A Practical Guide to Intensive Care Unit Management after Left Ventricular Assist Device Implantation

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KEYWORDS

• Left ventricular assist device (LVAD) • Post-operative ICU management • Right ventricular failure
• LVAD hemodynamics • LVAD troubleshooting • Anticoagulation and hemostasis
• Continuous-flow LVAD physiology • Pulmonary management after cardiac surgery

KEY POINTS

- Early intensive care unit (ICU) management after left ventricular assist device (LVAD) implantation is physiology-driven, with outcomes heavily influenced by hemostasis, afterload control, preload optimization, and right ventricular (RV) adaptation during the first post-operative days.
- Continuous-flow LVAD performance is highly preload-dependent and afterload-sensitive, requiring strict blood pressure control and frequent reassessment of volume status, RV function, and device parameters rather than reliance on pump flow alone.
- Acute RV failure is a dominant cause of early morbidity, driven by increased venous return and septal shift after LV unloading; prompt recognition and targeted management of preload, afterload, LVAD speed, and pulmonary vascular resistance are critical.
- Anticoagulation requires staged initiation, balancing bleeding risk against thrombotic complications, with goal-directed correction of coagulopathy and cautious escalation from parenteral to oral therapy.
- Ventilatory and supportive ICU strategies directly affect LVAD performance, as positive-pressure ventilation, excessive positive end-expiratory pressure, and hypoxia increase RV afterload and reduce LVAD preload, underscoring the importance of lung-protective ventilation, early extubation, nutrition, and mobilization.

INTRODUCTION

Durable left ventricular assist devices (LVADs) improve survival and quality of life in patients with stage D heart failure who require short-term or long-term mechanical circulatory support.[1–3] Contemporary 1 year survival after LVAD implantation is comparable to that of heart transplantation.[4] However, morbidity remains high, with the greatest risk of death and major adverse events occurring within the first 30 days after implantation.[5]

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Abbreviations

CI	cardiac index
ICU	intensive care unit
LVAD	left ventricular assist device
MAP	mean arterial pressure
PAPi	pulmonary artery pulsatility index
PEEP	positive end-expiratory pressure
PI	pulsatility index
RV	right ventricular

The immediate post-operative intensive care unit (ICU) period following LVAD implantation is characterized by physiologic vulnerability. Because early ICU management decisions may have disproportionate effects on short-term and long-term outcomes, care during this phase is delivered via a structured, multidisciplinary approach with coordination among critical care, heart failure cardiology, cardiothoracic surgery, perfusion, and nursing teams. Priorities include hemostasis, hemodynamic stabilization, LVAD speed optimization, and monitoring of right ventricular (RV) function.[6] Following stabilization, management goals shift to respiratory management with early extubation, infection prevention, nutrition, mobilization, LVAD education, and transition to the next level of care (**Table 1**).

This practical guide outlines contemporary management of the acute post-implant LVAD patient, emphasizing physiology-driven critical care and a systematic approach to identifying and managing LVAD-related complications from the operating room through discharge from critical care.

HEMOSTASIS AND ANTICOAGULATION

Hemostasis

Coagulation management following LVAD implantation requires balancing post-operative bleeding risk against preservation of hemodynamic stability and future transplant candidacy. Peri-operative hemostasis is frequently impaired by preexisting antithrombotic therapy, end-organ dysfunction, nutritional frailty, and cardiopulmonary bypass-related physiologic stress, creating a pro-hemorrhagic early post-operative state.[7,8]

Initial management focuses on meticulous surgical hemostasis and early hemodynamic optimization. Maintaining relatively low arterial and venous pressures in the immediate post-operative period reduces microvascular bleeding and supports clot stabilization, particularly in patients with tissue fragility or cardiac cachexia. Persistent bleeding should prompt early differentiation between surgical bleeding and systemic coagulopathy through multidisciplinary evaluation.[9,10]

Correction of coagulopathy should be goal-directed rather than empirical. Key targets include normalization of INR and PTT, fibrinogen greater than 2 g/L, platelet count greater than 100,000/μL, and adequate ionized calcium. Viscoelastic testing, when available, facilitates targeted therapy and limits unnecessary transfusion. Adjunctive agents such as desmopressin, low-dose protamine, and antifibrinolytics may be used selectively, while a low threshold for surgical re-exploration is essential when bleeding persists despite correction of coagulopathy.[10]

Blood product transfusion remains a cornerstone of early post-operative hemostasis but must be judicious. Excessive transfusions may increase central venous pressure, worsen RV distension, and contribute to alloantibody sensitization in patients who may later undergo transplantation.[7,8]

Once hemostasis is achieved, anticoagulation can be cautiously initiated with close reassessment, recognizing that device-related acquired von Willebrand syndrome and platelet dysfunction continue to influence bleeding risk throughout LVAD support.[9]

Anticoagulation in the Immediate Post-operative Phase

Systemic anticoagulation is typically deferred until adequate hemostasis is achieved. Chest tube output trends and coagulation parameters should guide initiation. In patients with normal coagulation, unfractionated heparin may be started 12 to 24 hours post-operatively or when chest tube drainage is consistently less than 50 mL/hr. Unfractionated heparin is preferred because of its short half-life and reversibility. Conservative initial targets are recommended, with a PTT goal of 40 to 50 seconds for the first 24 hours.[11,12] If no overt bleeding occurs, the heparin dose is escalated to a PTT of 50 to 60 seconds after 24 hours, then 55 to 65 seconds after an additional 24 hours. In patients with heparin intolerance or heparin-induced thrombocytopenia, direct thrombin inhibitors such as argatroban or bivalirudin are acceptable alternatives but require careful monitoring due to limited reversibility and sensitivity to hepatic or renal dysfunction.[13]

Transition to Long-Term Anticoagulation

Between post-operative days 3 and 5, if there is no evidence of bleeding and chest tubes have been removed, warfarin may be initiated with unfractionated heparin overlap. Warfarin remains the standard oral anticoagulant for contemporary LVAD platforms, targeting an INR of 2.0 to 3.0. Parenteral anticoagulation is continued until a therapeutic INR is achieved.[12,14,15] Antiplatelet therapy is

Table 1
Goals of intensive care unit care following left ventricular assist device implantation

Domain	Primary Goals	Key Interventions/ Considerations
Hemostasis and Anticoagulation	Balance hemorrhagic and thrombotic risk	Monitor coagulation labs Coagulopathy should be corrected in the early post-operative period Monitor chest tube output Initiate protocolized anticoagulation
Hemodynamic Assessment and Management	Maintain systemic perfusion Optimize preload and afterload	Monitor MAP Monitor filling pressures Monitor end-organ perfusion Titrate fluids, vasopressors and inotropes
Monitoring and Optimizing Right Ventricular Function	Preserved RV function Prevent RV failure	Optimize preload and afterload Manage pulmonary vascular resistance Inotropes and pulmonary vasodilators, as needed Early recognition of RV-LVAD mismatch
Optimizing LVAD Performance	Ensure proper device function Prevent complications	Monitor pump flow, power, and alarms Monitor for PI events Adjust pump speed, as indicated
Ventilator and Respiratory Management	Maintain oxygenation/ ventilation Reduce RV afterload Enable early extubation	Lung-protective ventilation Minimize intrathoracic pressure Early extubation when hemodynamically stable
Nutrition	Optimize nutritional support to promote recovery and wound healing while minimizing volume-related and gastrointestinal complications	Initiate early enteral nutrition when hemodynamically stable Ensure adequate caloric and protein delivery while avoiding overfeeding Monitor for ileus or impaired gastrointestinal absorption
Mobilization	Minimize deconditioning and facilitate functional recover	Initiate early physical therapy as clinically feasible Secure driveline and LVAD equipment prior to activity Monitor for suction events, arrhythmias, or hypotension during mobilization
Multidisciplinary Care & Transition Planning	Coordinate multidisciplinary care Prepare for ICU discharge	Integrate cardiology, surgery, critical care, perfusion, nursing, nutrition, mobilization, and patient/ caregiver education

Abbreviations: ICU, intensive care unit; LVAD, left ventricular assist device; MAP, mean arterial pressure; RV, right ventricle; SIRS, systemic inflammatory response syndrome.

device-specific, and routine aspirin use is no longer recommended for patients supported with HeartMate 3 LVADs.[16]

Chronic Anticoagulation

Patients are generally maintained on indefinite warfarin therapy with a target INR of 2.0 to 3.0. Anticoagulation strategies should be reassessed in response to bleeding, hemolysis, arrhythmias, invasive procedures, or changes in device performance. Close multidisciplinary coordination is essential to minimize bleeding while reducing pump thrombosis risk[11,14] (**Fig. 1**).

