Controversies in extracorporeal membrane oxygenation: Immediate versus watchful waiting for venoarterial extracorporeal membrane oxygenation venting

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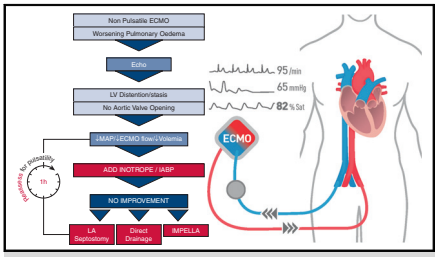
In patients in cardiogenic shock (CS), mechanical circulatory support (MCS) may improve systemic perfusion without increasing myocardial work. Restoration of oxygen delivery with reduced myocardial oxygen demand favors myocardial recovery. The hemodynamic effects of MCS have been extensively studied and elegantly reviewed previously. Pressure-volume loop simulations suggest that although venoarterial extracorporeal membrane oxygenation (VA-ECMO) may improve systemic perfusion, accompanying consequences on left ventricular (LV) dynamics may be detrimental.[1] In severe LV dysfunction, this has significant implications, potentially resulting in LV distension and pulmonary congestion.[2] Myocardial recovery may be limited, negatively affecting long-term prognosis.[2] A recent series reported 22% subclinical LV distension (LVD) and 7% overt LVD requiring immediate decompression. Myocardial recovery was found to be inversely related to the degree of LVD.[3] Increased pulmonary congestion after VA-ECMO initiation is similarly associated with poorer prognosis.[4] Other MCS devices may have superior LV unloading properties, but do not offer comparable cardiopulmonary support. The purpose of this article is to review strategies to prevent, recognize, and treat LVD, a key to maximizing the benefits of VA-ECMO.



Clinical features leading to LV vent insertion in patients on VA-ECMO.

CENTRAL MESSAGE

Controversies surrounding indications, timing, and LV venting modalities require randomized trial data and LV overload definition standardization.

PATHOPHYSIOLOGY OF LEFT VENTRICULAR DISTENSION

In VA-ECMO, blood is drained from the venae cavae or the right atrium, pumped through an oxygenator and reinfused into the arterial circulation. Blood going through the circuit thereby bypasses the right ventricle (RV), the pulmonary circulation, the left atrium (LA), and the LV. ECMO can completely support blood oxygenation, decarboxylation, and circulation, even in the absence of cardiopulmonary function. However, in the setting of incomplete LV unloading, a severely impaired LV may stop ejecting because of increased afterload caused by normalized systemic pressures. This may lead to blood stasis in the native heart-lung unit, with significant thrombotic risk.

If the patient's venous return exceeds the rate at which the blood is drained into the ECMO circuit, blood enters the RV and is ejected through the pulmonary circulation. LVD occurs when residual transpulmonary blood flow and bronchial venous return exceed LV ejection. LV end-diastolic pressure increases, leading to progressive LV dilatation, pulmonary congestion, and edema. Functional mitral regurgitation secondary to LV dilatation further aggravates this phenomenon. Significant aortic valve regurgitation will also contribute to LV distension. As LV wall tension increases, myocardial oxygen consumption increases, compromising subendocardial perfusion. This further impairs LV performance and potential myocardial recovery.

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The main determinants of detrimental LVD during VA-ECMO are LV afterload, LV contractility, LV preload, left-sided valve integrity, and left-right ventricular interactions.[5]

IDENTIFICATION OF LEFT VENTRICULAR DISTENSION

In the absence of a standardized definition, definitively diagnosing LVD can be elusive.[5] Direct measurement LA or LV end-diastolic pressure is rarely performed in VA-ECMO. Thus, clinicians rely on indirect signs of high LV pressures and distension (Table 1). Reduced arterial pulsatility is one of the earliest signs of LV overload on ECMO; a pulse pressure less than 10 mm Hg is usually considered worrisome. Absence of the dicrotic notch on the arterial line pressure waveform indicates failure of aortic valve opening, which also results in LVD. If a pulmonary artery catheter is in place, a progressive increase in pulmonary artery diastolic and occlusion (wedge) pressures may be observed. Such trends should prompt further investigation with an echocardiogram. Typical findings include increased LV end-diastolic diameter, increased E/e' ratio, spontaneous echocontrast or thrombus in the LV, and intermittent or absent opening of the aortic valve. Significant functional MR may also be present. Later signs of LVD include overt pulmonary edema with significant hypoxemic respiratory failure and characteristic radiographic findings. Severe LVD may result in refractory ventricular arrhythmias. Clinicians should not delay to intervene until such signs and symptoms develop because early identification of LV overload provides more management options, including less-invasive ones.

