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Venoarterial extracorporeal membrane oxygenation as a rescue strategy in high-risk pulmonary thromboembolism

Woo Jin Jang[1,*], Ik Hyun Park[2,*], Gyu Tae Park[1], Tae Wan Kim[3], Won-Young Kim[3]

[1]Division of Cardiology, Department of Internal Medicine, Chung-Ang University Hospital, Chung-Ang University College of Medicine, Seoul, Korea
[2]Division of Cardiology, Department of Internal Medicine, Samsung Changwon Hospital, Sungkyunkwan University School of Medicine, Changwon, Korea
[3]Division of Pulmonary and Critical Care Medicine, Department of Internal Medicine, Chung-Ang University Hospital, Chung-Ang University College of Medicine, Seoul, Korea

Acute high-risk pulmonary thromboembolism (PTE) is the most severe clinical manifestation of venous thromboembolism, carrying a substantial mortality rate, particularly when complicated by cardiogenic shock or cardiac arrest. Although current guidelines recommend systemic thrombolysis for high-risk PTE in the absence of contraindications, this approach is associated with a significant risk of major hemorrhage and may be precluded in patients with severe comorbidities, advanced frailty, or elevated perioperative risk. Consequently, venoarterial extracorporeal membrane oxygenation (VA-ECMO) has emerged as an important mechanical circulatory support strategy for this vulnerable cohort. By bypassing the obstructed pulmonary vascular bed, VA-ECMO facilitates rapid hemodynamic stabilization, mechanically unloads the strained right ventricle, and reverses systemic hypoperfusion. This review elucidates the pathophysiological rationale for right ventricular unloading in high-risk PTE and examines the evolving approach toward catheter-directed interventions and patient-tailored treatment strategies. Furthermore, we evaluate the clinical outcomes of VA-ECMO when deployed either as a stand-alone therapy bridge to recovery alongside systemic anticoagulation or as a bridge to definitive active thrombus clearance.

Key Words: cardiogenic shock; extracorporeal membrane oxygenation; pulmonary thromboembolism; right heart failure

Review Article

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Corresponding author
Woo Jin Jang
Division of Cardiology, Department of Internal Medicine, Chung-Ang University Hospital, Chung-Ang University College of Medicine, 102 Heukseok-ro, Dongjak-gu, Seoul 06973, Korea
Tel: +82-2-6299-1415
Fax: +82-2-6299-1415
Email: wj78914@gmail.com

