Prevention and Treatment of Right Ventricular Failure During Left Ventricular Assist Device Therapy

Amresh Raina, MD*, Maria Patarroyo-Aponte, MD

KEYWORDS

• Left ventricular assist device • Right ventricular failure • Morbidity • Mortality
• Risk stratification • Treatment • Prevention

KEY POINTS

- Right ventricular failure is a frequently encountered clinical problem due to the increasing utilization of left ventricular assist devices (LVADs) for end-stage heart failure and expansion of the patient population eligible for LVAD therapy.
- The true incidence of right ventricular failure post-LVAD implantation has been challenging to define because of varying definitions in the literature and switch from pulsatile to continuous flow technology.
- Postoperative right ventricular failure may be predicted by preoperative clinical, hemodynamic, and imaging variables, which have been combined into a variety of risk prediction algorithms, although right ventricular failure may also develop due to unanticipated intraoperative and postoperative factors.
- Early recognition of right ventricular failure is critical because early institution of medical therapy for right heart failure and/or right ventricular mechanical circulatory support is associated with superior outcomes versus delayed treatment.
- Randomized clinical trial data are needed to support the use of specific medical and device therapy in patients with right ventricular failure post-LVAD implant.

INTRODUCTION

Left ventricular assist devices (LVAD) are used with increasing frequency in patients with heart failure with reduced ejection fraction (HFrEF) and advanced heart failure

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Cardiovascular Institute, Allegheny General Hospital, 320 East North Avenue, Pittsburgh, PA 15212-4772, USA
* Corresponding author. Pulmonary Hypertension Program, Section of Heart Failure/Transplant/MCS & Pulmonary Hypertension, Allegheny General Hospital, Temple University School of Medicine, 320 East North Avenue, Pittsburgh, PA 15212-4772.
E-mail address: amresh.raina@ahn.org

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(HF) symptoms despite maximally tolerated medical therapy.[1] Improvements in device design with smaller continuous flow (CF) pumps have improved device durability, and newer centrifugal pumps hold the promise of reducing serious adverse events, such as pump thrombosis.[2,3] With the increasing utilization of CF LVAD therapy and expansion of the patient population potentially eligible for LVAD implantation, right ventricular (RV) failure after LVAD implantation is more commonly encountered in clinical practice. The true incidence of RV failure after LVAD implantation is difficult to firmly establish because of varying definitions of RV failure in single-center studies and the shift from utilization of pulsatile to CF devices. Thus, estimates of the incidence of RV failure vary widely over a range from 10% to 40%.[4–7]

Severe RV failure after LVAD implantation and particularly requirement with a right ventricular assist device (RVAD) is associated with a substantial increase in morbidity and mortality and less successful bridging to cardiac transplantation.[1,5,6,8,9] Given that more LVAD patients with RV failure will be encountered clinically and the substantial impact RV failure has on LVAD outcomes, an important ongoing focus will be on strategies to identify and ideally prevent RV failure. In those patients who do develop RV failure, developing a treatment paradigm to improve outcomes remains a focus of discussion and continued research. In turn, a thorough understanding of mechanisms that are involved in the development of RV failure is required in order to prevent and treat it. This article reviews the following:

- The physiology underlying the development of RV failure in LVAD patients
- Established RV failure risk prediction algorithms
- Intraoperative and postoperative measures to try to prevent RV failure
- Management of patients who develop RV failure despite preoperative risk stratification, medical optimization, and aggressive perioperative treatment

DEFINITION OF RIGHT VENTRICULAR FAILURE

RV failure after LVAD implantation has been challenging to consistently define in the literature because there have been a variety of definitions in different single-center studies and most RV failure has been defined in relation to the index hospitalization for LVAD implant. The most consistently used and updated definition from the INTERMACs database characterizes RV failure as mild, moderate, or severe/severe-acute predominantly based on signs of elevated central venous pressure (CVP) and duration of inotropic/vasodilator support, need for RVAD implant, or death from RV failure after LVAD implant.[1] However, there is increasing recognition of a subset of patients who survive the index LVAD hospitalization without meeting criteria for severe RV failure but who subsequently present much later in their clinical course with symptomatic RV failure. These patients with “late” RV failure also have substantially increased morbidity and mortality.[10,11]

MECHANISMS OF RIGHT VENTRICULAR FAILURE

The development of RV failure can occur for a variety of reasons: some secondary to patient-related factors evident before LVAD implantation, others due to factors occurring during the intraoperative or perioperative course and additional issues that develop in the immediate postoperative period.

