

Best Management Practices on Temporary Mechanical Circulatory Support: Joint Consensus Report of the PeriOperative Quality Initiative and the Enhanced Recovery After Surgery Cardiac Society



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ABSTRACT

BACKGROUND Effective use of temporary mechanical circulatory support (tMCS) mandates a multifaceted understanding of patient physiology, device technology, procedural techniques, patient-device interactions, and interdisciplinary collaboration. The consensus statement presented here endeavors to provide clinicians with a practical roadmap incorporating evidence-based best practices in several key areas that delineate the initial priorities in mechanical ventilation, anticoagulation, sedation, and monitoring for patients requiring tMCS.

METHODS With an interdisciplinary, international group of clinicians and through a structured literature review, a modified Delphi method was used to achieve consensus on best practices in tMCS.

RESULTS Nine key questions were developed with accompanying statements to direct areas that institutions and providers should prioritize to optimize care. These questions included: What expertise is required within the interdisciplinary team to optimize patient care? How should medical centers facilitate escalation of care when indicated? What is the optimal ventilation management strategy? What are the recommended gas exchange targets to preserve end-organ function? What is the recommended timing to start or resume anticoagulation? What anticoagulation agent and monitoring approach should be used routinely? What is the optimal strategy for patient comfort and device interactions? Can a patient on tMCS be mobilized? What routine monitoring needs to be performed?

CONCLUSIONS A comprehensive review is provided of key management strategies incorporating interdisciplinary team and evidence-based medical knowledge to improve patient outcomes while using tMCS.

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Abbreviations and Acronyms

aPTT	= activated partial thromboplastin time
ARDS	= acute respiratory distress syndrome
CS	= cardiogenic shock
ECMO	= extracorporeal membrane oxygenation
ECPR	= extracorporeal cardiopulmonary resuscitation
IABP	= intra-aortic balloon pump
ICU	= intensive care unit
IMV	= invasive mechanical ventilation
LPV	= lung protective ventilation
LV	= left ventricle
LVAD	= left ventricular assist device
tLVAD	= temporary left ventricular assist device
MCS	= mechanical circulatory support
PCWP	= pulmonary capillary wedge pressure
POQI	= PeriOperative Quality Initiative
RV	= right ventricle
SDM	= shared decision-making
tMCS	= temporary mechanical circulatory support
VA	= venoarterial
VAD	= ventricular assist device
VV	= venovenous

The evidence-based propagation of shock teams has propelled temporary mechanical circulatory support (tMCS) to become an essential tool for acute decompensated cardiogenic shock (CS) management.^{1,2} Clinicians may be challenged to select the most appropriate tMCS devices and implement best practices to optimize patient outcomes. Effective use of tMCS mandates a multifaceted understanding of patient physiology, device technology, procedural techniques, patient-device interactions, and interdisciplinary collaboration, thus making it a challenging endeavor. Recent randomized controlled trial data suggest that the implementation of best practices may be essential to ensure favorable outcomes in patients who require tMCS.³

For related articles, see pages 194, 202, 213

This is the third document of 3 reports linked through an executive summary. These reports present the fundamental principles of patient and institutional factors that influence the use of tMCS. We acknowledge that this is a vast body of literature and have endeavored to distill it into practical guidance for providers caring for these vulnerable patients. To achieve this, we have organized the content into 3 distinct areas.

First, we provide an overview of the definitions of CS and the indications for tMCS. Second, we present an algorithmic approach for the escalation and de-escalation of tMCS for patients in CS. Third, and the content of the current document, we aim to

apply enhanced recovery best practices for managing patients on tMCS in the intensive care unit (ICU).

Recognizing the rapid expansion of centers that provide tMCS therapies, particularly in the post-coronavirus disease era, we acknowledge that centers vary in volume and capacity. Therefore, it is essential for the reader to adapt and contextualize this content to their specific center or health care system. These reviews intend to offer actionable guidance for centers with varied tMCS capabilities.

MATERIAL AND METHODS

PeriOperative Quality Initiative (POQI) is a nonprofit organization that assembles international, multidisciplinary groups to develop consensus statements on key topics pertinent to perioperative medicine. From January 24 to 26, 2024, the 14th POQI meeting convened in person in conjunction with the Enhanced Recovery After Surgery (ERAS) Cardiac Society to address topics relevant to the management of CS and MCS. A group of experts was identified with clinical backgrounds in anesthesiology, cardiothoracic surgery, cardiology, and nursing, with a particular focus on CS and tMCS. For this effort, tMCS includes any nondurable device designed to support cardiac function, including intra-aortic balloon pump (IABP), left ventricular assist device (LVAD) and right ventricular (RV) assist device (RVAD), and venoarterial extracorporeal membrane oxygenation (VA-ECMO). This review was generated from a subgroup who appraised the best management practices for tMCS.

The POQI methodology has been previously described.⁴ Briefly, each POQI conference reviews 3 topics related to the central theme of that conference. The topics are selected by the POQI Board, and the conference directors based on the potential for developing clinical practice recommendations to improve patient care. The preconference phase consists of a literature review, identification of key questions to be assessed, and initial summary statements. The conference phase consists of sequential plenary and breakout sessions to debate and refine statements based on literature. All participants vote on key questions and statements in a final plenary session, with the vote recorded for transparency and to act as a subsequent record. After the conference, each workgroup finalizes its work for publication in a peer reviewed journal. Question 1 was revised for clarity during postconference manuscript writing. All delegates reviewed the final manuscripts before submission, and after publication, the manuscripts and figures

are made available on the POQI website (www.poqi.org).

RESULTS

Key Questions:

1. What are the required elements to establish goals and objectives for tMCS therapies?
2. How should medical centers facilitate escalation of care when indicated?
3. What is the optimal ventilation management strategy?
4. What are the recommended gas exchange targets to preserve end-organ function?
5. What is the recommended timing to start or resume anticoagulation?
6. What anticoagulation agent and monitoring approach should be used routinely?
7. What is the optimal strategy for patient comfort and device interactions?
8. Can a patient on tMCS be mobilized?
9. What routine monitoring needs to be performed?

COMMENT

Question 1: What are the required elements to establish goals and objectives for tMCS therapies?

Statement: Teams should be capable of providing shared decision-making (SDM) and palliative support services tailored to each center's expertise and resources.

Level of certainty: High

Rationale:

SHARED DECISION-MAKING. Despite its potential benefits, tMCS poses significant challenges in decision-making regarding goals of care due to its associated risks, resource intensiveness, and uncertain outcomes.⁵⁻⁷ The care goals for patients on tMCS should be individualized, considering the patient's prognosis, values, preferences, and overall clinical condition. SDM ensures that patients are well informed about their options, potential benefits, risks, and alternatives, enabling them to actively participate in making decisions that align with their preferences and values.^{8,9} Establishing realistic expectations and communicating effectively with patients and their families is essential. Although the primary goal of tMCS is to provide temporary support to allow for recovery of the underlying condition, the goals of care may include (but are not limited to):

1. Bridge to recovery: For some patients, this is the primary goal, and as such, aggressive treatment and ongoing support may be warranted to maximize the chances of a meaningful recovery.
2. Bridge to decision or definitive therapy: If the use of tMCS does not result in sufficient myocardial recovery to support hemodynamics and liberation from devices, the decision on candidacy for more advanced heart failure therapies, such as durable ventricular assist devices (VADs) or cardiac transplantation, will need to be considered.
3. Palliation and comfort: In situations where the prognosis is poor or recovery is unlikely, the focus may shift toward palliation and maximizing comfort. This involves symptom management, psychosocial support, and facilitating a peaceful and dignified end-of-life experience.

PALLIATIVE CARE/SUPPORTIVE CARE MEDICINE. Palliative care (ie supportive care medicine), focusing on improving quality of life and aligning medical care with patient preferences, is crucial in supporting patients and families throughout the tMCS journey.^{6,7} Palliative care should be integrated early into the care of patients on tMCS to effectively address physical, psychological, social, and spiritual needs. Palliative care involvement can:

1. Provide comprehensive information about prognosis, treatment options, and potential outcomes to help patients and families navigate complex medical decisions.
2. Help manage distressing symptoms, enhancing the patient's comfort and quality of life during ECMO support.
3. Provide psychosocial support through counseling, emotional support, and resources to address their psychosocial needs effectively.
4. Assist with transitions in goals of care.

Question 2: How should medical centers facilitate escalation of care when indicated?

Level of certainty: High

Statement: Teams should develop a partnership with a hospital system with advanced capabilities.

Rationale:

MODELS OF CARE. CS management requires an interdisciplinary team for optimal management, particularly when advanced mechanical support is required. Creating and maintaining an experienced interdisciplinary team may be difficult in

TABLE 1 Suggested Organized Interdisciplinary Approach

Approach	Minimally Acceptable ^a	Better	Best
Outcome assessment	Identification of goals of tMCS Involvement of patient or SDM in care decisions.	Identification of goals of tMCS. Patient or SDM involvement in care decisions. Palliative care involvement into patient management.	Identification of goals of tMCS. Patient or SDM involvement in care decisions. Palliative care involvement into patient management. Standardized multidisciplinary review of all tMCS cases.
Multicenter collaborative care	Partnership with high-volume center with advanced capabilities. Standardized communication tools to organize assessment and transfers.	Partnership with high-volume center with advanced capabilities. Standardized communication tools to organize assessment and transfers. Simultaneous multidisciplinary team discussion of referred cases at time of transfer.	Partnership with high-volume center with advanced capabilities. Standardized communication tools to organize assessment and transfers. Simultaneous multidisciplinary team discussion of referred cases at time of transfer. Feedback process to review outcomes between referring and reference centers.

^aThese suggestions represent minimally acceptable targets for short durations only, assuming stable hemodynamics that allow the ability to progress to better or best categories. Inability to progress to the better category should warrant consideration of transfer to a more experienced center. SDM, shared decision-making; tMCS, temporary mechanical circulatory support.

centers with lower CS and mechanical support volume. Data suggest that higher-volume centers have better outcomes for CS, nontraumatic out-of-hospital cardiac arrest, and acute myocardial infarctions,¹⁰⁻¹² which is in keeping with other disease states.¹³ Multiple models have been explored, with the hub-and-spoke model being a common method for organizing regional care in CS and advanced mechanical support.^{5,14,15} Regionalized care using a hub-and-spoke model decreases morbidity, mortality, and increased rates of explant from VADs.

A 2010-2014 large Nationwide Readmissions database study highlighted that patients admitted or transferred directly to or transferred to a hub hospital had lower mortality than in spoke hospitals.¹⁶ This study did not specifically address the organizational processes at play and simply classified hospitals a posteriori; outcomes may be linked to better organizational processes rather than simple access to advanced care.^{17(p20),18} However, single regional care and more contemporary network data have shown similar cardiovascular outcomes between patients initially presenting to a spoke or a hub hospital, although patients in hub hospitals received more advanced mechanical support.¹⁹ A standardized regional care model thus appears to allow appropriate escalation of care in patients with adequate outcomes and resource utilization. This is in line with other data that suggest higher-volume centers frequently use advanced support and revascularization techniques that may be linked to better outcomes if used early in the disease process.¹⁰

INTERHOSPITAL PARTNERSHIPS. We recommend that every center without high-volume advanced mechanical support capabilities (the referring center) develop a partnership with a high-volume center with access to advanced mechanical support and exit strategies, including transplant and durable VAD (the reference center). We recognize that creating a regional hub-and-spoke model may only be feasible in some cases, that this model may evolve, and that the evidence is equivocal, and we thus do not recommend a specific model.

