yang D, et al. Hydroxocobalamin for vasoplegia in cardiac surgery: a retrospective cohort analysis. Br J Anaesth 2025;135(4):870–7.
61. Khanna A, English SW, Wang XS, et al. Angiotensin II for the treatment of vasodilatory shock. N Engl J Med 2017;377(5):419–30.
62. Klijian A, Khanna AK, Reddy VS, et al. Treatment with angiotensin II is associated with rapid blood pressure response and vasopressor sparing in patients with vasoplegia after cardiac surgery: a post-hoc analysis of angiotensin II for the treatment of high-output shock (ATHOS-3) study. J Cardiothorac Vasc Anesth 2021;35(1):51–8.
63. Ghadimi K, Cappiello JL, Wright MC, et al. Inhaled epoprostenol compared with nitric oxide for right ventricular support after major cardiac surgery. Circulation 2023;148(17):1316–29.
64. Ghadimi K, Cappiello J, Cooter-Wright M, et al. Inhaled pulmonary vasodilator therapy in adult lung transplant: a randomized clinical trial. JAMA Surg 2022;157(1):e215856. https://doi.org/10.1001/jamasurg.2021.5856.
65. EL-Khuffash A, McNamara PJ, Breatnach C, et al. The use of milrinone in neonates with persistent pulmonary hypertension of the newborn - a randomised controlled trial pilot study (MINT 1): study protocol and review of literature. Matern Health Neonatol Perinatol 2018;4:24.
66. Alviar CL, Rico-Mesa JS, Morrow DA, et al. Positive pressure ventilation in cardiogenic shock: review of the evidence and practical advice for patients with mechanical circulatory support. Can J Cardiol 2020;36(2):300–12.
67. Grace MP, Greenbaum DM. Cardiac performance in response to PEEP in patients with cardiac dysfunction. Crit Care Med 1982;10(6):358–60.
68. Darrouj J, Karma L, Arora R. Cardiovascular manifestations of sedatives and analgesics in the critical care unit. Am J Ther 2009;16(4):339–53.
69. Gall SA, Olsen CO, Reves JG, et al. Beneficial effects of endotracheal extubation on ventricular performance. Implications for early extubation after cardiac operations. J Thorac Cardiovasc Surg 1988;95(5):819–27.
70. Writing Group for the PReVENT Investigators, Simonis FD, Serpa Neto A, Binnekade JM, et al. Effect of a low vs intermediate tidal volume strategy on ventilator-free days in intensive care unit patients without ARDS: a randomized clinical trial. JAMA 2018;320(18):1872–80.
71. Chang HC, Ho CH, Kung SC, et al. Maintenance of low driving pressure in patients with early acute respiratory distress syndrome significantly affects outcomes. Respir Res 2021;22(1):313.
72. Yamamoto R, Okazaki SR, Fujita Y, et al. Usefulness of low tidal volume ventilation strategy for patients with acute respiratory distress syndrome: a systematic review and meta-analysis. Sci Rep 2022;12:9331.
73. Tavazzi G, Costanza CNJC, Corradi CF, et al. PEEP trial and effect on hemodynamic in cardiogenic

shock. Eur Heart J Acute Cardiovasc Care 2024;13-(Supplement_1):zuae036–149.
74. Dongelmans DA, Hemmes SN, Kudoga AC, et al. Positive end-expiratory pressure following coronary artery bypass grafting. Minerva Anestesiol 2012; 78(7):790–800.
75. Rali AS, Tran LE, Auvil B, et al. Modifiable mechanical ventilation targets are associated with improved survival in ventilated VA-ECLS patients. JACC Heart Fail 2023;11(8 Pt 1):961–8.
76. Liu H, Wu X, Zhao X, et al. Intra-aortic balloon pump combined with mechanical ventilation for treating patients aged > 60 years in cardiogenic shock: retrospective analysis. J Int Med Res 2016;44(3):433–43.
77. Grant MC, Kanwar MK, Spelde AE, et al. Temporary mechanical circulatory support in cardiogenic shock: executive summary of the joint consensus reports of the PeriOperative quality initiative and the enhanced recovery after surgery cardiac society. Ann Thorac Surg 2025;120(2):194–201.
78. Spelde AE, Barron LM, Cangut B, et al. Escalation and De-escalation of temporary mechanical circulatory support: joint consensus report of the PeriOperative quality initiative and the enhanced recovery after surgery cardiac society. Ann Thorac Surg 2025;120(2):213–24.
79. Deschamps J, Obanor O, Clarizia N, et al. Best management practices on temporary mechanical circulatory support: joint consensus report of the PeriOperative quality initiative and the enhanced recovery after surgery cardiac society. Ann Thorac Surg 2025;120(2):225–43.
80. Grant MC, Brudney CS, Hernandez-Montfort J, et al. Definitions of cardiogenic shock and indications for temporary mechanical circulatory support: joint consensus report of the PeriOperative quality initiative and the enhanced recovery after surgery cardiac society. Ann Thorac Surg 2025;120(2):202–12.
81. Picado-Loaiza S, Ayala R, Ferreira ROM, et al. Early versus bail-out left ventricular unloading during venoarterial extracorporeal membrane oxygenation: a systematic review and meta-analysis. J Cardiothorac Vasc Anesth 2025;39(4):1015–25.
82. Pappalardo F, Schulte C, Pieri M, et al. Concomitant implantation of impella® on top of veno-arterial extracorporeal membrane oxygenation may improve survival of patients with cardiogenic shock. Eur J Heart Fail 2017;19(3):404–12.
83. Ramzy D, Soltesz EG, Silvestry S, et al. Improved clinical outcomes associated with the Impella 5.5 compared to the Impella 5.0 in contemporary cardiogenic shock and heart failure patients. J Heart Lung Transplant 2023;42(5):553–7.
84. Geller BJ, Sinha SS, Kapur NK, et al. Escalating and De-escalating temporary mechanical circulatory support in cardiogenic shock: a scientific statement from the American heart association. Circulation 2022;146(6):e50–68.
85. Pahuja M, Yerasi C, Lam PH, et al. Review of pathophysiology of cardiogenic shock and escalation of mechanical circulatory support devices. Curr Cardiol Rep 2023;25(4):213–27.
86. Eisenga J, McCullough K, Moubarak G, et al. The use of perioperative Impella 5.5 support in high-risk cardiac surgery: a retrospective cohort study. J Thorac Dis 2024;16(9):6045–51.
87. Saeed D, Maxhera B, Kamiya H, et al. Alternative right ventricular assist device implantation technique for patients with perioperative right ventricular failure. J Thorac Cardiovasc Surg 2015;149(3): 927–32.
88. Whitlock JP. Cardiac surgery unit advanced life support training: a 10-Year retrospective study examining patient mortality outcomes after implementation. Dimens Crit Care Nurs 2023;42(1):22–32.
89. Dunning J, Levine A, Ley J, et al. The society of thoracic surgeons expert consensus for the resuscitation of patients who arrest after cardiac surgery. Ann Thorac Surg 2017;103(3):1005–20.