Preoperative Risk Factors

One of the biggest risk factors for postoperative RV failure is preoperative RV dysfunction. Biventricular dysfunction is common in patients with end-stage HFrEF of both ischemic and nonischemic causes and can occur due to the development of chronic secondary pulmonary hypertension (PH), left-sided valvular heart disease, particularly mitral regurgitation (MR), but also due to extension of the primary myopathic process that affects the left ventricle (LV) to the RV.[12–16]

Several parameters have been used to describe RV function (**Table 1**). RV dysfunction was initially defined by invasive hemodynamic parameters from preoperative right heart catheterization. Low RV stroke work index, defined as (mean pulmonary artery pressure [PA] – right atrial pressure/stroke volume index) is one such metric that has been associated with RV failure after LVAD implantation in single-center studies.[5,17] An even simpler hemodynamic measurement that can be calculated at bedside is CVP to pulmonary capillary wedge pressure (PCWP) ratio, with a ratio greater than 0.63 associated with development of postoperative RV failure.[6] Finally, a recent study in 132 CF LVADs suggested pulmonary arterial pulsatility index (PA systolic–PA diastolic/CVP) was additional hemodynamic metric superior to CVP:PCWP ratio, RV stroke work index, or CVP alone in predicting RV failure.[18]

Beyond invasive hemodynamic parameters, there has been increasing interest in echocardiographic assessment of preoperative RV function in prediction of postoperative RV failure. A variety of echocardiographic parameters have been associated with RV failure, predominantly derived again from relatively small single-center studies. For example, Puwanant and colleagues[19] demonstrated that tricuspid annular plane systolic excursion (TAPSE) at a cutoff of 8 mm was highly sensitive for predicting postoperative RV failure in a single-center study of mixed pulsatile and CF LVADs. Subsequently, a variety of additional echocardiographic measures of RV function, such as qualitative assessment of severe RV dysfunction, RV global free wall strain, and right ventricular fractional area change (RVFAC), have all been associated with post-LVAD RV failure in relatively small single-center studies.[5,20–22]

In addition to measurements of RV function, anatomic measurements of the right atrium, RV, and LV have been associated with RV failure. RV/LV ratio on intraoperative transesophageal echocardiogram or preoperative transthoracic echocardiogram, left atrial diameter to LV end-diastolic dimension, and left atrial volume index have also been associated with RV failure.[22–25] Finally, severe tricuspid valve regurgitation is another indirect echocardiographic surrogate of RV function that has been associated with RV failure post-LVAD implant.[20,26]

Table 1
Assessment of right ventricular function

Echocardiography	• Qualitative assessment • Right atrium and RV size • RV/LV ratio • Left atrium volume index • TAPSE • RVFAC • RV global free wall strain • Tricuspid regurgitation severity
Hemodynamics	• RA/PCWP ratio • PA pulsatility index • RV stroke work index

However, preoperative RV dysfunction is clearly not the only explanation for the development of RV failure after LVAD implant. Indeed, in many patients with preoperative RV dysfunction before LVAD implantation, RV function can improve post-LVAD implant because of the decongestion of the LV, lowering of left heart–filling pressures, and decrease in secondary PH and tricuspid regurgitation.[27] Therefore, beyond the assessment of preoperative RV dysfunction using hemodynamics or echocardiographic variables, additional preoperative laboratory, demographic, and clinical variables have been evaluated in RV failure risk prediction scores.