MEDICAL MANAGEMENT OF LEFT VENTRICULAR DISTENSION

The objectives of medical management of LVD are to (1) increase LV contractility, (2) decrease LV preload, and (3) minimize LV afterload. Inotropes can be used to improve contractility and promote aortic valve opening and LV ejection. Maintaining LV ejection is ideal to promote LV decompression. The initial goal is to achieve adequate intravascular volume status to restore end-organ perfusion. Once such resuscitation is successfully accomplished, volume contraction may be sought. Positive end-expiratory pressure can be increased to reduce both LV preload and afterload, either with invasive or noninvasive mechanical ventilation. In patients with significant residual transpulmonary blood flow, it may be effective to increase extracorporeal blood flow (ECBF) to improve RV drainage. If this cannot be achieved by increasing pump speed, optimizing drainage cannula position or adding a second drainage cannula can be considered.[6] Reduced LV filling decreases LV diameter, resulting in reduced LV wall tension, one of the components of afterload.[7] This strategy will have to balance increased arterial flow, which increases LV afterload and possible LVD.

LV afterload can be decreased by reducing the mean arterial pressure. This is done by decreasing vasopressor doses

TABLE 1. Parameters to consider for left ventricular unloading (at any time during venoarterial extracorporeal membrane oxygenation)

	Parameters
Medical condition	Etiology of cardiogenic shock: acute myocardial infarction, chronic decompensated heart failure Myocardial stunning (post-ECPR) Congestive heart failure before VA-ECMO
Hemodynamic - Invasive arterial line - PA catheter - Conductance catheter	Low systemic arterial pulsatility (<10-30 mm Hg) High mean arterial pressure without pressors High diastolic pulmonary artery pressure (>20 mm Hg) High capillary wedge pressure (>25 mm Hg) High left atrial pressure High end-diastolic left ventricular pressure Need for high-flow support (>4 L/min)
Echocardiographic - TTE - TEE	No aortic valve opening Aortic regurgitation more than mild-moderate Absence of LV contractility Blood stagnation in left-sided cavities (smoke sign) Dilated left ventricle High LV filling pressure Significant mitral regurgitation
Radiographic	Pulmonary edema or signs of pulmonary congestion
Respiratory	Hypoxemia under VA-ECMO High end-tidal CO2 (high transpulmonary flow) Blood-tinged secretions in endotracheal tube

ECPR, Extracorporeal cardiopulmonary resuscitation; *VA-ECMO*, venoarterial extracorporeal membrane oxygenation; *PA*, pulmonary artery; *TTE*, transthoracic echocardiogram, *TEE*, transesophageal echocardiography; *LV*, left ventricular.

or introducing vasodilators. In patients with minimal to no residual transpulmonary blood flow, normal perfusion markers (lactates, SVO_2) and high ECBF relative to body surface area, the minimal ECBF required to provide sufficient end-organ perfusion, should be targeted. A higher than required ECBF increases arterial input impedance unnecessarily and demands operation at higher intravascular volume. Reducing ECBF often allows greater intravascular volume depletion to be tolerated. This may not always be possible early on, because high vasopressor requirements and persistent signs of hypoperfusion may necessitate higher ECMO flow rates. Excessive ECBF reduces LV filling and may adversely impact LV contractility and ejection. These interventions can be performed and titrated under echocardiographic monitoring to visualize the effect on aortic valve opening and LV size. Expired CO_2 may be used to track changes in transpulmonary blood flow.

INTRA-AORTIC BALLOON PUMP

The intra-aortic balloon pump (IABP) is used to reduce afterload and promote aortic valve opening. It has been demonstrated to be effective in reducing central venous pressure, pulmonary artery occlusion pressure, and pulmonary edema in patients on VA-ECMO. Its main advantages are inexpensive cost, wide availability, familiarity, possible bedside insertion, and relatively low complication rate. The main concern is decreased cerebral blood flow by interrupting retrograde aortic diastolic blood flow in some patients on peripheral VA-ECMO. Moreover, in the setting of relatively low systemic pressures and reduced pulsatility, balloon positioning close to visceral vessels may lead to significant abdominal organ hypoperfusion, especially in older patients with significant peripheral vascular disease. Despite these limitations and limited LV unloading effect,[8] a recent meta-analysis reports the use of IABP to be associated with improved patient survival in VA-ECMO.[9]

ATRIAL SEPTOSTOMY

By creating a breach in the interatrial septum, a left-to-right shunt can indirectly decompress the LA via the ECMO drainage cannula placed in the RA. The procedure results in a mean decrease of 15 mm Hg in LA pressure.[10] This has been shown to reduce inotropic support, improve LV function, and facilitate ECMO weaning, with a low complication rate.[11] Left atrial septostomy decompresses the LA, only indirectly unloading the LV.

