Medical Management and Mechanical Circulatory Support Escalation in Right Ventricular Failure

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KEYWORDS

• Right ventricular failure • Cardiogenic shock • Mechanical circulatory support
• Hemodynamic assessment • RV–PA coupling • Pulmonary hypertension

KEY POINTS

- Right ventricular failure is a distinct and underrecognized condition associated with high morbidity and mortality.
- Clinical presentation differs from left ventricular failure, with predominant venous congestion and reduced left ventricular preload.
- Diagnosis relies on imaging and invasive hemodynamics, including central venous pressure/pulmonary capillary wedge pressure ratio and pulmonary artery pulsatility index.
- Management focuses on optimizing preload, afterload, contractility, and rhythm.
- Early, physiology-guided escalation to mechanical circulatory support is critical in refractory shock.

Right ventricular (RV) failure is a distinct clinical entity with a significant impact on morbidity and mortality across diverse etiologies. Traditionally, the term "congestive heart failure" de facto refers to left ventricular (LV) failure. However, LV dysfunction can progress to involve the RV (biventricular failure), or a patient may develop primary RV failure (RVF). Recognition of RV failure can be challenging and is often quite delayed, as many clinical and laboratory features are synonymous with and often occurring in the setting of LV failure. The presence of RV failure as a feature or driver of "congestive heart failure" is far more than semantic, however. In a broad range of routinely encountered pathologies including acute myocardial infarction (MI), pulmonary embolism (PE), acute decompensated heart failure (AHDF) and cardiogenic shock (CS), the presence of RV dysfunction or failure is universally associated with higher morbidity and mortality.[1–4] Considering the significantly elevated risk that RV failure indicates in our patients, the prompt identification of RV failure and rapid deployment of appropriate management guided by physiologic principles is critical. In this article, we will highlight the diagnostic challenges and therapeutic complexities unique to RV failure, distinguishing it from LV failure. In addition, we will provide a structured, practical approach to medical management and mechanical circulatory support (MCS) strategies for various clinical presentations of RV failure.

Key morphologic differences between the LV and RV: The LV is a thick-walled, conical muscular structure capable of generating systemic arterial pressure and maintaining blood flow to the high resistance systemic circulation. Failure of the

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Abbreviations

ADHF	acute decompensated heart failure
CS	cardiogenic shock
CTEPH	chronic thromboembolic pulmonary hypertension
CVP	central venous pressure
JVP	jugular venous pressure
LV	left ventricular
MCS	mechanical circulatory support
MI	myocardial infarction
PA	pulmonary artery
PAPi	pulmonary artery pulsatility index
PCWP	pulmonary capillary wedge pressure
PE	pulmonary embolism
RA	right atrial
RV	right ventricular

left-sided pump results in decreased forward flow, increased filling pressure (left ventricular end diastolic pressure), and resultant pulmonary congestion—causing typical symptoms of orthopnea and paroxysmal nocturnal dyspnea. In contrast, the RV is a thin-walled, crescentic-shaped structure which is more compliant by design—and capable of accommodating volume more effectively, while pumping blood to the low resistance pulmonary circulation.[5] Like its counterpart, RV becomes dysfunctional when contractility is affected by ischemia or myocardial inflammation. However, due to being thin walled—when there is a rapid increase in RV afterload it results in RV–PA (pulmonary artery) uncoupling leading to RV failure[6] (**Fig. 1**).

CLINICAL PRESENTATION OF RIGHT VENTRICULAR FAILURE

In contrast to symptoms of pulmonary congestion (orthopnea, paroxysmal nocturnal dyspnea) from left heart failure, RV failure manifests as signs of venous hypertension and reduced LV preload.[7,8] Peripheral edema in dependent areas, progressive over weeks to months and resultant weight gain are the most common presenting symptoms. Hepatic venous congestion presents as abdominal fullness, bloating, and right upper quadrant discomfort. Congestion of the gut results in early satiety and nausea. Exertional syncope is a symptom of advanced RV failure resulting from a significant reduction in LV preload.

Clinical signs include elevated jugular venous pressure (JVP), pitting edema, ascites, hepatomegaly, and positive hepatojugular reflux. Split S2 or loud P2 may be present if pulmonary hypertension (PH) is present. RV S3 and holosystolic murmur of tricuspid regurgitation can be heard. *Kussmaul sign* is one of the classic bedside findings that support RV failure or decreased RV compliance. During inspiration, there is an increase in venous return to the right side of the heart which can be accommodated by a normal RV, but a failing or stiff RV cannot accommodate this and the resultant increase in right atrial (RA)/RV pressure is seen as rise in JVP.[9]

Diagnosis of RV failure: In addition to clinical context, the diagnosis of RV failure is made using imaging (echocardiography, cardiac MRI). The severity of RV failure is further stratified by hemodynamic assessment using right heart catheterization (**Table 1**).

In cases where imaging is equivocal or technically difficult, invasive hemodynamics via Swan-Ganz catheter can help differentiate shock phenotypes.[13,14] Moreover, even when RV dysfunction is apparent, invasive hemodynamics help in titration or selection of therapy.

