Escalation Strategies in Cardiogenic Shock

From Early Recognition to Advanced Support

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KEYWORDS

• Cardiogenic shock • Escalation time to support • Heart failure • Acute myocardial infarction

KEY POINTS

- Cardiogenic shock has high mortality; survival depends on rapid recognition and timely escalation (time-to-escalation).
- Early phenotyping within 60 to 90 minutes using invasive hemodynamics guides escalation and device choice.
- Standardized transfer processes and cardiogenic shock coordinators improve safety during interhospital transfer.
- Escalation to temporary mechanical circulatory support should be phenotype-driven, balancing benefits with device-related risks; recovery-focused outcomes are essential.

INTRODUCTION

Despite major advances in acute cardiovascular care, cardiogenic shock (CS) continues to carry high mortality and remains a leading cause of cardiovascular decline and critical illness.[1,2] CS is defined by primary cardiac pump failure resulting in systemic hypoperfusion, progressive hemodynamic compromise, and multisystem organ dysfunction.[3,4] Although acute myocardial infarction (AMI) historically accounted for most cases, contemporary cohorts reveal a rising burden of acute and chronic heart failure–related CS (HF-CS).[5,6] Additional etiologies, including postcardiotomy shock, postcardiac arrest, severe valvular dysfunction, arrhythmia-mediated shock, peripartum cardiomyopathy, and myocarditis highlight the heterogeneous and rapidly evolving CS population.[7]

Despite these shifts in epidemiology, outcomes remain poor. Across modern mechanical circulatory support (MCS) trials, 180-day mortality consistently exceeds 50% irrespective of device or strategy.[8] Survivors experience substantial morbidity, characterized by prolonged intensive care unit stays, high resource utilization, need for invasive hemodynamic monitoring, vasoactive support, organ support, and recurrent decompensation.[7,9–11] Even after initial stabilization, rehospitalizations for both cardiac and noncardiac causes

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Abbreviations

ACC	American College of Cardiology
AMI	acute myocardial infarction
CS	cardiogenic shock
CSWG	Cardiogenic Shock Working Group
HF-CS	heart failure–related CS
HRT	heart replacement therapy
IABP	intra-aortic balloon pump
mAFP	microaxial flow pumps
MCS	mechanical circulatory support
PAC	pulmonary artery catheter
POCUS	point-of-care ultrasound
SCAI	Society for Cardiovascular Angiography and Interventions
TMCS	temporary mechanical circulatory support

remain frequent,[12,13] and survivors of CS continue to experience substantial long-term morbidity, complications, and elevated late mortality.[14]

The divergence between AMI-CS and HF-CS further underscores the urgency of timely escalation. While AMI-CS benefits from well-established systems of care modeled on ST-segment elevation myocardial infarction (STEMI) pathways, featuring early recognition, rapid transfer, and standardized reperfusion strategies,[15] HF-CS often presents with insidious hemodynamic decline, recurrent preshock episodes, and delayed recognition, all of which contribute to greater vulnerability to irreversible end-organ injury.[16,17] Evidence to guide escalation in HF-CS remains limited, with Altshock-2 representing one of the few randomized studies in this population.[18] Consequently, best practices are derived primarily from high-quality registry data.[19,20]

Across etiologies, a consistent theme has emerged: timely, structured escalation is the central determinant of survival. Early recognition, rapid hemodynamic profiling, standardized activation of shock teams, and coordinated transfer to centers capable of advanced MCS and multidisciplinary care are now recognized as essential components of CS management.[21,22] Integrating invasive hemodynamics with metabolic and perfusion biomarkers (*hemometabolics*) enables more precise identification of perfusion failure and earlier initiation of temporary MCS. The concept of *time-to-escalation*, analogous to door-to-balloon metrics in STEMI, has emerged as a critical quality marker for CS systems of care. Reducing delays between shock recognition and definitive escalation may limit end-organ injury, increase hemodynamic stability, and expand the window for myocardial recovery.[23,24]

This article provides a contemporary, systems-based framework for escalation in non-extracoproreal membrane oxygenation (ECMO) CS, emphasizing early recognition, standardized shock team activation, interhospital transfer logistics, device-specific considerations, and recovery-focused decision-making. Through synthesis of emerging evidence and real-world experience, we outline a comprehensive approach to escalation that spans the continuum from initial presentation to remission-phase assessment and long-term recovery.