IMPELLA

The Impella (AbioMed, Danvers, Mass) is an axial flow pump inserted under fluoroscopic guidance via central or peripheral access and placed across the aortic valve. Multiple sizes are available, providing blood flows ranging from 2.5 L/min to 6 L/min. By directly suctioning blood from the LV, superior LV decompression is possible. The Impella can reduce LVD and stasis, and improve systemic blood flow, myocardial oxygen imbalance, pulmonary congestion, and RV performance. The Impella also allows reduction in VA-ECMO flow, facilitating RV function assessment, and eventually staged weaning. The main drawback of the device is cost and migration tendency, with ensuing hemolysis.

A multicenter international retrospective study compared 255 patients treated with VA-ECMO in combination with Impella with 255 propensity-matched patients supported with VA-ECMO alone. The authors reported the use of Impella with VA-ECMO to be associated with decreased mortality (hazard ratio, 0.79; 95% CI, 0.63-0.98; $P = .03$), despite an increased rate of complications such as bleeding, hemolysis, ischemic complications, and renal replacement therapy (RRT).[12] In a smaller propensity-matched cohort, the Impella in combination with ECMO was associated with a lower hospital mortality (47% vs 80%, $P < .001$) despite an increased rate of RRT (48% vs 19%, $P = .02$) and hemolysis (76% vs 33%, $P = .004$).[13] LV unloading would appear to be paramount to LV recovery and VA-ECMO survival. Although these data are intriguing, reconciling additional complications on VA-ECMO with improved survival may also reflect selection bias.

DECOMPRESSION CANNULAE

A drainage cannula can be added in any part of the right to left circulation to unload the LV: pulmonary artery (PA), pulmonary veins (PVs), LA, LV. The additional drainage cannula is connected to the venous drainage limb of the circuit. When LVD occurs during VA-ECMO initiation in the operating room, it is efficient to vent the LV via the apex or the LA via the right superior PV. In case of biventricular failure, a PA vent can decompress both right ventricular afterload and LV preload. Monitoring of the blood flow in the PA vent and the flow in the native pulmonary bed is important to avoid pulmonary vascular bed thrombosis in case of absence of pulmonary flow, especially if the patient is not anticoagulated.

Various other surgical techniques have been described outside of this common scenario, including direct LA or pulmonary venous drainage through a standard or mini thoracotomy or direct transapical LV vent placement through left minithoracotomy. Direct LV venting on the ECMO circuit may provide a better reduction in pulmonary diastolic pressure compared with Impella.[14] It is also a less-expensive option. Additional drainage cannulas can be added percutaneously. Most commonly used approaches include transeptal LA drainage or pulmonary artery drainage. For percutaneous pulmonary artery venting, a 10F to 15F catheter is inserted via the right internal jugular vein into the pulmonary artery[15] under fluoroscopic guidance. Major complications include cardiac or pulmonary artery perforation during the insertion and subsequent cannula

dislodgement. Percutaneous decompression cannula are simple and convenient options in patients on peripheral VA-ECMO, but there is dislodgement risk. Obtaining optimal drainage and decompression may be limited by smaller catheter sizes compared with direct surgical venting.

INFLUENCE OF THE ARTERIAL CANNULATION SITE ON LEFT VENTRICULAR DISTENSION

Axillary Arterial Return Cannula

Axillary cannulation, with its anterograde aortic blood outflow, could attenuate the impact of VA-ECMO on LV afterload. In an elegant study, using Doppler evaluation of the descending thoracic aortic blood flow, Andrei and colleagues[16] demonstrated that in an axillary-femoral configuration, the ECMO flow and the native LV outflow were additive. In comparison, in a femoro-femoral configuration, ECMO flow and the native LV outflow were competitive. In a series of 174 patients who underwent right axillary VA-ECMO, only 9 patients (5%) required LV venting.[17] However, axillary cannulation requires more complex surgical skills, making it particularly challenging to use in the setting of cardiac arrest or salvage cannulation. Further, hyperemia in the upper extremity may be problematic with prolonged support, resulting in compartment syndrome.

Central Cannulation

Central cannulation may improve cardiac drainage and anterograde flow in the proximal aorta and prevent "Harlequin syndrome." Although we could not find direct hemodynamic comparisons in patients on peripheral versus central VA-ECMO, computational geometrical model suggest differences in blood flow distribution and aortic wall shear stress.[18] However, central cannulation in the setting of postcardiotomy cardiogenic shock (PCCS) is associated with higher rates of bleeding and acute kidney injury requiring hemofiltration. A recent meta-analysis found no difference in overall survival compared with peripheral cannulation.