A partnership with a high-volume center requires specific conditions for success and significantly benefits the referring center (Table 1). These conditions for success include:

1. A common and shared definition of CS. Multiple CS classifications can be used with variable benefits and drawbacks, notably the Society for Cardiovascular Angiography and Interventions and the Interagency Registry for Mechanically Assisted Circulatory Support.^{20,21} We do not recommend a specific classification but recommend a shared classification across centers. This allows for clear and consistent communication between centers, improving performance over time.
2. A standardized communication protocol to communicate with the interdisciplinary team at the high-volume center. Multiple studies in cardiovascular care have shown that rapid initiation of appropriate therapy improves outcomes.²²⁻²⁴ Efficient communication

TABLE 2 Suggested Practices for Mechanical Ventilation and Metabolic Targets

Variable	Minimally Acceptable ^a	Better	Best
Ventilation	Lung protective ventilation Tidal volumes 4–10 mL/kg IBW PEEP 3–10 cm H ₂ O Plateau pressure <28 cm H ₂ O Driving pressure <16 cm H ₂ O	Spontaneous ventilation mode Pressure support ventilation Volume support ventilation Ensure minimal patient effort to prevent patient-induced lung injury	Negative pressure ventilation Extubated patient Tracheostomized patient
Metabolic targets	O ₂ sat >88% pH 7.25–7.5 Pco ₂ 30–60 mm Hg	O ₂ 92%–100% pH 7.35–7.45 Pco ₂ 35–45 mm Hg	O ₂ 92%–96% PaO ₂ <150 mm Hg pH 7.35–7.45 Pco ₂ 35–45 mm Hg
Sedation	Moderate to deep sedation RASS –4 to –5 Intermittent and/or opioid infusions Intermittent and/or nonbenzodiazepine sedative infusions No neuromuscular blockade	Light sedation RASS 0 to –2 Intermittent opioids Intermittent and/or low-dose infusions of nonbenzodiazepine sedatives	No sedation RASS 0 to –1 Intermittent opioids Intermittent nonbenzodiazepine sedatives

^aThese suggestions represent minimally acceptable targets for short durations only, assuming stable hemodynamics that allow the ability to progress to better or best categories. Inability to progress to the better category should warrant consideration of transfer to a more experienced center. IBW, ideal body weight; PEEP, positive end-expiratory pressure; RASS, Richmond Agitation-Sedation Scale.

necessitates transferring information to all stakeholders from a common source.¹⁷ We recommend a standardized protocol to rapidly initiate a transfer of information to all stakeholders simultaneously.

3. A standardized interdisciplinary team at the referring and reference hospital. The interdisciplinary team may be variable, depending on local processes and resources. It should ideally include a cardiothoracic surgeon, a cardiovascular anesthesiologist, a cardiovascular intensivist, a heart failure cardiologist, and a perfusionist.^{17,25,26} We recommend that the interdisciplinary team be systematically activated and consulted to assess every referred case, as it may improve outcomes.^{1,27}
4. A feedback process to review outcomes between the referring and reference hospital. This process needs to report back to the referring center to allow progressive improvement in the standardized process, including referral appropriateness, timing, and transfer process. We recommend implementing a regular multi-center review of cases, with frequency as dictated by local resources. This should form the basis of a quality review process initiative at both the referring and reference hospitals.

Question 3: What is the optimal ventilation management strategy?

Statement: Ventilation strategies should promote optimal gas exchange, minimize lung injury, and promote patient-ventilator synchrony.

Level of certainty: Moderate

Rationale: An awake and extubated patient is the optimal strategy during tMCS and is possible in more than one-third of patients (Table 2).²⁸ Spontaneous breathing without excessive effort is highly beneficial because it improves ventilation-perfusion ratio matching, increases RV preload and decreases RV afterload, and provides diaphragmatic protection.^{29,30} Low respiratory muscle effort can result in diaphragmatic weakness in up to 64% of patients by 24 hours of invasive mechanical ventilation (IMV)³¹⁻³³ and impair ventilator weaning. Extubated or spontaneously breathing IMV VA-ECMO is possible and desirable. A retrospective analysis of 231 CS patients on VA-ECMO demonstrated a decrease in ventilator-associated pneumonia, need for tracheostomy, renal replacement therapy, and risk of mortality at 60 days and 1 year in the 39% of “awake” patients (<50% of ECMO without mechanical ventilation).²⁸

No comparable evidence exists for spontaneous IMV during tMCS without an oxygenator in CS, but adjacent population data are informative. A meta-analysis of 2916 patients with non-IMV and CS demonstrated reduced in-hospital mortality and lowered intubation rates without adverse cardiac events.³⁴ Acute respiratory distress syndrome (ARDS) data have shown that increasing CO₂ elimination reduces respiratory drive, muscle effort, and transpulmonary pressures in spontaneously breathing patients with severe ARDS^{35,36} and makes awake state or spontaneous breathing possible.³⁷ Resolving lung

injury or the presence of an oxygenator should thus prompt attempts at spontaneous breathing if excessive effort can be controlled.^{28,38}

Spontaneous breathing may be impractical in the early period because hemodynamic instability and respiratory failure often accompany the insertion of tMCS and may preclude awake insertion or early extubation. Patients with tMCS and on IMV should be managed with lung protective ventilation (LPV) until extubation is possible.³⁹ Cardiovascular patients benefit from preload and afterload unloading from IMV. Still, they are more susceptible to adverse hemodynamic adverse effects of aggressive LPV, usually reserved for ARDS.⁴⁰ Contemporary evidence has shifted LPV to driving pressure- and respiratory system compliance-led ventilation strategies, with better associated outcomes in ARDS^{41,42} and uninjured lungs.⁴³ Evidence for LPV-positive end-expiratory pressure titration in CS and tMCS is derived from ARDS literature; it is reasonable to adopt similar principles with attention to preload vs afterload dependent states for hemodynamic impact.³⁹

Patients treated with tMCS without an oxygenator should be managed with LPV with similar targets as patients with acute cardiovascular conditions or after cardiac surgery. Tandem Heart (LivaNova) and left-sided microaxial-flow pumps are more susceptible to higher ventilatory pressures that affect RV function, reduce LV and RV preload, and reduce device efficiency and total cardiac output.⁴⁴ Similarly, RVADs and right-sided microaxial-flow pumps can be affected by decreased preload and increased afterload, and additional reductions in native RV output may negatively affect patients.³⁹ Evidence for specific variables adjustment is nonexistent, and alterations should be made based on a careful hemodynamic and echocardiographic evaluation. Outcome data are limited to small nonrandomized trials in early phases of CS showing no definite survival advantage but some hemodynamic benefits in the IMV + IABP compared with the IABP-only group.^{45,46}

LPV is more easily achieved in patients supported with tMCS with an oxygenator. Central VA-ECMO and RVAD with an oxygenator allow low volume and low fraction of inspired oxygen LPV because there are often complete oxygenation and CO₂ clearance capabilities. Peripheral VA-ECMO requires lower native lung minute ventilation due to the lower amount of native lung blood transit but remains at risk of differential oxygenation.⁴⁷

A retrospective Extracorporeal Life Support Organization registry study of 2226 CS non-extracorporeal

cardiopulmonary resuscitation (ECPR) VA-ECMO patients found that survival to discharge was higher in the low intrapulmonary pressure ventilation group, with peak inspiratory pressure <30 cm H₂O (adjusted odds ratio, 1.69; 95% CI, 1.21-2.37; *P* = .0021).⁴⁸ This aligns with venovenous (VV)-ECMO literature, in which additional purported benefits relevant to cardiac patients include decreased systemic inflammatory response, reduced organ dysfunction, and beneficial cardiovascular effects of higher positive end-expiratory pressure while maintaining protective ventilatory settings.^{35,36}

Question 4: What are the recommended gas exchange targets to preserve end-organ function?

Statement: Acid-base balance should be normalized to promote end-organ functional recovery and reverse cellular anoxia.

Level of certainty: Moderate

Rationale: Patients on tMCS without an oxygenator should be managed using current best practices for critically ill patients; in patients with an oxygenator, oxygenation and acid-base status should be normalized to optimize cardiac and respiratory function (Table 2). Current evidence demonstrates a clear association between acidemia and mortality in CS; efforts to normalize acid-base status should be performed as limited by the patient's cardiopulmonary status.⁴⁹ Outcome data for VA-ECMO has shown an association between Pco₂ <30 mm Hg and >60 mm Hg with in-hospital mortality.⁵⁰ In the same population, large reductions (>20 mm Hg) in Paco₂ over 24 hours were associated with composite major intracranial events, regardless of the initial Paco₂.⁵⁰ Retrospective data in non-ECPR VA-ECMO populations has demonstrated an association of high levels of oxygen (PaO₂ >150) with mortality³⁷⁻³⁹ and with time-dependent exposure with neurologic outcomes.³⁹ The randomized controlled trial outcome data for IMV for ICU patients suggests hyperoxia is harmful.⁵¹ This is consistent with data on severe septic shock, acute myocardial infarction, out-of-hospital cardiac arrest, and ECPR.⁵²⁻⁵⁶ Although expert consensus would agree that hypoxia should be avoided in CS, data showing worse outcomes in CS and negative data in VA-ECMO are lacking.⁵⁷ Given these findings, we suggest targeting an oxygen saturation >90% rather than a Po₂ target and avoiding hyperoxia (>150 mm Hg).⁵⁸ We suggest targeting a normal pH first and a normal Pco₂ second and avoiding rapid changes in Pco₂.

Question 5: What is the recommended timing to start or resume anticoagulation?

Statement: Anticoagulation must be initiated as soon as the benefits (ie, avoiding thrombus formation) outweigh the systemic risks (ie, hemorrhage).

Level of certainty: High

Rationale: The interaction of blood with non-endothelial surfaces promotes activation of the coagulation cascade. As such, patients and devices are at risk of catastrophic hemorrhagic and thrombotic complications.

Ideally, avoiding anticoagulation for tMCS would mitigate hemorrhagic concerns and other agent-specific complications. A single-center retrospective observational study of VA-ECMO patients not receiving anticoagulation reported significantly lower overall complication rates, fewer hemorrhagic complications, fewer packed red blood cell and platelet transfusion requirements, and lower heparin-induced thrombocytopenia incidence.⁵⁹ They reported no statistically significant increase in thrombosis or mortality.⁵⁹ However, the absolute mortality risk was 10% higher in patients not anticoagulated.⁵⁹ Additionally, there was likely substantial selection bias, because only 35% of the VA-ECMO cases during the review period did not receive anticoagulation.⁵⁹ Few centers have adopted routine anticoagulant-free VA-ECMO due to thrombosis risk. This risk is likely lower in other tMCS systems due to the absence of a membrane oxygenator.

The ideal approach for ECMO anticoagulation is unknown, because coagulation profile differences exist in VA-ECMO and VV-ECMO concerning thrombin generation and total heparin dose.⁶⁰ After evaluating major bleeding, thromboembolic events, and mortality, a systematic review and meta-analysis could not determine the optimal VA-ECMO anticoagulation strategy due to study quality and heterogeneity.⁶¹ Another systematic review reported comparable circuit and patient thrombosis complications for patients receiving continuous anticoagulation and anticoagulation-free ECMO, with the caveats of retrospective data and inconsistent outcome reporting.⁶²

Microaxial-flow pumps have the unique need among tMCS devices to require a constant infusion of a heparinized purge solution to prevent blood entry in the motor housing. Systemic anticoagulation should be added to prevent catheter surface thrombosis if anticoagulation is subtherapeutic despite the purge solution. Recent data have shown that the effect of the purge solution relies more on the negative anion charge of

heparin rather than its anticoagulation properties.⁶³ As such, a dextrose and bicarbonate infusion was shown to be safe as an alternative when the risk of systemic anticoagulation is deemed excessive in a single-center study of 43 patients.⁶³ Extensive data supports the safety of withholding anticoagulation for IABPs while in 1:1 ratio, and for short durations of 1:2 and 1:3 ratios.⁶⁴

Accordingly, we advise initiating anticoagulation as soon as the benefit of avoiding thrombotic complications outweighs the systemic risks of hemorrhage in devices that require it (Table 3). An interdisciplinary team approach is recommended for optimal patient care.

Question 6: What anticoagulation agent and monitoring approach should be used routinely?

Statement: The first-line agent for anticoagulation should be unfractionated heparin.