INITIAL RECOGNITION AS THE GATEWAY TO TIMELY ESCALATION

CS is a time-dependent state of circulatory failure in which early recognition must be explicitly linked to escalation readiness to alter the trajectory of end-organ injury and mortality. Recognition in this context extends beyond identification of hypotension and encompasses timely clinical suspicion, rapid phenotypic characterization, and early identification of patients at risk for imminent deterioration. Building on the *door-to-support* paradigm introduced by Esposito and Kapur,[25] contemporary evidence demonstrates that delays in recognizing evolving shock physiology, rather than delays in treatment alone, drive progression to irreversible organ dysfunction. Reductions in time from presentation to escalation are associated with improved tissue perfusion, lower cumulative exposure to vasopressors and inotropes, fewer complications, enhanced likelihood of myocardial recovery, and reduced long-term morbidity.[14]

Recent guidance from the American College of Cardiology (ACC) Solution Set Oversight Committee, articulated in the 2025 ACC Expert Consensus Statement on the Evaluation and Management of Cardiogenic Shock, formally operationalizes this concept by emphasizing early suspicion and triage through the *SUSPECT* framework and recommending that initial phenotyping occur within the first 60 to 90 minutes of clinical contact.[23] This early phenotyping window is critical, as contemporary registry data demonstrate that progression in Society for Cardiovascular Angiography and Interventions (SCAI) shock stage within the first 6 to 12 hours is associated with the steepest increase in mortality, underscoring a narrow temporal window during which recognition can meaningfully influence outcomes before irreversible deterioration occurs (**Fig. 1**).[26–28]

Early identification therefore requires a high index of suspicion, particularly because hypotension is neither necessary nor sufficient for the diagnosis of CS. Hypotension, narrow pulse pressure (<25%), tachycardia, and clinical signs of congestion or hypoperfusion should prompt

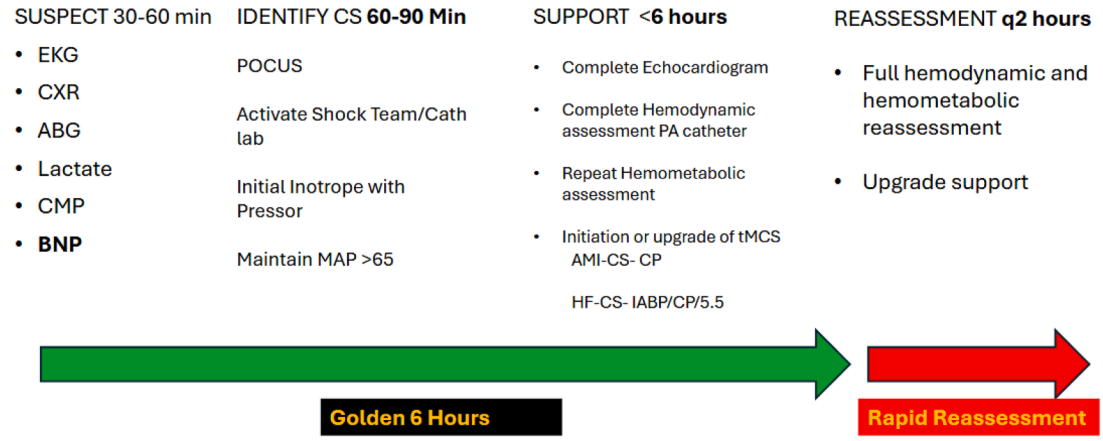

Fig. 1. Time to escalation in CS.

immediate evaluation and escalation-oriented triage.[23] Overreliance on blood pressure thresholds delays recognition of patients with preserved systolic blood pressure but impaired cardiac output and tissue hypoperfusion, a presentation frequently observed in HF-CS. Late recognition is consistently associated with higher rates of cardiac arrest, acute kidney injury, respiratory failure, mesenteric ischemia, and acute limb ischemia, each of which independently confers excess mortality.[29–31]

Initial diagnostic evaluation should proceed in parallel and be designed to rapidly define escalation risk. Electrocardiography, laboratory testing (comprehensive metabolic panel, arterial blood gas, lactate, and natriuretic peptides), and point-of-care ultrasound (POCUS) together refine early phenotyping and help distinguish cardiogenic from mixed shock states. Elevated lactate, metabolic acidosis, worsening renal function, and evidence of venous congestion identify patients with impaired systemic perfusion and high risk for rapid progression. While POCUS is valuable for bedside screening, its ability to define filling pressures, cardiac output, and biventricular involvement is limited. Comprehensive transthoracic echocardiography may be constrained by timing, availability, or imaging windows in unstable patients, making early invasive hemodynamic assessment a critical tool for precise diagnosis and escalation planning.[32]

Complete hemodynamic assessment using a pulmonary artery catheter (PAC) enables objective characterization of ventricular involvement, filling pressures, cardiac output, and systemic perfusion, and has been associated with improved survival across CS etiologies.[33,34] PAC-guided therapy is also linked to more rapid lactate clearance in AMI-CS, reinforcing its role in identifying effective escalation strategies rather than serving solely as a diagnostic adjunct.[34] Early invasive hemodynamic profiling facilitates accurate SCAI staging and identifies patients most likely to progress during the early high-risk window when escalation has the greatest potential to alter outcomes.