Key Practical Considerations

- *Before leaving the OR:* fully reverse anticoagulation, targeting INR less than or equal to 1.3, PTT less than or equal to 35 sec, platelets greater than 100,000/μL, and fibrinogen greater than 2 g/L
- *Initiate IV heparin* at 12 to 24 hours postoperatively or once chest tube output is less than 50 mL/hr sustained over 2 to 3 hours
  - First 24 hours: target *PTT 45 to 50 seconds*
  - Next 24 hours: target *PTT 50 to 60 seconds*
  - Thereafter: *target PTT 55 to 65 seconds* if no bleeding
- *Transition to oral* anticoagulation*:* start warfarin on post-operative day 3 to 5 after chest tube removal and bleeding stabilization, overlapping with heparin
- *Chronic anticoagulation target*: maintain *INR 2.0 to 3.0*; discontinue heparin once INR is stable and therapeutic

HEMODYNAMIC ASSESSMENT AND MANAGEMENT

Left Ventricular Assist Device Impact on Hemodynamics

Continuous-flow LVADs fundamentally alter cardiovascular physiology by decoupling systemic perfusion from native left ventricular contractility, rendering cardiac output predominantly dependent on loading conditions.[7,17,18] Flow is generated by transfer of kinetic energy to blood via a rapidly rotating impeller rather than by positive displacement, and therefore is not delivered as a fixed stroke volume.[14,18]

At a given pump speed, LVAD flow is primarily determined by the trans-pump pressure differential (ΔP), defined as the difference between left ventricular pressure at the inflow and aortic pressure at the outflow (ΔP = LVP − AoP).[18–20] This pressure head represents the hydraulic load opposing pump flow and varies inversely with flow. Physiologic conditions that increase ΔP—such as elevated afterload or reduced preload—decrease LVAD flow, whereas conditions that reduce ΔP—such as vasodilation or increased preload—augment flow.[17,18,20] The relationship between pump flow (Q) and pressure head (H) is described by the device-specific H-Q curve, with a family of curves corresponding to different operating speeds.[14,19,20] Accordingly, contemporary continuous-flow LVADs are relatively preload-dependent and highly afterload-sensitive. At a fixed pump speed, flow is governed primarily by loading conditions—preload and afterload—rather than intrinsic myocardial contractility. From a clinical standpoint, elevations in mean arterial pressure (MAP) or reductions in intravascular volume lead to decreased pump flow, whereas vasodilation or volume repletion increase flow without change in pump speed[7,17,18,20] (**Fig. 2**).

Post-operative Hemodynamic Goals

The primary objective of post-operative hemodynamic management following LVAD implantation is to maintain adequate end-organ perfusion while minimizing venous congestion and facilitating RV adaptation to altered loading conditions. Early post-operative physiology is highly dynamic, shaped by bleeding risk, vasoplegia, systemic inflammatory stress, and evolving RV function. As a result, continuous reassessment of perfusion, congestion, and RV performance is essential. A structured, physiology-based management strategy helps reduce venous congestion, prevent end-organ dysfunction, and shorten length of stay.[21,22]

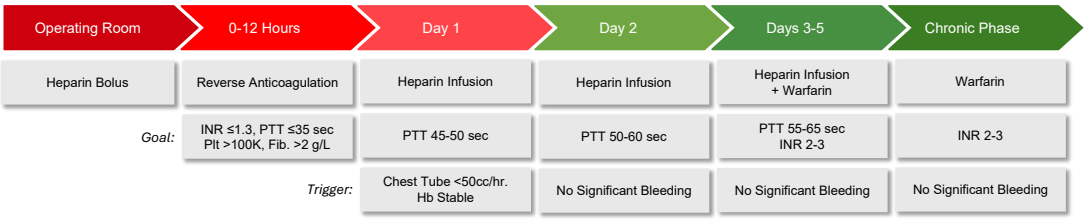

Fig. 1. Anticoagulation timeline: OR to discharge. Fib, fibrinogen; INR, international normalized ratio; Plt, platelet count; PTT, partial prothrombin time.

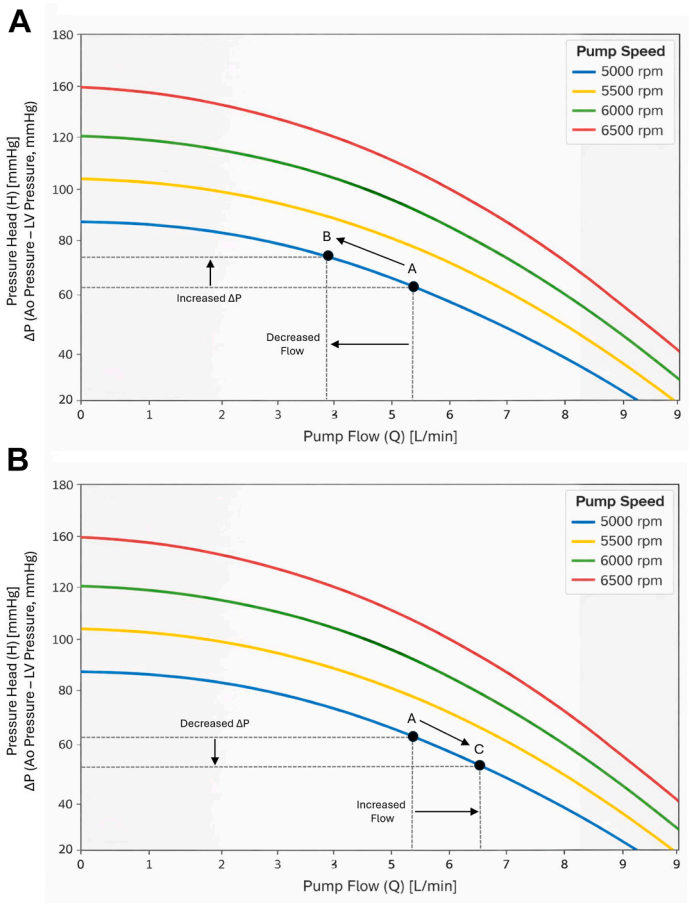

Fig. 2. Effect of loading conditions on LVAD pressure–flow relationships. (*A*) (Increasing afterload or decreasing preload): At a constant pump speed (*blue curve*), an increase in afterload or a decrease in preload (higher ΔP) shifts the operating point from A to B, resulting in a reduction in pump flow despite no change in LVAD speed. (*B*) (Decreasing afterload or increasing preload): Conversely, a reduction in afterload or an increase in preload (lower ΔP) shifts the operating point from A to C along the same speed curve, leading to an increase in pump flow. This figure illustrates the intrinsic afterload sensitivity and preload dependence of continuous-flow LVADs. LVAD, left ventricular assist device.

Mean Arterial Pressure and Afterload

In the immediate post-operative period, systemic afterload should be titrated to maintain an MAP of 60 to 70 mm Hg until surgical hemostasis is achieved. Once bleeding risk stabilizes, a MAP target of 65 to 85 mm Hg is generally appropriate to support LVAD flow and end-organ perfusion. Sustained MAP values above 85 mm Hg should be avoided, as excessive afterload can impair LVAD flow and increase bleeding risk.[21–23] Vasoplegia is common after continuous-flow LVAD implantation and may persist for hours to days.[24] In this setting, an intravenous vasopressin infusion is often preferred because it increases systemic vascular resistance while exerting relatively minimal effects on pulmonary vascular resistance and RV afterload.[25]

Cardiac Output and Perfusion

Adequate forward flow is fundamental to meeting post-operative metabolic demands. A cardiac index (CI) greater than or equal to 2.2 L/min/m^2 is commonly used as a minimum target for perfusion; however, higher targets may be necessary in vasoplegia or inflammatory states, where systemic oxygen demand is increased and effective perfusion may be inadequate despite apparently acceptable LVAD flow. Mixed venous oxygen saturation (SvO_2) should generally exceed 60%, with lower values indicating impaired oxygen delivery even when filling pressures and arterial pressures appear satisfactory.