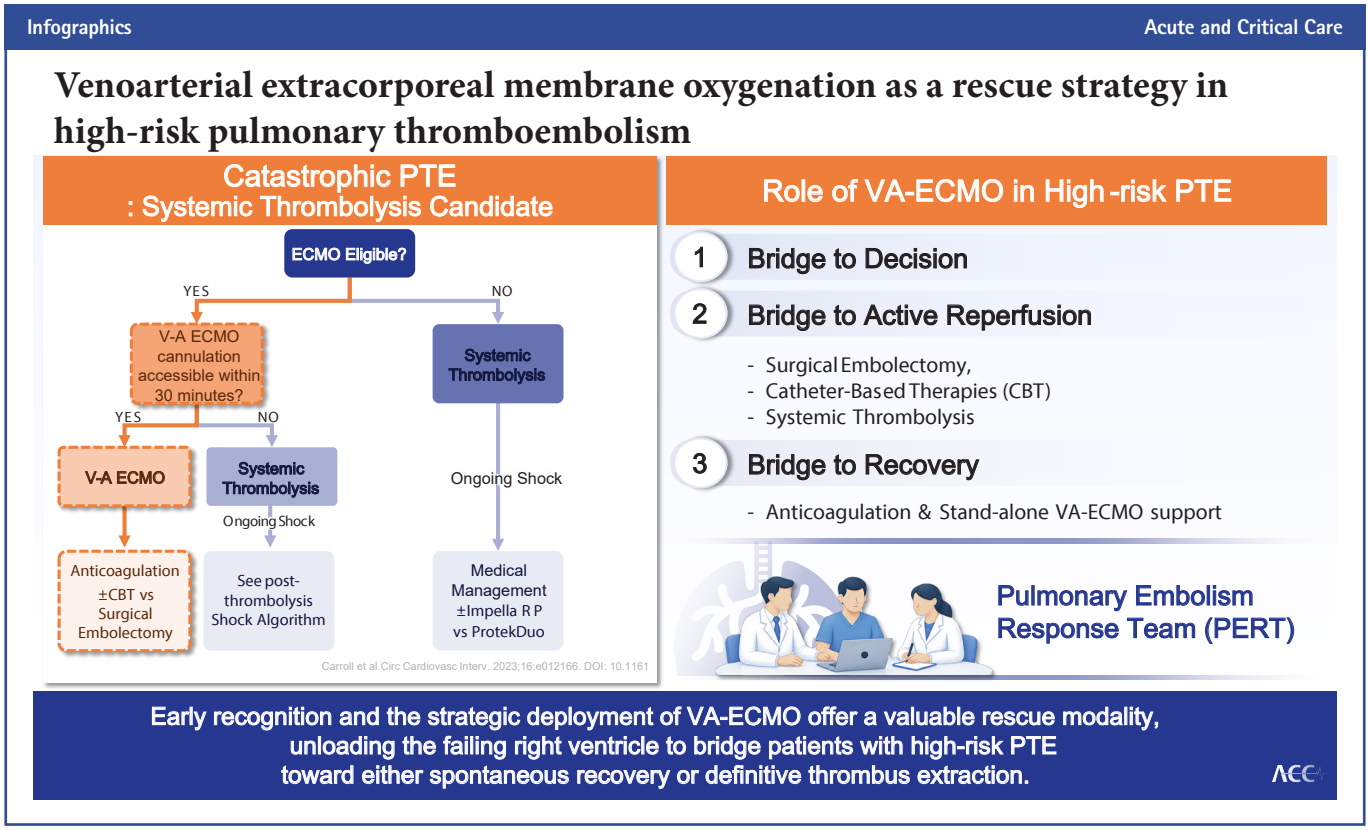
*These authors contributed equally to this work as co-first authors.

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INTRODUCTION

Acute high-risk pulmonary thromboembolism (PTE) is a life-threatening cardiovascular emergency with an estimated annual global incidence of 1 per 1,000 individuals [1]. As the most severe clinical manifestation of venous thromboembolism, high-risk PTE is characterized by profound hemodynamic instability—manifesting as sustained hypotension, end-or-



gan hypoperfusion, or cardiac arrest—concomitant with acute right ventricular (RV) strain [2,3]. Hemodynamic collapse in this setting confers a dire prognosis; 30-day mortality for high-risk PTE ranges 16%–46%, escalating to 50%–80% in patients who deteriorate into cardiac arrest [3,4].

Although contemporary guidelines recommend systemic thrombolysis for high-risk PTE in the absence of contraindications [2], this intervention carries a substantial risk of catastrophic hemorrhagic complications [5]. Furthermore, affected patients frequently present with serious medical comorbidities or advanced frailty, rendering the perioperative risk of surgical embolectomy prohibitively high [6]. Consequently, evolving clinical practices are expanding the role for mechanical circulatory support (MCS) and advanced catheter-based interventions [7].

While large-scale randomized controlled trials are currently lacking and societal guidelines typically relegate venoarterial extracorporeal membrane oxygenation (VA-ECMO) to a conditional rescue therapy [2,8], emerging observational and retrospective registry data highlight the growing utility of this approach. VA-ECMO provides rapid cardiopulmonary stabilization, effectively interrupting the pathophysiological spiral of RV failure and myocardial ischemia [7,9]. Notably, the 2026 American Heart Association/American College of Cardiology (AHA/ACC) guidelines for the Evaluation and Management of Acute Pulmonary Embolism redefined high-risk PTE as category E (cardiopulmonary failure), explicitly delineating the role of VA-ECMO in patients with persistent hypotension or cardiac arrest [8]. Accordingly, this review examines the hemodynamic rationale, optimal integration strategies, and intensive care management of VA-ECMO in the setting of high-risk PTE.

