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Comprehensive Review

Management of Post–Myocardial Infarction Right Ventricular Failure

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ABSTRACT

Right ventricular failure (RVF) due to an acute myocardial infarction (MI) has been associated with high morbidity and mortality. Initial treatment is guided by early recognition and prompt revascularization. Current management of post-MI RVF is built upon expert consensus and is also informed by RVF from other etiologies, including massive pulmonary embolism, left ventricular assist device–associated right ventricular dysfunction, postcardiotomy shock, etc.; this speaks to the limited data available on the specific management of RVF in acute MI. The goal of this review is to discuss the current literature on the pathophysiology, general management considerations, interventional management, hemodynamic monitoring, medical management, and mechanical circulatory support of MI-induced RVF.

Introduction

Acute right ventricular (RV) failure may present as a rapidly progressive syndrome that can occur in the setting of several different situations, including but not limited to massive pulmonary embolism, acute myocardial infarction (MI) involving right coronary artery (RCA) occlusion, and left ventricular assist device (LVAD) implantation.[1-3] Right ventricular failure (RVF) after MI is defined by hypotension and/or hypoperfusion due to decreased RV contractility and, ultimately, reduced right-sided cardiac output. MI affecting the right ventricle has been associated with high morbidity and mortality.[3-11] Post-MI RVF can lead to systemic hypotension due to reduced RV function from myocardial ischemia with interventricular dependence due to pericardial constraint, resulting in underfilling of the left ventricle. The degree of the left ventricular (LV) myocardium subtended by the RCA and baseline LV function can also influence the clinical presentation of RV MI. If RVF develops, there is an associated in-hospital mortality of up to 17%.[12-15] Approximately 5% of acute MI-induced cardiogenic shock cases are primarily due to RVF.[16] In a report from the SHOCK Registry, it was found that RV shock was associated with a higher mortality than the mortality associated with LV shock despite patients with RVF being younger in age and having lower prevalence of multivessel disease and anterior MI.[16] In a study of 200 patients admitted for acute inferior MIs, the in-hospital mortality and major complications were higher in patients with inferior ST-elevation MI with RV involvement than in those without, with the former defined by ST elevation (≥1 mm) in lead V_{4R}.[11] Another study of patients with inferior MI independently came to the same conclusion.[17] In addition, patients with RVF due to RV MI were found to have higher mortality than that in those with predominant LV failure.[16] Taken all together, these data highlight the importance of being aware of RVF in the setting of acute MI. In this review, we discuss the pathophysiology of acute post-MI RVF, current literature on management, and outcomes.

Pathophysiology of acute post-MI RVF and complications

With normal physiologic function, the right ventricle is a thin-walled chamber that pumps its output into a low-pressure, high-capacitance vascular bed. Therefore, the right ventricle is extremely sensitive to acute changes in loading conditions compared with the left ventricle.[18,19] Under ischemic conditions, the right ventricle becomes stiff, leading to reduced peak systolic pressure; increased end-diastolic volume; and, in turn, increased diastolic pressure. This pressure overload leads to a leftward shift of the interventricular septum due to pericardial constraint, decreasing LV end-diastolic volume, compliance, and filling. Thus, acute MI RVF results in reduced RV systolic and diastolic function (Central Illustration). With reduced diastolic function and

Abbreviations: IABP, intra-aortic balloon pump; LV, left ventricle; MCS, mechanical circulatory support; MI, myocardial infarction; PAC, pulmonary artery catheter; PAPi, pulmonary artery pulsatility index; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure; RVF, right ventricular failure; VA-ECMO, venoarterial-extracorporeal membrane oxygenations.

Keywords: cardiogenic shock; mechanical circulatory support; myocardial infarction; right ventricular failure; STEMI.

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dilatation, RV volume overload leads to increased right atrial (RA) pressure and systemic (eg, hepatic and renal) congestion. The combination of these effects can ultimately lead to underfilling of the left side of the heart and cardiogenic shock.[16]

Proximal occlusion of the RCA leads to anterolateral RV infarction via the acute marginal arteries[17] and is the predominant location of occlusion that results in RVF after MI.[16] Additional locations of occlusion are the left dominant circumflex artery and distal RCA, which can lead to posterior RV wall infarction.[6,20,21] Compromised coronary flow leads to poor RV perfusion, decreased contractility, and RV dysfunction. The left coronary system has flow occurring primarily during diastole and may fall sharply or even reverse direction during systole because of the extravascular compressive forces of the thick LV myocardium. In contrast, the flow in the RCA is less phasic because the right ventricle generates much less force of contraction than that generated by the left ventricle; as a result, RV perfusion occurs throughout the cardiac cycle in systole and diastole. Hypotension in the setting of an RCA occlusion further compromises RV perfusion, and wall stress increases. Consequently, poor RV output leads to decreased LV preload and cardiac output despite an intact LV systolic function.[3,5,22,23] Furthermore, an RCA infarct that causes papillary muscle dysfunction can lead to ischemic mitral regurgitation, resulting in increased RV afterload, further compromising an already diminished cardiac output.