Predicting Right Ventricular Failure: Risk Scores

Using multivariate analysis, a variety of preoperative variables have been combined into RV failure risk prediction scores. Unfortunately, many of these algorithms were developed in the era of pulsatile flow LVADs, have used varying definitions of RV failure, have predominantly been derived from single-center studies without large validation cohorts, and have had modest predictive value.[4–6,20,26,28]

The Michigan RV failure risk score was perhaps the first of these algorithms to be developed and incorporated preoperative variables, including vasopressor requirement, renal dysfunction, elevated bilirubin, and aspartate aminotransferase levels. In this cohort, most patients had pulsatile flow LVADs. Area under the curve (AUC) for this risk score was 0.73.[28] Fitzpatrick and colleagues[5] studied a cohort of 167 patients with predominantly pulsatile flow LVADs and developed a score that predicted the need for biventricular mechanical circulatory support. Components to the score included preoperative cardiac index, RV stroke work index, severe RV dysfunction by qualitative assessment on echocardiogram, preoperative creatinine, and previous cardiac surgery. Another study by Drakos and colleagues[4] studied 175 patients again with predominantly pulsatile LVADs and showed that preoperative intra-aortic balloon pump, inotrope dependency destination therapy LVAD indication, β-blocker use, ACE inhibitor use, elevated pulmonary vascular resistance (PVR), and obesity were associated with RV failure. AUC for this score was similar to the Michigan score at 0.74.

In the more contemporary CF LVAD era, Kormos and colleagues[6] developed a preoperative score using the HeartMate II bridge to transplant data and found that CVP:PCWP ratio greater than 0.63, need for preoperative ventilatory support, or elevated blood urea nitrogen (BUN) greater than 39 mg/dL were associated with RV failure. Kato and colleagues[23] evaluated 111 patients with predominantly CF devices and found that LV diastolic dimension, left ventricular ejection fraction, left atrial diameter divided by LV diastolic dimension, bilirubin, and RV stroke work index were associated with RV failure with an AUC of 0.789. Atluri and colleagues[20] evaluated a contemporary cohort of 218 CF LVADs and found the elevated CVP greater than 15 mm Hg, qualitative severe RV dysfunction, preoperative intubation, severe tricuspid regurgitation, and preoperative tachycardia were associated with RV failure. Vivo and colleagues[25] and Kukucka and colleagues[24] evaluated a relatively simple echocardiographic metric of RV-to-LV end-diastolic ratio from either transthoracic echocardiogram or transesophageal echocardiogram at the time of LVAD implant with similar AUC of 0.68 and 0.74 to the more complex scores. Aissaoui and colleagues[29] combined several echocardiographic metrics, including RVFAC, basal RV end-diastolic diameter, RV tissue Doppler systolic velocity, and TAPSE with INTERMACs implant classification and found that a score greater than 3 predicted the occurrence of right ventricular failure with 89% sensitivity and 74% specificity. Bellavia and colleagues[30] recently published a meta-analysis of observational studies evaluating RV failure after LVAD implant with overall incidence of 35%. In this study, the investigators reported that the principal risk factors associated with RV failure were clinical

factors (need for mechanical ventilation, renal replacement therapy), biochemical markers (international normalized ratio and N-terminal prohormone of brain natriuretic peptide), hemodynamic measures (RV stroke work index and CVP), and echocardiographic measurements (preimplant moderate to severe RV dysfunction assessed qualitatively or a greater RV/LV diameter). Most recently, Kashiyama and colleagues[31] showed that addition of liver stiffness (a measure closely related to right-sided filling pressures) to other parameters can predict RV failure after LVAD with an AUC of 0.89.

Unfortunately, one of the problems with risk prediction scores is that they have not been well validated outside of their derivation cohorts, and their predictive value in validation cohorts is likely lower than in the derivation cohorts. Pettinari and colleagues[32] evaluated 3 of the previously published RV risk scores in a series of 59 LVAD implants and found that the risk scores were not significantly different in patients who needed temporary RV mechanical support versus those who did not. Kalogeropoulos and colleagues[33] evaluated 6 of the currently available RV failure risk prediction models and found that all of these algorithms performed modestly when applied to external populations outside their original derivation cohort. A more sophisticated approach to RV failure risk prediction may be using a Bayesian prognostic model that incorporates multiple preoperative risk factors as well as their interaction (**Table 2**). The AUC of the Bayesian model using data from the large INTERMACs registry was 0.90 for acute (<48 hours after implant) RV failure, 0.84 for early (48 hours to 14 days) RV failure, and 0.88 for late (>14 days) RV failure after LVAD implantation.[34]

Intraoperative and Perioperative Factors

In some patients, RV dysfunction can develop de novo after LVAD implantation, and in others, mild or moderate preoperative RV dysfunction can progress to frank RV failure due to factors that develop in the operating room or in the immediate perioperative period.