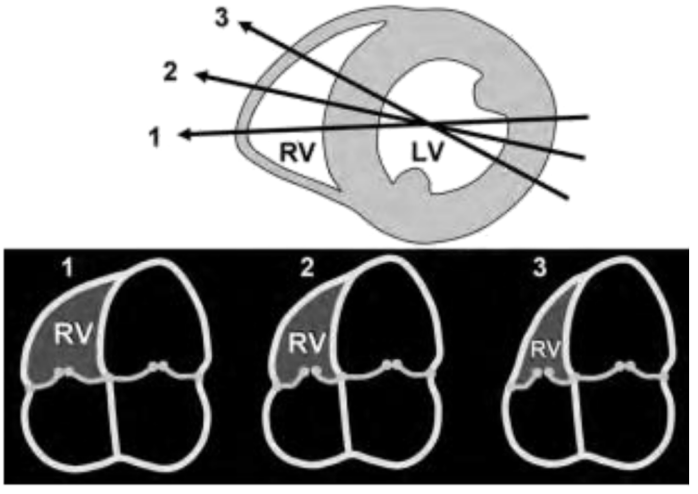

Fig. 1. Comparison of RV and LV structure and muscular composition. (Lawrence G. Rudski et al., Guidelines for the Echocardiographic Assessment of the Right Heart in Adults: A Report from the American Society of Echocardiography: Endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography, Journal of the American Society of Echocardiography, 23 (7), 2010, 685-713, https://doi.org/10.1016/j.echo.2010.05.010.)

Table 1
Imaging features of right ventricular failure

Modality	Findings
Echocardiography	• RV dilation • RV systolic function ○ Annular function: reduced TAPSE (<17 mm) or RV S′ velocity (<9.5 cm/s)[10,11] ○ Fractional area change <35% ○ RV strain • RV pressure overload: septal flattening (D-shaped LV) in systole • McConnell sign: decreased free wall contractility with apical sparing[12] • Elevated RA pressure: IVC plethoric and noncollapsing
Computed tomography (CT)	Mostly used to identify right ventricular strain in patients with acute pulmonary embolism. Features suggesting RV strain are: • Contrast reflux in IVC • RV/LV size ratio > 0.9 • Septal bowing toward LV
Cardiac MRI	• Gold standard for assessment of RV function and RV dimensions • Dilated RV – RV end-diastolic volume index > 110 mL/m^2 in males or > 100 mL/m^2 in females • Tissue characterization may help identify etiology—eg, late gadolinium enhancement at RV insertion point seen in PH. • Can be used to calculate shunt fraction when RV dysfunction is attributed to intracardiac shunt (Qp:Qs > 1.5 represents hemodynamically significant left-to-right shunt

Abbreviations: IVC, inferior vena cava; Qp:Qs, intracardiac shunt fraction; TAPSE, tricuspid annular plane systolic excursion.

Differentiation of Isolated Left Ventricular Failure from Right Ventricular Failure

- Central venous pressure/pulmonary capillary wedge pressure (CVP/PCWP) ratio: Primary LV dysfunction results in backward transmission of pressure to pulmonary circulation and RV involvement is secondary—RV preload is normal or only modestly elevated. In contrast, primary RV dysfunction results in a normal PCWP and a markedly elevated CVP[13] (**Table 2**).
- Pulmonary artery pulsatility index (PAPi): With progressive dysfunction, the RV struggles to generate pulmonary pressure and this results in systemic venous congestion. Both these variables are captured in the PAPi[15] which is calculated as (PA systolic – PA diastolic)/CVP. Utility of PAPi in assessment of RV failure has been demonstrated in RVMI,[16] post left ventcricular assist device (LVAD) RV failure[17] and cardiogenic shock phenotyping[15] (**Table 3**).
- RV-PA coupling: the relationship between RV contractility and afterload can be assessed invasively through pressure-volume loops or noninvasively by echocardiographic surrogates such as tricuspid annular plane systolic excursion /PASP ratio. Preserved RV-PA coupling demonstrates the ability of RV to maintain adequate stroke volume despite increased afterload, while uncoupling portends worse outcomes.[18,19]

Optimizing right ventricular performance: the pillars of management

- *Preload:* The RV is more sensitive to both underfilling and overdistension. Contradictory

Table 2
Filling pressures in the failing ventricle(s)

Predominant LV Failure	CVP < 14 mm Hg	PCWP >18 mm Hg	CVP/PCWP <0.5
Predominant RV failure	CVP > 14 mm Hg	PCWP < 18 mm Hg	CVP/PCWP >0.86
Biventricular failure	CVP > 14 mm Hg	PCWP > 18 mm Hg	CVP/PCWP >0.86