Recognition patterns vary substantially by CS phenotype. AMI-CS is often identified rapidly due to overt electrocardiographic and biomarker abnormalities, whereas HF-CS frequently presents with insidious symptoms and attenuated diagnostic signals, resulting in delayed recognition despite comparable or greater physiologic severity.[35] This disparity in diagnostic signal strength, rather than differences in underlying risk, contributes to delayed escalation and poorer outcomes in HF-CS populations. Observational data from the Detroit Cardiogenic Shock Initiative, National Cardiogenic Shock Initiative, and DanGer Shock trial demonstrate that earlier identification of high-risk hemodynamics, reflected by anticipatory deployment of temporary mechanical circulatory support (tMCS) before percutaneous coronary intervention, is associated with improved outcomes, underscoring the importance of recognizing escalation thresholds before overt collapse occurs.[36–38]

Once hemodynamic phenotyping is established, escalation readiness becomes the dominant determinant of outcome. Accurate characterization of left-versus right-sided failure, biventricular involvement, filling pressures, cardiac output, and systemic perfusion is essential to guide timely selection of the optimal tMCS strategy.[39] Recognition without access to escalation pathways is insufficient; diagnostic clarity must be paired with systems capable of delivering rapid transfer, coordinated escalation, and advanced support. Across CS phenotypes, time-to-lactate clearance remains

a robust marker of effective perfusion restoration and treatment response,[40,41] reinforcing that early recognition must be inseparable from early escalation, continuous invasive monitoring, and iterative hemodynamic optimization to improve outcomes.[42]

TRANSFER AND SYSTEMS OF CARE

While early recognition is essential, the safe, efficient, and timely interhospital transfer of patients requiring escalation remains equally crucial. Many patients initially present to institutions lacking advanced tMCS platforms, cardiac surgery, or transplant capabilities. For these individuals, transfer represents a period of heightened vulnerability, occurring precisely when patients are at the greatest risk of clinical deterioration.

Data from 3268 patients in the Cardiogenic Shock Working Group (CSWG) registry demonstrate that more than half of patients experience worsening SCAI stage within the first 24 hours after diagnosis, and early deterioration is strongly associated with higher in-hospital mortality.[24] Because interhospital transfer typically occurs within this same high-risk interval, the window for physiologic instability, device malfunction, and end-organ injury is substantial.[24,27,28] Patients requiring transfer generally have higher comorbidity burdens, more advanced organ failure, and more frequent need for critical care interventions, factors that compound the inherent transport risks.[43–45]

Transfer of patients supported with tMCS adds further complexity. Bleeding, already one of the most common MCS-related complications, may be exacerbated by movement, catheter angulation, or disruption of vascular access sites.[46] Malposition of large-bore arterial or venous cannulas during transport increases the risk of hemolysis, vascular injury, arrhythmia, and loss of circulatory support. In addition, transport personnel may have limited familiarity with device troubleshooting, raising the likelihood of unrecognized alarms or suboptimal device flows during extended transfers.

These vulnerabilities underscore the need for standardized, protocol-driven transfer pathways. A structured *shock transfer bundle* offers a mechanism to reduce preventable complications by delineating predeparture stabilization steps, device-specific safety checks, hemodynamic assessment targets, and communication expectations between sending and receiving teams (**Box 1**). While comprehensive tMCS training for all transport teams may not be feasible, having clear, device-specific checklists with assigned intervals for reassessment can improve safety. Equally important is ensuring transport personnel have immediate access, via phone or telehealth, to clinicians familiar with the patient's device and anatomy.[47,48]

Box 1
Impella CP transfer checklist

- Mean arterial pressure (MAP) greater than 65 and heart rate (HR) greater than 60 on acceptable doses of vasopressors/inotropes
- Stable Sp02 greater than 92%
- Absence of Impella position alarms unless position confirmed by echo
- Catheter secured with securement device
- Catheter insertion placement assessed before transfer and reassessed upon admission.
- Limb ischemia addressed
- Absence of suction alarms
- Bleeding is controlled
- Hemolysis, if present, has been addressed via echo for positioning and valve interaction
- Handoff communication between cardiogenic shock attending, and STAT MDOC has been completed

Developed by Ramanan R, Guyette FX, Kunz N. Approved by Cardiogenic Shock Steering Committee 4/2025. UPMC Presbyterian Hospital, Pittsburgh, PA.

Dedicated CS coordinators, where available, are poised to play a central role in this process. Continuous 24/7 CS coordinator coverage, such as implemented at Medical University of South Carolina, allows a coordinator who is already involved in the initial multidisciplinary shock call to serve as the primary contact during transport.[49] This ensures continuity, enables rapid escalation if complications arise, and facilitates efficient communication with the receiving clinical team. Beyond transfer logistics, coordinators contribute meaningfully to quality improvement, protocol adherence, education, and cross-institutional learning. As the field evolves, these roles may expand to encompass the responsibilities of *implementation scientists*, helping to disseminate best practices across systems of care.[49] (**Box 2**).