Suspected low cardiac output in an LVAD recipient should trigger a multimodal evaluation integrating echocardiography, invasive hemodynamics, and LVAD parameters, each contributing complementary information regarding preload, afterload, ventricular function, and device performance. Serum lactate and lactate clearance provide additional insight into global tissue perfusion and should normalize or decline with adequate support. When guiding escalation or de-escalation of therapy, trends in CI, SvO_2, and lactate are more informative than isolated measurements[21–23] (**Fig. 3**).

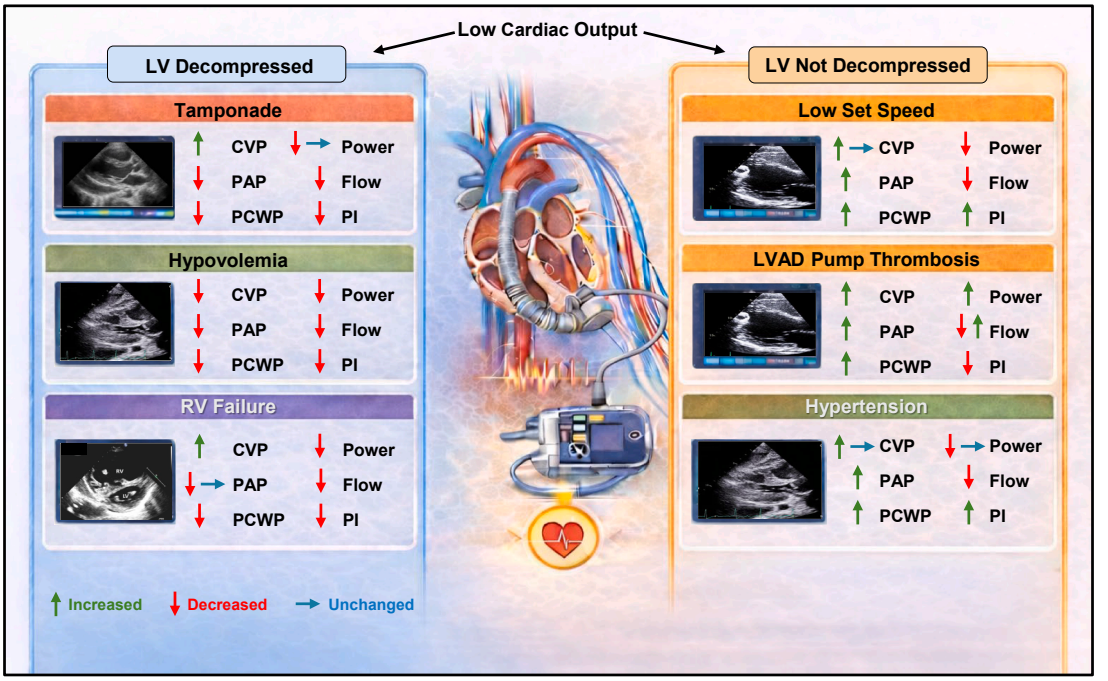

Fig. 3. Multimodality assessment of low cardiac output after LVAD implantation. Integrated echocardiographic, invasive hemodynamic, and LVAD console data used to differentiate common causes of low cardiac output in continuous-flow LVAD patients. LV cavity size (decompressed vs not decompressed) is determined by echocardiography; CVP, PAP, and PCWP are obtained from invasive hemodynamic monitoring; and LVAD power, flow, and PI are derived from the LVAD console. Etiologies are stratified as LV decompressed (tamponade, hypovolemia, RV failure) or LV not decompressed (low pump speed, pump thrombosis, hypertension). Arrows denote direction of change relative to baseline (*green* = increased, *red* = decreased, *blue* = unchanged). CVP, central venous pressure; LVAD, left ventricular assist device; PAP, pulmonary artery pressure; PCWP, pulmonary capillary wedge pressure; PI, pulsatility index; RV, right ventricular.

Use of Inotropic Support

RV dysfunction after LVAD implantation may improve with effective left ventricular unloading, although severe pre-existing RV failure may not fully recover. During the early post-operative period, many patients require temporary inotropic support as the right ventricle adapts to increased preload and altered septal geometry. Milrinone is commonly used because of its combined inotropic and pulmonary vasodilatory effects, whereas dobutamine or epinephrine may be preferred when hypotension limits the use of phosphodiesterase-3 inhibitors. Maintenance of sinus rhythm and atrioventricular synchrony is essential for optimizing RV output and systemic perfusion.[22,26]

Right Ventricular Afterload and Pulmonary Vasodilators

Selective pulmonary vasodilators, including inhaled nitric oxide and prostacyclins, are often initiated intraoperatively or early in the post-operative period to reduce RV afterload while minimizing systemic hypotension. Elevated pulmonary artery pressures may be acceptable provided that cardiac output, SvO_2, and LVAD flows remain adequate and venous congestion is controlled. In contrast, low pulmonary artery pressures accompanied by a reduced CI and declining SvO_2 may reflect worsening RV failure rather than hemodynamic improvement. Persistent pulmonary hypertension should prompt distinction between pre-capillary and post-capillary physiology, ideally guided by pre-operative right heart catheterization data. Difficulty weaning inhaled pulmonary vasodilators may signal evolving RV dysfunction or fixed pulmonary vascular disease.[22,27]

Preload

Optimizing preload requires integration of clinical examination, invasive hemodynamic monitoring, echocardiography, and markers of tissue perfusion. Central venous pressure targets should be individualized, although values of approximately 8 to 12 mm Hg are commonly acceptable in

patients without severe RV dysfunction. Pulmonary capillary wedge pressure targets typically range from 8 to 15 mm Hg, recognizing that higher values may be necessary in patients with impaired ventricular compliance. Echocardiographic evidence of RV dilation, septal flattening, or worsening tricuspid regurgitation should prompt avoidance of further volume loading.[22,26,27]

Response to Acute Hemodynamic Instability

When hypotension is reported in a patient with a continuous-flow LVAD, the first priority is to verify blood pressure accuracy, as oscillometric measurements are often unreliable in low-pulsatility states. Doppler-derived MAP correlates closely with invasive arterial measurements and should be used routinely for assessment. Once accuracy is established, hypotension—defined as a MAP below 60 mm Hg—should prompt immediate evaluation of preload, afterload, RV function, LVAD performance, SvO_2, and serum lactate. Echocardiography plays a central role in identifying reversible causes of hemodynamic instability. Early, physiology-directed intervention is critical to preventing progression to RV failure and multiorgan dysfunction.[22,27]

Key practical considerations

- *Perfusion goals:* target MAP 60 to 70 mm Hg initially, then 65 to 85 mm Hg after hemostasis; avoid greater than 85 mm Hg. Target CI greater than 2.2 L/min/m^2, SvO_2 greater than or equal to 60%, and normalizing or declining lactate
- *Vasoplegia:* common post-operatively; vasopressin preferred to increase SVR with minimal RV afterload; may add norepinephrine as needed
- *RV support:* anticipate temporary inotropic support; use milrinone as tolerated, dobutamine or epinephrine if hypotension limits PDE-3 inhibition; maintain sinus rhythm and AV synchrony
- *Pulmonary vasodilators:* inhaled nitric oxide or prostacyclins for RV afterload reduction; may accept elevated PA pressures if CI, SvO_2, and LVAD flows are preserved; suspect RV failure if low PA pressures are accompanied by low CI/SvO_2
- *Preload management:* individualize targets; typical ranges CVP ~8 to 12 mm Hg and PCWP ~8 to 15 mm Hg; avoid volume loading in the setting of RV dilation, septal flattening, or worsening tricuspid regurgitation on echocardiography
- *Response to hemodynamic instability:* define hypotension as MAP less than 60 mm Hg (Doppler assessment preferred); evaluate preload, afterload, RV function, LVAD flow, SvO_2, and lactate; use echocardiography early to identify reversible causes

RIGHT VENTRICULAR FUNCTION AFTER LEFT VENTRICULAR ASSIST DEVICE IMPLANTATION

Pre-operative Right Ventricular Risk Stratification and Biventricular Support Planning

Pre-operative assessment of RV reserve is an essential component of patient selection for durable LVAD implantation and should precede implantation in all candidates. Post-operative RV failure remains common and contributes disproportionately to early morbidity and mortality.[26–28] Accordingly, pre-operative risk stratification must not only identify patients at increased risk but also inform a structured strategy for perioperative support.