Level of certainty: Moderate

Statement: The therapeutic efficacy of systemic anticoagulation should be monitored with activated partial thromboplastin time (aPTT) or anti-Xa levels according to center capabilities.

Level of certainty: Moderate

Rationale:

ANTICOAGULATION AGENTS. The low cost, very rapid onset of anticoagulation, presence of an antidote for reversibility, and familiarity render heparin the most commonly used anticoagulant for tMCS, and heparin is endorsed by the Extracorporeal Life Support Organization to support ECMO.⁶⁵ Heparin is associated with notable adverse effects, however. Even subtherapeutic administration can result in hemorrhage. Additional considerations include the development of heparin resistance, which necessitates higher heparin dosing and possible antithrombin replenishment, and heparin-induced thrombocytopenia and thrombosis.⁶⁶

The preponderance of literature investigating alternatives to heparin for tMCS anticoagulation specifically focuses on ECMO anticoagulation. Extrapolation to other tMCS devices is potentially fraught with assumptions, because the presence of an oxygenator may create increasing thrombotic risk. Direct thrombin inhibitors, specifically bivalirudin and argatroban, are the main alternative anticoagulation agents studied in ECMO and are most regularly used when heparin-induced thrombocytopenia is suspected. Their advantages include more stable anticoagulation with less dose adjustment and no antithrombin interactions, obviating any need for replenishment. Although direct thrombin

TABLE 3 Suggested Anticoagulation Monitoring and Targets			
Variable	Acceptable	Better	Best
Monitoring	Plasma-based monitoring (aPTT, anti-Xa)	Plasma-based monitoring (aPTT, anti-Xa) Lab-based viscoelastic assays	Plasma-based monitoring (aPTT, anti-Xa). Point-of-care viscoelastic assays
	Microaxial-Flow Pumps		ECMO/tRVAD/tLVAD
Targets	aPTT: 55 and 80 seconds anti-Xa: 0.3-0.5 Target lower range (0.2-0.3) + bicarbonate purge if bleeding concerns.		aPTT: 60-80 seconds anti-Xa: 0.3-0.7 Target lower range (0.2-0.3) if bleeding concerns.
Timing			
No bleeding risk	Device + systemic anticoagulation upon device insertion.		Systemic anticoagulation upon device insertion
Bleeding risk^a	Device only anticoagulation (full or half purge). Delay systemic anticoagulation for short duration (<48 hours). Consider bicarbonate purge only if higher risk.		Target lower range anticoagulation. Consider no anticoagulation for short duration (<12 hours) if higher risk. Maintain higher flows (>2.5 LPM).
Active bleeding	Bicarbonate purge only. No systemic anticoagulation.		Stop anticoagulation for entire duration of active bleeding. Maintain higher flows (>2.5 LPM).
^a Bleeding risk should be determined through an interdisciplinary approach of all involved stakeholders. aPTT, activated partial thromboplastin time; ECMO, extracorporeal membrane oxygenation; LPM, liters per minute; tLVAD, temporary left ventricular assist device; tRVAD, temporary right ventricular assist device.			

inhibitors have shorter half-lives (argatroban, 39-51 minutes; bivalirudin, 25 minutes) compared with heparin (60-90 minutes), there is no established antidote for rapid direct thrombin inhibitor reversal, which has caused concerns preventing widespread adoption.⁶⁷

Argatroban has been suggested as a safe anticoagulant for adult ECMO patients, but the limited available data preclude definitive conclusions.^{67,68} Bivalirudin, which has more robust data, was studied in a systematic review of 16 retrospective studies, including 558 adults and 116 pediatric patients.⁶⁹ The preponderance of studies either favored bivalirudin or showed no difference between bivalirudin and heparin for thrombosis risk.⁶⁹ A separate meta-analysis of 9 retrospective studies evaluating bivalirudin for alternative anticoagulation showed decreased mortality (odds ratio, 0.65; 95% CI, 0.44-0.95; $P = .03$) and thrombosis events (odds ratio, 0.55; 95% CI, 0.37-0.83; $P = .004$) in the bivalirudin group compared with heparin⁷⁰; major bleeding, ECMO duration or circuit intervention events were not statistically different between groups.⁷⁰ Bivalirudin was purported to be safe for postcardiotomy ECMO, with fewer bleeding complications and lower blood product transfusion requirements.⁷¹ In circumstances of blood stasis, bivalirudin should be used with wariness, because localized proteolysis can promote intracardiac thrombus and circuit access ports thrombosis.⁷² Bivalirudin with a bicarbonate purge has been explored in a few Impella 5.0 and 5.5 (Abiomed)

studies with similar results to heparin-based therapies.^{73,74}

ANTICOAGULATION MONITORING. Activated clotting time is the most widely used monitoring study available as point-of-care testing. It is influenced by multiple technical and clinical factors and is not standardized between devices. Activated clotting time is unreliable for monitoring heparin anticoagulation in adult ECMO patients and is not recommended.^{75,76}

The aPTT is ubiquitous for unfractionated heparin monitoring and can also be used for direct thrombin inhibitor monitoring. The typical therapeutic aPTT target is 1.5 to 2 times above baseline measurement, based on a single study and without prospective randomized controlled trial data in ECMO patients (Table 3).⁶⁵ aPTT alone is inadequate for assessing heparin anticoagulation or accurately measuring the heparin effect in diagnosing heparin resistance.⁶⁶

Centers with anti-Xa assay access may prefer this test because it specifically reflects the unfractionated heparin effect. Most programs use 0.3 to 0.7 IU/mL for the therapeutic target range, although numerous centers successfully use a lower therapeutic range to mitigate hemorrhagic complications. Because heparin binds anti-thrombin, catalyzing factor Xa inhibition, the anti-Xa assay is the most specific measure of the heparin effect. Although anti-Xa directly measures the heparin effect, it does not reflect the overall hemostasis.⁶⁶ This test is only available in some centers and needs to be standardized between

TABLE 4 Suggested Team Model for Awake Patient and Device Management and Mobilization

Team Member	Minimally Acceptable ^a	Better	Best
Physician	Physician coordinating management of patient's care, including sedation, ventilation, and patient-device interactions.	Physician coordinating management of patient's care, including sedation, ventilation and patient-device interactions.	Physician coordinating management of patient's care, including sedation, ventilation, and patient-device interactions.
Bedside staff	One staff member to monitor for systemic issues with the patient, including vital signs, neurologic status (including presence of pain agitation/anxiety, etc), ventilator dyssynchrony (if applicable), labs etc. One to manage patient interactions with the ECMO circuit, including potential adverse events (ie, bleeding), and monitor of inadvertent manipulation of device.	One staff member to monitor for systemic issues with the patient, including vital signs, neurologic status (including presence of pain agitation/anxiety, etc), ventilator dyssynchrony (if applicable), labs etc. One to manage patient interactions with the ECMO circuit, including potential adverse events (ie, bleeding), and monitor of inadvertent manipulation of device.	One staff member to monitor for systemic issues with the patient, including vital signs, neurologic status (including presence of pain agitation/anxiety, etc), ventilator dyssynchrony (if applicable), labs etc. One to manage patient interactions with the ECMO circuit, including potential adverse events (ie, bleeding) and monitor of inadvertent manipulation of device.
ECMO specialist		Medical professional adept at managing patient-device interactions.	Medical professional adept at managing patient-device interactions.
Mobility team			Physical therapists/occupational therapists specialized in mobilizing patients with mechanical devices. Mobility team operates on a set protocol clearly delineating steps for mobilization and how to proceed if complications arise during mobilization.

^aThese suggestions represent minimally acceptable targets for short durations only, assuming stable hemodynamics that allow the ability to progress to better or best categories. Inability to progress to the better category should warrant consideration of transfer to a more experienced center. ECMO, extracorporeal membrane oxygenation.

assays. Finally, anti-Xa is unable to monitor direct thrombin inhibitors.

Clot integrity and fibrinolysis are not reflected in aPTT and anti-Xa assays. On the other hand, viscoelastic hemostatic assays, such as thromboelastography and rotational thromboelastography, are whole-blood point-of-care coagulation assays capable of assessing clotting time, clot strength and amplitude, and clot stability (fibrinolysis). Viscoelastic assays are recommended to reduce bleeding and blood product transfusion requirements after cardiac surgery.⁷⁷⁻⁷⁹ Although new studies are emerging, currently, viscoelastic data are insufficient to predict hemorrhage and thrombosis reliably in the context of tMCS. Nonetheless, it may provide additional valuable information to guide therapy in combination with anti-Xa.^{80,81}

Question 7: What is the optimal strategy for patient comfort and device interactions?

Statement: Sedative agents should be minimized as tolerated, with a focus on symptom control and improving awake comfort.

Level of certainty: Moderate

Rationale: The ideal level of sedation for a patient supported with tMCS allows an awake and

comfortable state. Targeting a light degree of sedation has been a longstanding recommendation in critically ill patients by expert societies (Table 4).^{82,83} Optimal sedation strategies that allow for awake interaction with a patient's environment are linked with reduced time on mechanical ventilation, rates of delirium, and ICU length of stay.^{82,84} The concept of awake comfort in the critically ill is fundamental for patients supported with tMCS, because it assists with:

1. Neurologic assessment: Hemorrhagic cerebral vascular accidents are a known complication for patients supported with tMCS. Emerging data suggest an abundant subclinical microembolic burden in patients supported with tMCS (VA-ECMO, IABP, microaxial-flow pump devices), which may play a role in the neurologic injury.⁸⁵ Awake comfort allows continuous neurologic assessment to identify major macrovascular events and potentially treat them rapidly.
2. Liberation from ventilation: Patients with lung injury supported by tMCS with an oxygenator may be liberated from IMV if

sedation is weaned, reducing adverse events associated with prolonged ventilation.⁸⁶

3. Hemodynamic optimization: Lighter sedation may limit vasoplegia related to sedatives. Reducing hypotensive episodes may reduce fluid administration and vasopressor initiation, both of which are linked to poor outcomes in CS and tMCS.⁸⁷
4. Patient involvement: Patient participation in discussions regarding goals of care should be prioritized and can be facilitated by light sedation.⁸⁴

Achieving awake comfort with adequate symptom control in patients supported with tMCS is feasible, although avoiding deleterious patient-device interactions requires special considerations. “Awake ECMO” has been well-documented in patients receiving VV-ECMO, including patients with cardiac compromise.⁸⁸⁻⁹⁰ Although data for tMCS devices for CS are more limited, VV-ECMO literature that includes bifemoral cannulation may have some crossover to this population. Smaller case reports and series have shown successful awake VA-ECMO for a short (<2 weeks) duration.^{84,91,92} Other tMCS strategies are usually for single-organ concerns, and more stable cannulation strategies may make awake strategies feasible. Contemporary studies of microaxial-flow pumps and IABPs have moved past the concept of awake comfort and are now focused on mobilization and ambulation strategies, with obvious feasibility implications.⁹³⁻⁹⁵ tMCS strategies that lend themselves well to awake comfort include central cannulation strategies (central biventricular assist devices) and strategies that avoid femoral access (such as axillary VA-ECMO, microaxial-flow pumps, and IABPs).^{93,96-98} Femoral or bifemoral strategies remain compatible with awake comfort goals.^{88,99}

Strategies for achieving awake comfort with tMCS devices should include titration of sedatives based on a validated sedation/anxiety/agitation scale, such as the Richmond Agitation-Sedation Scale and the Critical-Care Pain Observation Scale.^{100,101} These 2 scales should be used synergistically, emphasizing treating pain, anxiety, and agitation while promoting wakeful interaction with the patient’s surroundings and optimizing patient-device interactions. Achieving the ideal awake state is dictated by local resources, team logistics, and the providers’ comfort level. An experienced team and appropriate bedside monitoring are essential to safely support awake patients on tMCS and avoiding inadvertent line or device manipulation.

