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REVIEW ARTICLE

Postoperative care after left ventricular assist device implantation: considerations for the cardiac surgical intensivist

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Abstract
Heart failure is a leading cause of morbidity and mortality, the incidence of which is predicted to continue to increase as the population ages. Left ventricular assist devices (LVADs) in particular have emerged as important therapies for the support of patients with advanced heart failure needing short- or long-term mechanical circulatory support. With over 5000 implantations per year, LVADs are the most commonly used durable devices worldwide. In this article, we provide an overview of the intensive care management of patients with LVADs during the early post-implantation period.

Keywords Heart failure · Intensive care management · Left ventricular assist devices

Introduction

Mechanical circulatory assist devices were originally designed to mimic human physiology and generate pulsatile flow. By default, pulsatile devices had multiple moving parts making them prone to complications including device failure. These devices were eventually replaced by continuous flow devices. There are two types of continuous flow left ventricular assist devices (LVADs): axial and centrifugal. Axial flow devices have a rotating impeller (rotor in a pipe) that propels blood forward, whereas centrifugal pumps spin creating a centrifugal force that moves blood tangentially away from the pump blades and towards the outflow cannula. The HeartMate 3 (Abbott, Lake County, IL, USA), has a fully magnetically suspended pump propeller thus reducing friction between the blood and the device and has shown improved survival without disabling strokes and reoperation compared to its predecessor, the Heartmate II [1]. As of 2021, only the HeartMate 3 is available for implantation in the United States as a bridge to transplant or destination therapy [2].

Significant effort has been made to stratify risk prior to implantation of an LVAD. The Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) classifies heart failure patients according to disease severity. Profile 1 signifies the highest acuity of cardiogenic shock, whereas Profile 7 describes patients with compensated heart failure and minimal symptoms. As one may predict, Profile 1 implantations have the lowest survival rates — 74%. The majority of implantations are in either Profile 2 or Profile 3 patients and have a 1-year survival of 82% with the lowest readmission rates [2, 3].

A thorough understanding of LVAD parameters is pivotal for the operation of the device as well as the management of patients in the post-implantation period.

Speed

Titration of speed is performed by changing the revolutions per minute (RPMs) of the device and aims to achieve adequate end-organ perfusion while maintaining normal right ventricle (RV) and interventricular septum (IVS) geometry. Initial speed is set and titrated under direct intraoperative transesophageal echocardiography (TEE) visualization to ensure the IVS maintains its natural curvature into the RV or be slightly midline, but never bowing into the left ventricle (LV). At optimal speed, the aortic valve (AV) should open every few beats at most and any mitral regurgitation (MR) present pre-insertion should be minimal. Excessive speed may result in flows the RV cannot maintain and shift the IVS into the LV, distorting RV geometry, impairing RV contractility, and causing tricuspid regurgitation (TR), all of which

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will further worsen RV stroke volume. Once the patient leaves the operating room, subsequent speed changes require follow-up transthoracic echocardiography (TTE) to ensure the IVS remains in a midline position. The range of LVAD speeds are system specific; HeartMate III: 3000–9000 RPM, HeartWare Ventricular Assist Device (HVAD) 2400–3200 RPM, HeartMate II: 6000–15,000 RPM [2].

Power

The amount of current and voltage the device needs to achieve the set speed is directly measured by the device in Watts (W). A normal range is 3–7 W, and the exact power varies according to the pump speed and flow [4]. Formation of a thrombus in the pump itself can result in increases in power, typically above 10–12 W as the device attempts to maintain the set speed. On the other hand, a thrombus in the inlet or outflow cannulas reduces pump flow and results in a decrease in power [5, 6]. A decrease in power, flows, and pulsatility may also result from tamponade, right ventricular failure, or hypovolemia [6]. TTE or TEE is typically used to narrow the diagnosis and guide therapy (see Fig. 1 for an approach to the LVAD patient in shock).