PATHOPHYSIOLOGY AND CONVENTIONAL LIMITATIONS IN HIGH-RISK PTE

The 2026 AHA/ACC Clinical Categories

The 2026 AHA/ACC guidelines introduced a conceptual shift by replacing traditional risk-based stratifications with the Acute Pulmonary Embolism Clinical Categories, designated A through E (Table 1) [8]. This novel classification taxonomy incorporates clinical severity, cardiac biomarker profiles, RV imaging, and hemodynamic parameters to optimize prog-

nostic precision and therapeutic decision-making. Category E represents the highest-risk cohort and constitutes the primary indication for VA-ECMO deployment. Specifically, category E1 patients are characterized by persistent hemodynamic instability despite optimal initial resuscitation, whereas category E2 patients are those who have progressed to refractory cardiogenic shock or cardiac arrest, thereby necessitating extracorporeal cardiopulmonary resuscitation. This granular classification facilitates individualized management strategies, underscoring that patients in categories D and E require immediate intensive care admission and emergent evaluation for advanced reperfusion modalities [8].

Hemodynamic Collapse and Ventricular Interdependence

The primary mechanism driving hemodynamic collapse in high-risk PTE is the acute mechanical obstruction of the pulmonary vascular bed coupled with profound hypoxia-induced pulmonary vasoconstriction [1]. This abrupt escalation in pulmonary vascular resistance and RV afterload precipitates rapid RV dilation. The consequential reduction in antegrade pulmonary blood flow critically impairs left ventricular (LV) preload, culminating in severe systemic hypoperfusion. Concurrently, the inability of the RV to eject against extreme afterload generates elevated right atrial pressures, profound systemic venous congestion, and subsequent multi-organ dysfunction [2].

A critical pathophysiological mechanism exacerbating this hemodynamic compromise is ventricular interdependence. Massive RV dilation induces a leftward bowing of the interventricular septum, which mechanically impedes LV diastolic compliance and further diminishes LV stroke volume. Given rapid anatomical and functional shifts, continuous hemodynamic and echocardiographic monitoring is paramount. Classical echocardiographic indices of severe RV strain in this setting include marked RV chamber enlargement, McConnell's sign, and a significantly reduced tricuspid annular plane systolic excursion (TAPSE <16 mm) [2].

KEY MESSAGES

- Acute high-risk pulmonary thromboembolism complicated by hemodynamic instability and right ventricular strain is associated with a 30-day mortality rate of 16%–46%, escalating to 50%–80% in cases of cardiac arrest.
- Observational data suggest that venoarterial extracorporeal membrane oxygenation can effectively halt the devastating cycle of right ventricular dysfunction and ischemia, serving as an effective bridge to recovery or active thrombus removal.
- An evolving clinical trend is increased reliance on mechanical circulatory support and catheter-based therapies to mitigate the bleeding risks of systemic thrombolysis; however, prospective randomized trials remain necessary to validate these strategies and refine patient selection.

Limitations of Systemic Thrombolysis and Surgical Embolectomy

Although contemporary guidelines advocate systemic thrombolysis as the primary intervention for high-risk PTE in the absence of absolute contraindications [2], this pharmacological thrombolysis fundamentally alters the hemostatic balance, conferring a substantial risk of catastrophic hemorrhage. Keller et al. [5] demonstrated in large-scale, real-world registry data that systemic thrombolysis is independently associated

Table 1. The 2026 AHA/ACC acute pulmonary embolism clinical categories

AHA/ACC PTE category	Clinical description	Subcategories and characteristics
A	Subclinical	Incidental finding, asymptomatic
B	Symptomatic (low clinical severity score)	B1: subsegmental
		B2: non-subsegmental
C	Symptomatic (elevated clinical severity score)	C1: normal RV/biomarkers
		C2: abnormal RV or biomarkers
		C3: both abnormal
D	Incipient cardiopulmonary failure	D1: transient/relative hypotension responsive to fluids
		D2: normotensive shock
E	Cardiopulmonary failure	E1: recurrent/persistent hypotension with cardiogenic shock
		E2: refractory shock or cardiac arrest

AHA/ACC: American Heart Association/American College of Cardiology; PTE: pulmonary thromboembolism; RV: right ventricle.

with elevated rates of major bleeding, most notably intracranial hemorrhage. Consequently, only a minority of hemodynamically unstable patients ultimately receive systemic thrombolysis in routine clinical practice.