Existing literature suggests that the extracorporeal circuit changes the pharmacodynamics and pharmacokinetics of highly lipophilic medications, which require special consideration.¹⁰² Sedative options should preferentially use short-acting agents that can be titrated quickly for symptom control and awake patient comfort. No data support a specific combination or dose-range for tMCS. Suggested options include:

1. Opioids: Limited data suggest that hydromorphone-based sedation in ECMO patients (compared with fentanyl) may result in less delirium, better symptom control, and less overall opioid and adjunct sedative use, especially if required for many days.^{103,104}
2. Sedatives: Propofol, dexmedetomidine, and ketamine are safe for patients requiring tMCS and will not negatively impact device function.¹⁰⁵⁻¹⁰⁷
3. Antipsychotics: Antipsychotics are safe in patients with tMCS support. Although data regarding their effectiveness for symptomatic control in this population are limited, they may aid in reducing sedative infusion use to achieve symptomatic control.^{108,109}
4. Neuromuscular blockade: Neuromuscular blockade should be used only in patients with severe cardiopulmonary interactions causing hemodynamic instability or worsening lung injury.

Question 8: Can a patient on tMCS be mobilized?

Statement: tMCS-supported patients may be mobilized with a protocolized approach involving the interdisciplinary team.

Level of certainty: Moderate

Rationale: Mobilization in the critically ill refers to physical activity performed to an intensity that can bring about physiological changes/prevent negative impacts of immobilization. There are two overarching levels of mobilization in the critically ill tMCS patient:

1. In-bed mobilization: This represents frequent position changes in bed and passive range of motion techniques. Frequent position changes in the bed are generally safe in patients with tMCS and are necessary to avoid pressure-induced tissue injuries.¹¹⁰⁻¹¹² Passive range of motion tactics used by bedside staff or physical therapists are also safe in patients with tMCS devices.^{112,113} These techniques have been linked to reduced muscle wasting in the critically ill.^{114,115} In-bed mobilization

should be limited to patients who are unable to participate in or who have a contraindication to advanced mobilization.

2. **Advanced mobilization:** This represents tactics to liberate a patient from a constant bed-bound state. This level of mobilization is associated with improved long-term outcomes in patients, most notably in functional independence scores.^{116–118} Two potential guides for characterizing mobility in this setting include the ICU Mobility Scale and the Johns Hopkins Highest Level of Mobility Scale.^{119,120} Although they have yet to be validated fully in patients with tMCS, their relevance as an overall guide cannot be understated.

Mobilization at both levels is feasible for appropriate patients. In-bed mobilization is easier to achieve and should be attempted for all patients with tMCS devices (Table 4). Special attention must be paid to the MCS equipment during in-bed mobilization to avoid harm to the patient. Advanced mobilization can also be attempted in patients with tMCS, with the degree of mobilization dependent on institutional capabilities, type of MCS device and its configuration/interaction with the patient, and provider/patient comfort. Other practice guidelines support advanced mobility of tMCS patients.¹²¹ Specific data for each tMCS strategy is as follows:

1. **IABP:** The current evidence supports advanced mobility with an IABP in place in the appropriate clinical scenario. Most published work supports advanced mobility in the setting of IABP placed in the axillary position.^{97,98,122} Some studies even suggest that high levels of activity (per the Johns Hopkins Highest Level of Mobility Scale) are possible with the IABP in an axillary position and appropriate support staff, although there is a higher risk of malpositioning.⁹⁸ Advanced mobility is linked to successful bridging to advanced therapies and improved survival rates.^{97,122} An IABP through femoral access may also be compatible with advanced mobility, although it requires a highly experienced and staffed medical team with protocolized mobilization checklists.
2. **Microaxial-flow pump:** Robust evidence supports advanced mobilization with these devices in the axillary position. Multiple studies have shown successful and safe advanced mobilization, with some institutions achieving a 90% ambulation rate.^{94,95,123} Early mobilization has been linked to better

recovery after bridge to durable LVAD or orthotopic heart transplant,^{94,95,123} improved recovery, and home discharge rates.^{93,123}

3. **VA-ECMO:** The data to support the advanced mobilization of these patients is limited. In a systematic review of 109 patients, 58.5% ambulated successfully. The data showed that centers that performed these tasks successfully were the most experienced ECMO centers.⁸⁸

The level of mobility achieved by a patient requiring tMCS depends on local resources, logistics, and comfort level. Increasing degrees of advanced mobility are achievable in patients with IABP and microaxial-flow pump devices. Advanced mobility in patients with tMCS devices with a lower degree of supportive evidence (ie, VA-ECMO) is best reserved for centers with experience and an appropriate staffing model, which includes a mobility team of specialists able to coordinate this complex care. It may be prudent to consider transfer to one of these centers if a prolonged time requiring such support is expected.

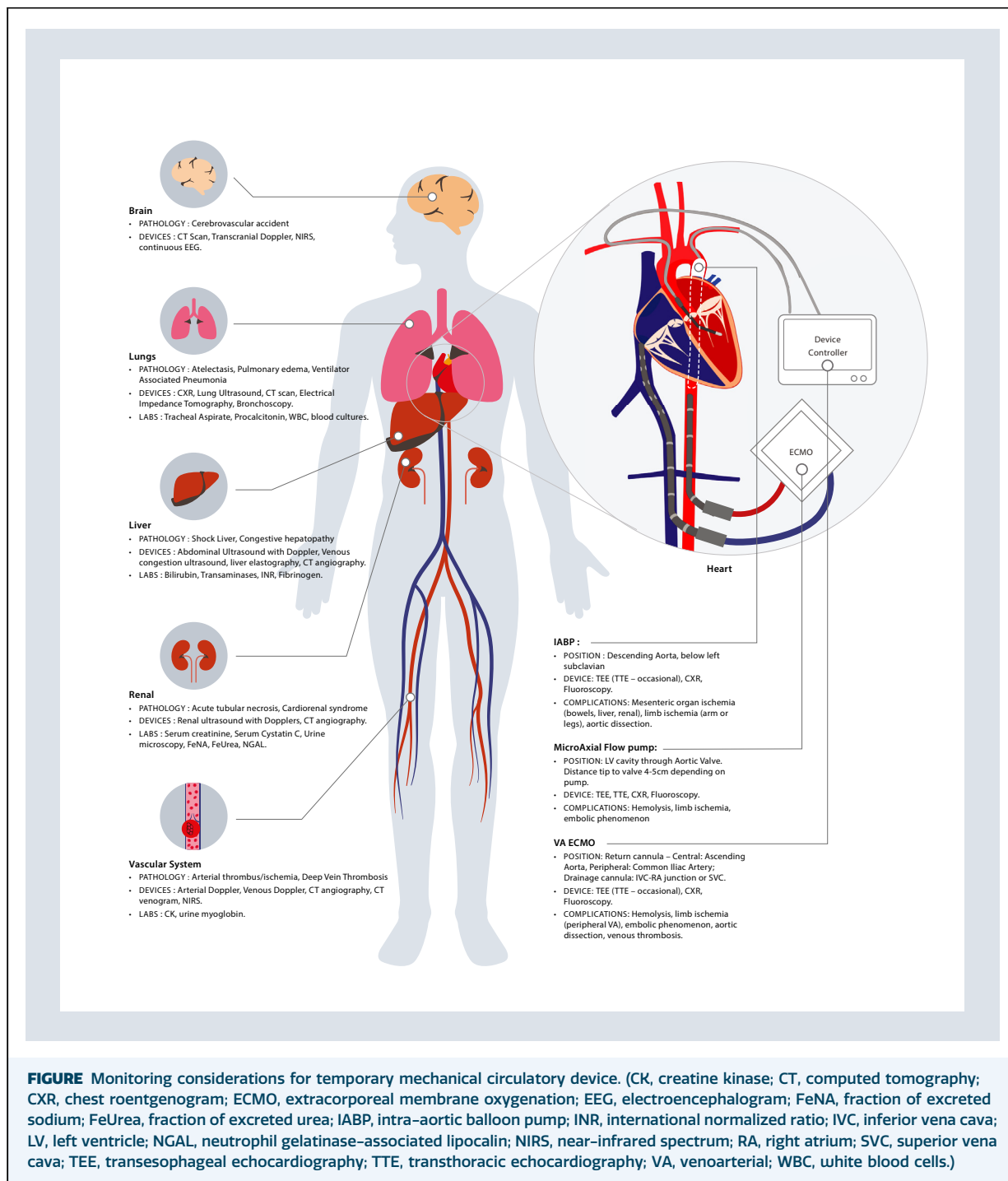
Question 9: What routine monitoring needs to be performed?

Statement: Monitoring and clinical reassessment is required to optimize patient-device interactions.

Level of certainty: High

Rationale: Temporary mechanical support is associated with significant morbidity (Figure). Monitoring strategies must be exhaustive, standardized, and targeted to identify early complications and guide proper management. We strongly advocate using standardized checklists and protocols to improve the quality of care provided.¹²⁴ Using checklists improved team performance during simulated ECMO emergencies in one randomized controlled trial and has wide overarching evidence in other associated fields.¹²⁵ Assessment checklists for tMCS devices must include patient-specific assessments (per standard ICU care), routine tMCS-specific laboratory testing, and routine circuit or tMCS device checks. Protocols should include emergency or critical event management protocols, such as pump failure, circuit thrombosis, air embolism, and cannula displacement.¹²⁶

It is advised that all mechanical support devices and their components be assiduously secured and monitored frequently for hemorrhage, infection, hypoperfusion/ischemia, hemolysis, and hyperperfusion. Institutions should devise specific protocols for device placement and positioning within the patient's setting, including ICU,



operating room, and transport situations. These protocols are essential to ensure maximum patient safety by avoiding the risk of accidental device disruptions that could lead to sudden interruption of proper tMCS support functions.

Statement: Monitoring protocols need to be device-specific.

Level of certainty: High

Rationale:

MONITORING CONSIDERATIONS FOR VA-ECMO DIFFERENTIAL OXYGENATION. Differential oxygenation, commonly referred to as North-South syndrome or Harlequin syndrome, can occur in the setting of peripheral cannulation for VA-ECMO.¹²⁷ Persistent respiratory failure in the setting of cardiac recovery leads to deoxygenated blood from the native circulation perfusing more proximal branches of the aorta and with oxygenated blood from the arterial cannula

oxygenating distal branches. Arterial saturations must be monitored from the right radial or brachial artery because they are the most proximal aortic branch that can be monitored.¹²⁴ Continuous cerebral oximetry is advised, if locally available, because a difference between the hemispheres or a progressive trend to lower bilateral cerebral oximetry may allow early detection.^{124,128-130}

LV DISTENSION. Thebesian and bronchial veins drain into the LV, which may cause progressive LV distension with elevated LV end-diastolic pressure and pulmonary edema, decreasing coronary perfusion with subendocardial edema, and LV thrombus with insufficient LV ejection.^{131,132} Patients with aortic valve insufficiency, inadequate systemic venous drainage, arrhythmias, and systemic arterial hypertension are at risk of developing LV distention.¹³¹ It will commonly present as refractory ventricular arrhythmias, pulmonary edema, pulmonary hemorrhage, refractory hypoxemia, or simply an elevated pulmonary capillary wedge pressure (PCWP). Insufficient LV ejection or LV unloading may be evident in the absence of aortic valve opening or insidious if there is evidence of aortic valve opening with higher LV end-diastolic pressure. The data supporting invasive and noninvasive indications for LV unloading are mainly based on expert opinion or extrapolated from ancillary data.^{133,134}