Durable LVAD implantation is still performed at most centers using full sternotomy and cardiopulmonary bypass. Cardioplegia can, despite myocardial protection, lead to relative stunning of the myocardium, particularly the RV, which is not directly unloaded by the LVAD postoperatively. In addition, cardiopulmonary bypass can cause cytokine release, systemic inflammatory response syndrome (SIRS), as well as elevations in PVR, which can stress an already dysfunctional RV.[35] Moreover, other intraoperative problems, such as myocardial ischemia, air embolism to the right coronary artery, mechanical compression of the PA, and tamponade, can all contribute to RV failure.[7]

LVAD circulatory physiology can also contribute to RV failure after implant. After LVAD implantation, there is increased flow from the LV/LVAD and increased venous return to the RV, resulting in an increase in RV preload. In addition, after LVAD

Table 2
Performance of Bayesian risk score for predicting right ventricular failure after left ventricular assist device implantation

Clinical Endpoint	Accuracy (%)	AUC (%)	Sensitivity (%)	Specificity (%)	No. of Variables
Acute RVF (<48 h)	97.3	90.3	80.0	99.7	33
Early RVF (>48 h to <14 d)	91.2	83.5	67.2	98.7	34
Late RVF (>14 d)	94.5	88.3	75.1	99.7	34

implantation, there is often loss of the septal contribution to overall RV function with postoperative paradoxic septal motion. In these patients, despite the objective decrease in ventricular afterload, the CVP remained unchanged and the ratio between CVP and PCWP worsened early after LVAD, suggesting poor RV adaptation early after LVAD with progressive improvement over time.[36] Finally, the increased venous return to the RV can in some instances, particularly with aggressive or rapid titration of LVAD speed, result in the interventricular septum being pulled toward LV, worsening right heart dimensions and tricuspid regurgitation.[25,27,36–38]

A variety of other perioperative or immediate postoperative factors can contribute to right HF. Acute hypoxemia perioperatively can result in pulmonary vasoconstriction, worsening PVR, and associated RV dysfunction. Acute renal dysfunction with secondary increase in CVP as well as metabolic and/or respiratory acidosis can contribute to a lack of vasopressor responsiveness.[39] Redo sternotomy with increased risk of perioperative bleeding and transfusion requirement has been associated with SIRS and worsening RV function. Sustained atrial and in particular ventricular tachyarrhythmias can also worsen RV function.[40]

PREVENTION OF RIGHT VENTRICULAR FAILURE

Preoperative Optimization

Other than patient selection and identification of patients at risk for RV failure using some of the risk stratification tools detailed above, preoperative optimization in patients who present with decompensated HF is imperative to prevent postoperative RV failure. The essential principles of preoperative management include optimization of preload, afterload, and contractility. Typically, diuretics should be used to maintain CVP less than 15 mm Hg if feasible. Inotropes such as milrinone or dobutamine should be used cautiously to optimize cardiac index and hemodynamics before LVAD implantation. These agents also have vasodilatory affects and so may improve systemic vascular resistance and to a lesser extent PVR. In patients who remain hemodynamically compromised, temporary mechanical support devices, such as an intra-aortic balloon pump (although associated with RV failure in some studies) or other percutaneous devices, such as Impella, may be used to optimize end-organ function. Correction of coagulopathy is also important in terms of minimizing postoperative bleeding and transfusion requirement. In patients with critical cardiogenic shock and INTERMACS 1 clinical profile, consideration should be made of bridging with temporary mechanical circulatory support or venoarterial extracorporeal membrane oxygenation (ECMO) to stabilize hemodynamics end-organ function before consideration of durable LVAD.

Intraoperative Optimization

As detailed above, a variety of intraoperative factors may result in worsening right heart function. Surgical technique with limitation of cardiopulmonary bypass time and careful myocardial preservation can help to diminish risk of RV stunning with cardiopulmonary bypass. Careful attention to bleeding can limit the requirement for transfusion, which may prevent SIRS and transfusion-related lung injury. Meticulous attention to proper deairing could also help to avoid air embolism to the right coronary artery. With close attention to acid-base balance and ventilatory status, acidosis can be avoided as can hypoxemia and hypercapnia. Delayed sternal closure can also be used in LVAD patients, particularly patients with previous cardiac surgery and coagulopathy, and this may ameliorate risk of cardiac tamponade and mechanical compression of the RV and PA.