A central consideration is the balance between an LVAD-first strategy—with predefined criteria for escalation to temporary right-sided support—and planned biventricular mechanical circulatory support in patients with limited RV reserve. Observational data consistently demonstrate worse outcomes when RV failure necessitates delayed or emergent RVAD implantation following isolated LVAD placement, compared with cases in which biventricular support is planned or instituted early.[29] These findings underscore the importance of proactive strategy selection. Multidisciplinary discussion should occur before surgery, with explicit agreement on hemodynamic thresholds for intraoperative or early post-operative RVAD implantation. These criteria should be established a priori rather than determined reactively in the setting of hemodynamic deterioration.[27]

Risk stratification for post-operative RV failure is best conceptualized as 3 complementary domains: hemodynamic, echocardiographic, and clinical or biochemical.[26] Hemodynamic assessment via right heart catheterization remains foundational. Elevated right atrial pressure, increased pulmonary vascular resistance, and reduced pulmonary artery pulsatility index (PAPi) reflect impaired RV-pulmonary circulation coupling and diminished RV reserve.[28,30] Among these, PAPi is the most readily accessible and clinically actionable single metric. A value below approximately 1.0—particularly when accompanied by echocardiographic evidence of RV dysfunction—should prompt formal multidisciplinary consideration of planned biventricular support.[30]

Several predictive tools have been developed to estimate the risk of post-operative RV failure,

including the Right Ventricular Failure Risk Score, EUROMACS-RHF score, Michigan score, and PAPi-based assessments, with emerging interest in echocardiographic strain parameters.[31,32] While these models demonstrate moderate discriminatory performance, their principal utility lies in structuring clinical reasoning and facilitating communication across multidisciplinary teams. They should not be used as standalone decision algorithms.[26]

In addition to formal risk assessment, clinicians must recognize disease substrates in which isolated LVAD therapy—or mechanical circulatory support more broadly—may be unlikely to yield durable benefit. These include advanced infiltrative cardiomyopathies, inflammatory cardiomyopathies, severe fixed pulmonary hypertension, and marked frailty with multiorgan dysfunction. In such contexts, transplant-first strategies or palliative approaches should be prioritized within a shared decision-making framework.[27,28]

Finally, the risk of allosensitization following implantation of durable mechanical circulatory support warrants consideration in transplant-eligible patients. Device implantation and peri-operative exposure to blood products may increase the risk of anti-HLA antibody formation, potentially complicating donor matching and prolonging wait times.[33–37] In patients likely to proceed to transplantation, these immunologic considerations further reinforce the need for deliberate pre-operative planning and multidisciplinary coordination.[27]

Key Practical Considerations

- *Early multidisciplinary discussion is essential*: define anticipated RV support strategy (LVAD alone vs planned RVAD/BiVAD) prior to implantation
- *Systematic RV risk stratification is mandatory:* integrate hemodynamics, echocardiography, and clinical profile to assess RV reserve
- *Avoid delayed RVAD implantation:* in high-risk patients, planned or concomitant RV support is preferred over rescue RVAD after clinical deterioration

Post-operative Monitoring and Optimization of Right Ventricular Function

Following optimization of left-sided hemodynamics in LVAD patients, a dedicated assessment of RV function is essential, as RV dysfunction is common after LVAD implantation and remains a major determinant of early morbidity and mortality despite apparently adequate LVAD support. Because acute RV failure is a leading cause of adverse post-operative outcomes, a structured approach to early recognition and targeted hemodynamic management is critical to preserving systemic perfusion and optimizing recovery.

RV failure after LVAD implantation is frequently multifactorial, with contributing mechanisms that include atrial and ventricular arrhythmias, pre-existing or progressive tricuspid regurgitation, ischemic myocardial injury, and increased pulmonary vascular resistance. In the immediate post-operative period, however, acute RV failure is driven predominantly by an abrupt increase in venous return and leftward interventricular septal shift following left ventricular unloading. Together, these changes increase RV preload while reducing the septal contribution to RV contractility, resulting in diminished forward flow and impaired RV performance[28] (**Fig. 4**).

Early recognition of acute RV failure relies on hemodynamic evidence of elevated central venous pressure with low cardiac output despite appropriate LVAD speed settings. This pattern is often accompanied by a reduced PAPi and echocardiographic findings consistent with RV dysfunction. Continuous monitoring of mixed or central venous oxygen saturation and serum lactate is essential, as declining SvO_2 and rising lactate suggest inadequate RV-supported systemic perfusion.[26] Early bedside transthoracic or transesophageal echocardiography should be performed to assess RV size and function, interventricular septal position, and the severity of tricuspid regurgitation, while excluding reversible mechanical contributors such as cardiac tamponade or excessive left ventricular unloading[27]

Initial management of post-operative acute RV failure focuses on preload optimization while avoiding RV overdistension, most commonly through cautious diuresis or ultrafiltration when central venous pressure is markedly elevated.[4] LVAD speed adjustment is a critical early intervention, as excessive left ventricular unloading can exacerbate leftward septal shift, distort RV geometry, and worsen tricuspid regurgitation, thereby further impairing RV performance.[5] Reduction of RV afterload should be prioritized using inhaled pulmonary vasodilators, including inhaled nitric oxide or prostacyclin, particularly in the setting of elevated pulmonary vascular resistance or hypoxemia.[2] Inotropic support with agents such as milrinone or dobutamine is frequently required to augment RV contractility and forward flow, with agent selection guided by systemic blood pressure and renal function. Mechanical ventilation strategies should minimize increases in pulmonary vascular resistance by avoiding hypoxia, hypercapnia, and excessive positive end-expiratory pressure[27] (**Fig. 5**).

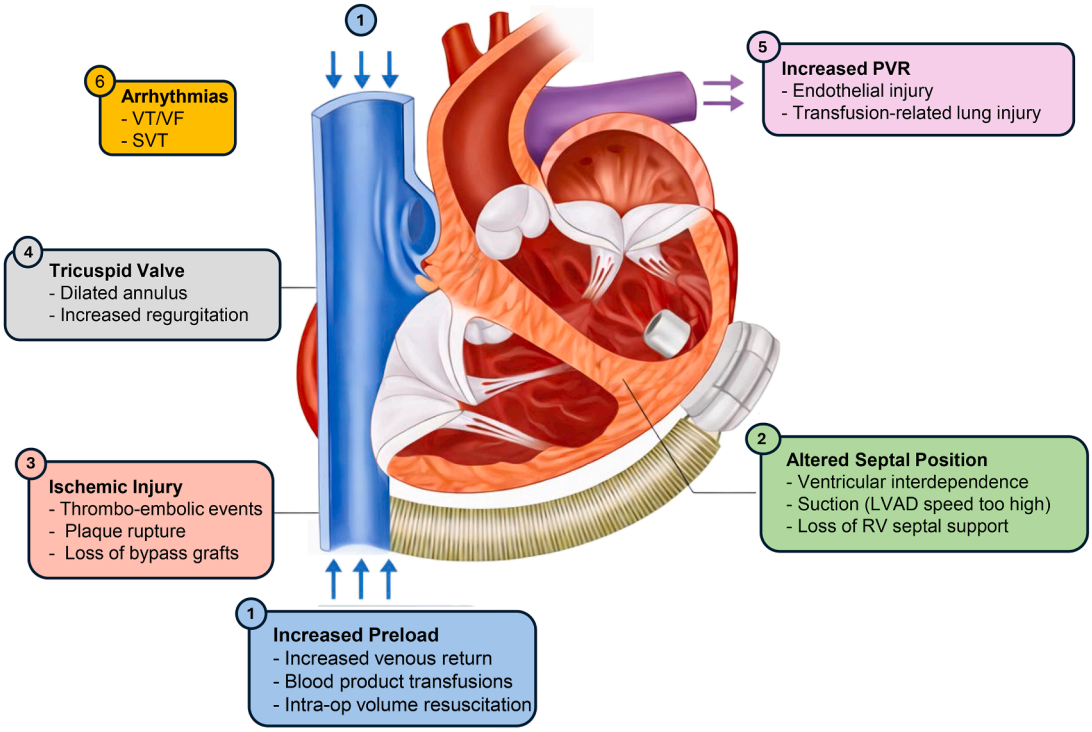

Fig. 4. Mechanisms of RV failure after LVAD implantation. An abrupt increase in preload and abnormal intraventricular septal function are primary mechanisms of acute RV failure after LVAD implantation, with additional potential contributions from atrial and ventricular arrhythmias, pre-existing or progressive tricuspid valve regurgitation, ischemic myocardial injury, and increased pulmonary vascular resistance. LVAD, left ventricular assist device; RV, right ventricular.