Authors have suggested that an arterial pulse pressure ranging from <8 to 15 mm Hg depending on the study should prompt evaluation for inadequate LV unloading.¹³⁵⁻¹⁴⁰ In a study of 106 patients on VA-ECMO a pulse pressure <15 mm Hg predicted a native cardiac output of <1 L/min with reasonable accuracy.¹³⁶ A study of 98 patients found a lower pulsatility index (pulse pressure/mean arterial pressure) was associated with spontaneous echo contrast and a higher risk of intracardiac thrombus.¹⁴⁰ In the same study by Mourad and colleagues,¹³⁶ end-tidal CO₂ <14 mm Hg had a stronger predictive value than pulse pressure. Combining both indicators may improve accuracy in predicting inadequate unloading. PCWP >15 to 18 mm Hg or a pulmonary artery diastolic pressure ≥25 mm Hg may be indicative of inadequate LV unloading, assuming there is RV pulsatility.¹³⁴ A study of 12 patients with peripheral VA-ECMO and an IABP showed patients with a PCWP >15 mm Hg had the most reduction in PCWP and echocardiographic markers of inadequate unloading upon onset of IABP.¹⁴¹ In a study of 121 patients with peripheral VA-ECMO, a combination of pulmonary

artery diastolic pressure ≥25 mm Hg and pulmonary edema on chest roentgenogram was used as a definition of subclinical LV distension¹³⁴; the difference between PCWP and pulmonary artery diastolic pressure between these studies may be related to the presence of a transpulmonary gradient.¹³⁴ There was no significant difference in survival between groups, but the group with LV distension criteria had higher rates of need for device transition and lower myocardial recovery. Overall, pulmonary artery catheter measurements should be used as a screening tool to prompt further investigations, because the correlation between PCWP and LV end-diastolic pressure may not always be accurate.^{134,137}

Echocardiography should be used to assess for LV markers indicative of poor LV unloading. Moderate to severe aortic or mitral insufficiency indicates the need for an unloading strategy, because it will inevitably lead to inefficient LV unloading, poor cardiac recovery, and pulmonary edema.¹³¹ The absence of intermittent aortic valve opening indicates blood stasis within the LV cavity and a high risk of thrombosis, regardless of anticoagulation status. This is generally associated with a low pulse pressure, pulsatility index, and spontaneous echo contrast, which are associated with a higher risk of intracardiac thrombus and stroke.¹⁴⁰ An LV outflow tract velocity-time integral <10 cm and LV ejection fraction < 0.20 to 0.25 are frequently used as a marker of inadequate unloading; this is mostly extrapolated from some limited ECMO weaning protocols and load studies.^{142,143} Increased LV end-diastolic diameter and LV end-systolic diameter have been used as markers of inadequate unloading; no study has shown a clinically meaningful threshold to determine adequate decompression.¹⁴¹ Echocardiography should also eliminate confounding complications that may cause poor LV ejection, such as tamponade physiology and RV failure. Pericardial effusions may limit cardiac pulsatility without causing low flow on VA-ECMO. In some cases, severe RV failure may lead to limited LV filling and ejection due to a lack of significant transpulmonary flow.

OXYGENATOR INTEGRITY. The circuit oxygenator should be inspected visually daily for thrombus, which may be an indicator of potential oxygenator failure. The correlation between visually apparent thrombus burden and oxygenator function is of generally limited value, but a high thrombotic burden may indicate a need for more frequent membrane assessment. Measurement of

postoxygenator blood gases should be performed at least daily to assess for membrane function. A postmembrane Po_2 of >250 mm Hg indicates a functioning oxygenator.¹⁴⁴ An increase or a transmembrane pressure of >30 mm Hg suggests worsening oxygenator function and may be superior to other measurements.¹⁴⁴ Daily laboratory values that need to be assessed for oxygenator and circuit integrity include lactate dehydrogenase, haptoglobin, fibrinogen, and platelets.^{144,145} Multiple suggestive algorithms have been published to detail strategies to distinguish systemic fibrinolysis or inflammatory situations from oxygenator or circuit-associated complications.¹⁴⁵

LIMB HYPERPERFUSION. Limb hyperperfusion is a complication specific to peripheral VA-ECMO. Limb hyperperfusion can occur in peripheral cannulation, typically in smaller arteries or those cannulated through a chimney graft anastomosed to the artery.¹⁴⁶ Cannula placement or technical problems with anastomosis of a chimney graft to the cannulated artery may result in preferential flow to the ipsilateral limb. Venous drainage impediment from a hematoma compressing the vein or from a deep vein thrombosis may lead to venous congestion. This complication requires prompt recognition and management because it can lead to compartment syndrome and potentially limb loss.

MONITORING CONSIDERATIONS FOR IABP POSITIONING. IABP position should be checked daily and after any major patient movement. Position can be assessed simply with chest radiography to ensure location of radiopaque marker is identified to be in the correct location. Transesophageal echocardiography can also confirm positioning. Patients with short stature are at risk of mesenteric or renal artery occlusion from an IABP. Although the selection of IABP dimensions should have accounted for this at insertion, specific attention to this potential complication is needed in unexplained hyperlactatemia and acute kidney injury despite good organ perfusion.

MONITORING CONSIDERATIONS FOR MICROAXIAL-FLOW PUMPS AND MICROAXIAL-FLOW DEVICES POSITIONING. The position of the microaxial-flow device should be checked after any major movement of the patient or when clinically indicated. Proper documentation of the device's external measurement markers should be verified at every assessment as determined by institutional protocols. We recommend minimum verification at every shift change of the bedside personnel. Indications to check positioning include changes in the placement signal on the device console or device

alarm indicating malposition. Malposition may cause dysrhythmias or trigger a low-flow event.¹⁴⁷ The position is assessed with transthoracic or transesophageal echocardiography. The Impella 5.5 is ideally positioned with the inlet tip ~ 5 cm from the aortic valve.¹⁴⁸ The Impella CP is ideally positioned with the inlet tip ~ 4 cm from the aortic valve.¹⁴⁸ The inflow should be in the mid-LV cavity and free from interacting with the mitral valve or subvalvular apparatus. In the event of a suction event within the LV, the device power level should be turned down, and urgent echocardiography should be undertaken to determine the position to guide any necessary adjustments.

CONCLUSION. Management practices for temporary mechanical support should be tailored to the available material and personnel resources to optimize care within the system's limits. Expertise is required for advanced and optimal management strategies, and patients may need to be transferred to a high-volume center.

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REFERENCES

1. Taleb I, Koliopoulou AG, Tandar A, et al. Shock team approach in refractory cardiogenic shock requiring short-term mechanical circulatory support: a proof of concept. *Circulation*. 2019;140:98–100. <https://doi.org/10.1161/CIRCULATIONAHA.119.040654>
2. Moghaddam N, van Diepen S, So D, Lawler PR, Fordyce CB. Cardiogenic shock teams and centres: a contemporary review of multidisciplinary care for cardiogenic shock. *ESC Heart Fail*. 2021;8:988–998. <https://doi.org/10.1002/ehf2.13180>
3. Møller Jacob E, Engstrøm Thomas, Jensen Lisette O, et al. Microaxial flow pump or standard care in infarct-related cardiogenic shock. *N Engl J Med*. 2024;390:1382–1393. <https://doi.org/10.1056/NEJMoa2312572>
4. Miller TE, Shaw AD, Mythen MG, Gan TJ, Perioperative Quality Initiative (POQI) I Workgroup. Evidence-based perioperative medicine comes of age: the Perioperative Quality Initiative (POQI): The 1st Consensus Conference of the Perioperative Quality Initiative (POQI). *Perioper Med (Lond)*. 2016;5:26. <https://doi.org/10.1186/s13741-016-0055-y>
5. van Diepen S, Katz JN, Albert NM, et al. Contemporary management of cardiogenic shock: a scientific statement from the American Heart Association. *Circulation*. 2017;136:e232–e268. <https://doi.org/10.1161/CIR.0000000000000525>
6. Grouls A, Dobbie K, Neuendorf K, et al. Top ten tips palliative care clinicians should know about working with adults receiving extracorporeal membrane oxygenation. *J Palliat Med*. 2023;26:1270–1276. <https://doi.org/10.1089/jpm.2023.0017>
7. Peeler A, Davidson PM, Gleason KT, et al. Palliative care utilization in patients requiring extracorporeal membrane oxygenation: an observational study. *ASAIO J*. 2023;69:1009–1015. <https://doi.org/10.1097/MAT.0000000000002021>
8. Mihalj M, Carrel T, Urman RD, Stueber F, Luedi MM. Recommendations for preoperative assessment and shared decision-making in cardiac surgery. *Curr Anesthesiol Rep*. 2020;10:185–195. <https://doi.org/10.1007/s40140-020-00377-7>
9. Gainer RA, Buth K, Begum J, Hirsch GM. A formalized shared decision-making process with individualized decision aids for older patients referred for cardiac surgery. *Can J Surg*. 2024;67:E7–E15. <https://doi.org/10.1503/cjs.004922>
10. Shaefi S, O'Gara B, Kociol RD, et al. Effect of cardiogenic shock hospital volume on mortality in patients with cardiogenic shock. *J Am Heart Assoc*. 2015;4:e001462. <https://doi.org/10.1161/JAHA.114.001462>
11. Kontos MC, Wang Y, Chaudhry SI, Vetrovec GW, Curtis J, Messenger J. Lower hospital volume is associated with higher in-hospital mortality in patients undergoing primary percutaneous coronary intervention for ST-segment-elevation myocardial infarction. *Circ Cardiovasc Qual Outcomes*. 2013;6:659–667. <https://doi.org/10.1161/CIRCOUTCOMES.113.000233>
12. Sasaki K, Koeda Y, Yoshizawa R, et al. Comparing in-hospital outcomes for acute myocardial infarction patients in high-volume hospitals performing primary percutaneous coronary intervention vs. regional general hospitals. *Circ J*. 2023;87:1347–1355. <https://doi.org/10.1253/circj.CJ-23-0188>
13. Ross JS, Normand SLT, Wang Y, et al. Hospital volume and 30-day mortality for three common medical conditions. *N Engl J Med*. 2010;362:1110–1118. <https://doi.org/10.1056/NEJMsa0907130>
14. Jaroszewski DE, Kleisli T, Staley L, et al. A traveling team concept to expedite the transfer and management of unstable patients in cardiopulmonary shock. *J Heart Lung Transplant*. 2011;30:618–623. <https://doi.org/10.1016/j.healun.2010.11.018>
15. Tehrani BN, Truesdell AG, Sherwood MW, et al. Standardized team-based care for cardiogenic shock. *J Am Coll Cardiol*. 2019;73:1659–1669. <https://doi.org/10.1016/j.jacc.2018.12.084>
16. Lu DY, Adelsheimer A, Chan K, et al. Impact of hospital transfer to hubs on outcomes of cardiogenic shock in the real world. *Eur J Heart Fail*. 2021;23:1927–1937. <https://doi.org/10.1002/ehf.2263>
17. Alviar CL, Hall S, Mebazaa A. Outcomes of patients with cardiogenic shock in hub and spoke centers: the importance of protocol standardization at a network level. *J Card Fail*. 2024;30:576–579. <https://doi.org/10.1016/j.cardfail.2024.01.007>
18. Warren AF, Rosner C, Gattani R, Truesdell AG, Proudfoot AG. Cardiogenic shock: protocols, teams, centers, and networks. *US Cardiol*. 2021;15:e18. <https://doi.org/10.15420/usc.2021.10>
19. Tehrani BN, Sherwood MW, Rosner C, et al. A standardized and regionalized network of care for cardiogenic shock. *JACC Heart Fail*. 2022;10:768–781. <https://doi.org/10.1016/j.jchf.2022.04.004>
20. Naidu SS, Baran DA, Jentzer JC, et al. SCAI SHOCK Stage Classification expert consensus update: a review and incorporation of validation studies: this statement was endorsed by the American College of Cardiology (ACC), American College of Emergency Physicians (ACEP), American Heart Association (AHA), European Society of Cardiology (ESC) Association for Acute Cardiovascular Care (ACVC), International Society for Heart and Lung Transplantation (ISHLT), Society of Critical Care Medicine (SCCM), and Society of Thoracic Surgeons (STS) in December 2021. *J Am Coll Cardiol*. 2022;79:933–946. <https://doi.org/10.1016/j.jacc.2022.01.018>
21. Stevenson LW, Pagani FD, Young JB, et al. INTERMACS profiles of advanced heart failure: the current picture. *J Heart Lung Transplant*. 2009;28:535–541. <https://doi.org/10.1016/j.healun.2009.02.015>
22. Henry TD, Unger BT, Sharkey SW, et al. Design of a standardized system for transfer of patients with ST-elevation myocardial infarction for percutaneous coronary intervention. *Am Heart J*. 2005;150:373–384. <https://doi.org/10.1016/j.ahj.2005.01.059>
23. Marcusohn E, Reiner Benaim A, Ronen S, Kerner A, Beyar R, Almog R. Door to balloon time in primary percutaneous coronary intervention in ST elevation myocardial infarction: every minute counts. *Coron Artery Dis*. 2022;33:341–348. <https://doi.org/10.1097/MCA.0000000000001145>
24. Esposito ML, Kapur NK. Acute mechanical circulatory support for cardiogenic shock: the “door to support” time. *F1000Res*. 2017;6:737. <https://doi.org/10.12688/f1000research.11150.1>
25. Senman B, Jentzer JC, Barnett CF, et al. Need for a cardiogenic shock team collaborative-promoting a team-based model of care to improve outcomes and identify best practices. *J Am Heart Assoc*. 2024;13:e031979. <https://doi.org/10.1161/JAHA.123.031979>
26. Brusca SB, Caughron H, Njoroge JN, Cheng R, O'Brien CG, Barnett CF. The shock team: a multidisciplinary approach to early patient phenotyping and appropriate care escalation in cardiogenic shock. *Curr Opin Cardiol*. 2022;37:241–249. <https://doi.org/10.1097/HCO.0000000000000967>
27. Héron FX, Beurton A, Oddos C, et al. Multidisciplinary cardiogenic shock team approach improves the long-term outcomes of patients suffering from refractory cardiogenic shock treated with short-term mechanical circulatory support. *Eur Heart J Acute Cardiovasc Care*. 2023;12:821–830. <https://doi.org/10.1093/ehjacc/zaad108>