In patients with baseline LV dysfunction, pulmonary vascular resistance and impedance may be increased, with a decrease in pulmonary compliance; this can lead to an already vulnerable right ventricle to be without the reserve necessary to withstand an acute insult, such as RV MI.[24-31] When such a right ventricle suffers from a proximal RCA occlusion, right-sided output may decrease, leading to diminishing LV preload and ultimately impairing cardiac output. LV systolic dysfunction directly influences RV function because of septal contribution to RV stroke volume and sharing of myofibers between chambers.[16,32,33]

General management considerations

The treatment of RVF is dependent upon early recognition, assessment of its severity, and management in an efficient and multidisciplinary manner.[34-36] Initial severity assessment can be performed in multiple settings, including at the first medical contact, at the emergency department, at the cardiac catheterization laboratory, and in the post-intervention care environment. Effective triage is essential regardless of location. The clinical presentation of acute post-MI RVF can vary, and rapid assessment of the severity is essential. This includes clinical history, physical examination with a complete set of vital signs, and the acquisition and interpretation of a 12-lead electrocardiogram. Acute physical examination findings include jugular venous distension with a prominent V wave and a Kussmaul sign. ST-segment elevation in leads II, III, and aVF with reciprocal ST-segment depression in the lateral leads can suggest RV MI. This should prompt right-sided leads V_{4R}, V_{5R}, and V_{6R} to be obtained. RV MI can demonstrate ST elevation in V_{4R} through V_{6R} (Figure 1).[37] Early attention should include determining the stability of the patient to ensure transfer to a facility capable of appropriate care escalation (eg, primary percutaneous intervention, mechanical circulatory support [MCS] devices, and availability of cardiac surgery support). Laboratory evaluation should include basic laboratory values, including a renal/hepatic function panel, lactate, and cardiac injury biomarkers. Unfortunately, there are no laboratory values specific for RVF.[38] When possible, a bedside point-of-care-echocardiography should be considered because this can provide crucial information, such as underlying LV dysfunction, relevant valvular disorders, and/or mechanical complications.[39,40] However, this should not be prioritized in a manner that would lead to a significant delay in performing percutaneous coronary intervention (PCI).

Interventional management

It is a class I recommendation to perform PCI within 90 minutes of a ST-segment elevation MI (STEMI) when possible.[41] If a hospital is unable to perform PCI on-site, immediate recognition of a MI should lead to transferring to a PCI capable facility, specifically one with on-call staff 24 hours a day, 7 days a week. In patients with cardiogenic shock from MI, successful PCI is beneficial, with superior outcomes seen with revascularization in the SHOCK trial.[42-44] During coronary catheterization, the interventional issues that may arise specifically due to RV infarction include avoiding jailing of the RV marginal branches, if possible, and considering balloon angioplasty alone of the marginal branches if jailing compromises flow in a sizable marginal branch.

Hemodynamic monitoring

Invasive hemodynamic measurements can be obtained with a pulmonary artery catheter (PAC).[45] The PAC can be helpful in the diagnosis and management of patients with RVF and cardiogenic shock.[46-52] A recent study by Garan et al[53] indicated that complete PAC hemodynamic data was associated with lower in-hospital mortality than that in those with incomplete PAC hemodynamics across all Society for Cardiovascular Angiography & Interventions stages of shock. Of note, the ESCAPE trial did not provide information on using PACs in cardiogenic shock.[45] Beyond individual PAC parameters, comparison of right- and left-sided filling pressures and estimation of vascular bed compliance can lend further insight into individual chamber performance metrics. For example, a ratio of RA pressure to pulmonary capillary wedge pressure (PCWP) of >0.86 has been associated with RVF due to an acute MI.[54] RV stroke work can also be calculated on the basis of the readings from the PAC. It is based on the mean pulmonary artery (PA) pressure, RA pressure, and a true estimate of stroke volume from the cardiac output.[55,56] An RV stroke work of <10 is an indicator of post-MI RVF.[57] The pulmonary artery pulsatility index (PAPi) is the ratio of PA pulse pressure divided by the RA pressure and provides an estimate for RV