Transport systems remain ripe for innovation. Real-time physiologic data transmission, including vital signs, tMCS console parameters, hemodynamic trends, arrhythmia monitoring, and vasoactive dosing, could substantially enhance safety by enabling remote oversight from the receiving team during transfer. Current transport monitors lack these capabilities, but next-generation platforms,

Box 2
Best practices in escalation in cardiogenic shock

1. Early Identification process: *SUSPECT* CS, use POCUS
2. Activate Shock team (Tier 3) and Cath laboratory (Tier 1 and 2)
3. Early use of Inotropes with Pressors Goal MAP greater than 65
4. Hemometabolic and Hemodynamic monitoring (PA catheters preferred)
5. tMCS placement (CP in AMI, 5.5. first device vs IABP/CP in HF-CS)
6. Order sets for all patients on MCS
   - Nursing communication to identify complications, adverse events, and anticoagulation
7. Rapid upgrade to 2nd device (requires surgical availability and expertise)
   - Recommend 24/7 availability upgrade to Impella 5.5. to avoid delays in care, VA ECMO requirement

including artificial inelligence (AI)-enabled remote monitoring and electronic heart record (EHR)-integrated shock alert systems (Fig. 2), may transform the interhospital transfer paradigm.

Altogether, the interhospital transfer period represents one of the most precarious phases of CS care. Standardized transfer bundles, device-specific checklists, real-time communication pathways, and the evolving role of CS coordinators offer promising strategies to improve the safety and reliability of transfers within regionalized shock networks.

ESCALATION TO TEMPORARY MECHANICAL CIRCULATORY SUPPORT: DEVICE-SPECIFIC AND SYSTEMS-LEVEL CONSIDERATIONS

tMCS represents a central escalation strategy in CS, providing partial or full circulatory support as a bridge to recovery, decision, or heart replacement therapy (HRT).[50] Optimal escalation requires integration of patient phenotype, hemodynamic profile, anticipated trajectory, and institutional capability. Importantly, the benefit of tMCS is highly time-dependent and must be weighed against device-specific risks, underscoring the need for structured escalation pathways informed by invasive hemodynamic assessment.

INTRA-AORTIC BALLOON PUMP

The intra-aortic balloon pump (IABP) remains the most commonly used t-MCS device in the United States for both AMI-CS and HF-CS.[1,2,51,52] Its continued use reflects ease of insertion, small cannula profile (typically 8 French), relatively low complication rates, and feasibility of deployment at hospitals without advanced surgical infrastructure.[30,53,54] These features make IABP a readily accessible escalation option in early or evolving shock (Fig. 3).

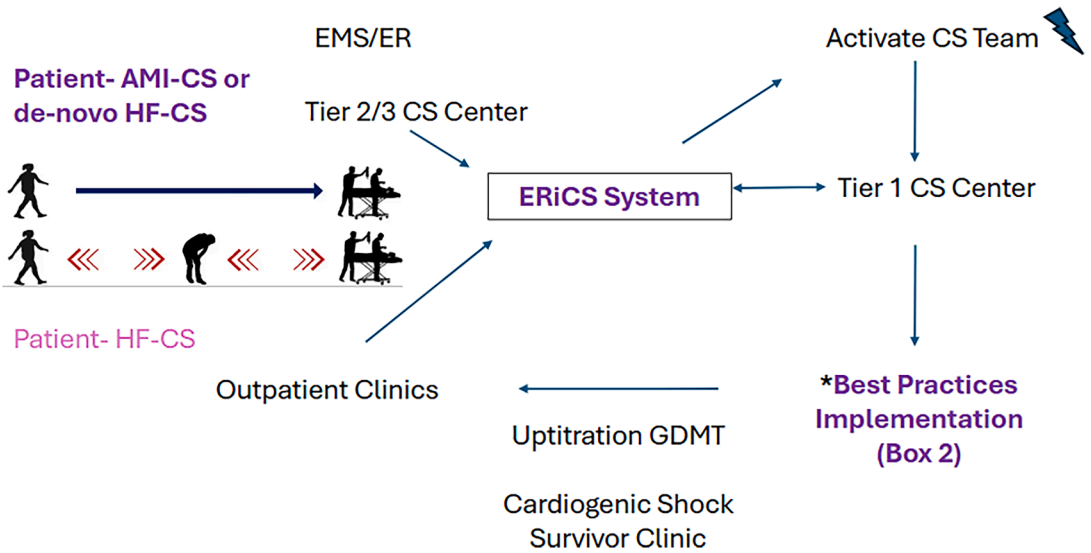

Fig. 2. Escalation using systems of care with HEHR Recognition of Cardiogenic Shock: *ERoCS* System. Fig. 2 depicts a future system with incorporation of an EHR based alert system with escalation and best practices implementation. (*Image Courtesy* Developed in coordination with Christopher Horvat, MD, MHA University of Pittsburgh, Department of Pediatrics.)