CHALLENGING CLINICAL SCENARIOS

Specific Considerations in Venoarterial Extracorporeal Membrane Oxygenation for Postcardiotomy Cardiogenic Shock

PCCS requiring VA-ECMO is associated with a high rate of mortality and morbidity,[19-21] and such patients are often excluded from ECMO trials.[12] Several factors may impact the decision-making pertaining to LV unloading in the context of PCCS: (1) availability of direct access to the cardiac chambers or great vessels; (2) frequent bleeding and need for chest reexploration (43%); (3) availability of alternative cannulation sites (LV apex, pulmonary artery vent, left atrium vent through the right superior pulmonary vein, LV vent through the mitral valve); and (4) timing of VA-ECMO insertion (before leaving the operative room or later). Recent literature suggests that an IABP is used in 12% to 100% of cases, invasive LV venting is performed in 8% of cases (pulmonary vein 6%, apex 1%), and Impella in 0.6% of the cases of VA-ECMO for PCCS.[19-21] The low rate of active LV venting in VA-ECMO for PCCS may be explained by the high rate of bleeding, leading to intravascular volume depletion, which is less likely to be associated with LV distension, at least in the acute setting, or by the high rate of IABP use. Central cannulation, performed in 30% of cases, may offer the advantage of better venous drainage and antegrade aortic flow.[22] LV venting and individual venting strategies were not associated with survival or myocardial recovery in the setting of PCCS.[19-21] Whether potential benefits in terms of improved LV recovery outweigh the additional bleeding risk remains to be determined in this particular patient population.

Mitral Valve Prosthesis in the Setting of Venoarterial Extracorporeal Membrane Oxygenation

Mitral prosthesis thrombosis (tissue and mechanical) in VA-ECMO is a devastating complication, almost invariably resulting in intracardiac thrombosis, pulmonary edema, multiorgan failure, stroke, and death. Anecdotal cases of successful mitral prosthesis removal have been described.[23] In a series of 63 patients with PCCS supported with VA-ECMO after mitral valve replacement, hospital mortality was 72%, intracardiac thrombus being one of the most common causes of death. LV venting was barely used. The authors concluded that clinicians should try to use partial ECMO support to allow some antegrade flow through the mitral valve if tolerated, or use LV venting if full flow is required.

EVIDENCE-BASED APPROACH TO LEFT VENTRICULAR UNLOADING

A recent meta-analysis suggested that LV unloading could improve the rate and the time of myocardial recovery.[24] However, this is still to be confirmed in randomized clinical trials (RCTs) in which myocardial recovery should be an important outcome. In the absence of RCTs, formulating recommendations pertaining to LV unloading is challenging. Even the exact incidence of LVD in patients on VA-ECMO is unclear in the absence of a standardized definition. Moreover, the impact of varying degrees of LVD severity on myocardial recovery or systemic complications is not well understood. These issues greatly limit our ability interpret the available observational literature. The REVERSE trial (NCT03431467) is currently randomizing patients with CS to early VA-ECMO with Impella versus VA-ECMO alone. It should provide some guidance in the future. While waiting for the results, we will try to address the 2 most pressing questions:

Do all Patients on Venoarterial Extracorporeal Membrane Oxygenation Require Left Ventricular Unloading?

A recent systematic review with meta-analysis found mechanical LV unloading to be associated with improved survival for VA-ECMO for patients in cardiogenic shock.[25] However, in the ARREST trial,[26] the only RCT published on VA-ECMO, no patient underwent LV unloading. Moreover, the reported use of mechanical LV unloading varies widely in the literature, ranging from 5% to 50%.[17,27,28] This probably reflects heterogeneity in definitions, patient populations, and management strategies between studies. Belohlavek and colleagues[28] described that although some LV overload may occur in up to 70% of patients on VA-ECMO, urgent decompression is undertaken in only approximately 10% of patients, whereas an additional 20% might need a delayed unloading intervention. These studies likely translate the current perception that routine mechanical LV unloading is probably not mandatory in all patients.

We also think that the approach to LV unloading should be individualized. First, LV unloading is probably unnecessary in the absence of LV failure, when, for instance, ECMO is used in the setting of isolated RV failure or massive pulmonary embolism. Conversely, in patients with significant LV failure, clinicians should seek to unload the LV as soon as possible.

Whether or not mechanical unloading should be used in all such patients remains a matter of debate. Mechanical LV unloading is associated with an increased rate of complications that might offset its benefits. Bleeding, access site ischemia, RRT, and abdominal compartment syndrome requiring a laparotomy are more common in patients treated with Impella and VA-ECMO compared with VA-ECMO alone.[12] In our experience, with an appropriately sized and positioned venous drainage cannula that allows efficient RV drainage, a carefully titrated fluid strategy, a low blood pressure target, and a judicious use of inotropes, the LV can be "medically" unloaded in most patients. Such an approach requires diligent monitoring for signs of LVD to be able to rapidly intervene and avoid the deleterious effects of overt distension (Figure 1). For the patients in more LV failure, in whom LV ejection is not achievable, the need for further support and LV unloading should be recognized early.