28. Montero S, Huang F, Rivas-Lasarte M, et al. Awake venoarterial extracorporeal membrane oxygenation for refractory cardiogenic shock. *Eur Heart J Acute Cardiovasc Care*. 2021;10:585–594. <https://doi.org/10.1093/ehjacc/zuab018>
29. Yoshida T, Rinka H, Kaji A, et al. The impact of spontaneous ventilation on distribution of lung aeration in patients with acute respiratory distress syndrome: airway pressure release ventilation versus pressure support ventilation. *Anesth Analg*. 2009;109:1892–1900. <https://doi.org/10.1213/ANE.0b013e3181bbd918>
30. Goligher EC, Brochard LJ, Reid WD, et al. Diaphragmatic myotrauma: a mediator of prolonged ventilation and poor patient outcomes in acute respiratory failure. *Lancet Respir Med*. 2019;7:90–98. [https://doi.org/10.1016/S2213-2600\(18\)30366-7](https://doi.org/10.1016/S2213-2600(18)30366-7)
31. Levine S, Nguyen T, Taylor N, et al. Rapid disuse atrophy of diaphragm fibers in mechanically ventilated humans. *N Engl J Med*. 2008;358:1327–1335. <https://doi.org/10.1056/NEJMoa070447>
32. Jaber S, Petrof BJ, Jung B, et al. Rapidly progressive diaphragmatic weakness and injury during mechanical ventilation in humans. *Am J Respir Crit Care Med*. 2011;183:364–371. <https://doi.org/10.1164/rccm.201004-0670OC>
33. Demoule A, Jung B, Prodanovic H, et al. Diaphragm dysfunction on admission to the intensive care unit. Prevalence, risk factors, and prognostic impact—a prospective study. *Am J Respir Crit Care Med*. 2013;188:213–219. <https://doi.org/10.1164/rccm.201209-1668OC>
34. Vital FMR, Ladeira MT, Atallah AN. Non-invasive positive pressure ventilation (CPAP or bilevel NPPV) for cardiogenic pulmonary oedema. *Cochrane Database Syst Rev*. 2013;CD005351. <https://doi.org/10.1002/14651858.CD005351.pub3>
35. Mauri T, Grasselli G, Suriano G, et al. Control of respiratory drive and effort in extracorporeal membrane oxygenation patients recovering from severe acute respiratory distress syndrome. *Anesthesiology*. 2016;125:159–167. <https://doi.org/10.1097/ALN.0000000000001103>
36. Karagiannidis C, Lubnow M, Philipp A, et al. Autoregulation of ventilation with neurally adjusted ventilatory assist on extracorporeal lung support. *Intensive Care Med*. 2010;36:2038–2044. <https://doi.org/10.1007/s00134-010-1982-6>
37. Kim NE, Woo A, Kim SY, et al. Long- and short-term clinical impact of awake extracorporeal membrane oxygenation as bridging therapy for lung transplantation. *Respir Res*. 2021;22:306. <https://doi.org/10.1186/s12931-021-01905-7>
38. Haji JY, Mehra S, Doraiswamy P. Awake ECMO and mobilizing patients on ECMO. *Indian J Thorac Cardiovasc Surg*. 2021;37(suppl 2):309–318. <https://doi.org/10.1007/s12055-020-01075-z>
39. 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;36:300–312. <https://doi.org/10.1016/j.cjca.2019.11.038>
40. Brochard L, Slutsky A, Pesenti A. Mechanical ventilation to minimize progression of lung injury in acute respiratory failure. *Am J Respir Crit Care Med*. 2017;195:438–442. <https://doi.org/10.1164/rccm.201605-1081CP>
41. 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;22:313. <https://doi.org/10.1186/s12931-021-01912-8>
42. Fan E, Del Sorbo L, Goligher EC, et al. An official American Thoracic Society/European Society of Intensive Care Medicine/Society of Critical Care Medicine Clinical Practice Guideline: mechanical ventilation in adult patients with acute respiratory distress syndrome. *Am J Respir Crit Care Med*. 2017;195:1253–1263. <https://doi.org/10.1164/rccm.201703-0548ST>
43. Writing Group for the PREVENT Investigators, Simonis FD, Serpa Neto A, 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;320:1872–1880. <https://doi.org/10.1001/jama.2018.14280>
44. Schwartz BG, Ludeman DJ, Mayeda GS, Kloner RA, Economides C, Burstein S. Treating refractory cardiogenic shock with the TandemHeart and Impella devices: a single center experience. *Cardiol Res*. 2012;3:54–66. <https://doi.org/10.4021/cr121u>
45. Kontoyannis DA, Nanas JN, Kontoyannis SA, Stamatelopoulos SF, Mouloupoulos SD. Mechanical ventilation in conjunction with the intra-aortic balloon pump improves the outcome of patients in profound cardiogenic shock. *Intensive Care Med*. 1999;25:835–838. <https://doi.org/10.1007/s001340050960>
46. Liu H, Wu X, Zhao X, Zhu P, Han L. Intra-aortic balloon pump combined with mechanical ventilation for treating patients aged > 60 years in cardiogenic shock: retrospective analysis. *J Int Med Res*. 2016;44:433–443. <https://doi.org/10.1177/0300060515621443>
47. Deschamps J, Singh G, Grunau B, Van Diepen S. Approach to Troubleshooting, Complications, and Resuscitation for Patients on ECMO. In: Vranckx P, ed. *Association for Acute Cardiovascular Care – Handbook on Mechanical Circulatory Support*. European Society of Cardiology, 2022: 272–289. Accessed April 17, 2024. <https://www.escardio.org/static-file/Escardio/Subspecialty/ACCA/Publications/ACVC%20Handbook%20MCS%202022/ESC-HandbookMCS-IMPELLA%20TROUBLESHOOTING%20AND%20RESUSCITATION.pdf>
48. Rali AS, Tran LE, Auvil B, et al. Modifiable mechanical ventilation targets are associated with improved survival in ventilated VA-ECLS patients. *Heart Fail*. 2023;11:961–968. <https://doi.org/10.1016/j.jchf.2023.03.023>
49. Jentzer JC, Schrage B, Patel PC, et al. Association between the acidemia, lactic acidosis, and shock severity with outcomes in patients with cardiogenic shock. *J Am Heart Assoc*. 2022;11:e024932. <https://doi.org/10.1161/JAHA.121.024932>
50. Diehl A, Burrell AJC, Udy AA, et al. Association between arterial carbon dioxide tension and clinical outcomes in venoarterial extracorporeal membrane oxygenation. *Crit Care Med*. 2020;48:977–984. <https://doi.org/10.1097/CCM.0000000000004347>
51. Barrot L, Asfar P, Mauny F, et al. Liberal or conservative oxygen therapy for acute respiratory distress syndrome. *N Engl J Med*. 2020;382:999–1008. <https://doi.org/10.1056/NEJMoa1916431>
52. Shou BL, Ong CS, Zhou AL, et al. Arterial carbon dioxide and acute brain injury in venoarterial extracorporeal membrane oxygenation. *ASAIO J*. 2022;68:1501–1507. <https://doi.org/10.1097/MAT.0000000000001699>
53. Demiselle J, Wepler M, Hartmann C, et al. Hyperoxia toxicity in septic shock patients according to the Sepsis-3 criteria: a post hoc analysis of the HYPER25 trial. *Ann Intensive Care*. 2018;8:90. <https://doi.org/10.1186/s13613-018-0435-1>
54. Kim TY, Kim DH, Kim SC, et al. Impact of early hyperoxia on 28-day in-hospital mortality in patients with myocardial injury. *PLoS One*. 2018;13:e0201286. <https://doi.org/10.1371/journal.pone.0201286>
55. Kashiura M, Yasuda H, Kishihara Y, et al. Association between short-term neurological outcomes and extreme hyperoxia in patients with out-of-hospital cardiac arrest who underwent extracorporeal cardiopulmonary resuscitation: a retrospective observational study from a multicenter registry. *BMC Cardiovasc Disord*. 2022;22:163. <https://doi.org/10.1186/s12872-022-02598-6>
56. Bonnemain J, Rusca M, Ltaief Z, et al. Hyperoxia during extracorporeal cardiopulmonary resuscitation for refractory cardiac arrest is associated with severe circulatory failure and increased mortality. *BMC Cardiovasc Disord*. 2021;21:542. <https://doi.org/10.1186/s12872-021-02361-3>
57. Munshi L, Kiss A, Cypel M, Keshavjee S, Ferguson ND, Fan E. Oxygen thresholds and mortality during extracorporeal life support in adult patients. *Crit Care Med*. 2017;45:1997–2005. <https://doi.org/10.1097/CCM.0000000000002643>
58. Lim HS. Cardiogenic shock: failure of oxygen delivery and oxygen utilization. *Clin Cardiol*. 2016;39:477–483. <https://doi.org/10.1002/clc.22564>
59. Wood KL, Ayers B, Gosev I, et al. Venoarterial-extracorporeal membrane oxygenation without routine systemic anticoagulation