While VA-ECMO serves as a viable rescue strategy following thrombolytic failure, the requisite large-bore arterial cannulation in the immediate post-thrombolysis window introduces a formidable risk of life-threatening access-site hemorrhage [8,10]. Notably, the 2026 AHA/ACC guidelines emphasize that invasive procedures performed subsequent to fibrinolytic administration frequently precipitate severe complications. Therefore, a rigorous evaluation of the hemorrhagic risk-benefit profile—ideally facilitated by a multidisciplinary Pulmonary Embolism Response Team (PERT)—must be a central component of the decision-making process [8].

Surgical embolectomy remains a definitive alternative when systemic thrombolysis is contraindicated or unsuccessful. However, as Keeling et al. [6] demonstrated, although surgical intervention improves survival in carefully selected cohorts, patients presenting with high-risk PTE frequently possess significant medical comorbidities or advanced frailty, rendering the perioperative mortality risk prohibitively high. This creates a critical therapeutic gap for patients who face unacceptable hemorrhagic risk with systemic thrombolysis yet are too frail to tolerate open surgical intervention.

ROLE OF VA-ECMO AND CATHETER-BASED THERAPIES

Hemodynamic Rationale for VA-ECMO

When high-risk PTE is complicated by refractory cardiogenic shock or cardiac arrest, the early initiation of MCS is crucial. VA-ECMO has become established as a premier modality for managing biventricular or isolated RV failure concomitant with severe respiratory compromise [7]. Hobohm et al. [9] reported a rapid increase in VA-ECMO utilization for hospitalized patients with high-risk PTE, underscoring its efficacy in achieving immediate hemodynamic stabilization.

Physiologically, VA-ECMO functions by draining deoxygenated venous blood from the right atrium or inferior vena cava and returning oxygenated blood to the iliofemoral arterial system. This configuration simultaneously unloads the strained RV, restores systemic perfusion, and corrects refractory hypoxemia [7]. Although current guidelines cautiously position VA-ECMO primarily as a rescue therapy [2,8], an expanding body of retrospective data and recent meta-analyses indicate its utility as a short-term, stand-alone mechanical support strategy. This "bridging" approach provides a critical window for endogenous fibrinolysis under systemic anticoagulation, thereby circumventing the compounded bleeding risks associated with adjunctive thrombolytic therapies [11,12]. Anticoagulation management is vital; while an activated clotting time of 160–180 seconds is typically targeted during the cannulation phase, maintenance therapy frequently relies on achieving an activated partial thromboplastin time of 60–80 seconds or 1.5 to 2.5 times the upper limit of normal, contingent upon institutional protocols.

Despite its capacity for rapid stabilization, the application of VA-ECMO as an isolated "bridge to recovery"—relying exclusively on endogenous fibrinolysis and systemic anticoagulation—warrants careful patient selection. This conservative strategy is highly situational, generally reserved for patients with absolute contraindications to active reperfusion (e.g., active intracranial hemorrhage or recent major neurosurgery) or those exhibiting rapid, spontaneous clinical and echocardiographic resolution of RV dysfunction shortly after cannulation. Conversely, for the majority of patients—particularly those presenting with a massive proximal thrombus burden, persistent RV failure, or an inability to tolerate initial ECMO weaning trials—active reperfusion via catheter-directed therapies or surgical embolectomy remains imperative to mechanically alleviate the pulmonary obstruction.

Regarding comparative outcomes, recent retrospective registries and meta-analyses suggest that a combined approach of VA-ECMO followed by active reperfusion may be associated with better survival than VA-ECMO alone [13]. However, the interpretation of these findings is heavily confounded by inherent selection bias. Observational "ECMO-alone" cohorts disproportionately include patients with prolonged cardiac arrest, severe anoxic brain injury, or profound hemodynamic instability precluding transport for subsequent interventional procedures. Consequently, the decision to pursue conservative ECMO management versus integration with active reperfusion must be meticulously individualized. This highlights the indispensable role of a multidisciplinary PERT in continuously reassessing the patient's clinical trajectory and modulating

therapeutic strategies accordingly.