When Should Mechanical Left Ventricular Unloading Be Used?

It is unclear whether LV venting strategies should be used immediately at the time of cannulation or added in select patients demonstrating signs of LVD after a period of observation on VA-ECMO alone. In an early study by Schrage and colleagues,[29] there was a trend toward a lower mortality in patients with concomitant VA-ECMO and Impella

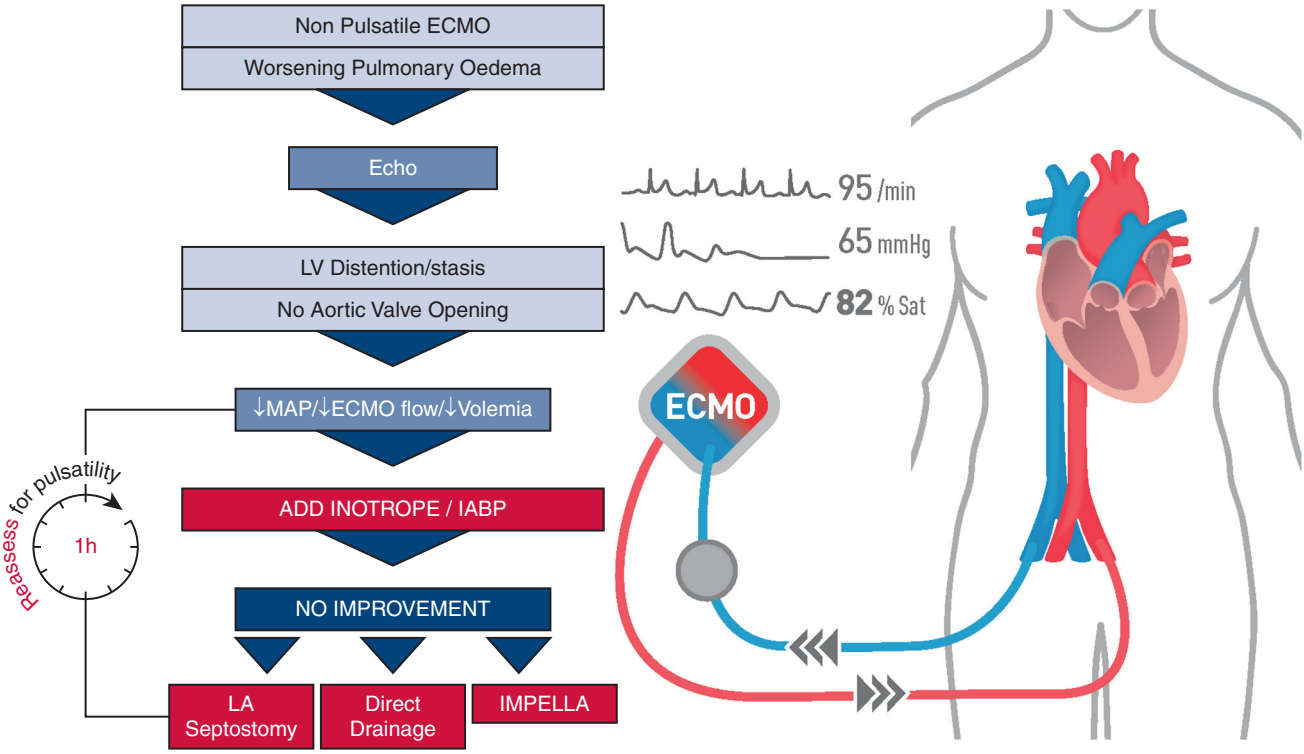

FIGURE 1. Clinical features leading to LV vent insertion in patients on VA-ECMO. *ECMO*, Extracorporeal membrane oxygenation; *LV*, left ventricular; *Sat*, saturation; *MAP*, mean arterial pressure; *IABP*, intra-aortic balloon pump; *LA*, left atrial.