Pump flow

Unlike power, which is directly measured, pump flow measured in liters per minute (LPM) is a calculated value based on Poiseuille's law and uses the set pump speed, power, as well as the hematocrit of the patient as a marker of blood viscosity. The higher the speed and the power, the higher the calculated flow. Hematocrit may be assumed on some devices or may be input by a staff member to more accurately calculate flow. The pump flow can change according to pressure differentials across the pump. LVADs are preload dependent and afterload sensitive [7]. For example, left ventricular contraction increases blood flow through the pump and thus pump flow increases, whereas an increase in systemic vascular resistance lowers the pump flow. A suck-down or suction event is a significant decrease in the pump flow, power, and pulsatility due to the contact of the LVAD with the LV cavity causing an abrupt decrease in preload. The etiologies are multifactorial and may include hypovolemia, RV failure, tamponade, or LVAD inflow cannula malposition and may be accompanied by arrhythmias.

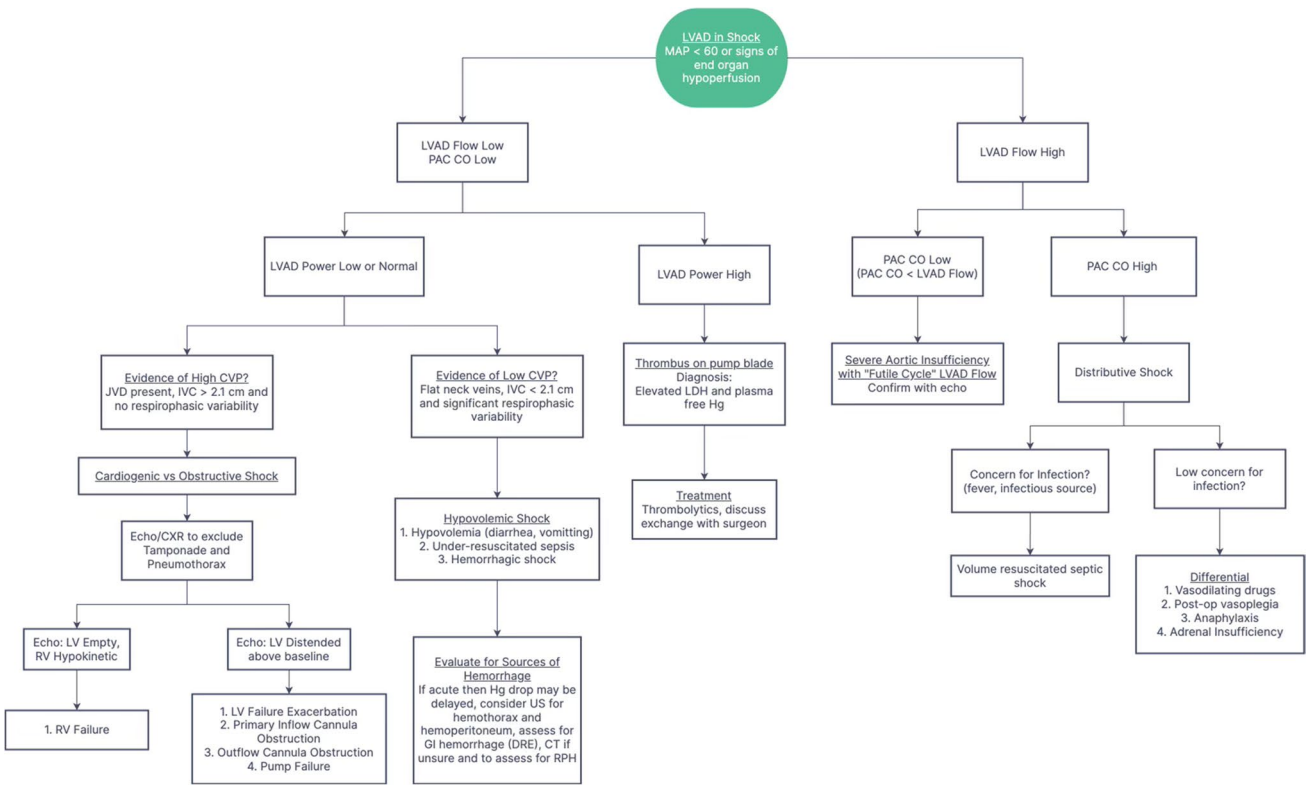

Fig. 1 Approach to the LVAD patient in shock

Pulsatility index (PI)