Post-Impella CP Insertion Order Set Template		
Domain	**Order / Parameter**	**Details**
Communication to Nursing	Impella Performance Level	Maintain performance level at **P*****. All performance level changes must be made by MD*** or mechanical support team only***.
Communication to Nursing	Hemodynamic Profile Assessment	Level patient at time of hemodynamic measurements. Assess PA catheter placement measurements and notify attending MD if catheter movement **>5 cm**.
Communication to Nursing	Impella Catheter Position	Measure Impella catheter insertion site **every 2 hours and after ambulation**. Notify attending MD of any changes in catheter position.
Dressing Care	Insertion Site Dressing	Occlusive, sterile dressing change **weekly** at insertion site per ICU procedure***. Increase frequency if drainage present. Dressing should allow visualization of measurements and access to repositioning button.
Vital Signs	Vital Signs with Site & Neurovascular Checks	Temperature and neuro checks **Q2H**. Assess Impella insertion site for bleeding or hematoma. Check pedal pulses with Doppler to assess leg perfusion. Notify attending MD immediately for any loss of pulses or decreased perfusion.
Nursing Documentation	Q2H Documentation	Document **Impella performance level, Impella flow, purge pressure, and motor current** every 2 hours.
Hemodynamic Monitoring	Invasive Hemodynamics	**SvO_2, PAS/PAD, PA(M), CO/CI, SVR/SVRI** measured at **Q2H, Q4H, Q8H, and Q24H** intervals as ordered.
Notify Attending Service***	Clinical Deterioration	Notify attending physician and consider **Cardiogenic Shock Team activation***** for: MAP <60, rising lactate, PAPi <1, CPO <0.6, Impella CP running >P7, hematuria, LDH >2× ULN or plasma-free Hgb >30, loss of limb pulses, insertion site bleeding/hematoma, new oliguria unresponsive to fluid challenge in 1 hour, or unanticipated need for dialysis.
Notify Attending Service***	Device Alarms	Notify **mechanical support team (pager ____)***** for alarms and/or device-related questions.
Notify Other	Manufacturer Support	Notify **Abiomed Clinical Support** for alarms/questions: **1-800-422-8666** or local representative.
Impella Maintenance	Side-Arm Flush	Maintain normal saline flush via pressurized (**300 mmHg**) flush bag on Impella red side-arm port.
Impella Maintenance	Side-Arm Port Use	Side-arm port of sheath must remain capped and **not used** for blood draws or infusions.
Notify Attending Service***	Pump Position or Suction Alarms	Notify attending MD and, for PUH patients, **Mechanical Support team***** for suction events or incorrect pump position alarms.

Fig. 3. Order set template. *** Indicate areas where manual entry is required, or where center-specific protocols may be added to the template.

Randomized trials including IABP-SHOCK II and Altshock-2 did not demonstrate a mortality benefit for IABP in unselected AMI-CS and HF-CS populations, respectively.[18,55] However, these neutral findings do not preclude a role for IABP as an escalation strategy in selected phenotypes. Observational data suggest potential benefit in patients with severe mitral regurgitation, those requiring preoperative stabilization before coronary artery bypass grafting, and advanced HF patients awaiting transplantation.[56] In the CSWG registry, patients with HF-CS supported with femoral IABP demonstrated lower mortality and complication rates compared with those with AMI-CS, likely reflecting differences in ischemic burden and ventricular reserve.[57]

Axillary IABP placement at experienced centers has been associated with higher rates of successful bridge to HRT without an increase in complications, highlighting the influence of access strategy and institutional expertise on escalation success.[58] Emerging hemodynamic markers, including aortic pulsatility index and diastolic blood pressure augmentation, have been proposed as predictors of favorable response to IABP support.[59,60] While these findings require prospective validation, they suggest that refined phenotyping may improve patient selection for IABP escalation.

MICROAXIAL FLOW PUMPS (IMPELLA)

Microaxial flow pumps (mAFP), including Impella 2.5, CP, RP, and surgically implanted Impella 5.0/5.5, provide incremental levels of circulatory support via percutaneous or surgical approaches and are increasingly used as primary escalation platforms in CS.[1,2,51,52] The DanGer Shock trial demonstrated a survival benefit associated with early Impella support in AMI-CS, leading to guideline endorsement with a Class II recommendation and Level of Evidence A.[38,61] These data support early escalation to mAFP before irreversible end-organ injury occurs.