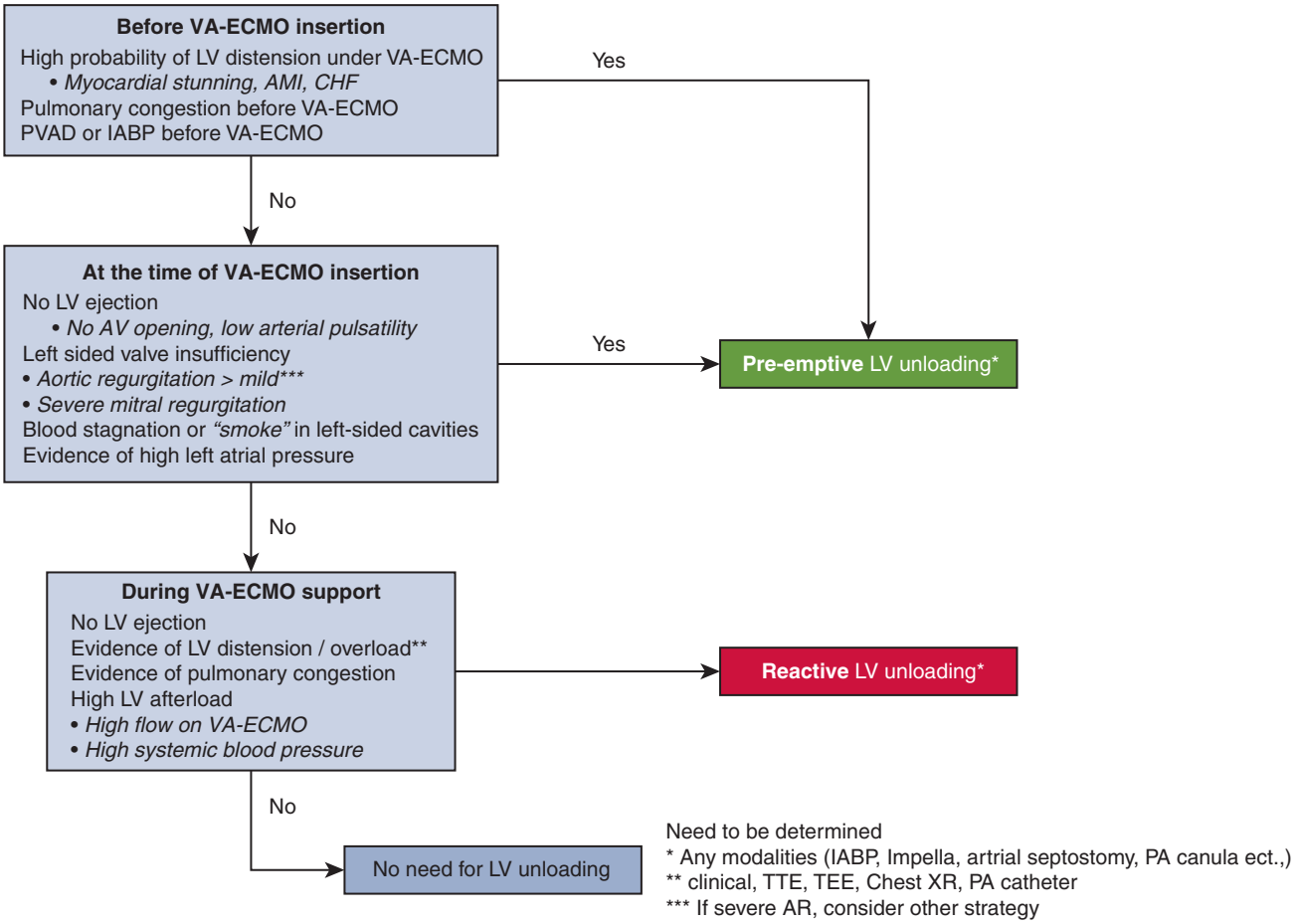

FIGURE 2. Considerations for the timing of LV unloading in patients on VA-ECMO. *VA-ECMO*, Venoarterial extracorporeal membrane oxygenation; *LV*, left ventricle; *AMI*, acute myocardial infraction; *CHF*, congestive heart failure; *PVAD*, percutaneous ventricular assist devices; *IABP*, intra-aortic balloon pump; *AV*, atrioventricular; *PA*, pulmonary artery; *TTE*, transthoracic echocardiogram; *TEE*, transesophageal echocardiography; *XR*, x-ray; *AR*, aortic regurgitation.

insertion (61%) compared with delayed Impella insertion (80%). Na and colleagues[30] found prophylactic transseptal drainage to be associated with a lower mortality compared to an as-needed approach. In a more recent study, Schrage and colleagues[12] found that the effect of adding an Impella to VA-ECMO on 30-day mortality might be significantly better when inserted early (HR, 0.76; 95% CI, 0.60-0.97; $P = .03$) but less clearly when delayed (HR, 0.77; 95% CI, 0.51-1.16; $P = .22$). In a retrospective single-center study, Char and colleagues[27] found no difference in survival between patients with pre-ECMO unloading (at the time of ECMO insertion or before ECMO) versus reactive unloading (in response to clinical criteria).