If hemodynamic stability cannot be achieved despite optimization of preload, afterload, LVAD speed, and inotropic therapy, early escalation to temporary RV mechanical support should be considered, as delayed intervention is associated with worse clinical outcomes.[26]

Key practical considerations

- *Recognition:* Suspect RV failure when CVP greater than 15 mm Hg, CI less than 2.2 L/min/m^2, PAPi less than 1.5, SvO_2 less than 60%, and/or rising lactate

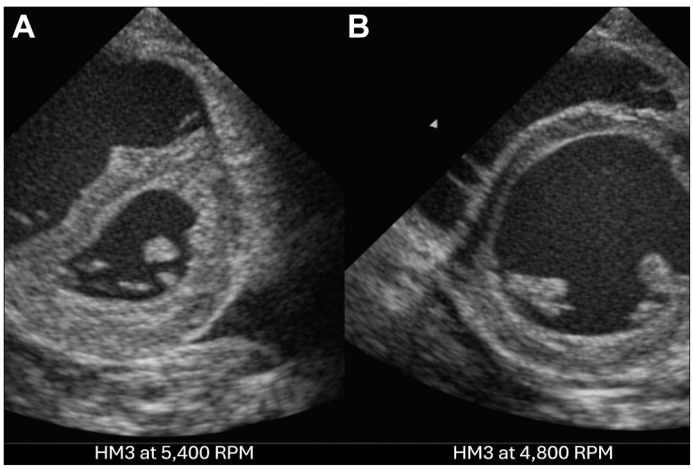

Fig. 5. RV failure after LVAD Implantation. Early after LVAD implantation, low-flow alarms prompted multimodal evaluation demonstrating elevated central venous pressure, reduced mixed venous oxygen saturation, low pulmonary capillary wedge pressure, and low LVAD power, flow, and PI. Parasternal short-axis echocardiography showed RV dilation, left ventricular underfilling, and leftward septal shift (*A*). Following LVAD speed reduction and administration of inhaled nitric oxide, ventricular geometry, and septal position normalized (*B*). LVAD, left ventricular assist device; PI, pulsatility index; RV, right ventricular.

- *Immediate assessment:* Perform early TTE/TEE to assess RV size and function, septal position, and tricuspid regurgitation severity; exclude tamponade and excessive LV unloading
- *Preload:* Avoid RV overdistension; if CVP is markedly elevated, favor diuresis or ultrafiltration over volume administration
- *LVAD speed:* Reduce speed if septal shift toward the LV, RV dilation, or worsening tricuspid regurgitation is present
- *Afterload reduction:* Initiate inhaled nitric oxide or prostacyclin for elevated pulmonary vascular resistance, hypoxemia, or RV failure physiology
- *Inotropes:* Use milrinone or dobutamine to augment RV contractility; consider epinephrine if hypotension limits inodilator use
- *Ventilation:* Minimize RV afterload by avoiding hypoxia, hypercapnia ($PaCO_2$ >45 mm Hg), and excessive positive end-expiratory pressure
- *Escalation:* If instability persists despite optimization, consider temporary RV mechanical support early

Transfusion-Associated Right Ventricular Failure and Prophylactic Inotrope Strategy

Peri-operative transfusion is an often-underrecognized contributor to early RV failure after LVAD implantation. Patients with pre-operative coagulopathy, prior sternotomy, or significant intraoperative bleeding frequently require substantial volumes of packed red blood cells, fresh frozen plasma, and/or platelets in the immediate peri-operative period. Although transfusions are often necessary to achieve hemostasis, the cumulative volume load can acutely increase right-sided filling pressures and promote RV dilation, thereby impairing RV contractile efficiency and worsening interventricular septal geometry in the setting of continuous LV unloading. This hemodynamic stress is particularly deleterious in patients with pre-existing RV dysfunction, elevated pulmonary vascular resistance, or other established risk factors for post-LVAD RV failure.[26–28]

Early RV decompensation may occur despite apparently adequate LVAD flows. Clinicians should therefore maintain a high index of suspicion for evolving RV dysfunction in heavily transfused patients during the early post-operative period.[26]

In this setting, a proactive strategy with a lower threshold for initiating or intensifying inotropic support is reasonable. Early administration of inotropic therapy, titrated in parallel with ongoing transfusion, may augment RV contractility and improve pulmonary circulation, thereby mitigating the hemodynamic impact of rapid volume loading[27,28]. This anticipatory approach may preserve RV performance and reduce progression to overt RV failure requiring escalation to temporary right-sided mechanical circulatory support.[26,27]

Key practical consideration

- Heavy peri-operative transfusion burden increases RV preload and may precipitate RV failure; consider early or prophylactic inotropic augmentation rather than waiting for hemodynamic decompensation

LEFT VENTRICULAR ASSIST DEVICE PUMP PARAMETERS AND TROUBLESHOOTING

In the immediate post-operative period following LVAD implantation, management relies on accurate interpretation of device-derived parameters amid rapidly evolving cardiovascular physiology. Unlike conventional hemodynamic monitors, LVADs do not directly measure intravascular volume status or cardiac output; instead, displayed parameters reflect the dynamic interplay between pump function, native ventricular performance, and loading conditions. A foundational understanding of these parameters is essential to translate device data into meaningful physiologic assessment and informed bedside decision-making.

Modern continuous-flow LVAD management relies on 4 interdependent parameters: *pump speed, flow, power, and pulsatility index (PI)*. Interpreted collectively rather than in isolation, these variables enable rapid recognition of common clinical problems, including hypovolemia, pump thrombosis, systemic hypertension, and RV failure.

Core Left Ventricular Assist Device Parameters

Pump speed (RPM)

Pump speed is the only LVAD parameter that clinicians can directly adjust and is typically maintained as a fixed setting once optimized. Speed titration is intended to ensure adequate systemic perfusion while preserving RV function and normal interventricular septal geometry. Initial optimization is performed intraoperatively under transesophageal echocardiographic guidance, with the goal of maintaining a midline interventricular septum.

The HeartMate 3 LVAD generally operates within a speed range of 4700 and 6200 RPM, depending on loading conditions and physiologic requirements. Excessive pump speeds may result in left ventricular over-unloading, leftward septal

shift, RV distortion, worsening tricuspid regurgitation, and impaired RV contractility. Apparent reductions in displayed speed most often reflect automated suction protection algorithms rather than true device malfunction. After ICU admission, any pump speed adjustment should be undertaken cautiously and guided by echocardiographic and hemodynamic data, rather than reflexive escalation without concurrent assessment of preload and afterload.

Pump flow (L/min)

Pump flow is an estimated, rather than directly measured, parameter. Device-specific algorithms incorporate pump speed, trans-pump pressure differentials, and blood viscosity, rendering flow highly preload-dependent and afterload-sensitive. Increases in preload or native ventricular contractility augment flow, whereas elevations in systemic vascular resistance reduce it.

Low-flow states most commonly arise from hypovolemia, RV failure, cardiac tamponade, inflow cannula obstruction or malposition, or severe systemic hypertension. In contrast, high-flow states are typically associated with distributive physiology, including sepsis, anemia, arteriovenous shunting, or significant aortic regurgitation. Abrupt reductions in pump flow accompanied by concurrent decreases in power and pulsatility are characteristic of suction events, reflecting acute preload collapse and often occurring in association with arrhythmias or mechanical inflow obstruction.[27,38,39]

Pump power (watts)

Pump power reflects the electrical energy required to maintain the set pump speed and is directly measured in watts (W), with typical values ranging from approximately 3 to 7 W depending on operating speed and flow. As with other LVAD parameters, trends in power are substantially more informative than isolated measurements.