Table 3
Critical cutoffs in the pulmonary artery pulsatility index

PAPi > 2	Normal RV Function
PAPi < 1	Severe RV failure in acute myocardial infarction related cardiogenic shock
PAPi < 1.85	RV failure post LVAD implantation

to traditional teaching (in the context of RVMI or acute PE): more preload is not always better.[13,20] Optimization of preload involves careful assessment of CVP and analyzing where the RV is operating on its Frank-Starling curve. The goal is not a normal CVP, but the lowest CVP (5–10 mm Hg) that maintains adequate cardiac output and organ perfusion. When CVP is low (<5 mm Hg), volume loading may be appropriate but should be small, incremental, and reassessed frequently. Elevated CVP greater than 14 mm Hg indicates RV congestion and necessitates decongestion via diuresis or ultrafiltration

- *Afterload:* As discussed before, the RV is poorly equipped to tolerate acute increase in afterload.[21,22] Decreasing RV afterload includes (a) identification and management of reversible causes of increased pulmonary vascular resistance such as hypoxia, acidosis, or excessive positive end-expiratory pressure (PEEP). Similarly, in acute PE, anticoagulation and clot reduction therapy lowers the RV afterload. (b) Pulmonary vasodilators such as inhaled nitric oxide or epoprostenol.[23] These are selectively delivered to well-ventilated alveoli and help improve V/Q mismatch, reverse hypoxic vasoconstriction, and improve RV-PA coupling.
- *Contractility:* Inotropes can be considered once preload is optimized and afterload is minimized. Milrinone has a dual benefit of increasing RV contractility and reduction of pulmonary vascular resistance (PVR) and is particularly useful in PH. However, inotropes should be used with caution as they cause increased myocardial oxygen demand, tachyarrythmias, and hypotension[24] (**Table 4**).

Importance of rhythm control in optimizing right ventricular function

- Atrioventricular synchrony: The RV is highly preload-dependent and relies on atrial contraction to maintain adequate end diastolic volume. Loss of atrioventricular synchrony is magnified when other loading conditions are not optimal—for example, high RV afterload or reduced contractility from ischemia. Thus, patients with RV dysfunction may benefit from restoration of sinus rhythm over rate control alone.
- Impact of arrhythmias (tachyarrhythmias, bradyarrhythmia) on RV filling and output:
  - Tachyarrhythmias result in shortened diastolic filling time, inadequate RV preload and increased myocardial oxygen demand.
  - Bradyarrhythmias result in fixed or minimally augmentable RV stroke volume and inadequate forward flow.
- Strategies for rhythm and rate control (eg, cardioversion, antiarrhythmics, pacing)
  - DC cardioversion—preferred strategy in the setting of hemodynamic instability. Results in rapid restoration of atrial contribution and prevents worsening of hemodynamics from prolonged ischemia or low output.
  - Anti-arrhythmic medications—Amiodarone is favored in RV failure, it is effective for both atrial and ventricular arrhythmias and has minimal negative inotropy.[25]
  - Rate control therapies—beta blockers or non-dihydropyridine calcium channel blockers should be avoided due to their negative inotropy. If rhythm control cannot be achieved, digoxin may provide the added benefit of positive inotropy along with rate control.
  - Pacing: atrial or atrioventricular (AV) sequential pacing can help restore AV synchrony, improve RV filling and cardiac output.

Intensive care unit (ICU) considerations

Sedation: Propofol is a commonly used anesthetic for general anesthesia, procedural sedation, ICU sedation, and as an inducing agent for intubation. Propofol can cause rapid decrease in preload and has a negative inotropic effect. This can result in hemodynamic instability in patients with severe RV dysfunction. In patients with RV dysfunction, etomidate and ketamine are better tolerated as induction agents for intubation.[26]

Dexmedetomidine is a selective alpha 2 agonist, used as an ICU sedative and anxiolytic. It can cause bradycardia and hypotension which is not well tolerated in RV dysfunction. Intravenous morphine is used as a sedative and anxiolytic. In addition to anxiolysis, it can cause venodilation and rapid decrease in RV preload. Even though it might be a desired effect in patients with acute pulmonary edema due to MI, administration of

Table 4
Vasoactive medications and their cardiovascular effects

Ino-Dilators	Mechanism of Action	Effects
Milrinone	PDE3 inhibitor	• Increases myocardial contractility and causes vasodilation in pulmonary and systemic circulation. • Can cause profound hypotension
Dobutamine	Beta 2 agonist	• Increases myocardial contractility with systemic vasodilation. • Vasodilatory effects are not as pronounced as milrinone • Can cause sinus tachycardia, atrial fibrillation
Ino-pressors	**Mechanism of Action**	**Effects**
Epinephrine	Alpha and beta 1,2 agonist	• Increases heart rate, myocardial contractility, and vasoconstriction. • Can cause lactic acidosis through beta 2 stimulation
Norepinephrine	Predominant alpha agonist, mild beta agonist	• Vasoconstriction with mild increase in myocardial contractility
Dopamine	Beta-1 and dopamine receptor agonists. Alpha agonist at high doses	• Increases heart rate, myocardial contractility and vasoconstriction • Can cause tachycardia, increase vascular resistance (ventricular afterload)
Vasopressors	**Mechanism of Action**	**Effects**
Vasopressin	V1 receptor agonist	• Systemic vasoconstriction • Pulmonary vasodilator at low doses • Preferred vasopressor, used in conjunction with an inodilator
Phenylephrine	Pure alpha agonist	• Vasoconstriction in the pulmonary and systemic circulation • Increase LV/RV afterload