The Evolving Role of Catheter-Based Therapies

To bridge the therapeutic gap between systemic thrombolysis and surgical embolectomy, there has been a gradual transition toward a greater role for catheter-based therapies (CBT). Giri et al. [7] emphasized that interventional modalities represent a vital evolution in the management of severe PTE. The primary mechanistic approaches of CBT encompass mechanical fragmentation, rheolytic therapy, catheter-directed thrombolysis (CDT), and aspiration embolectomy.

Currently, the majority of CBT interventions involve CDT or large-bore aspiration therapy. Ultrasound-facilitated CDT and large-bore aspiration have demonstrated substantial efficacy in rapidly alleviating RV strain and restoring pulmonary perfusion. In contrast, rheolytic therapies warrant extreme caution due to a high incidence of procedural complications, most notably profound bradycardia and hemoglobinuria [14].

Although CBT provides a minimally invasive alternative, its application necessitates rigorous patient selection and is frequently optimized when performed under MCS. A critical limitation arises in patients presenting with impending or established cardiac arrest. In such peri-arrest scenarios, the stand-alone use of CBT is precluded by logistical challenges during active resuscitation and a paucity of definitive evidence [2,7,8]. Consequently, VA-ECMO remains the premier strategy for immediate circulatory stabilization. Furthermore, recent data suggest that a hybrid approach—initial stabilization with VA-ECMO followed by CBT—may confer superior clinical outcomes compared to isolated VA-ECMO support [6,15].

Role of the PERT

A PERT comprises multidisciplinary experts and serves a central role in initial risk stratification and formation of individualized management paradigms for patients with high-risk PTE [15,16]. Contemporary PERT models meticulously evaluate the patient's clinical and hemorrhagic risk profiles to determine the optimal reperfusion therapy, navigating the complex decision-making matrix between systemic thrombolysis, CBT, and surgical embolectomy [8].

In the setting of profound cardiogenic shock, the failure of initial systemic thrombolysis presents a severe clinical dilemma; the heightened hemorrhagic risk associated with subsequent large-bore cannulation significantly complicates the emergent initiation of VA-ECMO support. Despite these formidable challenges, VA-ECMO frequently serves as an indispensable bridge to definitive therapeutic interventions [8,11]. In such complex clinical scenarios, a PERT facilitates the decision-making process for integrated strategies—such as VA-ECMO combined with CBT—to achieve immediate stability while addressing the limitations and hemorrhagic risks of conventional therapies [8,15].

Integrated Decision-Making Framework

To translate these diverse therapeutic modalities into a cohesive clinical paradigm, we propose a structured management algorithm for high-risk PTE, directed by continuous multidisciplinary PERT evaluation (Figure 1). Upon the recognition of a patient with high-risk PTE presenting with profound hemodynamic instability, the immediate triage decision hinges on the trajectory of the patient's shock state and the presence of absolute contraindications to systemic thrombolysis. In patients devoid of contraindications who maintain a viable systemic perfusion pressure, systemic thrombolysis remains the primary guideline-directed intervention. Conversely, if the patient exhibits absolute contraindications to fibrinolysis, rapidly progresses to refractory cardiogenic shock, or experiences impending or established cardiac arrest, an "ECMO-first" stabilization strategy must be immediately prioritized.

Following successful VA-ECMO cannulation, the PERT must expeditiously determine the definitive reperfusion strategy. As previously delineated, while a stand-alone VA-ECMO approach relying exclusively on systemic anticoagulation may suffice for a highly selected minority exhibiting rapid spontaneous recovery, the vast majority of patients will require active mechanical clot extraction. Selection among advanced reperfusion modalities while on ECMO is dictated by institutional expertise, local resource availability, and patient-specific cardiopulmonary anatomy. CBT, particularly large-bore aspiration, is generally favored as the immediate subsequent intervention due to its minimally invasive profile and high procedural success rates when performed under MCS. In contrast, open surgical embolectomy is typically reserved for patients who fail CBT, harbor a right heart thrombus in transit, or require concomitant structural cardiac interventions (e.g., closure of a patent foramen ovale).