A new or progressive rise in pump power should raise concern for pump thrombosis, as greater energy is required to maintain target speed in the presence of flow obstruction. In contrast, low pump power is most commonly observed in low-flow states—such as hypovolemia, RV failure, cardiac tamponade, or inflow cannula obstruction—where impaired left ventricular filling limits pump output.[40,41]

Pulsatility index

The PI represents the ratio of the maximum-to-minimum pump power differential relative to average pump power and is calculated as:

$$PI = Power_{max} - Power_{min}/Power_{average}$$

Pump power is measured over 15 second intervals and averaged to generate the PI. This index reflects variability in pump power across the cardiac cycle, which directly corresponds to variations in pump flow.[41]

Clinically, PI serves as a surrogate for native LV contractility. Greater systolic–diastolic variation in flow and power occurs with stronger LV contraction, resulting in higher PI values. Accordingly, PI increases with improved LV contractility or volume resuscitation and decreases with hypovolemia, RV failure, worsening LV function, or excessive pump speeds. A low or rapidly declining PI often signals reduced native LV output or excessive unloading and may represent an early marker of hemodynamic compromise. Very low PI values increase the risk of suction events, whereas higher PI values generally indicate improved LV contractility or increased volume loading.[6,42,43]

Suction events

Suction events occur when excessive left ventricular unloading results in partial or complete collapse of the LV cavity around the inflow cannula, typically during abrupt changes in loading conditions. Common precipitants include hypovolemia, RV failure, tachyarrhythmias, and inflow cannula malposition, all of which impair LV filling and predispose to intermittent or sustained suction physiology. Recognition is based on integrating device parameters and clinical findings, including low-flow states, reduced PI, intermittent flow variability, and signs of hypoperfusion or hypotension, often in the absence of intrinsic pump malfunction. Immediate management focuses on reversing the underlying cause, with priority given to restoring adequate preload through volume resuscitation, optimizing RV function, and correcting arrhythmias, while avoiding empiric pump speed adjustments unless performed in conjunction with the implanting VAD team.[27,44]

High-Yield Troubleshooting Using Left Ventricular Assist Device Parameters

Interpretation of LVAD parameters should be pattern-based rather than focused on isolated values (**Table 2**). Hypovolemia or active bleeding typically presents with concurrent reductions in pump flow, power, and PI, reflecting diminished preload. Management should prioritize volume resuscitation and evaluation for occult bleeding, including gastrointestinal sources.

Pump thrombosis should be suspected when pump power rises disproportionately, often in association with variable flow and a low PI. This pattern warrants immediate evaluation—including INR, lactate dehydrogenase, and plasma-free

Table 2
High-yield troubleshooting using left ventricular assist device parameters

Clinical Scenario	Power	Flow	PI	Clinical Action
Hypovolemia/bleeding (decreased preload)	Low	Low	Low	Decrease MAP (Typical goal 65–85 mm Hg)
LVAD Thrombosis	High	Variable	Low	Check INR, LDH, plasma-free Hb Echocardiogram Urgent LVAD team consult
Hypertension (increased afterload)	Normal, or Mildly Decreased	Low	Increased	Decrease MAP (Typical goal 65–85 mm Hg)
Right Heart Failure	Low	Low	Low	Assess volume status—target CVP <15 mm Hg Adjust inotropic support Add pulmonary vasodilator (iNO or epoprostenol)

Abbreviations: ICU, intensive care unit; LVAD, left ventricular assist device; MAP, mean arterial pressure; RV, right ventricle; SIRS, systemic inflammatory response syndrome.

hemoglobin—and urgent consultation with the LVAD team that implanted the device.

Systemic hypertension (elevated afterload) commonly manifests as reduced pump flow with normal or only mildly decreased power and increased pulsatility. Management centers on controlled reduction of MAP, typically targeting 65 to 85 mm Hg.

RV failure or cardiac tamponade is characterized by a low-flow, low-power, low-pulsatility state due to impaired LVAD preload. In these settings, urgent echocardiography is essential to assess RV size and function and to evaluate for pericardial effusion, thereby guiding timely escalation of inotropic support, pulmonary vasodilator therapy, or surgical intervention when indicated.[27,43]

Left Ventricular Assist Device Alarms

A structured, tiered approach to LVAD alarms improves bedside recognition and response by distinguishing informational alarms from urgent ones that require immediate intervention. Informational alarms typically reflect non-critical system notifications such as transient flow variations or pulsatility changes and should prompt clinical assessment and trend evaluation without immediate escalation if the patient remains stable. In contrast, urgent alarms including sustained low-flow states, pump stoppage, or power disconnection require immediate bedside evaluation with simultaneous assessment of patient hemodynamics and device function, as delays may result in rapid clinical deterioration. Initial response should prioritize the patient over the device, including assessment of perfusion, mental status, and MAP, followed by verification of driveline integrity and power source. Escalation to the implanting VAD team is recommended for any persistent or unexplained alarm, recurrent low-flow events, suspected pump thrombosis, or hemodynamic instability, and should occur early in the course of evaluation to facilitate timely expert guidance.[27,44]

When an LVAD alarm occurs, the initial priority is a focused clinical assessment of the patient, not the device itself. This assessment should include evaluation of mental status, skin perfusion, urine output, and auscultation for the continuous pump "hum." The LVAD system should then be inspected to confirm adequate power delivery and the integrity of external components, including verifying connection to an active power source and carefully examining the driveline for disconnection or damage.

MAP should be measured using an aneroid sphygmomanometer with a handheld Doppler probe, recognizing that the Doppler opening pressure closely approximates true MAP. Target MAP values are typically 65 to 85 mm Hg unless otherwise specified by institutional protocols. Any concern regarding device malfunction, rising power requirements, suspected pump thrombosis, or uncertainty in alarm interpretation should prompt immediate communication with the implanting ventricular assist device team before proceeding with invasive evaluation or pump speed adjustment.

Key practical considerations

- Pump speed is fixed; all other LVAD parameters reflect underlying physiology.
- Do not assume low pump flow is due to hypovolemia until preload, afterload, and RV function have been systematically evaluated.

- A rise in pump power accompanied by declining PI should prompt immediate concern for pump thrombosis until proven otherwise.
- Device troubleshooting must always be integrated with a comprehensive clinical assessment of the patient.

VENTILATOR AND RESPIRATORY MANAGEMENT

Post-operative pulmonary complications—including atelectasis, pneumonia, and respiratory failure requiring reintubation—are common after cardiothoracic surgery and are associated with increased morbidity, prolonged mechanical ventilation, and longer hospital stays.[45–47] This risk is further amplified in LVAD recipients by systemic inflammatory and coagulation activation, pulmonary ischemia during cardiopulmonary bypass, and reperfusion-related pulmonary endothelial dysfunction.[48,49]

Post-operative respiratory management after LVAD implantation generally follows principles applied to other cardiothoracic surgical patients, with greater emphasis on cardiopulmonary interactions and RV function. Mechanical ventilation has important hemodynamic effects through changes in intrathoracic pressure, lung volumes, ventilation–perfusion matching, and pulmonary vascular resistance. Excessive airway pressures, lung overdistension, and aggressive positive end-expiratory pressure (PEEP) increase RV afterload, reduce RV preload, and ultimately decrease LVAD flow. A lung-protective ventilatory strategy is recommended beginning intraoperatively and continuing through the early ICU course, using tidal volumes less than or equal to 6 to 8 mL/kg predicted body weight, plateau pressures less than 30 cm H_2O, and driving pressures less than 15 cm H_2O to optimize oxygenation while minimizing adverse hemodynamic effects.

Mild hypercapnia may be acceptable when clinically appropriate, as permissive hypercapnia can reduce airway pressures and mitigate adverse RV loading conditions, provided pulmonary vascular resistance and acid–base status remain acceptable. Ventilator settings should be individualized and frequently reassessed in conjunction with invasive hemodynamic measurements, LVAD parameters, and the patient's overall clinical trajectory.