The PI is calculated utilizing the variation in power and it corresponds to the degree of variation of flow through the pump. Conceptually, PI can be thought of as the LV's contribution to native cardiac output (CO). As such, the PI fluctuates with changes in LV preload, afterload, and native LV function. The PI increases with improvement in LV or RV function and resuscitation of hypovolemia. It decreases with worsening LV function, low LV preload from hypovolemia or RV failure, and excessive pump speeds [6, 8]. The HeartMate 3 has a programmed pulsatility, which results in a rhythmic deceleration and acceleration of the rotor by 2000 RPMs every 2 s which is designed to eliminate stasis within the device and thus decrease the incidence of pump thrombosis and embolic events [2, 5].

Dedicated intensive care unit (ICU) team

When planning perioperative care for patients undergoing LVAD implantation, just as with any cardiac surgical patient, care should be led by an intensivist team providing comprehensive care day and night. Several studies have identified a reduction in postoperative morbidity, ventilator time, and even early mortality with 24-h intensivist care in comparison to an in-house trainee with back-up attending coverage [9, 10]. In general, intensivists are comfortable with critically ill patients, will actively promote progress day and night, and are more familiar with subtle signs of decline therefore can intervene early to prevent complications. In addition to a reduction of postoperative mechanical ventilation times, Benoit et al. [10] also found a reduced rate of surgical site infections, ICU readmission, and surgical postponements due to inadequate ICU bed availability. Though LVAD patients were excluded from these study populations due to significant improvements in surgical technique and LVAD technology over the study period, end-stage heart failure patients have similar, if not higher, indices of illness and perioperative risk.

Preop optimization

One of the key predictors of outcome after LVAD insertion is the condition of the patient preoperatively. This requires a multidisciplinary discussion of the LVAD candidate including the status of comorbid conditions, frailty assessment, fitness for surgery, and goals for optimization. Comorbid conditions must be maximally medically managed. Often advanced heart failure patients have difficulty maintaining adequate control of conditions like chronic kidney disease and RV dysfunction, and these may contribute to significant morbidity postoperatively. It is reasonable to admit the outpatient candidate preoperatively for optimization, and the inpatient candidate to the ICU to utilize hemodynamics derived from continuous pulmonary artery catheter (PAC) monitoring to guide optimization of volume status and to support perfusion prior to surgery. Suboptimal end-organ perfusion despite inotropic or inodilator support may require bridging to durable mechanical support with an intra-aortic balloon pump or temporary mechanical support such as Impella (Abiomed, Danvers, MA) [11].

Operating room to ICU handoff

In addition to details about patient history, the operation, and anesthetic management, the intraoperative transesophageal echo report is critical. The ICU team will anticipate potential issues associated with specific echocardiographic findings detailed in Table 1. RV function before and after LVAD insertion should be discussed with mention of specific inotrope doses and presence of pulmonary vasodilators at the time of assessment. Quantitative RV assessment, such as RV free wall strain, a load-independent parameter,

Table 1 Intraoperative TEE findings and ICU anticipatory guidance

Echo finding	ICU anticipation
RV dysfunction and inotropic support	Avoid hypoxia and acidosis. Caution with inotrope wean
Aortic insufficiency (AI)	LVAD flow may be higher than PAC-derived CO due to futile cycle regurgitant flow. Any AI more than mild is generally repaired. The AV may be oversewn to prevent opening
Mitral stenosis	Rare, should be fixed intraoperatively
Mitral regurgitation (MR)	Should improve with LVAD support. Persistent MR may indicate insufficient LV unloading
Patent foramen ovale (PFO)	Risk for stroke, hypoxemia from right-to-left shunting if LVAD flow and right atrial pressure are too high
LV thrombus	Higher risk for stroke
LV hypertrophy or significant LV diastolic dysfunction	Higher left atrial pressure may be needed to maintain adequate LV preload. LVAD may be prone to suction events, especially with suboptimal inflow cannula positioning
RV hypertrophy or diastolic dysfunction	Higher right atrial pressure may be needed to maintain adequate RV preload