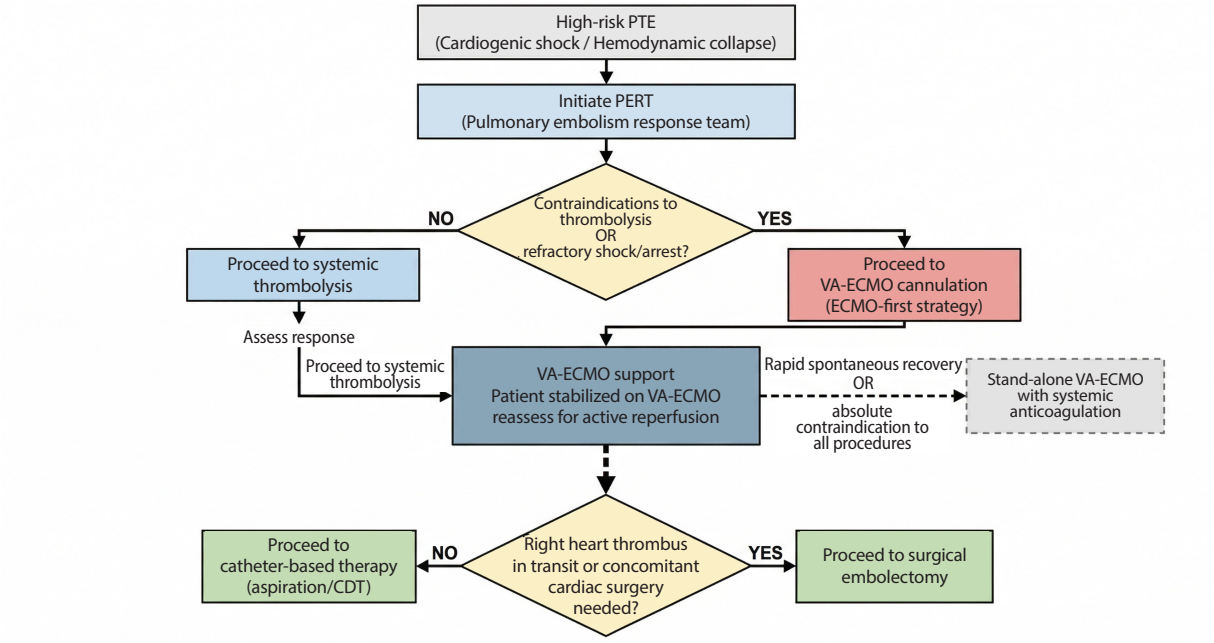


Figure 1. Step-by-step decision pathway for the management of high-risk pulmonary thromboembolism (PTE). PERT: pulmonary embolism response team; VA-ECMO: venoarterial extracorporeal membrane oxygenation; CDT: catheter-directed thrombolysis.

CLINICAL IMPLICATIONS AND INTENSIVE CARE MANAGEMENT

Optimal Ventilation and Positive End-Expiratory Pressure Strategies

In intubated patients with high-risk PTE, cardiopulmonary interactions during positive pressure mechanical ventilation exert a profound influence on RV hemodynamics. Sinha et al. [17] demonstrated that optimal ventilatory management necessitates the application of low positive end-expiratory pressure, typically restricted to 5–7 cm H_2O. This conservative positive end-expiratory pressure strategy mitigates excessive elevations in intrathoracic pressure, thereby preventing the exacerbation of RV afterload and myocardial strain. Furthermore, the fraction of inspired oxygen (FiO_2) should be carefully titrated. While prolonged hyperoxia (>60% FiO_2) should be avoided when clinically feasible to mitigate oxygen toxicity, higher FiO_2 is often temporarily necessary during early acute stabilization. Furthermore, the VA-ECMO circuit fundamentally alters standard oxygenation dynamics by creating a dual circulation. Consequently, FiO_2 titration requires a highly individualized approach that balances the avoidance of hyperoxia with the critical need to maintain adequate systemic oxygenation, which is necessary to induce pulmonary vasodilation and further reduce RV afterload [17].

