Review Article

Facilitating Noncardiac Surgery for the Patient with Left Ventricular Assist Device: A Guide for the Anesthesiologist

Abstract

The introduction of left ventricular assist device (LVAD) has improved survival rates for patients with end-stage heart failure. Two categories of VADs exist: one generates pulsatile flow and the other produces nonpulsatile continuous flow. Survival is better for patients with continuous-flow LVADs. With improved survival, more of such patients now present for noncardiac surgery (NCS). This review, written for the general anesthesiologists, addresses the perioperative considerations when the patient undergoes NCS. For best outcomes, a multidisciplinary approach is essential in perioperative management of the patient.

Keywords: *Afterload, continuous-flow, nonpulsatile, power consumption, preload, pulsatile, pump speed*

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Introduction

Durable left ventricular (LV) assist devices (LVADs) are implanted in patients with chronic heart failure refractory to maximal medical therapy including cardiac resynchronization therapy. Such recipients may have ischemic cardiomyopathy, dilated cardiomyopathy, myocarditis, postpartum cardiomyopathy, and refractory arrhythmias.[1] The LVAD functions as a pump, drawing blood into an inflow cannula that is inserted through the apex of the left ventricle and returning the blood to the ascending aorta through an outflow graft. Essentially, the LVAD offloads the left ventricle and assists with cardiac output.

Methods of search

PubMed and Cochrane databases from 1990 until 2017 were searched without restriction on languages. Abstracts from the citations were scrutinized and determined for inclusion into the review. Each included article was then examined for additional references.

Development of indications for ventricular assist device implantation

Early pumps introduced in the 1990s were pulsatile and noisy. Before 2008, all VADs implanted in the United States outside the clinical trial setting delivered pulsatile flow via an electrically (Novacor, HeartMate XVE) or pneumatically (HeartMate IP, Thoratec IVAD/PVAD) driven volume displacement pump.[2] The pumps were either implanted in the preperitoneal space (Heartmate XVE, Novacor LVAD) or carried extracorporeally (Thoratec PVAD). With technological advancements, current second- and third-generation LVADs implanted worldwide are small nonpulsatile pumps driven by an impeller in an axial (Thoratec HeartMate II) or centrifugal (HeartWare HVAD and HeartMate 3 LV assist system [LVAS]) fashion. Implantation of the HeartMate II (HMII) LVAD requires a preperitoneal pocket to be created under the left rectus abdominis muscle. The HVAD and HeartMate 3 LVAS (HM3) are designed to be attached directly to the LV apex and small enough to fit within the pericardial cavity. Due to the size of these nonpulsatile continuous-flow (CF) VADs, they can be used in smaller sized patients. These smaller sized patients include mostly women and men of small stature. CF-LVADs have rotating impellers which pump blood at a fixed speed. Patients with these devices do not have a palpable pulse, and arterial bleeding is nonpulsatile as well. Adequacy of circulatory volume and right ventricular (RV) function have a significant impact on LVAD flow and therefore cardiac output.[3] Pump speed of the device is optimized to balance both outflow and hemolytic shear forces on red blood cells,

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while RV function depends both on contractility and intravascular volume.[4] Electrical power for the device is supplied using portable batteries carried extracorporeally and connected via a driveline cable tunneled from the LVAD through the abdominal wall. When the patient is at rest, sleeping, or nonambulant, the batteries are housed in a mobile power unit which will continue to power the device via wall power supply.[5]

The 2001 REMATCH trial involving 129 patients in 20 centers showed a 48% reduction of all-cause mortality over the study period of 30 months in the 68 patients randomized to LVAD implantation, compared to the 61 patients randomized to optimal medical management.[6] With the approval of the CF HMII for destination therapy (DT) in 2010, there has also been a 10-fold increase in approved LVADs implanted for lifelong support in transplant-ineligible patients.[2] Accordingly, LVADs are not only a temporary respite for end-stage heart failure patients while awaiting definitive heart transplant (bridge-to-therapy [BTT]) but also as DT in themselves.[7,8] For some patients, the LVAD is a bridge-to-decision where the heart is supported until the patient recovers sufficiently to be eligible for heart transplant (also referred to as bridge-to-candidacy), or a bridge-to-recovery where the patient's heart has recovered sufficient contractile function so that the LVAD is no longer necessary and can be explanted.[2,9]