Abbreviation: PDE-3, phosphodiesterase-3.

morphine can result in hemodynamic deterioration and adverse events in the setting of RVMI.[26]

Ventilation strategies and hypoxia: Positive pressure ventilation decreases RV preload, and can increase RV afterload at very high levels of positive pressure although it should be noted that this exhibits a U-shaped relationship and there initially is a reduction in PVR due to opening of alveoli and favorable tethering of pulmonary vasculature.[26] Understandably, mechanical ventilation can thus potentiate further hemodynamic instability with high levels of PEEP support. Use of high flow oxygen can avoid the need for positive pressure ventilation in some patients. Positive pressure should be avoided if possible or used with caution and minimized in patients with RV failure.

DISEASE-SPECIFIC APPROACH TO RIGHT VENTRICULAR FAILURE

Acute Myocardial Infarction Related Right Ventricular Cardiogenic Shock

Acute RV dysfunction should be suspected in an inferior wall ST elevation MI. Right-sided ECG should be routinely performed, the presence of ST elevation in V4r suggests RVMI.

Medical therapy: Routine use of sublingual nitrates should be avoided in RVMI, as the venodilation effect of nitrates will cause an abrupt drop in RV preload and results in an exaggerated hypotensive response. The recommended approach is to rapidly establish coronary perfusion by performing percutaneous coronary intervention (PCI) of the culprit artery.[27] Initial fluid boluses can be helpful to maintain adequate preload to the RV. If there is persistent hypotension despite fluid bolus, starting norepinephrine or dopamine is recommended.[28] After revascularization and initial hemodynamic stabilization, transition can be made to an inotropic agent to improve RV contractility and optimize preload to keep CVP around 5 to 10 mm Hg.

MCS considerations: If there is persistent hypotension or a need for vasopressor requirement, post PCI, PA catheter-guided hemodynamic risk stratification should be done to determine the phenotype as either LV shock or biventricular shock. CVP greater than 14 mm Hg, cardiac power output (CPO) less than 0.6, and PAPi less than 1 are

suggestive of severe RV failure. RV dysfunction is often missed or overlooked in cardiogenic shock patients with anterior wall STEMI involving the left anterior descending or left main coronary artery. If the hemodynamic parameters for RV function are not optimized despite adequate LV support, MCS escalation might be required to restore tissue perfusion. Persistently elevated CVP greater than 14 mm Hg and PAPi less than 1 despite inotrope support should prompt tMCS escalation with either Impella RP flex or venoarterial extracorporeal membrane oxygenation (VA-ECMO) depending on availability (**Fig. 2**).

Acute Pulmonary Embolism Related Right Ventricular Cardiogenic Shock

Acute, large-burden PE or saddle PE results in hypoxia and an abrupt increase in RV afterload leading to acute RV systolic dysfunction, reduced RV stroke volume (decrease in LV preload) leading to hypotension. The RV strain pattern can be identified on the CT chest with contrast as contrast reflux in inferior vena cava, RV/LV ratio greater than 0.9.[29] Serum biomarkers such as elevated serum troponin and brain natiuretic peptide can point toward ongoing RV strain. Bedside echocardiography can immediately assess and quantify the severity of RV dysfunction.[30] Progressive RV systolic dysfunction and hypoxia results in lactic acidosis, sinus tachycardia, and systemic hypotension.

Medical therapy: Immediate therapeutic anticoagulation is the mainstay of treatment. In addition to anticoagulation, catheter-directed thrombolytics or large bore mechanical thrombectomy can augment clot reduction and has shown to stabilize RV function in intermediate risk PE. Systemic thrombolytics were historically recommended as class I indication in patients with hemodynamic instability[30]; however, the recently updated 2026 AHA/ACC Guideline for the Management of Acute Pulmonary Embolism in Adults lists systemic thrombolytics as a class IIa indication for patients with overt cardiopulmonary failure (category E1/E2). In patients with CS (category E1), systemic thrombolytics share a class IIa indication alongside mechanical thrombectomy, catheter-directed thromboylsis, and surgical embolectomy. In patients with refractory cardiogenic shock or cardiac arrest (category E2), systemic thrombolytics stand alone as a class IIa recommendation.[31] Vasopressors and/or inotropes carry a class I indication across cardiopulmonary failure states in these guidelines if warranted clinically.

MCS considerations: In patients with hemodynamically unstable PE defined as systemic hypotension (systolic blood pressure [SBP] < 90 mm Hg) or in cardiac arrest requiring rapid restoration of circulation or a need for vasopressor to maintain SBP greater than 90 mm Hg, tMCS can be considered. VA-ECMO can be used in patients with catastrophic PE (impending cardiogenic shock, cardiac arrest, high flow oxygen requirement). In the 2026 AHA/ACC Guideline for the Management of Acute Pulmonary Embolism in Adults, VA-ECMO carries a specific class IIa recommendation in patients with refractory cardiogenic shock or cardiac arrest (category E2). In selected patients, after clot reduction therapy has been achieved and if the patient has persistent shock, RV assist devices like Protek Duo can be used (**Fig. 3**).