- decreases adverse events. *Ann Thorac Surg*. 2020;109:1458–1466. <https://doi.org/10.1016/j.athoracsur.2019.08.040>
60. Cartwright B, Bruce HM, Kershaw G, et al. Hemostasis, coagulation and thrombin in venoarterial and venovenous extracorporeal membrane oxygenation: the HECTIC study. *Sci Rep*. 2021;11:7975. <https://doi.org/10.1038/s41598-021-87026-z>
61. Sy E, Sklar MC, Lequier L, Fan E, Kanji HD. Anticoagulation practices and the prevalence of major bleeding, thromboembolic events, and mortality in venoarterial extracorporeal membrane oxygenation: a systematic review and meta-analysis. *J Crit Care*. 2017;39:87–96. <https://doi.org/10.1016/j.jcrc.2017.02.014>
62. Olson SR, Murphree CR, Zonies D, et al. Thrombosis and bleeding in extracorporeal membrane oxygenation (ECMO) without anticoagulation: a systematic review. *ASAIO J*. 2021;67:290–296. <https://doi.org/10.1097/MAT.0000000000001230>
63. Bergen K, Sridhara S, Cavarocchi N, Silvestry S, Ventura D. Analysis of bicarbonate-based purge solution in patients with cardiogenic shock supported via Impella ventricular assist device. *Ann Pharmacother*. 2023;57:646–652. <https://doi.org/10.1177/10600280221124156>
64. Pucher PH, Cummings IG, Shipolini AR, McCormack DJ. Is heparin needed for patients with an intra-aortic balloon pump? *Interact Cardiovasc Thorac Surg*. 2012;15:136–139. <https://doi.org/10.1093/icvts/ivs017>
65. McMichael ABV, Ryerson LM, Ratano D, Fan E, Faraoni D, Annich GM. 2021 ELSO adult and pediatric anticoagulation guidelines. *ASAIO J*. 2022;68:303–310. <https://doi.org/10.1097/MAT.0000000000001652>
66. Chlebowski MM, Baltagi S, Carlson M, Levy JH, Spinella PC. Clinical controversies in anticoagulation monitoring and antithrombin supplementation for ECMO. *Crit Care*. 2020;24:19. <https://doi.org/10.1186/s13054-020-2726-9>
67. Geli J, Capoccia M, Maybauer DM, Maybauer MO. Argatroban anticoagulation for adult extracorporeal membrane oxygenation: a systematic review. *J Intensive Care Med*. 2022;37:459–471. <https://doi.org/10.1177/0885066621993739>
68. Kim YS, Lee H, Yang JH, et al. Use of argatroban for extracorporeal life support in patients with nonheparin-induced thrombocytopenia: analysis of 10 consecutive patients. *Medicine (Baltimore)*. 2018;97:e13235. <https://doi.org/10.1097/MD.00000000000013235>
69. Navaei A, Kostousov V, Teruya J. Is it time to switch to bivalirudin for ECMO anticoagulation? *Front Med*. 2023;10:1237601. <https://doi.org/10.3389/fmed.2023.1237601>
70. Li DH, Sun MW, Zhang JC, Zhang C, Deng L, Jiang H. Is bivalirudin an alternative anticoagulant for extracorporeal membrane oxygenation (ECMO) patients? A systematic review and meta-analysis. *Thromb Res*. 2022;210:53–62. <https://doi.org/10.1016/j.thromres.2021.12.024>
71. Ranucci M, Ballotta A, Kandil H, et al. Bivalirudin-based versus conventional heparin anticoagulation for postcardiotomy extracorporeal membrane oxygenation. *Crit Care*. 2011;15:R275. <https://doi.org/10.1186/cc10556>
72. Ranucci M. Bivalirudin and post-cardiotomy ECMO: a word of caution. *Crit Care*. 2012;16:427. <https://doi.org/10.1186/cc11314>
73. Bashline M, DiBridge J, Klass WJ, et al. Outcomes of systemic bivalirudin and sodium bicarbonate purge solution for Impella 5.5. *Artif Organs*. 2023;47:361–369. <https://doi.org/10.1111/aor.14428>
74. Kazmi H, Milkovits AE. Use of systemic bivalirudin and an anticoagulant-free purge solution in a percutaneous left ventricular assist device in a patient with heparin-induced thrombocytopenia. *J Pharm Pract*. 2021;34:662–664. <https://doi.org/10.1177/0897190020930530>
75. Hohlfelder B, Kelly D, Hoang M, et al. Activated clotting times demonstrate weak correlation with heparin dosing in adult extracorporeal membrane oxygenation. *Am J Ther*. 2022;29:e385–e393. <https://doi.org/10.1097/MJT.0000000000001113>
76. Atallah S, Liebl M, Fitousis K, Bostan F, Masud F. Evaluation of the activated clotting time and activated partial thromboplastin time for the monitoring of heparin in adult extracorporeal membrane oxygenation patients. *Perfusion*. 2014;29:456–461. <https://doi.org/10.1177/0267659114524264>
77. Karkouti K, Callum J, Wijeyesundera DN, et al. Point-of-care hemostatic testing in cardiac surgery: a stepped-wedge clustered randomized controlled trial. *Circulation*. 2016;134:1152–1162. <https://doi.org/10.1161/CIRCULATIONAHA.116.023956>
78. 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;112:981–1004. <https://doi.org/10.1016/j.athoracsur.2021.03.033>
79. Task Force on Patient Blood Management for Adult Cardiac Surgery of the European Association of Cardio-Thoracic Surgery (EACTS) and the European Association of Cardiothoracic Anaesthesiology (EACTA), Boer C, Meesters MI, et al. 2017 EACTS/EACTA guidelines on patient blood management for adult cardiac surgery. *J Cardiothorac Vasc Anesth*. 2018;32:88–120. <https://doi.org/10.1053/j.jvca.2017.06.026>
80. Colman E, Yin EB, Laine G, et al. Evaluation of a heparin monitoring protocol for extracorporeal membrane oxygenation and review of the literature. *J Thorac Dis*. 2019;11:3325–3335. <https://doi.org/10.21037/jtd.2019.08.44>
81. Panigada M, E. Iapichino G, Brioni M, et al. Thromboelastography-based anticoagulation management during extracorporeal membrane oxygenation: a safety and feasibility pilot study. *Ann Intensive Care*. 2018;8:7. <https://doi.org/10.1186/s13613-017-0352-8>
82. Devlin JW, Skrobik Y, Gélinas C, et al. Clinical practice guidelines for the prevention and management of pain, agitation/sedation, delirium, immobility, and sleep disruption in adult patients in the ICU. *Crit Care Med*. 2018;46:e825. <https://doi.org/10.1097/CCM.0000000000003299>
83. 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) [published correction appears in *Ann Thorac Surg*. 2024;118:524–525. <https://doi.org/10.1016/j.athoracsur.2024.06.006>] *Ann Thorac Surg*. 2024;117:669–689. <https://doi.org/10.1016/j.athoracsur.2023.12.006>
84. Mori M, McCloskey G, Geirsson A, et al. Improving outcomes in INTERMACS category 1 patients with pre-LVAD, awake venous-arterial extracorporeal membrane oxygenation support. *ASAIO J*. 2019;65:819–826. <https://doi.org/10.1097/MAT.0000000000000908>
85. Caturegli G, Kapoor S, Ponomarev V, et al. Transcranial Doppler microemboli and acute brain injury in extracorporeal membrane oxygenation: a prospective observational study. *JTCVS Tech*. 2022;15:111–122. <https://doi.org/10.1016/j.jtc.2022.07.026>
86. Deng L, Xia Q, Chi C, Hu G. Awake veno-arterial extracorporeal membrane oxygenation in patients with perioperative period acute heart failure in cardiac surgery. *J Thorac Dis*. 2020;12:2179–2187. <https://doi.org/10.21037/jtd.2020.04.38>
87. Fong KM, Au SY, Ng GWY, Leung AKH. Positive fluid balance and mortality in adult patients treated with extracorporeal membrane oxygenation: a retrospective study. *J Intensive Care Soc*. 2020;21:210–220. <https://doi.org/10.1177/1751143719862240>
88. Cucchi M, Mariani S, De Piero ME, et al. Awake extracorporeal life support and physiotherapy in adult patients: a systematic review of the literature. *Perfusion*. 2023;38:939–958. <https://doi.org/10.1177/02676591221096078>
89. Paternoster G, Bertini P, Belletti A, et al. Venovenous extracorporeal membrane oxygenation in awake non-intubated patients with COVID-19 ARDS at high risk for barotrauma. *J Cardiothorac Vasc Anesth*. 2022;36:2975–2982. <https://doi.org/10.1053/j.jvca.2022.03.011>
90. Rabie AA, Elhazmi A, Azzam MH, et al. Expert consensus statement on venovenous extracorporeal membrane oxygenation ECMO for COVID-19 severe ARDS: an international Delphi study. *Ann Intensive Care*. 2023;13:36. <https://doi.org/10.1186/s13613-023-01126-9>
91. Kim HA, Kim YS, Cho YH, Kim WS, Sung K, Jeong DS. Implementation of venoarterial extracorporeal membrane oxygenation in nonintubated patients. *J Chest Surg*. 2021;54:17–24. <https://doi.org/10.5090/kjctcs.20.070>