Mechanistically, mAFP devices unload the left ventricle by reducing left ventricular end-diastolic pressure, decreasing myocardial oxygen demand, increasing mean arterial pressure, and improving systemic perfusion.[62] These physiologic effects make mAFP particularly attractive in patients with predominant left-sided failure and persistent hypoperfusion despite pharmacologic support. However, escalation to mAFP carries device-specific risks, including hemolysis, limb ischemia, bleeding, and acute kidney injury.[30,63] Anticoagulation strategies, purge solution composition, and vascular access management remain heterogeneous across institutions, with optimal protocols still under investigation.[30,64]

Impella 5.5 has emerged as a preferred escalation platform in HF-CS, offering high flow rates, stable axillary positioning, lower hemolysis burden, and the ability to ambulate and engage in prehabilitation.[65,66] Registry data demonstrate prolonged support durations exceeding 14 days with high rates of successful transition to HRT and low overall adverse event rates.[67] While enthusiasm for early or primary escalation to Impella 5.5 is growing, particularly in advanced HF-CS, comparative effectiveness data remain limited.

SYSTEMS-BASED DRIVERS OF ESCALATION SUCCESS AND COMPLICATION BURDEN

Successful escalation to tMCS is not determined by device selection alone but is strongly influenced by system-level capability, including institutional experience, procedural infrastructure, and access to multidisciplinary expertise. Across CS phenotypes, observational analyses demonstrate lower mortality and higher utilization of guideline-directed invasive therapies at high-volume tertiary centers with continuous availability of advanced interventional cardiology, cardiothoracic surgery, and hybrid operating room resources.[68–70] These associations likely reflect the ability of such centers to deliver timely escalation, manage device-related complications, and adapt support strategies as shock physiology evolves.

tMCS provides short-term hemodynamic augmentation ranging from hours to weeks and may serve as a bridge to decision, recovery, or durable heart replacement therapy.[50] Devices offer variable degrees of circulatory support and differential effects on myocardial oxygen consumption, left ventricular unloading, wall stress, and coronary perfusion, necessitating dynamic reassessment rather than static device assignment.[50] Importantly, escalation strategies must balance hemodynamic benefit against device-associated complications, which remain a major determinant of outcomes and often serve as the clinical trigger for support intensification rather than de-escalation.

The DanGer Shock trial underscored this principle by demonstrating that early Impella CP support in ST-STEMI-CS was associated with improved survival but at the cost of increased major bleeding and vascular complications. The excess bleeding risk translated into a number needed to harm of approximately 11 to 14, depending on bleeding definition, highlighting that insufficient flow support, prolonged exposure to smaller-profile devices, or delayed escalation may amplify complication burden.[38] These findings have informed contemporary escalation paradigms favoring early transition from IABP or Impella CP to higher-flow platforms, such as surgically implanted Impella 5.5, in patients with persistent hypoperfusion or rising complication risk, particularly in centers with appropriate surgical capability (Fig. 4).

Complication-driven escalation is therefore increasingly recognized as a core component of

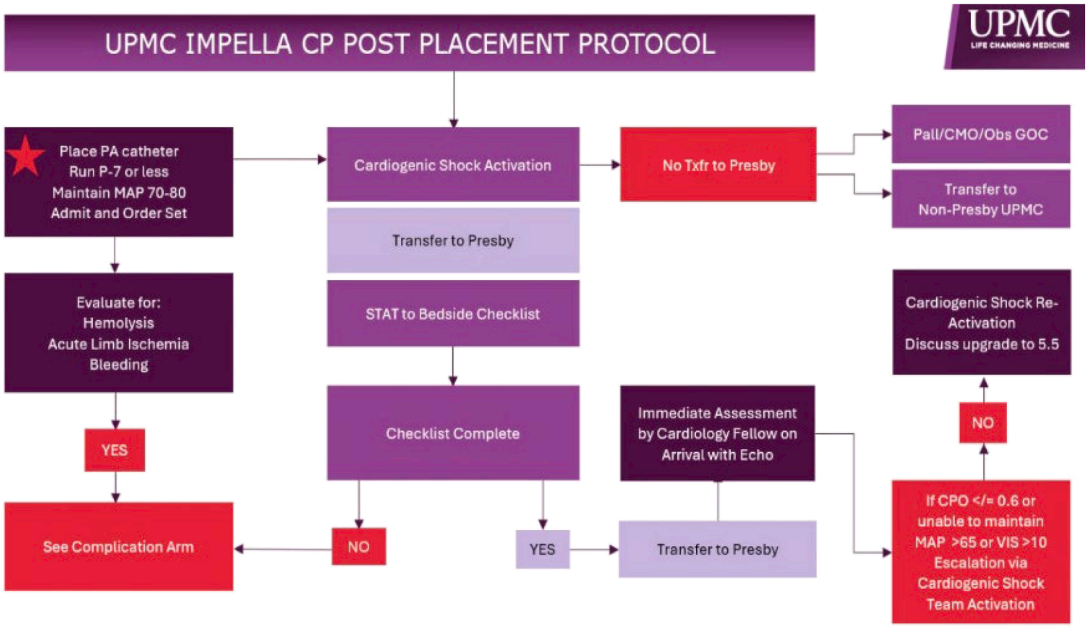

Fig. 4. Impella CP post placement protocol.