Heart Failure-Related Biventricular Cardiogenic Shock

The mainstay of therapy for RV failure in de novo or acute-on-chronic heart failure related cardiogenic shock (HF-CS) is optimization of loading conditions for the right ventricle. Whether or not this can be achieved pharmacologically or with device therapy is the main question and is difficult to predict with MCS often warranted to normalize LV loading conditions or support end-organ function regardless of RV function.

Medical therapy: Volume removal should be pursued aggressively to normalize both RV filling pressures and pulmonary pressures to optimize RV preload and afterload. Inotropic support may assist with the initial stage of shock management as well as with persistent RV dysfunction despite optimization of RV loading conditions.

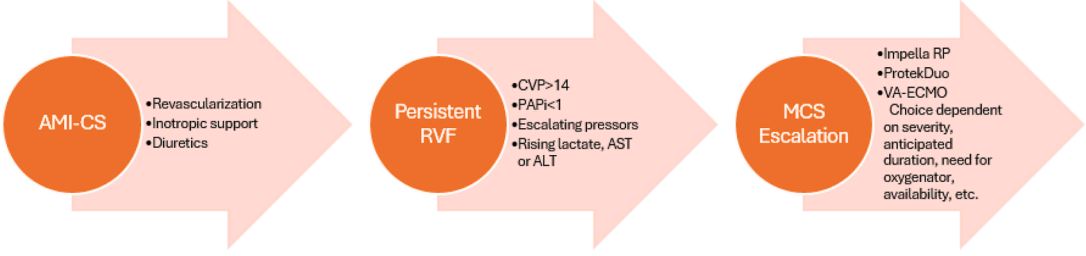

Fig. 2. Escalation in acute myocardial infarction related carrdiogenic shock (AMI-CS) with RV failure.

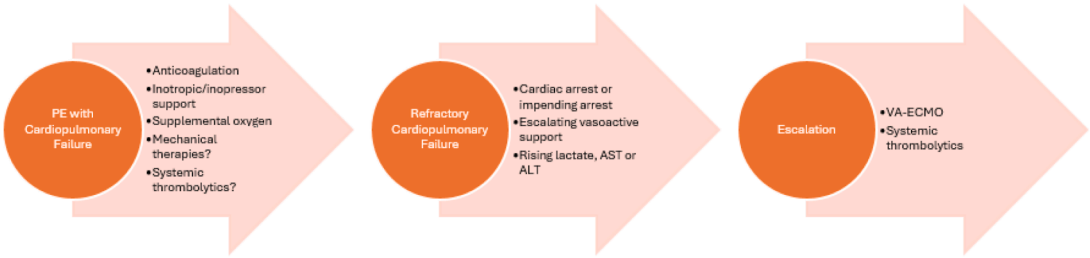

Fig. 3. Escalation in acute pulmonary embolism with RV failure.

Vasopressors and inopressors may be required in the initial management period and should be utilized with RV loading conditions in mind, but the ongoing use of vasopressors and failure to clear shock despite best-achieved loading conditions and inotropy should prompt teams to upgrade to MCS if appropriate and not otherwise contraindicated.

MCS considerations: The authors recommend early implantation of MCS to restore end-organ perfusion, as an ever-increasing base of literature suggests the first 24 hours of shock management is critical.[32] For SCAI B and C cardiogenic shock with RV involvement, an attempt at medical stabilization with inotropic support and aggressive diuresis is reasonable in the first hours of management. We recommend implantation of MCS in appropriate candidates for SCAI D and E stages of cardiogenic shock with RV involvement as well as SCAI C cardiogenic shock that fails to rapidly improve with initial medical interventions.

With biventricular failure, biventricular support would seem to be the obvious answer. The most rapidly deployable biventricular support is VA-ECMO and this is a mainstay of initial stabilizing therapy for advanced stages of shock. Depending on the patient, support with IABP or Impella 5.5 may facilitate adequate pulmonary decongestion to improve RV function with or without inotropic support but resolution of the shock state may lag behind that which can be achieved with VA-ECMO. Additionally, these devices are often deployed with VA-ECMO to unload or vent the left ventricle and provide a platform for step down of MCS intensity as the shock state stabilizes. In the medium term, biventricular support with biventricular Impella has been described, but there is limited evidence to support this practice. Adequate LV unloading, including with Impella 5.5 and inotropic support, is often sufficient to stabilize RV function and is commonplace during the workup and preoperative period for patients undergoing heart transplant or durable LVAD placement.

In cases of refractory RV failure, surgically implanted biventricular VADs have been described, again with very limited evidence. A Total Artificial Heart (TAH) by (SynCardia) remains FDA approved as well for biventricular support as a bridge to transplant, with retrospective analysis of the INTERMACS registry suggesting reasonable survival or transplantation at 1 year in high-volume centers[33] (Fig. 4).

Pulmonary Hypertension-Related Right Ventricular Failure

For our purposes, we will focus on WHO group I and WHO group IV PH, though the principles herein could be applied to select WHO group III patients who are eligible for transplantation.