If a prophylactic venting strategy is to be adopted, data on factors predicting overt LVD are needed to facilitate patient selection. If a delayed strategy is adopted, a proactive approach with close monitoring of signs of LV overload should be advocated (Figure 2). In patients who do not respond to medical therapy, IABP is probably the least invasive mechanical unloading strategy.[31] More invasive or costly strategies may be used when IABP is contraindicated or insufficient. When this is the case, the approach should be selected on the basis of the clinical scenario (eg, postcardiotomy vs nonsurgical patient, isolated LV failure vs biventricular failure), patient characteristics (eg, available vascular access), candidacy for durable LVAD or transplant, center experience, and device availability.

CONCLUSIONS

The available literature cannot support strong evidence-based recommendations concerning the optimal strategy for LV unloading in patients receiving VA-ECMO. Controversies surrounding indications, timing, and LV venting modalities require randomized controlled trial data and standardization of LV overload definition and severity assessment. While waiting for further trial results, we advocate for an individualized approach to LV unloading.

Conflict of Interest Statement

The authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

References

1. Pahuja M, Schrage B, Westermann D, Basir MB, Garan AR, Burkhoff D. Hemodynamic effects of mechanical circulatory support devices in ventricular septal defect–results from a computer simulation model. *Circ Heart Fail*. 2019;12:e005981.
2. Donker DW, Brodie D, Henriques JPS, Broomé M. Left ventricular unloading during veno-arterial ECMO: a review of percutaneous and surgical unloading interventions. *Perfusion*. 2019;34:98-105.
3. Truby LK, Takeda K, Mauro C, Yuzefpolskaya M, Garan AR, Kirtane AJ, et al. Incidence and implications of left ventricular distention during venoarterial extracorporeal membrane oxygenation support. *ASAIO J*. 2017;63:257-65.
4. Distelmaier K, Wiedemann D, Lampichler K, Toth D, Galli L, Haberl T, et al. Interdependence of VA-ECMO output, pulmonary congestion and outcome after cardiac surgery. *Eur J Intern Med*. 2020;81:67-70.
5. Singh G, Hudson D, Shaw A. Medical optimization and liberation of adult patients from VA-ECMO. *Can J Cardiol*. 2020;36:280-90.
6. Lo Coco V, Swol J, De Piero ME, Massimi G, Chiarini G, Broman LM, et al. Dynamic extracorporeal life support: a novel management modality in temporary cardio-circulatory assistance. *Artif Organs*. 2021;45:427-34.
7. Vest AR, Heupler FA. Afterload. In: Anwaruddin S, Martin JM, Stephens JC, Askari AT, eds. *Cardiovascular Hemodynamics*. Totowa, NJ: Humana Press; 2013:29-51.
8. Donker DW, Brodie D, Henriques JPS, Broomé M. Left ventricular unloading during veno-arterial ECMO. *ASAIO J*. 2019;65:11-20.
9. Al-Fares AA, Randhawa VK, Englesakis M, McDonald MA, Nagpal AD, Estep JD, et al. Optimal strategy and timing of left ventricular venting during veno-arterial extracorporeal life support for adults in cardiogenic shock: a systematic review and meta-analysis. *Circ Heart Fail*. 2019;12:e006486.
10. Baruteau AE, Barnetche T, Morin L, Jalal Z, Boscamp NS, Le Bret E, et al. Percutaneous balloon atrial septostomy on top of venoarterial extracorporeal membrane oxygenation results in safe and effective left heart decompression. *Eur Heart J Acute Cardiovasc Care*. 2016;7:70-9.
11. Alhussein M, Osten M, Horlick E, Ross H, Fan E, Rao V, et al. Percutaneous left atrial decompression in adults with refractory cardiogenic shock supported with veno-arterial extracorporeal membrane oxygenation. *J Card Surg*. 2017;32:396-401.
12. Schrage B, Becher PM, Bernhardt A, Bezerra H, Blankenberg S, Brunner S, et al. Left ventricular unloading is associated with lower mortality in cardiogenic shock patients treated with veno-arterial extracorporeal membrane oxygenation: results from an international, multicenter cohort study. *Circulation*. 2020;142:2095-106.
13. Pappalardo F, Schulte C, Pieri M, Schrage B, Contri R, Soeffker G, et al. Concomitant implantation of Impella® on top of veno-arterial extracorporeal membrane oxygenation may improve survival of patients with cardiogenic shock. *Eur J Heart Fail*. 2017;19:404-12.