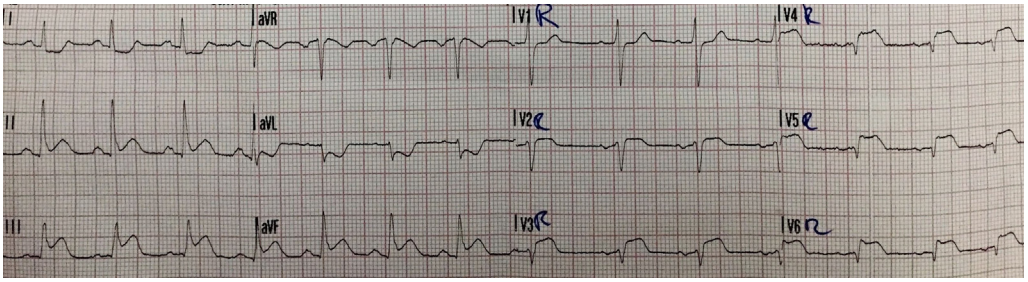


Figure 1.
An electrocardiogram of right ventricular myocardial infarction. (Leads V4-$V6_R$).[36]

Table 1. Hemodynamics formulas to assess right ventricular function in the setting of acute myocardial infarction right ventricular failure.

Hemodynamics formulas in post-MI vs post-LVAD RVF			
Hemodynamics	Formulas	Post-MI RVF	Post-LVAD RVF
Cardiac filling pressures	RAP ÷ PCWP	>0.86	>0.63
PAPi	(PASP − PADP) ÷ RAP	<1.0	<1.85
RV stroke work	(mPAP − RAP) × SV × 0.0136	<10	<15

LVAD, left ventricular assist device; MI, myocardial infarction; mPAP, mean pulmonary artery pressure; PAPi, pulmonary artery pulsatility index; PADP, pulmonary artery diastolic pressure; PASP, pulmonary artery systolic pressure; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure; RV, right ventricle; RVF, right ventricular failure; SV, stroke volume.

pulsatile load. With normalization to RA pressure, PAPi also indexes pulsatility to RV accommodation to load. A PAPi of <1.0 was found to be an indicator of RVF in the acute MI setting (Table 1).[58,59] These hemodynamic parameters allow us to classify cardiogenic shock into LV, RV, or biventricular phenotypes; however, these classifications exist on a spectrum and are not always in discrete categories. Interventricular interactions can result in dynamic clinical scenarios despite the territory served by the culprit vessel.[60,61] For instance, Lala et al[61] found that despite excluding patients with isolated RV shock in the SHOCK trial and registry, RV dysfunction was common in acute MI cardiogenic shock irrespective of the culprit coronary vessel, using the aforementioned well-established hemodynamic criteria. Finally, once clinical improvement and stability have been achieved, the PAC should be removed to minimize the risks of complications, including catheter-related thrombosis and infection.

Fluid management

Optimization of volume and fluid status is meant to assist with RV preload. Fluid boluses are recommended for cardiogenic shock due to RVF.[41,62] When dealing with a patient in cardiogenic shock, PAC use is recommended for objective data to guide management. Volume resuscitation can be used to achieve an RA pressure of approximately 15 to 20 mm Hg. However, excessive fluid resuscitation may overload the right ventricle, causing decreased contractility, worsened tricuspid regurgitation, increased leftward shift of the interventricular septum, reduced LV filling, and decreased cardiac output.[63,64] Therefore, with the assistance of a PAC, one must take care to ensure that the central venous pressure (CVP) is <20 mm Hg to avoid RV overload. Subsequently, venous and systemic congestion may occur, for which intravenous loop diuretics may be used. The presence of a PAC that can monitor the PCWP as well can help assess this in an objective fashion. As such, the use of a central venous line alone is discouraged because this would be incomplete hemodynamic profiling. Furthermore, loading conditions tailored to the patient result in improved cardiac output and can be assessed by using a PAC as well.[65]

Vasopressors and inotropes

Vasopressors and/or inotropes are used for patients with persistent hemodynamic instability and for management of cardiogenic shock to maintain end-organ perfusion, increase ventricular contractility and cardiac output, and reduce cardiac filling pressures.[66]

Norepinephrine has primarily been used to restore blood pressure and improve coronary perfusion without major effects on pulmonary vascular resistance[67] and is associated with a lower risk of arrhythmia. When compared with epinephrine, norepinephrine has been associated with improved renal outcomes.[68] Therefore, it can assist with maintenance of systemic blood pressure, if needed. The typical dose is 0.2 to 1.0 μg/kg/min.[38]