is preferable to qualitative assessment. Fractional area of change (FAC) is a reasonable alternative and preferable over tricuspid annular plane systolic excursion (TAPSE) which tends to underestimate RV function after pericardiotomy and does not correlate well with RV ejection fraction (EF) on cardiac magnetic resonance imaging (MRI) in patients with moderate or greater TR [12, 13]. Hemodynamics should be considered more important than the visual appearance of the RV in the decision to escalate RV support. Treat the patient, not the echo.

Postoperative bleeding

In patients with an indication for chronic therapeutic anticoagulation or dual anti-platelet therapy, long-acting anticoagulants and non-aspirin platelet inhibitors are withheld for 3–7 days preoperatively. Indicated aspirin is infrequently discontinued in anticipation of surgery. Anticoagulated patients at high risk of thrombotic complications are generally transitioned to short-acting intravenous anticoagulation. This medication can be stopped immediately prior to surgery, or it can be reversed intraoperatively.

Immediate post-surgical hemorrhage will necessitate higher transfusion and reoperation rates, which are associated with an increase in overall mortality rate after LVAD implantation [14]. Early identification of coagulopathic or surgical bleeding and management of hematologic derangements will minimize the need for blood product transfusion, reducing the risk of developing transfusion-related complications. To this end, if continued sanguineous chest tube output is present immediately postoperatively, early and frequent discussions with the operating surgeon are warranted to avoid complications. Similar thresholds for transfusion and reoperation are used for the post-LVAD patient as the general cardiac surgical patient. It is useful to perform goal-directed treatment rather than empiric product transfusion using coagulation efficiency testing or component tests, such as platelet aggregation, fibrinogen level, partial thromboplastin time (PTT), and international normalized ratio (INR). For persistent bleeding, desmopressin and antifibrinolytics are acceptable adjuncts but the use of vitamin K or factor concentrates should be discussed with the operating surgeon due to risk of pump thrombosis. Regardless of the etiology, significant retained blood should be evacuated as a retained hematoma is a nidus for infection that may require delayed evacuation and prolonged hospitalization [15].

Conversely, LVAD thrombosis can be a devastating complication with risk of LVAD failure or embolization causing stroke, or abdominal or limb ischemia. Each device manufacturer details the recommended thrombosis prevention strategy that generally includes the initiation of aspirin on the first postoperative day as well as therapeutic unfractionated heparin once hemostasis occurs. Patients are eventually transitioned to a vitamin K antagonist for long-term anticoagulation [15]. There is emerging data to suggest apixaban may be a safe alternative to warfarin [16].

Hemodynamic management

The goal of hemodynamic management postoperatively is to maintain tissue perfusion while achieving hemostasis and supporting RV function. To maintain perfusion, an appropriate balance of CO and systemic vascular resistance (SVR) must be achieved. Generally, this requires a combination of vasopressors and inotropes targeting a mean arterial pressure (MAP) of 60–70 mmHg and a cardiac index (CI) > 2.2.

Systemic vascular resistance

A goal MAP of 60–70 mmHg should be targeted until hemostasis is achieved, and subsequently a MAP of 60–80 mmHg. A MAP of > 85 mmHg should be avoided due to the potential for reduction in LVAD flow and bleeding complications.

Some patients will have vasoplegia and require vasopressors postoperatively for several hours to days. Generally, vasopressin is preferred over norepinephrine as it preferentially increases SVR and spares the pulmonary circulation thereby avoiding excessive RV afterload [17]. It may also elicit pulmonary vasodilation via a nitric oxide–dependent mechanism [17]. Vasopressin can be titrated along with norepinephrine. In severe cases of vasoplegia, consideration may be given to hydroxycobalamin as a 5-g bolus. We prefer hydroxycobalamin over methylene blue due to the nitric oxide (NO) scavenging effect of methylene blue, which may increase pulmonary vascular resistance (PVR).