In the operating room, a decision must often be made with regards to repair of the tricuspid valve because tricuspid regurgitation can worsen after LVAD implant because of leftward interventricular septal shift.[41] Moreover, severe tricuspid regurgitation has been associated with the development of postoperative RV failure.[20,26] Some studies have suggested that tricuspid repair is associated with decreased risk of RV failure and improved morbidity/mortality. However, a large meta-analysis suggested no benefit of routine tricuspid valve repair in terms of reducing early mortality or need for RVAD in patients with moderate to severe tricuspid regurgitation.[42]

With regards to MR, in general, with LV decompression with the LVAD, MR decreases, and consequently, there has not been much impetus to repair or replace the mitral valve at the time of LVAD implantation. However, a recent study suggested that patients with residual MR after LVAD implantation had larger postoperative RV dimensions, worse postoperative RV function, and adverse clinical outcomes (**Table 3**).[43] A recent small observational study also showed that overall survival and freedom from recurrent MR were significantly better in patients who underwent surgical repair of MR at the time of LVAD.[44] Despite these data, the role of mitral repair remains controversial in LVAD patients, and literature on this topic is still developing.

Cautious optimization of LVAD speed is critically important in the operating room immediately postoperatively. Overly aggressive titration of LVAD speed can cause shift of the ventricular septum toward the LV, impacting RV performance, and can also potentially cause ventricular tachycardia via contact of the ventricular septum to the LVAD inflow cannula. In the operating room, LVAD speed should be cautiously titrated using transesophageal echocardiographic guidance to optimize aortic valve opening and septal position.[24,39]

Postoperative Measures

In the immediate postoperative period, typically patients should be carefully managed using hemodynamic guidance. Inotropes such as dobutamine or milrinone are typically used to optimize cardiac output and LVAD filling. In patients with relative hypotension, epinephrine and to a lesser extent norepinephrine are commonly used.

Avoiding hypoxemia and hypercapnia is critically important in the postoperative period. In patients with severe intraoperative or perioperative hypoxemic,

Table 3
Relationship of residual mitral regurgitation after left ventricular assist devices implant to right ventricular dysfunction and clinical outcomes

Parameter	Preoperative Data (n = 69)	Residual MR Cohort (n = 14)	No Residual MR Cohort (n = 55)	*P* Value
RVEDD (mm)	48 ± 8	49 ± 6	45 ± 9	.04
TAPSE (mm)	14 ± 4	10 ± 2	12 ± 3	.02
RVFAC (%)	26 ± 12	29 ± 5	34 ± 9	.02
Tricuspid regurgitation jet (mm Hg)	38 ± 13	26 ± 7	25 ± 10	.79
PA systolic pressure (mm Hg)	51 ± 14	35 ± 9	34 ± 11	.85
RVOT VTI (cm)	12 ± 4	13 ± 4	16 ± 12	.13
Time from LVAD implant to first hospitalization (d)	—	62 ± 34	103 ± 112	.05
Time from LVAD implant to death (d)	—	80 ± 11	421 ± 514	.03

Abbreviations: RVEDD, right ventricular end diastolic dimension; VTI, velocity time integral.

consideration of venovenous ECMO should be made. Maintaining adequate oxygenation and ventilation is also important after extubation, and supplementary oxygen and/or noninvasive positive-pressure ventilation may be used in these situations.

Inhaled nitric oxide has been frequently used in patients with preoperative PH and/or RV dysfunction. A prospective, randomized clinical trial of inhaled nitric oxide in 150 patients undergoing LVAD implant with elevated PVR did not suggest overall benefit in terms of prevention of RV failure or mortality, but there was a signal toward benefit in patients with elevated PA pressures and PVR.[45] Unfortunately, although inhaled vasodilatory agents have the potential to decrease pulmonary pressures in the postoperative period with low incidence of RVF and improvement in survival when used early, there is a still lack of large randomized clinical studies to support their use as well as guidance regarding doses and agent selection.[46] In this regard, Sabato and colleagues[47] propose that the medical treatment of RV failure should be based on the presence or absence of elevated PVR. Thus, if RV failure occurs in the setting of normal PVR, inotropic agents such as dobutamine should be used, but if the PVR is elevated (>3 Wood units) or the patient has a transpulmonary gradient higher than 12 mm Hg, then an inhaled pulmonary vasodilator agent should be considered.