In cardiogenic shock, the Sepsis Occurrence in Acutely Ill Patients (SOAP II) trial demonstrated that dopamine, compared with norepinephrine, is associated with higher rates of arrhythmia and a higher risk of mortality; however, the clinical methodology of the study has raised concern.[69] The main hemodynamic effect of dopamine is dependent on the infusion dose. At lower doses (0.5-3 μg/kg/min), dopamine acts on dopamine 1 receptors in the renal, mesenteric, cerebral, and coronary beds, resulting in vasodilation and theoretically increased renal perfusion.[70] At higher doses (3-10 μg/kg/min), dopamine begins binding to β-1 adrenergic receptors, resulting in inotropy and increased cardiac output with variable effects on the heart rate. At doses of >10 μg/kg/min, dopamine additionally binds to α-1 receptors, causing increased systemic vascular resistance.[70]

Dobutamine predominantly acts on β-1 adrenergic receptors at a dose range of 2.5 to 15 μg/kg/min, resulting in improved contractility, and, therefore, increases cardiac output depending upon cardiac reserve. Dobutamine has a 3:1 ratio for β-1 to β-2 receptors and functions as an inodilator. At doses of >15 μg/kg/min, it may cause α-1 receptor–mediated vasoconstriction. In general, it can be used successfully when the systolic blood pressure (SBP) is >90 mm Hg; however, if used alone when the SBP is <90 mm Hg, it may aggravate arterial hypotension and should be considered add-on therapy with additional vasopressors.[38]

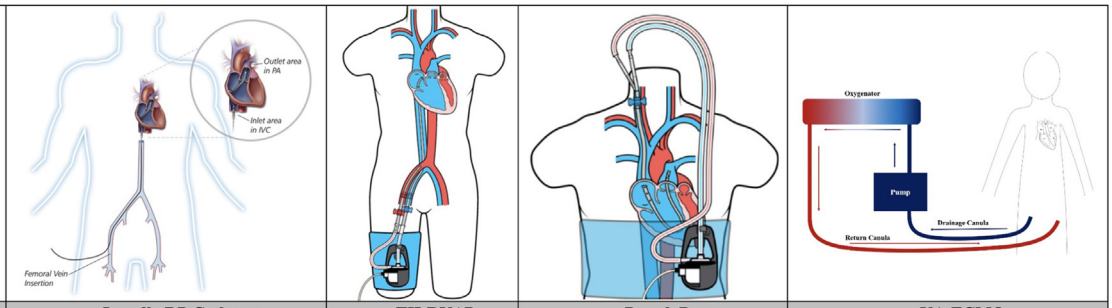
Additional medications that may be beneficial for RVF are phosphodiesterase III inhibitors, such as milrinone, another widely used inodilator. Pharmacologically, milrinone has a longer half-life than that of dobutamine; therefore, it can take longer to achieve the intended effect. An initial bolus of milrinone may be used to achieve the desired effect sooner; however, this can be associated with hypotension. Because of its longer half-life, milrinone is less titratable than dobutamine. As a result, it should be used with caution in patients with renal impairment. Until recently, robust evidence comparing both inodilators were sparse. The DOREMI trial showed no differences between milrinone or dobutamine in the composite primary or secondary outcomes in patients with cardiogenic shock; however, it should be noted that the trial exclusively included patients with LV dysfunction with a median left ventricular ejection fraction of 25% as the primary etiology of cardiogenic shock and not purely RVF-induced shock.[71] Similar to dobutamine, caution should be exercised with milrinone when the SBP is <90 mm Hg owing to concerns for hypotension.[38]

Reduction of RV afterload

Prostacyclin analogs and nitric oxide are the 2 major medications that assist with the reduction of RV afterload by acting as pulmonary arterial vasodilators. Intravenous prostacyclin analogs have been associated with systemic hypotension and have not been used for post-MI RVF.[63,65,72,73] Instead, inhaled prostacyclin can be used in right heart dysfunction in the setting of prior cardiothoracic surgery.[74] Similarly, inhaled nitric oxide can be considered and has mainly been used in patients after cardiac surgery.[75-77] There have been no studies on the use of inhaled prostacyclin or nitric oxide for post-MI RVF.