Cardiac output measurement and goals

A normal CI goal of > 2.2 may be insufficient in patients with significant vasoplegia and increased oxygen consumptive needs from their inflammatory state post-op. Thermodilution-derived CO is preferred over the Fick principle, which assumes a constant oxygen consumption less reliable in rapidly changing conditions like general anesthesia or inflammatory state. Unless severe TR is present, thermodilution measurements remain accurate [18, 19]. Assuming the AV opens and is not regurgitant, thermodilution CO should be slightly higher than the LVAD flow, which does not include native CO.

In the event a PAC is not available, and the AV does not open, pump flow estimates the true cardiac output. If the AV does open, the native cardiac output can be estimated with echocardiography using pulsed wave Doppler of the left ventricular outflow tract (LVOT). This is then added to

the pump flow to estimate the "true" cardiac output. Alternatively, if there are technical difficulties making the LVOT measurements invalid, the total cardiac output can be estimated with pulsed wave Doppler of the right ventricular outflow tract.

Inotropes

RV dysfunction identified pre-LVAD insertion is frequently due to LV failure and should improve with LVAD support. However, severe RV failure pre-LVAD may not recover [20]. Many patients will benefit from inotropes for hours to days postoperatively to support the RV as it adapts to its new loading parameters. Milrinone is the first-line inodilator, but dobutamine is also acceptable. On occasion, it either may cause hypotension and an increase in vasopressor requirement. In these situations, epinephrine may be preferred.

For the struggling RV, maintenance of sinus rhythm and atrioventricular synchrony is also important. Antiarrhythmics and cardioversion are used for arrhythmias associated with hypotension or reduced CI.

Pulmonary vasodilators

Inhaled pulmonary vasodilators such as epoprostenol or nitric oxide are frequently initiated intraoperatively and are better tolerated hemodynamically than intravenous forms. These medications are weaned as soon as tolerated in preparation for extubation if adequate LVAD flow and CI are maintained. Elevated pulmonary artery (PA) pressures, in the absence of a reduced CI, may be tolerated. Paradoxically, low PA pressures, in the setting of low CI, may be more concerning for RV failure, as the RV is unable to generate sufficient flow.

If elevated PA pressures persist, it is important to know whether the pulmonary hypertension (PH) is pre- or post-capillary in origin. Ideally, the presence of pre-capillary PH is known from the preoperative right heart catheterization (RHC). LVAD candidates frequently have post-capillary PH due to their left heart disease which improves with LVAD support. A transpulmonary gradient (TPG) over 12 mmHg, or a diastolic pressure gradient above 6 mmHg, may indicate the presence of concomitant pre-capillary PH, which may not improve post-LVAD [21]. If RHC hemodynamics are not available, checking a wedge pressure may be necessary to diagnose pre-capillary PH and those patients may struggle with weaning of inhaled pulmonary vasodilators and require transition to an oral phosphodiesterase inhibitor. Triggers for re-initiating inhaled pulmonary vasodilators may include abrupt increases in PA pressure associated with a decrease in CI, significant increase in central venous pressure (CVP), low pulmonary artery pulsatility index (PAPi), and echo signs of RV failure.

Inhaled pulmonary vasodilators may also be delivered non-invasively with a heated high flow nasal cannula system. This delivery method does pose a theoretical risk to staff exposed to drug and is prohibited in some institutions. These medications, while lifesaving, do carry some risks. First, thrombocytopenia, although mostly described with intravenous epoprostenols, can occur with inhaled forms [22]. Second, in patients with emphysematous lung disease, these medicines may increase blood flow to areas of physiological dead space worsening ventilation/perfusion (V/Q) matching and causing hypoxemia [23]. Finally, the tenuous LV may become overloaded with additional pre-load causing pulmonary edema. For these reasons, inhaled pulmonary vasodilators should be initiated after careful consideration of the entire clinical picture, as opposed to a reflexive response to elevated PA pressures.