WHO group 1, pulmonary arterial hypertension

Pulmonary arterial hypertension (PAH) is defined as high pressures in the pulmonary arteries stemming from increased pulmonary vascular resistance as a result of progressive endothelial dysfunction and vascular remodeling in the pulmonary vascular bed. While overall rare, PAH from any cause may progress to RV failure and shock in the presence or absence of another precipitating event. De novo cases of RV shock in PH are less common in the absence of a second hit as the indolent progression of pulmonary vascular tone usually comes with enough lead time that right heart failure symptoms present before hemodynamic collapse precipitates. Second hits are not necessarily hard to imagine though, with the risk of PE or transient RV ischemia from any cause of hypotension being common.

Medical therapy: Medical therapy for PAH-related RV failure is dependent on the baseline medical therapy for PH, the degree of RV failure, and the degree of systemic hypoperfusion present. Correction of hypoxemia and acidemia should be aggressive and may significantly improve pulmonary vascular resistance. Rapid pharmacologic mitigation of pulmonary vascular resistance is best achieved with the use of inhaled pulmonary vasodilators such as nitric oxide and epoprostenol, but these require eventual transition to long-term medications in the prostacyclin class.

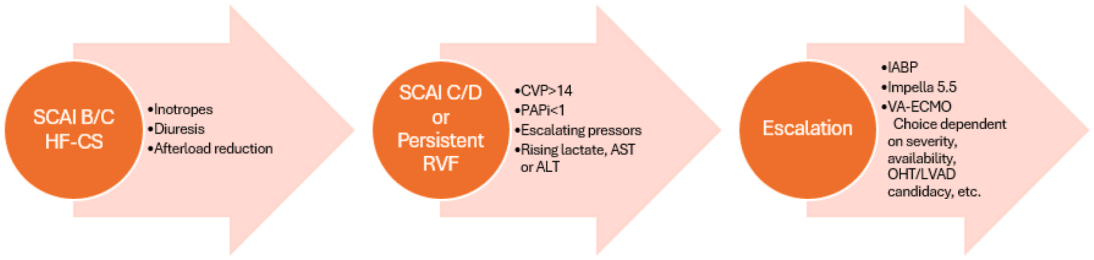

Fig. 4. Escalation in HF-CS with RV failure.

Depending on the severity of pulmonary pressures and risk stratification, triple therapy with a prostacyclin, endothelin receptor antagonist and phosphodiesterase type 5 receptor inhibitor may be warranted. Some patients may further warrant therapy with novel agents like sotatercept or clinical trial enrollment.

Inotropic support utilizing dobutamine, milrinone and less commonly dopamine is often necessary to stabilize patients with RV failure from PH, depending on the hemodynamic scenario.

For patients with systemic malperfusion and severe PH, vasopressors may be required. Maximization of coronary perfusion pressure to the RV is a key concept in this area. There is a paucity of high-quality evidence regarding first-line vasopressors in this scenario. Physiologic principles of limiting additional RV afterload as well as animal models would suggest vasopressin as a first-line pure vasopressor as it lacks receptors in the pulmonary circulation and minimally affects PVR. With concomitant low cardiac output states, inopressors such as dopamine, norepinephrine, or epinephrine may be reasonable initial therapy, with careful attention to dose-dependent PVR elevation. Phenylephrine is generally avoided as it offers pure vasoconstriction including in the pulmonary circulation and will worsen RV afterload.[34]

MCS considerations: The decision to deploy MCS in the setting of RV failure and shock secondary to PAH is challenging. Time permitting, many factors may be taken into account including transplant eligibility and overall reversibility of the degree of PH. The 2022 AATS Expert Consensus Document on MCS in Lung Transplantation suggests the use of VA-ECMO is a valuable option as a bridge to transplantation in end-stage PH evolving to RV failure (Class I; B-NR).[35]

Outside of a bridge to transplantation, the use of VA-ECMO in PAH-associated RV cardiogenic shock comes with many challenges and all of the usual complications of large-bore MCS devices. While the right ventricle has long demonstrated the ability to recover from acute insults, RV failure from PAH is generally an end-stage condition in the absence of a second insult and similar recovery cannot reasonably be expected. Similarly, while temporary and durable MCS for LV failure are increasingly used to facilitate loading of guideline-directed medical therapy for LV recovery, no such medical therapy exists for the RV. Any use of temporary or durable VAD for RV failure does not extend to PH-related RV failure for this and many other reasons. Indeed, no durable MCS devices are FDA approved for isolated RV failure from PAH and evidence in durable right-sided VAD is limited to etiologies of RV shock known to recover including postcardiotomy, RVMI, early primary graft dysfunction after transplant, and post-LVAD implantation.[36] TAH, while sometimes used as a bridge to transplant in biventricular failure, similarly does not extend to use in isolated RV failure. Physiologically, all these devices are well known to be very afterload sensitive and our inability to quickly and effectively reduce the PVR and thus RV afterload is the root of the problem.