Hemodynamic Monitoring and Support

The intensive care management of patients with high-risk PTE, particularly those supported by VA-ECMO, necessitates precise cardiopulmonary optimization. Continuous echocardiography should serve as the primary and safest modality to guide the nuanced titration of volume and vasoactive therapies. Although pulmonary artery catheterization (PAC) can provide comprehensive hemodynamic data, its application in this specific population mandates extreme caution. Advancing a PAC through an acutely dilated, compromised RV into an obstructed pulmonary vascular bed carries a substantial risk of inducing malignant arrhythmias or precipitating profound hemodynamic collapse. Furthermore, the retrograde flow dynamics inherent to peripheral VA-ECMO can significantly confound PAC-derived measurements [18]. Consequently, PAC insertion requires meticulous timing, must be performed by highly experienced personnel, and is best reserved for complex scenarios where echocardiographic evaluation proves insufficient to guide management. Optimizing preload is necessary, but volume overload must be strictly avoided to prevent further RV distension and the worsening of ventricular interdependence. Inotropic and vasopressor support, preferentially with dobu-

tamine and norepinephrine, should be carefully titrated to maintain systemic perfusion without exacerbating myocardial oxygen demand [2].

The fluid management strategy in acute RV failure must be highly restrictive. Although a cautious fluid challenge (<500 ml) may be considered in strictly hypovolemic patients, routine volume loading is profoundly detrimental in the setting of high-risk PTE. In the presence of elevated pulmonary vascular resistance, the RV operates on the flat portion of the Frank-Starling curve; consequently, excessive fluid administration rapidly overdistends the RV, sharply increasing wall tension and exacerbating myocardial ischemia. Furthermore, this acute overdistension worsens adverse ventricular interdependence by displacing the interventricular septum leftward, which paradoxically impairs LV preload and diminishes systemic cardiac output.

When pharmacological hemodynamic support is required, the precise selection and judicious titration of vasoactive agents are critical. Norepinephrine remains the first-line vasopressor. Its primary utility lies in restoring systemic mean arterial pressure, which is essential to re-establish the aorto-RV pressure gradient. This gradient drives critical coronary perfusion to the failing RV without significantly escalating pulmonary vascular resistance. If systemic cardiac output remains inadequate despite the optimization of systemic blood pressure, inotropic support with dobutamine may be introduced. While dobutamine enhances RV contractility and can induce mild pulmonary vasodilation, it must be administered with extreme caution. Tachycardia or tachyarrhythmias induced by excessive inotropic stimulation drastically increase myocardial oxygen demand and abbreviate the RV diastolic perfusion time, thereby accelerating the vicious cycle of ischemia and ventricular failure [19].

Complications and Limitations of Peripheral VA-ECMO

Despite its life-saving potential, the deployment of peripheral VA-ECMO is accompanied by a spectrum of severe complications and inherent limitations that demand vigilant intensive care monitoring. Among the most frequent and devastating morbidities is ipsilateral lower limb ischemia, driven by the occlusive nature of large-bore femoral arterial cannulation, which strictly mandates the prophylactic or early insertion of a distal perfusion catheter. Furthermore, mechanical shear stress and continuous blood exposure to the artificial circuit surface frequently precipitate hemolysis and device thrombosis. This generates a precarious anticoagulation challenge in the intensive care unit; clinicians must continuously balance the prevention of circuit failure against a profoundly amplified risk of catastrophic access-site and systemic hemorrhage. This hemorrhagic hazard is particularly prohibitive in patients cannulated shortly after the failure of systemic thrombolysis. Additionally, prolonged extracorporeal support inherently exposes critically ill patients to significant risks of cannula-related and nosocomial bloodstream infections.