modern CS management. Bleeding, hemolysis, limb ischemia, and inadequate unloading are not merely adverse events but physiologic signals indicating mismatch between patient requirements and support capacity. Early and serial invasive hemodynamic assessment is essential to guide these decisions, yet existing risk prediction models, including CardShock, IABP-SHOCK II, and the Inova Heart and Vascular Institute risk scores, predict mortality rather than recovery and lack external validation across mixed CS populations, particularly HF-CS.[68,71–73]

Accordingly, escalation frameworks increasingly emphasize structured reassessment, predefined triggers for device upgrade, and systems capable of executing higher-level support without delay. Prior scientific statements have outlined escalation and management schemas for device selection[50,74] building on this foundation, integrating complication-aware escalation pathways (**Fig. 5**), supported by real-time hemodynamics and surgical readiness, represents a critical opportunity to reduce preventable morbidity and optimize outcomes across CS phenotypes.

ESCALATION WITH A FOCUS ON RECOVERY

Escalation of care in CS, especially to tMCS, represents a critical transition point that influences subsequent clinical trajectories, including native heart recovery, progression to durable mechanical support or transplantation, and mortality. Contemporary cohort and registry studies demonstrate substantial heterogeneity in postshock outcomes among patients who survive the acute phase following escalation, underscoring that short-term survival alone does not capture the full impact of escalation strategies.[14] As a result, evaluation of escalation in CS increasingly incorporates longer-term measures of myocardial function, end-organ recovery, and downstream treatment pathways.

Across CS phenotypes, observational data suggest that early hemodynamic stabilization and ventricular unloading are associated with improved systemic perfusion and end-organ function.[75] In selected patient populations, these interventions may facilitate partial or complete recovery of ventricular function. Mechanical circulatory support devices, including microaxial flow pumps and

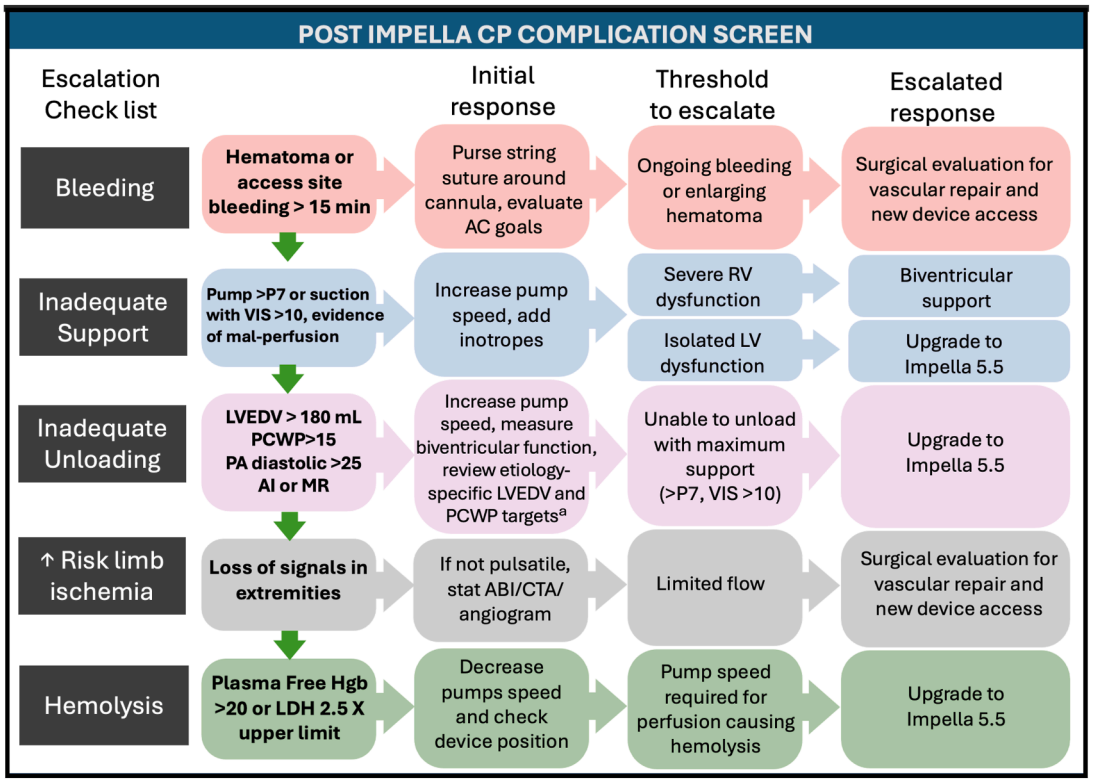

Fig. 5. Post Impella CP Complication Screen. [a]For acute on chronic HF-CS patients, achieving a left ventricular end-diastolic volume (LVEDV) within normal limits may not be possible in the acute setting. A higher pulmonary capillary wedge pressure (PCWP) target may be required initially. For AMI-CS, more aggressive targets (lower LVEDV and PCWP) are ideal.

surgically implanted ventricular assist platforms, reduce left ventricular filling pressures and myocardial oxygen demand while increasing cardiac output and mean arterial pressure.[76] However, recovery of native cardiac function remains variable and highly dependent on underlying etiology, severity and duration of shock, and degree of preexisting myocardial injury. Persistent hypoperfusion, failure of metabolic indices to improve, or progression of multiorgan dysfunction despite adequate support tend to predict adverse outcomes and limited potential for myocardial recovery.