In a de novo diagnosis of PAH-associated RV failure/shock with no background medical therapy, temporary MCS with VA-ECMO could be considered if initial medical therapy fails to stabilize RV performance as a bridge-to-decision and/or as a bridge to medical therapy. Theoretically, such support could provide time for initiation of pulmonary vasodilator therapy to improve PVR and allow optimization of loading conditions to improve RV performance and eventually allow decannulation. We propose this strategy only be deployed after extensive informed consent regarding potential complications and risks of treatment failure. In contrast to de novo diagnosis of PAH presenting as RV failure and shock, deploying MCS in a non-transplant eligible patient with end-stage RV failure from PAH despite maximal medical therapy should be considered a “bridge to nowhere” and should be avoided.

VA-ECMO is the ideal choice for decompensated RV failure with shock, as it directly unloads the failing right ventricle, reverses hypoxemia and hypoperfusion-related acidemia, maintains coronary perfusion pressure, and corrects impaired

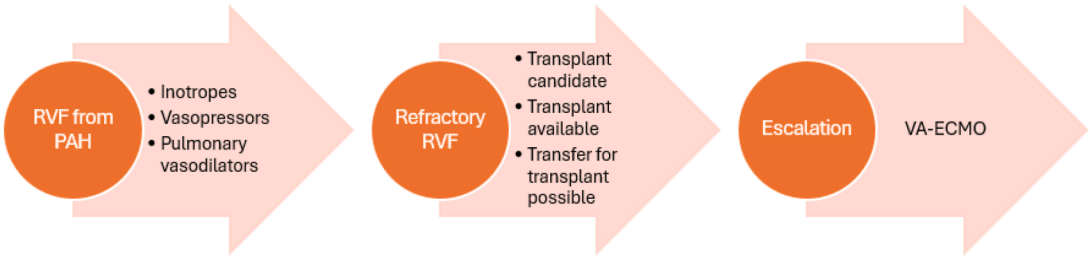

Fig. 5. Escalation in PAH with RV failure.

systemic blood flow. Dramatic improvements in RV mechanics can be expected, but exit strategies are challenging. Temporary right ventricular assist devices (RVAD) like Impella RP and ProtekDuo are a consideration and certainly are capable of unloading and moving blood through a failing right ventricle, but they still face marked afterload in the pulmonary circuit and do not provide as much flow as VA-ECMO, which can impact time to stabilization of the shock state. Membrane oxygenators can be added (RVAD-MO) to improve hypoxemia, but the same hemodynamic challenges remain (Fig. 5).

WHO group 4, chronic thromboembolic pulmonary hypertension

Medical therapy: The soluble guanylate cyclase inhibitor riociguat is the only medication approved for inoperable chronic thromboembolic pulmonary hypertension (CTEPH) or residual CTEPH after pulmonary endarterectomy (PEA). All patients should be systemically anticoagulated. Catheter-based lytic therapy or catheter-based thrombectomy can be considered if it is clear that there is acute-on-chronic disease. Inotropes, inopressors and vasopressors used as described in the preceding section on pulmonary arterial hypertension may provide enough support to temporize the situation until definitive treatment can be established.

MCS considerations: The collective understanding of the use of MCS in CTEPH is limited to retrospective analyses and case series. MCS could be deployed in patients reasonably eligible for PEA or transplantation as a bridge to those therapies. Access to these therapies is very limited, and receiving PH/shock centers should have mechanisms and relationships in place to facilitate consultation and transfer to such services prior to instituting MCS in these patients. Importantly, in the absence of compelling evidence, MCS as bridge to balloon pulmonary angioplasty (BPA) is not a viable strategy, principally as the treatment time required for BPA to provide meaningful reduction of pulmonary pressures is long and medical therapy cannot reasonably be assumed to be enough to facilitate MCS weaning.

As in PAH, VA-ECMO offers excellent unloading and optimization of the right ventricle during the period before PEA and is often left in place post-PEA given the length of cardiopulmonary bypass (CPB) and risk of hypothermic/ischemic/reperfusion driven RV dysfunction. A larger retrospective review demonstrated the viability of conversion to central VA-ECMO for patients with RV failure and CTEPH post-CPB.[37] Continued support with membrane oxygenation (via VA, VAV, or VV configuration) also provides adequate support in the event of significant hypoxemia driven by reperfusion pulmonary edema (Fig. 6).

Right Ventricular Failure Post-LVAD Implantation

RV failure following durable LVAD implantation continues to be a challenge despite advancements in LVAD technology. Post-LVAD RV failure can present as an early or late complication. We will focus on early RV failure as management

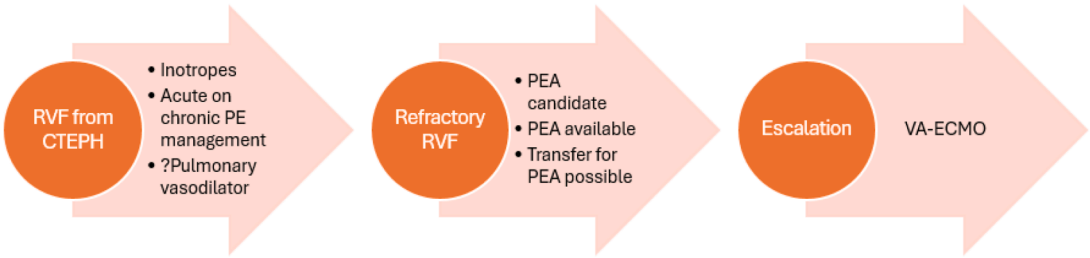

Fig. 6. Escalation in CTEPH with RV failure.