Early liberation from mechanical ventilation is encouraged once hemostasis is achieved and hemodynamics are stable or improving, as prolonged positive-pressure ventilation can impair venous return and RV performance. However, extubation should be approached cautiously, as transition to spontaneous breathing alters RV preload and afterload and may unmask or exacerbate RV dysfunction. Readiness for extubation should be assessed using standard criteria, including adequate oxygenation on minimal ventilatory support, successful spontaneous breathing trials with acceptable respiratory mechanics, preserved neurologic function, stable or decreasing vasoactive requirements, and no anticipated need for reoperation within 24 to 48 hours. During sedation weaning and the peri-extubation period, careful analgesia and sedation management are essential to avoid sympathetic surges that increase pulmonary vascular resistance, while also preventing hypoventilation, hypercapnia, or hypoxia due to oversedation. After extubation, high-flow nasal cannula or non-invasive ventilation may be used selectively to support gas exchange, with close clinical and hemodynamic monitoring and a low threshold for reintubation if respiratory or hemodynamic compromise develops.

Pulmonary rehabilitation should begin early and emphasize pulmonary hygiene and mobilization to reduce post-operative pulmonary complications. Incentive spirometry, deep-breathing exercises, directed coughing, early ambulation, and adequate analgesia are central components of care after extubation. Bronchodilator therapy and secretion-mobilization techniques may be used selectively in patients with bronchospasm, atelectasis, or impaired secretion clearance. Early, structured pulmonary rehabilitation complements lung-protective ventilation and supports sustained respiratory recovery in the post-LVAD population[50] (**Fig. 6**).

Key Practical Considerations

- *LVAD recipients are at high risk for pulmonary complications*
- *Lung-protective ventilation:* Use low tidal volumes and avoid excessive airway pressures to minimize lung injury and adverse RV loading (VT 6–8 mL/kg PBW, plateau <30 cm H_2O, driving pressure <15 cm H_2O)
- *Gas exchange:* avoid hypoxia to limit pulmonary vascular resistance and RV afterload
- *Permissive ventilatory strategy:* Accept mild hypercapnia and limit unnecessary hyperoxia to reduce airway pressures and pulmonary vascular resistance, while maintaining acceptable gas exchange.
- *Positive end-expiratory pressure (PEEP):* Use minimum PEEP required for oxygenation to avoid impaired RV preload, increased RV afterload, and reduced LVAD filling.

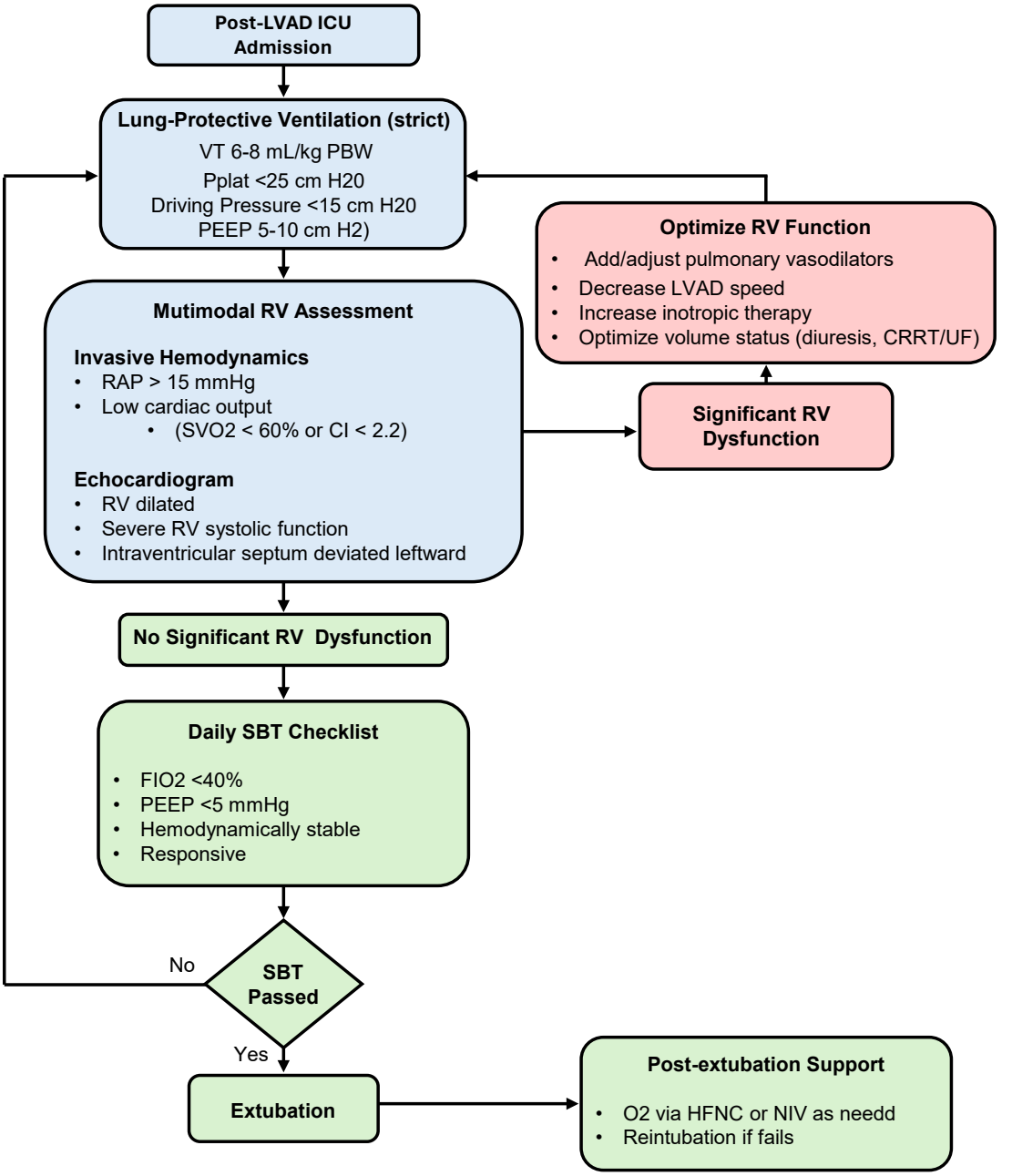

Fig. 6. Mechanical ventilation and extubation strategy after LVAD implantation. Algorithm for postoperative respiratory management emphasizing lung-protective ventilation (tidal volume 4–6 mL/kg predicted body weight, plateau pressure <25 cm H_2O, driving pressure <15 cm H_2O, moderate PEEP) and daily assessment for extubation readiness using a standardized SBT. Patients meeting criteria without evidence of significant RV dysfunction proceed to extubation. Failure of SBT or concern for RV dysfunction prompts multimodal evaluation (hemodynamics and echocardiography) and targeted optimization, including adjustment of LVAD speed, inotropic support, pulmonary vasodilators, and volume management, prior to reassessment. Post-extubation support with high-flow nasal cannula or non-invasive ventilation may be used selectively, with early reintubation for clinical deterioration. FIO2, fraction of inspired oxygen; HFNC, high flow nasal cannula; LVAD, left ventricular assist device; NIV, non-invasive ventilation; PEEP, positive end-expiratory pressure; Pplat, plateau pressure; RV, right ventricular; SBT, spontaneous breathing trial; VT, tidal volume.