Assessment of recovery potential following escalation relies on serial, multimodal evaluation rather than isolated measurements. Repeated transthoracic echocardiography provides longitudinal assessment of biventricular systolic function, loading conditions, and valvular competence, which, when interpreted alongside invasive hemodynamic data, helps contextualize myocardial performance during support.[76–78] In patients stable enough to undergo advanced imaging, cardiac magnetic resonance may offer additional prognostic information; in myocarditis and ischemic injury cohorts, limited late gadolinium enhancement has been associated with reversibility, whereas extensive scar burden correlates with poor functional recovery. Metabolic and end-organ trajectories, including lactate clearance, normalization of mixed venous oxygen saturation, declining natriuretic peptide levels, and improvement in renal and hepatic indices, are among the most consistently reported markers of effective perfusion restoration and are associated with improved short- and intermediate-term outcomes after escalation.[41,79]

Etiologic and genetic factors further influence recovery trajectories.[80,81] Observational studies in cardiomyopathy populations indicate that pathogenic variants associated with malignant or progressive myocardial disease (including LMNA, DSP, FLNC, and truncating TTN variants) are associated with reduced likelihood of sustained functional recovery, whereas inflammatory or stress-mediated cardiomyopathies more frequently demonstrate improvement in ventricular function following stabilization and unloading. At present, incorporation of genetic data into acute escalation decision-making remains limited, and their role is best considered in the context of longer-term prognostication rather than immediate escalation strategy.

Functional status and physical resilience are increasingly recognized as relevant considerations during prolonged t-MCS support. Contemporary series have reported that axillary ventricular assist platforms enable ambulation and participation in physical rehabilitation during extended support, with acceptable safety profiles and feasibility of transition to recovery or advanced therapies in selected patients.[58] While these observations suggest potential benefits related to preservation of conditioning, comparative effectiveness data remain limited, and causality cannot be inferred.

Despite growing clinical experience, substantial evidence gaps remain. No validated predictive model integrates imaging, hemodynamic, metabolic, end-organ recovery, and genetic data to reliably identify patients most likely to achieve native heart recovery following escalation. Comparative data evaluating early versus delayed escalation strategies outside of AMI-CS are limited, and definitions of pharmacologic non-response remain heterogeneous across studies and clinical practice. Addressing these gaps will require prospective, pragmatic trials and registry-based analyses that extend beyond short-term mortality to incorporate recovery-focused outcomes, including myocardial functional recovery, physical capacity, health-related quality of life, neurocognitive status, and long-term health-care utilization.[14] Emphasizing these patient-centered endpoints is essential to accurately characterize the full impact of escalation strategies and to inform decision-making that aligns survival with meaningful functional recovery.

SUMMARY

Outcomes in CS are strongly associated with the timeliness of recognition and the intensity of escalation. Observational data consistently demonstrate that delays in diagnosis, underuse of invasive hemodynamic assessment, and inconsistent access to timely tMCS are associated with early clinical deterioration and increased mortality. Escalation to advanced support should therefore be viewed as a trajectory-defining intervention that influences downstream stabilization, end-organ recovery, and candidacy for durable therapies rather than as a rescue strategy applied late in shock progression. However, current practice remains limited by heterogeneous escalation thresholds, variable definitions of pharmacologic nonresponse, and a lack of comparative data to guide escalation strategies across shock phenotypes. Advancing care will require prospective trials and registry-based learning health systems that emphasize early recognition, protocolized escalation, multidisciplinary management, and recovery-oriented endpoints beyond mortality, aligning survival with meaningful functional outcomes in patients with CS.

CLINICS CARE POINTS

- SUSPECT cardiogenic shock and obtain appropriate cardiac testing in patients with hypotension and/or hypoperfusion.
- Narrow pulse pressure in the setting of tachycardia can be a sign of cardiogenic shock.
- Point of care ultrasound should be utilized early in patients with suspected cardiogenic shock.
- Rapid activation of regional cardiogenic shock teams have shown survival benefit.
- Full hemodynamic assessment with PA catheter allows for phenotyping and aids in determination of appropriate determination of pharmacologic and mechanical support.
- Frequent reassessment of biomarkers and hemodynamics allows for appropriate escalation and de-escalation.

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