TREATMENT OF RIGHT VENTRICULAR FAILURE

Available Medical Treatment Options for Right Ventricular Failure Are Categorized

Acute and early right ventricular failure

While treating RV failure, therapy should be directed toward the same physiologic principles as in prevention namely optimization of preload, afterload, and contractility (Table 4). Device parameters should be reviewed carefully to understand the interaction between the LVAD and the patient's heart, including LV dimensions, septal position, aortic valve opening, MR, pump speed, and assessment of RV function.

Table 4
Treatment of right ventricular failure

	Acute RV Failure (<48 h)	Early RV Failure (48 h to <14 d)	Late RV Failure (>14 d)
RV preload	Optimize volume status with diuresis, ultrafiltration, transfusion	Optimize volume status with diuresis, ultrafiltration	Optimize volume status with diuresis
RV afterload	Inhaled nitric oxide	Inhaled prostanoid or oral PDE5 inhibitors	Oral PDE5 inhibitors
RV contractility	Epinephrine	Dobutamine or milrinone	Digoxin
Arrhythmias	Manage atrial and ventricular arrhythmias	Manage atrial and ventricular arrhythmias	Manage atrial and ventricular arrhythmias
Mechanical support	V-A and/or V-V ECMO, TandemHeart, RP Impella	TandemHeart, RP Impella	
LVAD parameters		Optimize LVAD parameters	Optimize LVAD parameters with echocardiographic and/or invasive hemodynamic ramp study

Patients with RV failure will typically have right heart congestion with elevated CVP and elevated right heart preload, impacting septal position and LVAD filling. Therefore, RV failure results in low indices of LVAD pulsatility on waveform analysis or pulsatility index. In this situation, typically aggressive diuresis and potentially ultrafiltration are required for volume removal to achieve CVP less than 15 mm Hg. In terms of lowering RV afterload, pulmonary vasodilating therapy, typically with inhaled nitric oxide or inhaled prostacyclins, is commonly used if PA pressures and PVR are elevated. Alternatively, phosphodiesterase type 5 (PDE5) inhibitors such as sildenafil may be used in patients who are not profoundly hypotensive, although the evidence supporting their use in the treatment of these patients is weak and mostly based on small studies and retrospective cohorts.[45,48–50] In terms of optimizing contractility and cardiac output, inotropic therapy, typically with dobutamine or milrinone, is often needed to optimize end-organ perfusion as well as LVAD filling. Epinephrine, norepinephrine, and dopamine are sometimes used in patients who are hypotensive.[41]

In patients with severe acute RV failure, consideration of early use of a temporary mechanical support device should be made. Studies have shown that planned or early RVAD implantation is associated with superior outcomes versus delayed or rescue placement, specifically with addition of in-line ECMO, which decreases 30-day mortality compared with patients that underwent RVAD alone.[51–53] Fortunately, there are an increasing number of options for temporary RVAD support. Centrally cannulated devices include centrifugal pumps, such as the CentriMag (Abbott, St Paul, MN) or Revolution (Sorin, Arvada, CO). However, there are now additional percutaneous RV support devices that are available, such as RV tandem heart (CardiacAssist, Pittsburgh, PA) or RP Impella (Abiomed, Danvers, MA) percutaneous devices.[54,55] The goal of temporary mechanical support placement is to support the failing RV and preserve LVAD filling and end-organ perfusion in the hopes that there will be gradual and consistent improvement in RV function, which will allow weaning of the RV support device, although in some patients use of temporary RVAD for more than 7 days with blood flow greater than 4 L/min has been associated with pulmonary hemorrhage.[56] In some patients, however, RV failure is persistent and consideration of a durable RVAD can be made in patients who are not likely to be emergently transplanted. In this situation, there have been numerous reports of utilization of a right-sided HeartWare CF device, cannulated either to the right atrium or to a portion of the RV and with outflow graft connected to the PA.[57,58]

Late right ventricular failure

The bulk of the established literature as detailed above has focused on the development of RV failure in the immediate postoperative period. However, there is much less known about the development of late RV failure, but this may be of increasing clinical importance. In some patients, late RV failure can develop secondary to inadequate unloading from the LVAD, which in turn can occur due to mechanical LVAD dysfunction, aortic or mitral regurgitant lesions, or inadequate blood pressure management. However, in other patients, primary RV dysfunction/RV failure occurs with normal LVAD function.