Beyond device-related morbidities, a fundamental limitation of VA-ECMO in high-risk PTE is the profound risk of futile support. In clinical scenarios where the massive pulmonary thrombus fails to undergo endogenous fibrinolysis and the patient is concurrently deemed ineligible for advanced catheter-based or surgical reperfusion, irreversible RV failure may ensue. This trajectory culminates in an inability to wean the patient from mechanical support without a viable long-term exit strategy. Consequently, a multidisciplinary PERT framework is imperative not only to mitigate mechanical complications but also to establish clear, early goals of care and to define strict clinical criteria for both successful decannulation and the withdrawal of futile support [20].

Re-evaluation and VA-ECMO Weaning Strategy

The primary objective of VA-ECMO in the management of high-risk PTE is to serve as a transient bridge to recovery, contingent upon adequate pulmonary reperfusion. Prior to initiating the weaning protocol, objective confirmation of pulmonary vascular recanalization is imperative. This is optimally achieved via follow-up computed tomography pulmonary angiography to document a quantitative reduction in thrombus burden, or through transesophageal echocardiography demonstrating restored antegrade pulmonary arterial flow. Upon confirmation of reperfusion, weaning readiness is assessed utilizing a rigorous multiparametric approach. Clinically, the patient must demonstrate sustained hemodynamic stability, characterized by adequate end-organ perfusion (e.g., normalized lactate clearance) and minimal to absent vasoactive requirements.

During a formal weaning trial—wherein extracorporeal flow is progressively decremented to 1.0–1.5 L/min under contin-

uous hemodynamic monitoring—echocardiography serves as the cornerstone for evaluating RV recovery. Criteria for successful weaning include the normalization of RV chamber dimensions, the resolution of leftward interventricular septal bowing, the absence of McConnell's sign, and the restoration of RV systolic indices toward normal (e.g., TAPSE ≥16–17 mm) [21]. Failure to maintain hemodynamic independence, or the echocardiographic reappearance of acute RV strain during the flow-reduction trial, necessitates the immediate resumption of full extracorporeal support and an urgent re-evaluation for residual or recurrent pulmonary obstruction.

CONCLUSIONS

Acute high-risk PTE is a catastrophic cardiovascular emergency characterized by profound hemodynamic collapse and severe RV strain. Given the prohibitive hemorrhagic risks and surgical morbidities associated with traditional systemic thrombolysis and surgical embolectomy, a gradual transition toward MCS and CBT is underway in clinical practice. Early recognition and the strategic deployment of VA-ECMO provide a valuable rescue strategy, by effectively unloading the failing RV and restoring systemic perfusion to bridge patients to recovery or definitive thrombus extraction.

Nevertheless, the application of this life-saving modality demands a highly sophisticated intensive care approach. Optimizing clinical outcomes relies upon tailored ventilatory strategies, continuous echocardiographic monitoring to strictly guide resuscitation, and the vigilant mitigation of severe device-related morbidities, most notably limb ischemia and amplified hemorrhagic complications. Furthermore, successful decannulation dictates a rigorous, multidisciplinary approach to objectively confirm pulmonary reperfusion and assess RV recovery during protocolized weaning trials. Future large-scale, prospective registries and randomized controlled trials are imperative to refine patient selection algorithms, standardize the optimal timing for these advanced interventions, and improve overall survival in this critically ill population.

CONFLICT OF INTEREST

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ORCID

Woo Jin Jang https://orcid.org/0000-0002-7421-308X
Ik Hyun Park https://orcid.org/0000-0001-7264-6087
Gyu Tae Park https://orcid.org/0009-0000-6653-4214
Tae Wan Kim https://orcid.org/0000-0001-9067-4129
Won-Young Kim https://orcid.org/0000-0002-6038-9818

AUTHOR CONTRIBUTIONS

Conceptualization: WJJ, IHP, GTP, TWK, WYK. Methodology: WJJ. Formal analysis: WJJ. Data curation: WJJ, IHP, GTP, TWK, WYK. Visualization: WJJ, GTP. Project administration: WJJ, IHP. Writing - original draft: WJJ, IHP. Writing - review & editing: WJJ. All authors read and agreed to the published version of the manuscript.

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