MCS in RV infarction

The right ventricle has the potential for recovery with the use of MCS. Therefore, MCS can be used for RVF refractory to medical therapy as a "bridging" therapy to recovery.[78] The decision to escalate to MCS in RVF should be based on clinical, laboratory, imaging, and



	Impella RP Catheter	TH-RVAD	Protek Duo	VA-ECMO
Inflow	RA	RA	RA	RA
Outflow	PA	PA	PA	FA
Flow range L/min	2-4	2-4	2-4	2-6

Figure 2.
Mechanical circulatory support devices in right ventricular failure. FA, femoral artery; IVC, inferior vena cava; PA, pulmonary artery, RA, right atrium; RVAD, right ventricular assist device; TH, TandemHeart; VA-ECMO, venoarterial extracorporeal membrane oxygenation. Images adapted from Pieri et al.[83]

hemodynamic variables and involve a cardiogenic shock team, if available. Although there are no well accepted criteria for the initiation of RV MCS, we propose early MCS initiation if the patient demonstrates signs and symptoms of poor organ perfusion and a cardiac index of <2.2 L/min/m^2, a PAPi of <1.0, or a cardiac power output of <0.6.[65,79,80]

Current options for percutaneous MCS for isolated RVF are the Impella RP catheter (Abiomed), TandemHeart right ventricular assist device (TH-RVAD) (LivaNova), ProtekDuo dual lumen cannula (LivaNova), and other RV bypass catheters, such as the dual lumen RV to PA cannula from Spectrum Medical and venoarterial (VA)–extracorporeal membrane oxygenation (ECMO) (Figure 2).[81]

The Impella RP device is a percutaneous, minimally invasive, single vascular–access microaxial-flow catheter. It is inserted percutaneously through the femoral vein using a 22F impeller mounted on an 11F catheter. Flow of the device is directly related to the revolutions per minute (RPM) of the impeller, and is indirectly related to the pressure gradient between the device inlet and outlet, also known as the pressure head (H).[82] Figure 3 demonstrates the head-capacity (H-Q) curve of a continuous flow pump based on Bernoulli's equation for the flow of fluids.[82] The H-Q curve is based on the revolutions per minute (RPM) in the numerator of this equation, with the denominator being the pressure head. Therefore, decreases in RPM will cause slower flow, whereas increases will lead to greater flow across the pump. Because the pressure head is the denominator, a larger pressure head will lead to slower flow, whereas a smaller pressure head will lead to faster flow across the pump. Given that right-sided pressures are lower than the left ventricle, the pressure head may be lower in the setting of acute RVF. Therefore, device flow will be higher for a given value of RPM. The Impella RP delivers blood from the inlet region of the right atrium through the cannula and into the outlet in the PA to restore right heart hemodynamics and reduce RV workload by directly bypassing the right ventricle.[83] In the setting of isolated acute RVF, this device will reduce RA pressure and augment RV flow and, ultimately, cardiac output. (Table 2).

The Impella RP was used successfully for RVF initially during cardiac surgery and LVAD placement.[84] The RECOVER RIGHT trial was conducted in 2015, and the Impella RP was approved for use through a humanitarian device exemption.[85] In this trial, the Impella RP was used for medically refractory RVF. It was a small prospective study that included 5 patients with acute post-MI RVF grouped with patients who underwent cardiotomy and cardiac transplant and a separate cohort of RVF after LVAD implantation.[85] The Impella RP device demonstrated improved CVP, cardiac index time to wean off inotrope and vasopressor support, and survival to 30 days or a hospital discharge rate of 73%; however, it is important to note that the survival achieved in the cohort of RVF after LVAD was 83%, whereas survival was 58% in the post-cardiotomy and post-MI group.[85] The US Food and Drug Administration examined postapproval outcome data on 42 Impella RP devices that were implanted and found a survival rate of 64%.[86] However, these studies excluded patients with acute MI with a history of unsuccessful

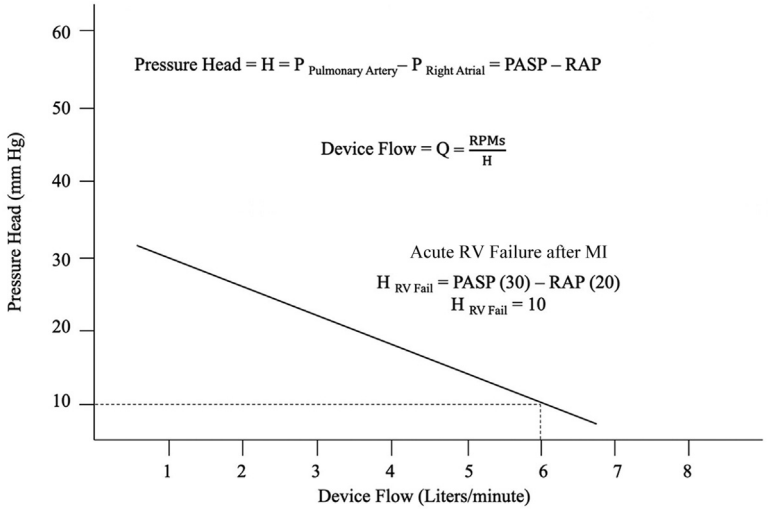
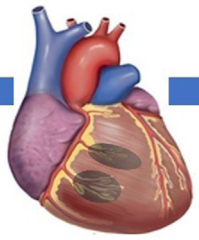


Figure 3.
Pressure head and flow (H-Q) curve for continuous flow pumps MI, myocardial infarction; PASP, pulmonary artery systolic pressure; RAP, right atrial pressure; RPM, revolutions per minute; RV right ventricular.