92. van Houte J, Donker DW, Wagenaar LJ, Slootweg AP, Kirkels JH, van Dijk D. Non-intubated recovery from refractory cardiogenic shock on percutaneous VA-extracorporeal membrane oxygenation. *Neth Heart J*. 2015;23:386–388. <https://doi.org/10.1007/s12471-015-0721-6>
93. Esposito ML, Jablonski J, Kras A, Krasney S, Kapur NK. Maximum level of mobility with axillary deployment of the Impella 5.0 is associated with improved survival. *Int J Artif Organs*. 2018;41:236–239. <https://doi.org/10.1177/039139881752575>
94. Bertoldi LF, Pappalardo F, Lubos E, et al. Bridging INTERMACS 1 patients from VA-ECMO to LVAD via Impella 5.0: de-escalate and ambulate. *J Crit Care*. 2020;57:259–263. <https://doi.org/10.1016/j.jcrc.2019.12.028>
95. Horman JYK, Mohan P, Patel S, et al. (767) Impella 5.5 as bridge to heart transplant improves mobility and length of stay after transplant. *J Heart Lung Transplant*. 2023;42:5337–5338. <https://doi.org/10.1016/j.healun.2023.02.781>
96. Downey P, Ragalie W, Gudzenko V, Ardehali A. Ambulatory central veno-arterial extracorporeal membrane oxygenation in lung transplant candidates. *J Heart Lung Transplant*. 2019;38:1317–1319. <https://doi.org/10.1016/j.healun.2019.09.006>
97. Umakanthan R, Hoff SJ, Solenkova N, et al. Benefits of ambulatory axillary intra-aortic balloon pump for circulatory support as bridge to heart transplant. *J Thorac Cardiovasc Surg*. 2012;143:1193–1197. <https://doi.org/10.1016/j.jtcvs.2012.02.009>
98. Bhimaraj A, Agrawal T, Duran A, et al. Percutaneous left axillary artery placement of intra-aortic balloon pump in advanced heart failure patients. *JACC Heart Fail*. 2020;8:313–323. <https://doi.org/10.1016/j.jchf.2020.01.011>
99. Skrzat JM, Santhanam GI, Olejer MS, Sando TA. Mobility checklist for patients with advanced heart failure and a femoral intra-aortic balloon pump. *J Acute Care Phys Ther*. 2024;15:27. <https://doi.org/10.1097/JAT.0000000000000230>
100. Sessler CN, Gosnell MS, Grap MJ, et al. The Richmond Agitation–Sedation Scale. *Am J Respir Crit Care Med*. 2002;166:1338–1344. <https://doi.org/10.1164/rccm.2107138>
101. Célinas C, Fillion L, Puntillo KA, Viens C, Fortier M. Validation of the critical-care pain observation tool in adult patients. *Am J Crit Care*. 2006;15:420–427.
102. Dzierba AL, Abrams D, Brodie D. Medicating patients during extracorporeal membrane oxygenation: the evidence is building. *Crit Care*. 2017;21:66. <https://doi.org/10.1186/s13054-017-1644-y>
103. Landolf KM, Rivosecchi RM, Gómez H, et al. Comparison of hydromorphone versus fentanyl-based sedation in extracorporeal membrane oxygenation: a propensity-matched analysis. *Pharmacotherapy*. 2020;40:389–397. <https://doi.org/10.1002/phar.2385>
104. Browder K, Wanek M, Wang L, Hohlfelder B. Opioid and sedative requirements in extracorporeal membrane oxygenation patients on hydromorphone versus fentanyl. *Artif Organs*. 2022;46:878–886. <https://doi.org/10.1111/aor.14125>
105. Lamm W, Nagler B, Hermann A, et al. Propofol-based sedation does not negatively influence oxygenator running time compared to midazolam in patients with extracorporeal membrane oxygenation. *Int J Artif Organs*. 2019;42:233–240. <https://doi.org/10.1177/0391398819833376>
106. Patel JS, Kooda K, Igreri LA. A narrative review of the impact of extracorporeal membrane oxygenation on the pharmacokinetics and pharmacodynamics of critical care therapies. *Ann Pharmacother*. 2023;57:706–726. <https://doi.org/10.1177/10600280221126438>
107. Farrokh S, Kim BS, Cho SM. Ketamine infusion for sedation in a patient on extracorporeal membrane oxygenation (ECMO). *Perfusion*. 2024;39:223–226. <https://doi.org/10.1177/02676591221134941>
108. DeGrado JR, Hohlfelder B, Ritchie BM, Anger KE, Reardon DP, Weinhouse GL. Evaluation of sedatives, analgesics, and neuromuscular blocking agents in adults receiving extracorporeal membrane oxygenation. *J Crit Care*. 2017;37:1–6. <https://doi.org/10.1016/j.jcrc.2016.07.020>
109. Kirigaya J, Iwahashi N, Terasaka K, Takeuchi I. Prevention and management of critical care complications in cardiogenic shock: a narrative review. *J Intensive Care*. 2023;11:31. <https://doi.org/10.1186/s40560-023-00675-2>
110. Pasek TA, Kitcho S, Fox S, et al. Preventing hospital-acquired pressure injuries by using a tiered protocol in children receiving ECMO in the pediatric intensive care unit. *Crit Care Nurse*. 2021;41:71–77. <https://doi.org/10.4037/ccn2021558>
111. Asiri S. Turning and repositioning frequency to prevent hospital-acquired pressure injuries among adult patients: systematic review. *Inquiry*. 2023;60:469580231215209. <https://doi.org/10.1177/00469580231215209>
112. Polastri M, Eden A, Loforte A, et al. Physiotherapy for patients on extracorporeal membrane oxygenation support: how, when, and who. An international EuroELSO survey. *Perfusion*. 2024;39:162–173. <https://doi.org/10.1177/02676591221133657>
113. Patnaik S, Nathan S, Bui A, Kar B, Gregoric ID, Gilley CB. Impact of structured physical therapy protocol among heart transplant recipients while on intra-aortic balloon pump in the pretransplant period. *Artif Organs*. 2023;47:198–204. <https://doi.org/10.1111/aor.14378>
114. Llano-Diez M, Renaud G, Andersson M, et al. Mechanisms underlying ICU muscle wasting and effects of passive mechanical loading. *Crit Care*. 2012;16:R209. <https://doi.org/10.1186/cc11841>
115. Brower RG. Consequences of bed rest. *Crit Care Med*. 2009;37:5422–5428. <https://doi.org/10.1097/CCM.0b013e3181b6e30a>
116. Zhang L, Hu W, Cai Z, et al. Early mobilization of critically ill patients in the intensive care unit: a systematic review and meta-analysis. *PLoS One*. 2019;14:e0223185. <https://doi.org/10.1371/journal.pone.0223185>
117. Wu RY, Yeh HJ, Chang KJ, Tsai MW. Effects of different types and frequencies of early rehabilitation on ventilator weaning among patients in intensive care units: a systematic review and meta-analysis. *PLoS One*. 2023;18:e0284923. <https://doi.org/10.1371/journal.pone.0284923>
118. Ko Y, Cho YH, Park YH, et al. Feasibility and safety of early physical therapy and active mobilization for patients on extracorporeal membrane oxygenation. *ASAIO J*. 2015;61:564–568. <https://doi.org/10.1097/MAT.0000000000000239>
119. Tipping CJ, Bailey MJ, Bellomo R, et al. The ICU Mobility Scale has construct and predictive validity and is responsive. A multicenter observational study. *Ann Am Thorac Soc*. 2016;13:887–893. <https://doi.org/10.1513/AnnalsATS.201510-717OC>
120. Hoyer EH, Friedman M, Lavezza A, et al. Promoting mobility and reducing length of stay in hospitalized general medicine patients: a quality-improvement project. *J Hosp Med*. 2016;11:341–347. <https://doi.org/10.1002/jhm.2546>
121. Assmann A, Beckmann A, Schmid C, et al. Use of extracorporeal circulation (ECLS/ECMO) for cardiac and circulatory failure –a clinical practice Guideline Level 3. *ESC Heart Fail*. 2022;9:506–518. <https://doi.org/10.1002/ehf2.13718>
122. Nishida H, Ota T, Onsager D, Grinstein J, Jeevanandam V, Song T. Ten-year, single center experience of ambulatory axillary intra-aortic balloon pump support for heart failure. *J Cardiol*. 2022;79:611–617. <https://doi.org/10.1016/j.jjcc.2021.11.010>
123. Schibilsky D, Lausberg H, Haller C, et al. Impella 5.0 support in INTERMACS II cardiogenic shock patients using right and left axillary artery access. *Artif Organs*. 2015;39:660–663. <https://doi.org/10.1111/aor.12529>
124. Lorusso R, Shekar K, MacLaren G, et al. ELSO interim guidelines for venoarterial extracorporeal membrane oxygenation in adult cardiac patients. *ASAIO J*. 2021;67:827–844. <https://doi.org/10.1097/MAT.00000000000001510>
125. Winters BD, Gurses AP, Lehmann H, Sexton JB, Rampersad CJ, Pronovost PJ. Clinical review: checklists—translating evidence into practice. *Crit Care*. 2009;13:210. <https://doi.org/10.1186/cc7792>
126. Stentz MJ, Wiepking MD, Hodge KA, Ramonell RP, Jabaley CS. Checklists improve team performance during simulated extracorporeal membrane oxygenation emergencies: a randomized trial. *Crit Care Explor*. 2021;3:e0404. <https://doi.org/10.1097/CCE.0000000000000404>

- 127.** Prisco AR, Aguado-Sierra J, Butakoff C, et al. Concomitant respiratory failure can impair myocardial oxygenation in patients with acute cardiogenic shock supported by VA-ECMO. *J Cardiovasc Transl Res.* 2022;15:217–226. <https://doi.org/10.1007/s12265-021-10110-2>
- 128.** Pozzebon S, Blandino Ortiz A, Franchi F, et al. Cerebral near-infrared spectroscopy in adult patients undergoing veno-arterial extracorporeal membrane oxygenation. *Neurocrit Care.* 2018;29:94–104. <https://doi.org/10.1007/s12028-018-0512-1>
- 129.** Khan I, Rehan M, Parikh G, et al. Regional cerebral oximetry as an indicator of acute brain injury in adults undergoing veno-arterial extracorporeal membrane oxygenation—a prospective pilot study. *Front Neurol.* 2018;9:993. <https://doi.org/10.3389/fneur.2018.00993>
- 130.** Risnes I, Wagner K, Nome T, et al. Cerebral outcome in adult patients treated with extracorporeal membrane oxygenation. *Ann Thorac Surg.* 2006;81:1401–1406. <https://doi.org/10.1016/j.athoracsur.2005.10.008>
- 131.** Dickstein ML. The Starling relationship and veno-arterial ECMO: ventricular distension explained. *ASAIO J.* 2018;64:497–501. <https://doi.org/10.1097/MAT.0000000000000660>
- 132.** Rajagopal K. Left ventricular distension in veno-arterial extracorporeal membrane oxygenation: from mechanics to therapies. *ASAIO J.* 2019;65:1–10. <https://doi.org/10.1097/MAT.0000000000000789>
- 133.** Cevasco M, Takayama H, Ando M, Garan AR, Naka Y, Takeda K. Left ventricular distension and venting strategies for patients on venoarterial extracorporeal membrane oxygenation. *J Thorac Dis.* 2019;11:1676–1683–1683.
- 134.** Truby LK, Takeda K, Mauro C, et al. Incidence and implications of left ventricular distention during venoarterial extracorporeal membrane oxygenation support. *ASAIO J.* 2017;63:257–265. <https://doi.org/10.1097/MAT.0000000000000553>
- 135.** Meani P, Delnoij T, Raffa GM, et al. Protracted aortic valve closure during peripheral veno-arterial extracorporeal life support: is intra-aortic balloon pump an effective solution? *Perfusion.* 2019;34:35–41. <https://doi.org/10.1177/0267659118787426>
- 136.** Mourad M, Eliet J, Zeroual N, et al. Pulse pressure and end-tidal carbon dioxide for monitoring low native cardiac output during veno-arterial ECLS: a prospective observational study. *Crit Care.* 2020;24:569. <https://doi.org/10.1186/s13054-020-03280-z>
- 137.** Donker DW, Brodie D, Henriques JPS, Broomé M. Left ventricular unloading during veno-arterial ECMO: a simulation study. *ASAIO J.* 2019;65:11–20. <https://doi.org/10.1097/MAT.0000000000000755>
- 138.** Su Y, Liu K, Zheng JL, et al. Hemodynamic monitoring in patients with venoarterial extracorporeal membrane oxygenation. *Ann Transl Med.* 2020;8:792. <https://doi.org/10.21037/atm.2020.03.186>
- 139.** Park BW, Seo DC, Moon IK, et al. Pulse pressure as a prognostic marker in patients receiving extracorporeal life support. *Resuscitation.* 2013;84:1404–1408. <https://doi.org/10.1016/j.resuscitation.2013.04.009>
- 140.** Unai S, Nguyen ML, Tanaka D, et al. Clinical significance of spontaneous echo contrast on extracorporeal membrane oxygenation. *Ann Thorac Surg.* 2017;103:773–778. <https://doi.org/10.1016/j.athoracsur.2016.07.019>
- 141.** Petroni T, Harrois A, Amour J, et al. Intra-aortic balloon pump effects on macrocirculation and microcirculation in cardiogenic shock patients supported by venoarterial extracorporeal membrane oxygenation. *Crit Care Med.* 2014;42:2075. <https://doi.org/10.1097/CCM.0000000000000410>
- 142.** Donker DW, Meuwese CL, Braithwaite SA, et al. Echocardiography in extracorporeal life support: a key player in procedural guidance, tailoring and monitoring. *Perfusion.* 2018;33(suppl):31–41. <https://doi.org/10.1177/0267659118766438>
- 143.** Aissaoui N, Luyt CE, Leprince P, et al. Predictors of successful extracorporeal membrane oxygenation (ECMO) weaning after assistance for refractory cardiogenic shock. *Intensive Care Med.* 2011;37:1738–1745. <https://doi.org/10.1007/s00134-011-2358-2>
- 144.** Vasques F, Sanderson B, Correa G, et al. Prevalence and indications for oxygenator circuit replacement in patients receiving venovenous extracorporeal membrane oxygenation. *ASAIO J.* 2023;69:849–855. <https://doi.org/10.1097/MAT.0000000000001977>
- 145.** Lubnow M, Philipp A, Foltan M, et al. Technical complications during veno-venous extracorporeal membrane oxygenation and their relevance predicting a system-exchange – retrospective analysis of 265 cases. *PLoS One.* 2014;9:e112316. <https://doi.org/10.1371/journal.pone.0112316>
- 146.** Chamogeorgakis T, Lima B, Shafii AE, et al. Outcomes of axillary artery side graft cannulation for extracorporeal membrane oxygenation. *J Thorac Cardiovasc Surg.* 2013;145:1088–1092. <https://doi.org/10.1016/j.jtcvs.2012.08.070>
- 147.** Balthazar T, Vandenbrielle C. Impella Troubleshooting and Resuscitation. In: Vranckx P, ed. *Association for Acute CardioVascular Care – Handbook on Mechanical Circulatory Support.* European Society of Cardiology; 2022:113–125. Accessed April 17, 2024. <https://www.escardio.org/static-file/Escardio/Subspecialty/ACCA/Publications/ACVC%20Handbook%20MCS%202022/ESC-HandbookMCS-IMPELLA%20TROUBLESHOOTING%20AND%20RESUSCITATION.pdf>
- 148.** Zein R, Patel C, Mercado-Alamo A, Schreiber T, Kaki A. A review of the Impella devices. *Interv Cardiol.* 2022;17:e05. <https://doi.org/10.15420/icr.2021.11>
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