- *Hemodynamic–ventilator interaction:* Positive-pressure ventilation decreases RV preload, increases RV afterload, and decreases LVAD preload; ventilator adjustments should be guided by clinical and hemodynamic data.
- *Peri-extubation:* balance analgesia and sedation to avoid sympathetic surges, hypoventilation, or hypercapnia
- *Early but cautious extubation:* Liberation once hemostasis is achieved and vasopressors are stable or decreasing, with close monitoring for RV dysfunction during transition to spontaneous breathing.
- *Post-extubation:* use HFNC or NIV selectively; reintubate early if respiratory or hemodynamic instability occurs
- *Rehabilitation:* initiate early pulmonary hygiene and mobilization; use bronchodilators and secretion clearance selectively

CARDIAC DYSRHYTHMIAS

Cardiac dysrhythmias are common early after LVAD implantation and may compromise hemodynamic stability, impair device performance, and precipitate RV failure. Atrial arrhythmias, particularly new-onset atrial fibrillation, are common after LVAD implantation and may impair RV filling, leading to reduced LVAD preload and decreased pump flow. Initial management prioritizes ventricular rate control with β-blockers, with digoxin as adjunctive therapy when needed; amiodarone may be added when adequate rate control cannot otherwise be achieved. Rhythm control strategies are generally reserved for patients in whom atrial arrhythmias compromise LVAD flow, RV performance, or systemic perfusion; in such cases, electrical cardioversion should be considered.[51,52]

Ventricular arrhythmias are also common after LVAD implantation and may occur despite apparently preserved systemic perfusion. Hemodynamically unstable ventricular tachyarrhythmias require immediate electrical cardioversion or defibrillation, both of which are safe and effective in patients supported with durable LVADs. In stable patients, evaluation should focus on reversible precipitants, including suction events, myocardial ischemia, RV dysfunction, and electrolyte disturbances, and antiarrhythmic therapy should be initiated as appropriate. When electrical therapy is required, standard pad positioning may be used; however, care should be taken to avoid direct overlap with the pump housing or driveline exit site, and continuous device function should be confirmed before and after shock delivery. Electrical cardioversion does not adversely affect LVAD performance, but post-shock assessment of pump parameters and right-sided hemodynamics is recommended[53,54] (**Table 3**).

Key Practical Considerations

- Manage dysrhythmias based on their effects on LVAD flow, RV filling, and systemic perfusion
- Atrial arrhythmias: Prioritize rate control with β-blockers ± digoxin; add amiodarone if rates remain uncontrolled. Consider electrical cardioversion when atrial arrhythmias impair LVAD performance, RV function, or perfusion
- Hemodynamically unstable ventricular arrhythmias require immediate cardioversion or defibrillation
- Hemodynamically unstable ventricular arrhythmias: Evaluate for reversible causes (suction events, ischemia, electrolyte abnormalities) and initiate antiarrhythmic therapy

Post-Left Ventricular Assist Device Renal Function

Acute kidney injury is a frequent complication after LVAD implantation, with meta-analytic data showing an incidence of 25% to 37% depending on the AKI definition applied.[55] Patients with preexisting renal dysfunction are at significantly higher risk of post-operative AKI, and baseline kidney impairment is consistently linked to increased mortality after LVAD implantation.[56] Hemodynamic instability, venous congestion, and reduced renal perfusion also contribute to AKI development in this population, in addition to non-hemodynamic factors such as neurohormonal activation and hemolysis.[57] Recovery of renal function after LVAD is highly variable and closely linked to RV performance. In particular, the development of right heart failure and persistent venous congestion is strongly associated with worsening or nonrecovery of renal function, underscoring the central role of cardiorenal interactions in this population.[58] Although LVAD implantation can improve forward flow and reduce congestion, resulting in early post-operative improvement in renal function in a subset of patients, long-term renal recovery remains inconsistent and unpredictable. In patients who develop severe AKI, renal replacement therapy is often required, and both continuous and intermittent modalities have been used. Continuous renal replacement therapy is generally preferred in the immediate post-operative period due to better hemodynamic tolerance and more controlled fluid management, a distinction that is particularly relevant in LVAD physiology. However, intermittent hemodialysis may be appropriate

Table 3
Arrhythmia Management

Arrhythmia	Physiologic Effect	Impact on LVAD	Initial Strategy	Initial Action	Antidysrhythmic Options	Cardioversion Criteria
Atrial Fibrillation	Loss of Atrial Contribution to Ventricular Filling	↓ Preload ↓ RV Output ↓ LV Flow	Rate control	β-Blocker add Digoxin as needed	Amiodarone (effective, minimal negative inotropy)	Low LVAD Flow Hypotension Decreased perfusion
Atrial Flutter	Loss of Atrial Contribution to Ventricular Filling	↓ Preload ↓ RV Output ↓ LV Flow	Rate control	β-Blocker add Digoxin as needed	Amiodarone (early catheter ablation if refractory)	Low LVAD Flow Hypotension Decreased perfusion
Ventricular Tachycardia	Gradual or Acute RV Failure	May be initially tolerated with eventual: ↓ Preload ↓ RV Output ↓ LV Flow	Rhythm Control	Amiodarone (if stable) Cardioversion (if unstable)	Amiodarone Lidocaine if refractory	Low LVAD Flow Hypotension Decreased perfusion RV Failure
Ventricular Fibrillation	Acute RV Failure	↓ Preload ↓ RV Output ↓ LV Flow	Rhythm Control	Defibrillation	Amiodarone (post-defibrillation)	Immediate

Abbreviations: LV, left ventricle; LVAD, left ventricular assist device; RV, right ventricle.

once patients stabilize, especially when long-term renal recovery remains uncertain.[59]

Driveline Management

Early driveline management is essential to prevent infectious complications, which remain a major source of morbidity in LVAD patients.[60] In the immediate post-operative period, strict driveline immobilization should be maintained to minimize traction and micro-motion at the exit site, which can predispose to tissue disruption and bacterial entry.[61] The driveline should be secured with appropriate anchoring devices and dressings, particularly during repositioning and early mobilization. Exit-site care should follow a sterile, standardized protocol with regular dressing changes and daily inspection for early signs of infection. Early surveillance is critical, as driveline infections often originate at the exit site and may initially present with subtle local findings before progressing to deeper infection or bloodstream involvement.[61,62]

NUTRITION

Early and proactive nutritional support is crucial after LVAD implantation. Enteral nutrition is preferred and should be initiated within 24 to 48 hours of achieving hemodynamic stability. This approach preserves gut integrity and reduces risk of infection. Close monitoring during initiation of feeding and advancement is essential, particularly for electrolyte disturbances associated with refeeding syndrome, which are more prevalent in patients with renal dysfunction. Micronutrient and vitamin deficiencies should be identified and promptly replenished to facilitate recovery and promote sternal wound healing. Glycemic control is a parallel priority, as postoperative hyperglycemia is associated with impaired wound healing and increased infection risk. Blood glucose levels should generally be maintained below 180 mg/dL. Early insulin infusion is often favored during the ICU course to allow precise titration while enteral and oral intake are evolving.[63,64]

Key Practical Considerations

- Route: enteral nutrition preferred; initiate within 24 to 48 hours of hemodynamic stability
- Advancement: monitor closely for refeeding syndrome (phosphorus, potassium, magnesium), especially with renal dysfunction
- Delivery: anticipate early underfeeding; involve nutrition support to minimize cumulative deficits
- Micronutrients: assess and replete to support recovery and sternal wound healing
- Glycemia: target glucose less than 180 mg/dL; use insulin infusion early as intake evolves

EARLY MOBILIZATION

Early mobilization is a key component of recovery following LVAD implantation and should begin as soon as hemodynamic stability and hemostasis permit. Prolonged immobility rapidly contributes to deconditioning, respiratory complications, and delayed functional recovery, whereas structured early rehabilitation is associated with improved physical capacity, fewer complications, and a smoother transition to long-term recovery. Mobilization should begin in the ICU, progressing from in-bed exercises and sitting at the edge of the bed to standing and ambulation, coordinated closely with physical therapy and nursing staff. Careful attention to LVAD driveline security and hemodynamic tolerance is essential as activity progresses. Early multidisciplinary rehabilitation should be viewed as an extension of ICU supportive care rather than a downstream intervention, with the goal of preserving functional reserve and facilitating overall recovery.[65,66.]

Key Practical Considerations

- Initiate mobilization once hemodynamics and hemostasis are stable
- Advance stepwise (bed → chair → stand → ambulate) while monitoring hemodynamics and LVAD parameters
- Secure the driveline and mobilize with coordinated ICU and physical therapy support

SUMMARY

Recovery following LVAD implantation is a multifaceted, resource-intensive process that requires coordinated multidisciplinary care in the ICU. Effective management of hemodynamics, RV function, respiratory status, and anticoagulation directly impacts survival and long-term outcomes. Early recognition of complications and timely intervention are essential to limit morbidity. Equally important are nutritional support and early mobilization, which promote functional recovery and improve overall outcomes. As LVAD technology continues to evolve, standardized, evidence-based ICU protocols combined with individualized patient care will remain central to optimizing recovery and quality of life in this vulnerable population.

DECLARATION OF AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

During the preparation of this work, the authors used Gen AI. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

DISCLOSURE

The authors have no disclosures relevant to this study.

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