14. Tepper S, Masood MF, Garcia MB, Pisani M, Ewald GA, Lasala JM, et al. Left ventricular unloading by Impella device versus surgical vent during extracorporeal life support. *Ann Thorac Surg*. 2017;104:861-7.
15. Avalli L, Maggioni E, Sangalli F, Favini G, Formica F, Fumagalli R. Percutaneous left-heart decompression during extracorporeal membrane oxygenation: an alternative to surgical and transeptal venting in adult patients. *ASAIO J*. 2011;57:38-40.
16. Andrei S, Tran-Dinh A, Provenchere S, Lortat-Jacob B, Ghodbane W, Montravers P, et al. A quantified description of the interactions between the native cardiovascular system and femoro-femoral versus femoro-axillary extracorporeal life support using descending thoracic aorta velocity time integral. *Artif Organs*. 2019;43:647-55.
17. Pisani A, Braham W, Brega C, Lajmi M, Provenchere S, Danial P, et al. Right axillary artery cannulation for venoarterial extracorporeal membrane oxygenation: a retrospective single centre observational study. *Eur J Cardiothorac Surg*. 2021;59:601-9.
18. Gu K, Zhang Y, Gao B, Chang Y, Zeng Y. Hemodynamic differences between central ECMO and peripheral ECMO: a primary CFD study. *Med Sci Monit*. 2016;22:717-26.
19. Lorusso R, Raffa GM, Alenizy K, Sluijpers N, Makhoul M, Brodie D, et al. Structured review of post-cardiotomy extracorporeal membrane oxygenation: part 1–adult patients. *J Heart Lung Transplant*. 2019;38:1125-43.
20. Biancari F, Dalen M, Fiore A, Ruggieri VG, Saeed D, Jonsson K, et al. Multicenter study on postcardiotomy venoarterial extracorporeal membrane oxygenation. *J Thorac Cardiovasc Surg*. 2020;159:1844-54.e6.
21. Biancari F, Perrotti A, Dalen M, Guerrieri M, Fiore A, Reichart D, et al. Meta-analysis of the outcome after postcardiotomy venoarterial extracorporeal membrane oxygenation in adult patients. *J Cardiothorac Vasc Anesth*. 2018;32:1175-82.
22. Rastan AJ, Dege A, Mohr M, Doll N, Falk V, Walther T, et al. Early and late outcomes of 517 consecutive adult patients treated with extracorporeal membrane oxygenation for refractory postcardiotomy cardiogenic shock. *J Thorac Cardiovasc Surg*. 2010;139:302-11.
23. Sandoval E, Ascaso M, Pereda D, Quintana E. Extracorporeal membrane oxygenation and mechanical valves: an unfriendly relationship. *J Thorac Cardiovasc Surg*. 2018;156:e103-5.
24. Kowalewski M, Malvindi PG, Zielinski K, Martucci G, Slomka A, Suwalski P, et al. Left ventricle unloading with veno-arterial extracorporeal membrane oxygenation for cardiogenic shock. Systematic review and meta-analysis. *J Clin Med*. 2020;9:1039.
25. Russo JJ, Aleksova N, Pitcher I, Couture E, Parlow S, Faraz M, et al. Left ventricular unloading during extracorporeal membrane oxygenation in patients with cardiogenic shock. *J Am Coll Cardiol*. 2019;73:654-62.
26. Yannopoulos D, Bartos J, Raveendran G, Walser E, Connett J, Murray TA, et al. Advanced reperfusion strategies for patients with out-of-hospital cardiac arrest and refractory ventricular fibrillation (ARREST): a phase 2, single centre, open-label, randomised controlled trial. *Lancet*. 2020;396:1807-16.
27. Char S, Fried J, Melehy A, Mehta S, Ning Y, Kurlansky P, et al. Clinical efficacy of direct or indirect left ventricular unloading during venoarterial extracorporeal membrane oxygenation for primary cardiogenic shock. *J Thorac Cardiovasc Surg*. June 18, 2021 [Epub ahead of print].
28. Belohlavek J, Hunziker P, Donker DW. Left ventricular unloading and the role of ECpella. *Eur Heart J Suppl*. 2021;23(Suppl A):A27-34.
29. Schrage B, Burkhoff D, Rübsamen N, Becher PM, Schwarzl M, Bernhardt A, et al. Unloading of the left ventricle during venoarterial extracorporeal membrane oxygenation therapy in cardiogenic shock. *JACC Heart Fail*. 2018;6:1035-43.
30. Na SJ, Yang JH, Yang JH, Sung K, Choi JO, Hahn JY, et al. Left heart decompression at venoarterial extracorporeal membrane oxygenation initiation in cardiogenic shock: prophylactic versus therapeutic strategy. *J Thorac Dis*. 2019;11:3746-56.
31. Lorusso R, Whitman G, Milojevic M, Raffa G, McMullan DM, Boeken U, et al. 2020 EACTS/ELSO/STS/AATS expert consensus on post-cardiotomy extracorporeal life support in adult patients. *Ann Thorac Surg*. 2021;111:327-69.

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