Again, there has been fairly little consensus with regards to what defines truly “late” RV failure. In a single-center study by Kapelios and colleagues,[10] at a median of 2.1 years after LVAD implant, 45% of patients developed signs or symptoms of right HF. In this study, there was no association between preoperative variables in those that developed late RV failure versus those who did not. A further 44% of the patients who developed late RV failure subsequently died during follow-up, illustrating the impact of late RV failure on outcomes. In another cohort studied by Takeda and

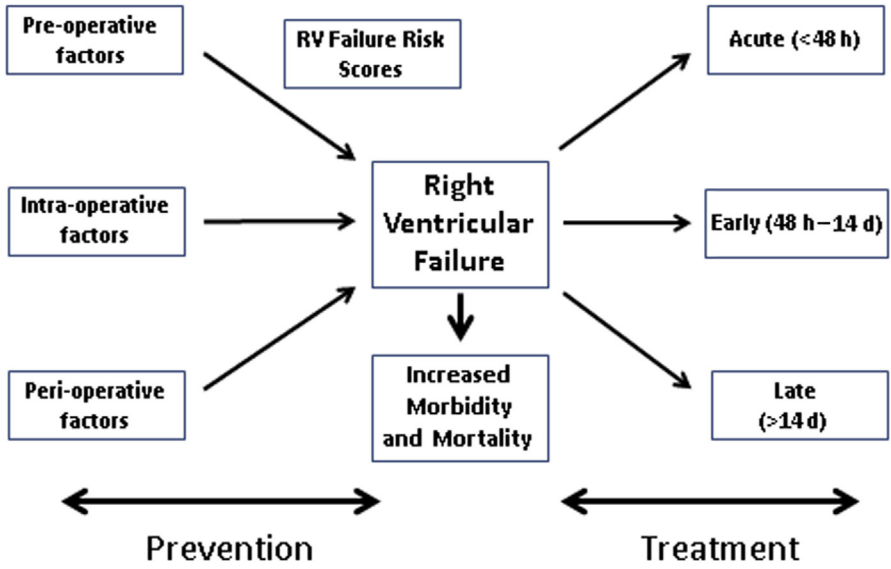

Fig. 1. RV failure in LVAD patients.

colleagues[11] with CF LVADs, 11% developed late right HF at a median of 99 days after discharge after LVAD implant. Several preimplant variables, including body mass index greater than 29, BUN greater than 41, and diabetes mellitus, were significant predictors of late right HF in this study. Of note, late RV failure during LVAD support has been also associated with worse 5-year posttransplant survival compared with patients who did not develop RV failure.[59]

In these patients, pump speed optimization after LVAD by echocardiographic and/or invasive hemodynamic RAMP study can lead to improvement in RVFAC and RV longitudinal peak systolic strain, suggesting that speed optimization in stable LVAD patients can improve RV function.

SUMMARY

Although CF LVADs offer the promise of smaller and more reliable devices designed for long-term hemodynamic support, RV failure is still a problem in more than one-third of patients early after LVAD implant (**Fig. 1**). RV failure either early or late after LVAD implant is associated with a substantial increase in morbidity and mortality, even for LVAD patients who are candidates for cardiac transplantation. Prevention of RV failure may be achieved through careful patient selection, through use of preoperative risk prediction tools to identify more appropriate LVAD candidates, and by careful preoperative optimization and perioperative management. Early recognition of RV failure is key to improving outcomes in these patients independent of the therapy used (either medical therapy or mechanical circulatory support or both). There is nonetheless a need for large, randomized studies to evaluate the currently available therapies for RV failure, including inhaled vasodilatory agents and temporary mechanical support.

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