↓Right Ventricular Systolic Function	
Physiologic Changes	**Hemodynamic Signs**
↓ Peak Right Ventricular Systolic Pressure ↓ Left Ventricular Preload ↓ Cardiac Output	Cardiac Index < 2.2 Cardiac Power Output < 0.6 PAPi < 1.0 RV Stroke Work < 10

↓Right Ventricular Diastolic Function	
Physiologic Changes	**Hemodynamic Signs**
↑ Right Atrial Pressure ↑ Right Ventricular End Diastolic Volume Right Ventricular Dilatation	RA Pressure > 15 RA Pressure/PCWP > 0.86

Central Illustration.
Physiologic changes and hemodynamic signs of acute right ventricular (RV) myocardial infarction leading to RV failure and cardiogenic shock PAPi, pulmonary artery pulsatility index; PCWP, pulmonary capillary wedge pressure; RA, right atrium.

RCA revascularization. One small study of 5 patients with acute MI complicated by unsuccessful revascularization of the RCA and refractory RVF reported that 80% of patients who underwent Impella RP placement had acute RV recovery, all patients had a reduction in lactic acid level and an increase in SBP, and all survived at the 30-day mark.[87] However, the US Food and Drug Administration recently reported a postapproval study assessment in which 28.6% of patients on Impella RP survived at 30 days after explant, hospital discharge, or the start of the next therapy.[86] Therefore, Impella RP can be considered in patients with RVF refractory to medical therapy and possibly serve as a bridge to recovery but requires further analysis for long-term benefit.

TH-RVAD is also a percutaneous option that uses an extracorporeal centrifugal pump to generate flow from 2 venous cannulas inserted into the bilateral femoral veins. One 21F inflow cannula is inserted into the right atrium while the other 21F cannula is inserted into the main PA after the pulmonic valve for outflow. The achieved flows of the TH-RVAD are similar to those of the Impella RP. It directly bypasses the right ventricle to reduce preload and increase the mean PA pressure and LV preload in the setting of isolated RVF.[88]

TH-RVAD has been previously used in RV support configuration based on data from small case series.[88] In the retrospective Tandem-Heart in Right Ventricular Support (THRIVE) study, 46 patients with RVF received the TH-RVAD. Implantation was associated with a significant decrease in RA pressure and improvement in cardiac output. The in-hospital mortality was 57%, with the lowest mortality observed in patients with RVF secondary to acute MI or after LVAD implantation.[89] A smaller study of 9 patients, 6 of whom had RVF due to acute MI, found that those with TH-RVAD had improved hemodynamics and a lower hospital mortality rate of 44%.[90] The TH-RVAD should be considered in the patient population with post-MI RVF as a bridge therapy to transplant, long-term MCS, or RV recovery.

Another RA-PA bypass percutaneous MCS strategy uses a single, dual lumen cannula that is placed through the right internal jugular vein (ProtekDuo).[91,92] The catheter is positioned as a PAC would be, with the distal outflow lumen containing a multifenestrated distal tip to deliver blood to the main PA. The blood flows through an external centrifugal pump that allows for possible splicing of an oxygenator, which can be useful in the setting of hypoxic failure.[59] This device also directly bypasses the right ventricle, allowing for RV support with the additional benefit of oxygenation for patients with pulmonary pathology.[59,92] Studies have shown benefits in RVF after LVAD implantation and decompensated severe pulmonary hypertension.[59,91,92] Currently, there are no studies examining the success of ProtekDuo dual lumen cannula for MI-induced acute RVF; however, there are case series on successful use for RVF after LVAD implantation.[93-95]

VA-ECMO can be used in cardiorespiratory failure or biventricular failure and allows for improvement of systemic oxygenation. It uses an extracorporeal centrifugal pump to displace blood from the vena cava or right atrium, through an oxygenator, and into the systemic arterial system.[96,97] In acute RVF, VA-ECMO allows for a decrease in RA pressure, RV preload, and RV cardiac output. However, because of placing blood into the arterial system, there will be an increase in the mean arterial pressure and LV afterload. If LV function is impaired, the increased LV afterload will increase LA and PA pressures. There are limited data on the use of VA-ECMO in acute RVF.[98-100] Therefore, larger clinical trials and studies may elucidate the effect of VA-ECMO in MI-induced acute RVF.