Volume

Euvolemia is a challenging endpoint to define, and maintaining it requires careful attention to the physical exam, PAC, and echocardiographic data. Dynamic assessment of volume responsiveness is preferred over static measurements. As an example, CI can be measured before and after a passive leg raise maneuver by thermodilution or with Doppler echo of the right ventricular outflow tract (RVOT). When addressing low CI or hypotension in the first postoperative hours, an empiric fluid challenge is reasonable. If unresponsive however, maintain a low threshold for increasing RV support. Volume loading should be avoided when there are echocardiographic signs of volume overload including RV dilation, flattening of the IVS, severe TR (secondary/functional), a plethoric inferior vena cava (IVC), hepatic vein flow reversal, and portal vein pulsatility.

Optimal filling pressures (CVP, wedge) will vary depending on the baseline RV and LV compliance. A wedge pressure of 8–12 mmHg may be insufficient in patients with grade 2 or 3 diastolic dysfunction. No single target CVP or wedge can be prescribed, rather goals are tailored to the individual and sometimes discovered via a trial-and-error method.

Management of acute hemodynamic instability

Accurate blood pressure monitoring is essential in managing patients with hemodynamic instability. Due to minimal or complete absence of pulsatility in continuous flow devices, the traditional auscultatory and oscillometric methods of measuring blood pressure often fail or are inaccurate. The Doppler ultrasound method to obtain an opening pressure is the preferred noninvasive technique, since it correlates closely with the arterial catheter MAP when pulse pressure is low [24]. If the pulse pressure increases with improvement

in heart function, the Doppler method loses its correlation [24] and oscillometry may be used.

In patients with continuous flow devices, hypotension is defined as MAP of less than 60 mmHg, by Doppler measurements or through invasive arterial pressure monitoring (see Fig. 1 for an approach to the LVAD patient in shock). TTE and TEE are frequently necessary in narrowing the diagnosis.

Temporary right ventricular assist devices (RVADs)

Some patients with severe RV failure post LVAD insertion require temporary RVAD support. This can be done surgically, or percutaneously with an Impella RP (Abiomed, Danvers, MA) or Protek Duo (LivaNova, London, UK). If higher device flows are needed or avoiding prolonged femoral venous access is advantageous, the latter may be preferred. Percutaneous RVADs have been associated with shorter times to insertion and shorter lengths of stay over surgical RVADs [25]. Preimplant characteristics associated with RVAD use include INTERMACS Profile 1 or 2, preoperative extracorporeal membrane oxygenation (ECMO) support, continuous renal replacement therapy, severe TR, and prior cardiac surgery [26]. Pre-existing RV failure out-of-proportion to LV failure may also predict RV failure post implant. An elevated TPG, a TAPSE less than 12.5 mm, and the absence of significant left atrial (LA) dilation are suggestive of pre-existing pre-capillary PH and RV failure, which may not recover with LVAD support [26, 27]. Instituted early, RVAD insertion does not negatively affect eventual transplantation or survival [28].

Mechanical ventilation

A lung protective ventilation strategy with tidal volume < 8 ml/kg, predicted body weight plateau pressure < 30 mmHg, and driving pressure < 15 mmHg is utilized from the intraoperative establishment of mechanical ventilation and is continued in the ICU [29, 30]. Liberation from mechanical ventilation occurs as early as possible and as long as basic ventilatory weaning criteria are met. The decision to extubate is made after establishing hemostasis, a reasonable vasopressor and/or inotrope requirement, and adequate cardiac indices with appropriate end-organ perfusion. As sedation is weaned, special attention is given to optimization of pain control to avoid abrupt increases in PVR which can have detrimental and long-lasting consequences on RV function. Alternatively, hypercapnia or hypoxia from oversedation or over-narcotization after liberation from mechanical ventilation will have similar effects on the PVR and must be avoided. Additionally, early detection and correction of metabolic acidosis is necessary, as the respiratory compensation capability may be inadequate. In these cases, noninvasive positive pressure ventilation or heated high flow nasal cannula is used with a low threshold for reinstating invasive mechanical ventilation.

Due to the relatively high occurrence of RV failure, inotropic support is typically maintained throughout the peri-extubation period. Similarly, hemodynamic monitoring using a PAC is continued after extubation to detect often subtle changes that may necessitate escalation of RV and/or pulmonary support.