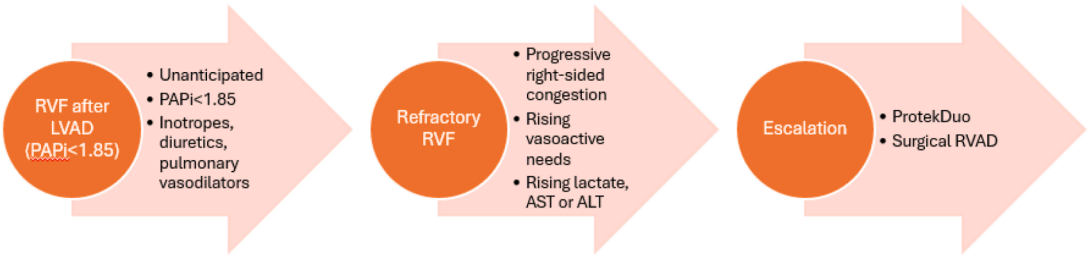

Fig. 7. Escalation in post-LVAD RVF where RV MCS was not instituted during LVAD operation.

strategies of later presentations center around inotropic support and/or heart transplantation. Physiologically complex, contributing factors to post-LVAD RV failure include but are not limited to: preoperative RV function, operative characteristics, perioperative volume administration, perioperative hypotension, arrhythmias, acidemia, mechanical ventilation, and increased return to the RV once the LVAD is flowing. Prior analysis of the INTERMACS registry noted a 24% rate of RV failure within 30 days of LVAD implantation and prognosis is clearly linked with patients free of RVF averaging a 2 year survival of 82% compared to 50% in those with RV failure.[36]

Medical therapy: Medical therapy for post-LVAD RV failure centers around optimization of loading conditions for the RV. Diuretic therapy targeting a CVP of 10 to 15 allows adequate RV output while minimizing wall tension and improving ventricular interdependence. Similarly, LVAD speed adjustment may be necessary to maintain a midline septal position to optimize ventricular interdependence. Optimizing coronary perfusion to limit any postoperative ischemia to the RV is important, often necessitating vasopressor use in the immediate postoperative period. Elevated pulmonary pressure is often encountered and can be problematic. Efforts toward reducing this RV afterload is focused on adequate diuresis and pulmonary vasodilator therapy if necessary, typically with inhaled epoprostenol or nitric oxide in the postoperative period. Positive pressure ventilation provides further RV afterload and both pressures and duration of mechanical ventilation should be limited. If the RV continues to inadequately fill the left ventricle despite these measures, inotropes can be deployed to increase RV output with dobutamine, milrinone, epinephrine, and dopamine often utilized depending on the rest of the hemodynamic profile. Arrhythmias should be aggressively managed with antiarrhythmic drugs and/or cardioversion. In pacemaker dependent patients, base heart rate may need to be increased if maximized RV stroke volume remains inadequate. With many facets of RV optimization to consider, emerging evidence suggests that protocolized management results in better outcomes post-LVAD as compared to usual care.[38]

MCS considerations: Failing medical management or in anticipation of such, temporary MCS for the RV may be necessary in the postoperative period. We suggest early deployment of either surgical or percutaneous RVAD support rather than delayed deployment after medical therapy has failed. The best time to decide to use RVAD support postoperatively is likely preoperatively. Careful assessment and understanding of underlying RV function is critical prior to implementation, in some cases aided by preoperative use of temporary LVAD like Impella 5.5 for cardiogenic shock. Evidence to support intraoperative deployment of RVAD support is also emerging, with retrospective analyses suggesting improved outcomes compared to delayed implantation.[39]

Intraoperative deployment allows the use of surgical RVAD (CentriMag) without re-do sternotomy or thoracotomy. Percutaneous RVAD (Impella RP, Protek Duo, Tandem Heart) can be deployed at any point. CentriMag and Tandem Heart are approved for 30 day use, Impella RP for 14 days, and Protek Duo for up to 24 hours although clinical scenarios often dictate longer use. Membrane oxygenators may be added to both Protek Duo and CentriMag if needed, which can prove useful in patients with pulmonary complications perioperatively (Fig. 7).

CLINICS CARE POINTS

- The diagnosis of RV failure requires high degree of clinical suspicion and careful hemodynamic assessment.
- The presence of RV failure adds significant morbidity and mortality to causitive pathologies.
- Management of RV failure is complex and involves rapid, disease-specific and hemodynamic-guided escalation of mechanical circulatory support in appropriate patients.

DISCLOSURES

The authors have nothing to disclose.

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