There have been limited data on the use of an intra-aortic balloon pump (IABP) in the setting of acute RV MI. In a retrospective study of 32 patients who had an acute inferior ST-elevation MI complicated by RVF requiring an IABP, those with an IABP that survived had improved SBP, diastolic pressure, and aortic pressure.[51] The mechanism is thought to

Table 2. Mechanical circulatory support device mechanisms and hemodynamics.

	Impella RP catheter	TH-RVAD	ProtekDuo	VA-ECMO
Mechanism	Percutaneous, catheter–based, directly bypasses the RV	Percutaneous vs surgical, directly bypasses the RV	Percutaneous, directly bypasses the RV	Percutaneous vs surgical, indirectly bypasses the RV
Pump	Intracorporeal, axial flow	Extracorporeal, centrifugal flow	Extracorporeal, centrifugal flow	Extracorporeal, centrifugal flow
RAP (mm Hg)	↓	↓	↓	↓
mPAP (mm Hg)	↑	↑	↑	↓/↔
PCWP (mm Hg)	↑	↑	↑	↓
LV preload	↑	↑	↑	↓
LV afterload	↔	↔	↔	↑↑
CO	↑	↑	↑	↓/↔

↑ indicates increase, ↓ indicates decrease, and ↔ indicates no change.
CO, cardiac output; LV, left ventricular; mPAP, mean pulmonary artery pressure; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure, RV, right ventricle; RVAD, right ventricular assist device; TH, TandemHeart; VA-ECMO, venoarterial extracorporeal membrane oxygenation.

Table 3. Considerations for weaning mechanical circulatory support.

Hemodynamic considerations	• MAP ≥ 65 mm Hg • RA < 10-15 mm Hg • PCWP ≤ 18 mm Hg • Pulse pressure > 20-30 mm Hg • Cardiac power > 0.6 W • Cardiac index > 2.2 L/min/m^2 • PAPi > 1-1.85
Labs and clinical considerations	• Lactate < 2.0 mmol/L • Urine output > 30 mL/h • Warm extremities • Resolution of mottling • Pao_2/FiO_2 > 200
Echocardiographic considerations	• LVEF > 20%-25% • No LV or RV distention • LVOT VTI > 10 cm • RVEF not severely reduced

MAP, mean arterial pressure; RA, right atrium; PCWP, pulmonary capillary wedge pressure; PAPi, pulmonary artery pulsatility index; FiO_2, fraction of inspired oxygen; LVEF, left ventricular ejection fraction; LV, left ventricular; RV, right ventricular; LVOT, left ventricular outflow tract; VTI, velocity time integral; RVEF, right ventricular ejection fraction.

be 2-fold: enhanced coronary flow and RV support through LV septal contractile contribution. In the setting of an occluded RCA, collateralization from the left coronary circulation may be augmented with an IABP to assist in perfusion of the RV free wall and benefit the right ventricle.[101] However, still, there are limited data on the use of an IABP in the setting of acute RV MI and RVF, with further larger studies required to examine the potential mechanism and outcomes. The advantages of IABP placement continue to be the widespread availability, ease of insertion, and relatively smaller bore access.

Univentricular support devices should be used with caution in those with underlying reduced LV function because overloading a vulnerable left ventricle can result in elevated PCWP, pulmonary edema, and even pulmonary hemorrhage.

Weaning MCS in RVF

In the setting of an acute MI with RVF, de-escalation of mechanical support can be considered with myocardial recovery after revascularization. To note, until recently, there have been no recommendations for MCS de-escalation in the setting of cardiogenic shock.[102] Weaning of support requires a prerequisite of clear improvement of end-organ function and hypoperfusion, minimal vasopressor or inotropic support, euvolemia, and evidence of, at minimum, partial myocardial recovery or improved contractility. The American Heart Association has proposed a de-escalation of MCS algorithm, which can be seen in Table 3.[102] These parameters include hemodynamic stability, improvement of filling pressures, normalization of lactate, adequate urine output, oxygenation, and echocardiographic assessment. Specific weaning protocols are device-dependent, and we propose a similar slow-weaning protocol (decrement by support level every 2-4 hours) as the preferred method, unless clinical circumstance forces a more rapid (5-15–minute interval) wean. To note, weaning for continuous flow devices should only be done down to a nadir flow level of >1.5 L/min to avoid thrombotic complications. Similarly, the European Society of Cardiology recommended VA-ECMO de-escalation as soon as possible after hemodynamic and metabolic stabilization, with particular mention of inotrope de-escalation and a serum lactate level of <2 mmol/L.[103]