Pulmonary hygiene and rehab

The risk of pulmonary complications, like respiratory failure, bronchospasm, respiratory infection, and atelectasis, is relatively high and has been reported in up to 55% of patients undergoing cardiopulmonary bypass [31]. Microaspiration of oropharyngeal flora can be reduced by using endotracheal tubes with subglottic secretion drainage systems. A recent meta-analysis showed a significant reduction in ventilator-associated pneumonia using this method [32]. It is recommended that incentive spirometry be used with deep breathing techniques, directed coughing, early mobilization, and optimal analgesia to prevent postoperative pulmonary complications [33].

During the early post-extubation period, beta-adrenergic agonists, like albuterol, are administered as bronchodilators with the additional benefit of improving the mucocilliary clearance [34, 35]. The administration of such bronchodilators can be through a nebulizer or with a high-frequency oscillatory device, such as the Metaneb system (Hill-Room, Chicago, IL, USA). The latter is used to mobilize secretions and treat or prevent atelectasis by delivering oscillations during both phases of the respiratory cycle. Such treatment with high-frequency oscillations and lung expansion can reduce pulmonary complications in high-risk patients [36].

Hypertonic saline aerosols have traditionally been used to stimulate a productive cough and reduce sputum viscoelasticity. In patients with chronic obstructive pulmonary disease, such aerosols can cause airway irritation and bronchoconstriction and therefore, are avoided [37]. Oral expectorants, like guaifenesin, reduce the viscosity of airway secretions and promote thinning of airway secretions through vagus nerve stimulation [38]. Caution should be taken when administering these agents in patients with poor cough due to pro-emetic side effects.

Nutrition

There is no need to withhold nutrition in the postoperative period after LVAD insertion. Extubated patients may begin oral intake as soon as deemed safe to swallow. Those who are expected to require mechanical ventilation beyond the immediate perioperative period or who fail ventilator weaning trials should have enteral nutrition started soon after maintaining reasonable hemodynamic stability and hemostasis. Care should be taken to recognize previously under-appreciated gastrointestinal dysfunction as a cause of intolerance to enteral nutrition or poor absorption. Longstanding preoperative heart failure is associated with reduced gastric perfusion which can lead to slow gastric emptying and suboptimal absorption [39, 40]. In the case of gastric ileus, early post-pyloric feeds should be instituted until patients are able to tolerate gastric nutrition, especially in those with pre-existing cachexia. Parenteral nutrition is a viable, but rarely needed, option [41].

Alterations in gastrointestinal mucosa and vasculature can be expected due to low flow from preoperative heart failure and low pulsatility after LVAD support is initiated. These, along with acquired von Willebrand syndrome and the need for anticoagulation and antiplatelet therapy, can contribute to the increased risk of gastrointestinal bleeding generally appearing after the immediate postoperative period [42]. While improvements in technology have significantly reduced the occurrence of gastrointestinal (GI) bleeding, it remains a common cause of readmission [43]. These episodes should be managed like GI bleeding in the anticoagulated, non-LVAD patient by holding anticoagulation, correcting the acquired coagulopathy if necessary, transfusion to maintain adequate perfusion and hemostasis, endoscopic evaluation, and intervention as appropriate. Therapeutic anticoagulation can be reinstituted, perhaps with a lower INR goal, if the risk of LVAD thrombosis is deemed higher than the risk of subsequent bleeding.

Conclusion

Critical care management of the postoperative LVAD patient is a clinically challenging encounter. Optimal outcomes are dependent on having a multidisciplinary team with effective communication between all team members. This includes a dedicated ICU team experienced in the hemodynamic management of postoperative cardiac surgical patients and those requiring mechanical circulatory support. Bedside echocardiography skills are crucial, as well as proficiency in managing patients in mixed shock. Optimal patient selection, preoperative optimization, immediate postoperative stabilization, and efficient progression in the areas of pulmonary hygiene, mobility, and nutrition are all essential for successful outcomes.

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