MCS considerations in RVF with LV failure

Although acute MI may involve the right ventricle, resulting in cardiogenic shock, this may be superimposed upon existing LV dysfunction. In addition, a scenario in which multivessel disease superimposed upon an RV infarction can further complicate the considerations of MCS because biventricular support may be needed. There is a myriad of strategies to facilitate biventricular support, each depending upon institutional and operator experience and comfort. VA-ECMO and Impella (EC-Pella) can be used in such a situation in which RV support is provided by unloading the right ventricle with venous drainage and an Impella device used to unload the left ventricle is used in combination. The latter is necessary to "vent" the left ventricle to avoid chamber distention, increased myocardial workload and pulmonary edema, and other undesirable physiologic consequences of pressurizing the aorta with VA-ECMO alone.[104-109] This strategy is necessary when severe hypoxia is also a dominant clinical feature. Another option is biventricular Impella use (BiPella) when hypoxic respiratory failure is not present. This involves placing an Impella RP with an Impella CP pump, a configuration that has been used successfully in several such circumstances.[110,111] In situations where LV support with an Impella is contraindicated (eg, mechanical aortic valve and LV thrombus), left-atrial venous arterial ECMO has demonstrated potential as management for patients with biventricular failure in cardiogenic shock with minimal direct complications from device implantation.[112] Lastly, ProtekDuo has been used with Impella 5.0/5.5 in patients with cardiogenic shock with biventricular failure with promising outcomes, including rapid extubation, mobilization, and physical exercise.[113] ProtekDuo can be used in concert with a left-sided TandemHeart system, although the large bore cannula burden in the vena cava and right atrium can become cumbersome. Advantages of the ProtekDuo device include mobility given its internal jugular access point and the ability to use an external oxygenator. However, there are still limited data on the use of these devices in the setting of acute MI, and the MCS choice depends heavily on the availability of resources in addition to operator preference.

Identifying RVF when a patient in cardiogenic shock is supported by a LV MCS device requires attention to right-sided filling pressures after LV support is initiated and absolute flow levels and patterns on the device. Rising CVP coupled with suction events or low-flow alarms on the LV support device are essential to monitor as warning signs for the development of RVF. We suggest continuous monitoring of the CVP/RA pressure with a PAC during LV support and with a CVP of >12 being an indicator of subclinical RVF. This parameter should prompt more aggressive decongestive therapies at a minimum if not strong consideration of RV MCS. If the CVP rise is accompanied by low flows and suction alarms on the LV device, pursuing concomitant RV MCS is prudent.[114] Interestingly, percutaneous LVAD support has not been shown to worsen RV adaptation, and in fact, RV load progressively declines during LV support.[115] This further implies that RVF occurring after surgical LVAD implantation is perhaps related to operative changes.

Conclusions

Post-MI RVF is associated with high morbidity and mortality. Initial intervention should be focused on revascularization based on the current American College of Cardiology/American Heart Association guidelines. In the setting of persistent RVF resulting in cardiogenic shock, a multidisciplinary team should be convoked for medical management and monitoring in the intensive care unit. MCS should be considered early as a bridge to recovery, and in some cases, advanced therapies should be considered. As more evidence becomes available, the use of MCS devices can play a major role in the management of acute MI RVF. However, the current MCS management of RVF after MI is highly dependent on institutional resources and operator preference, among other variables.

Currently, there are limited data on acute MI-induced RVF management. Many studies group RVF from MI with other common causes, including RVF after LVAD implantation, World Health Organization

group I or III pulmonary hypertension, and postcardiotomy RVF. Future directions include the need for investigation on MCS strategies and preferred agents for vasopressor and/or inotropic support for these patients, with emphasis on patients with RVF after MI. Preclinical animal models of RVF can also help to inform such clinical endeavors. Finally, more data will be needed on the use of the Impella RP device, TH-RVAD, ProTek dual lumen cannula, and VA-ECMO in patients with acute MI RVF. Current data on MCS devices in RVF after MI are scarce, and more real-world experience and data are needed. The right ventricle has a remarkable potential for recovery; therefore, it would stand to reason that adequate and timely support in the setting of RVF after MI can optimize recovery and decrease morbidity and mortality.

Declaration of competing interest

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethics statement

Institutional review board approval and informed consent were not required for this study as this is a review article.

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