Following the 2011 third Interagency Registry for Mechanically Assisted Circulatory Support report confirming greater survival with CF over pulsatile flow LVADs at 1 year (83% versus 67%) and 2 years (75% versus 45%), the HMII became the most common VAD implanted worldwide.[10,11] In nonrandomized observational 2015 ROADMAPS trial, patients with functionally limited noninotrope-dependent heart failure who were implanted with HMII had better outcomes compared to patients on medical therapy alone, even though the former group initially had more severe heart failure, lower baseline quality of life, more depressed individuals, and lower predicted Seattle Heart Failure model 12-month survival rates.[12] The primary composite endpoint was survival on original therapy with improvement in 6-min walk distance of at least 75 m at 12 months.[12] Seventy-seven percent of the patients who had LVAD improved to New York Heart Association I (NYHA I) (25%) or II (52%), compared to 0% to NYHA I and 29% to NYHA II in the group of patients who received optimal medical management for heart failure, and experienced more improvement in quality of life.[12] In addition, patients with CF-LVADs had fewer complications than those with pulsatile devices.[13] By the first half of 2010, more than 98% of all LVADs implanted were nonpulsatile and were placed inside the chest cavity.[14,15] Actuarial survival of all patients with continuous flow devices was 80% at 1 year, 70% at 2 years, 59% at 3 years, and 47% at 4 years.[16] The HM3 which has a Conformité Européene Mark approval in the European Union in 2015 has the impeller that is fully magnetically levitated in its housing. The wider gap between the impeller and housing reduces shear forces on the blood cells as they pass through the pump.[17] The HM3 was approved in 2017 by the Food and Drug Administration for BTT and bridge to recovery following the results of MOMENTUM 3 trial, which compared the performance of HM3 to the HMII in patients with advanced-stage heart failure.[18] The MOMENTUM 3 reported that 86% of the patients implanted with HM3, as compared to 77% of the patients with HMII, survived to 6 months without a disabling stroke and without the need to replace the pump. None of the patients with HM3 experienced pump thrombosis at 6-month postimplantation, while 10% of the patients with HMII did.[19]

Noncardiac Surgery for Patients with Left Ventricular Assist Device

With improved survival, there has been a surge in the number of patients supported by VADs requiring noncardiac surgical (NCS) procedures. Between 20% and 50% of patients with LVADs present for both elective and emergency NCS, including NCS during the indexed hospitalization for LVAD implantation, for conditions which may or may not be related to complications arising from the presence of LVADs.[12,20-22]

Interprofessional team approach

A multidisciplinary approach is essential in perioperative management of the patient. Due to the anatomical location of the VAD, all procedures should ideally be performed at centers with experience in implanting or managing patients with LVADs.[23] A cardiovascular surgeon should be informed before surgery and should be immediately available for consultation (Class 1, Level of evidence: C).[7] The noncardiac surgeon should ideally also have had experience operating in patients with LVADs before (Class 2, Level of evidence: C).[7] The patient who is still dependent on pharmacotherapy for heart failure, has major comorbidities, or is scheduled for major surgery with predicted significant hemodynamic changes should be cared for by the cardiac anesthesiologist. Noncardiac anesthesiologists can care for patients who are stable on their LVADs, do not require pharmacological support, and are scheduled for NCS under monitored anesthesia care (MAC), where significant hemodynamic fluctuations are not expected. However, a cardiac anesthesiologist should be informed about the NCS and be physically available for consultation.[24,25] Endoscopy and cystoscopy can be performed in their usual procedure rooms.[24]

Understanding pharmacotherapy for the patient with left ventricular assist device

Pharmacotherapy for heart failure

After LVAD implantation, patients will continue to receive heart failure medications (angiotensin-converting

enzyme [ACE] inhibitor, angiotensin receptor blocker [ARB], beta-blocker, hydralazine, and nitrates) for blood pressure (BP) management (Class I, Level of evidence: C).[7] Adverse events have been seen with mean arterial pressure (MAP) >110 mmHg in CF-LVAD, or systolic BP >140 mmHg and diastolic BP >90 mmHg in pulsatile pumps.[16] High peripheral vascular tone in such hypertension increases the afterload and limits the pump output at any given speed.[26] The constant high pressure on the aortic valve (AV) may also cause or worsen aortic regurgitation, and in-pump thrombosis is associated with MAP >90 mmHg.[27-29] Elevated BP in patients with CF-LVAD was independently associated with a greater risk of subsequent stroke.[30] The ISHLT 2013 guidelines recommend a target of MAP <80 mmHg in CF pumps, and systolic BP <130 mmHg and diastolic BP <85 mmHg in pulsatile pumps (Class IIb, Level of evidence: C).[7] However, there is no strong evidence for a specific target BP level and titration of heart failure medications and antihypertensive therapy is best optimized for the individual.[31]

Both ACE inhibitor and ARB have additional benefit of risk reduction in patients with vascular disease and diabetes mellitus. Atenolol or bisoprolol can be prescribed for rate control in patients with tachyarrhythmias. Loop diuretics and thiazides are useful for the management of volume overload (Class I, Level of evidence: C).[32] Patients with existing gout may experience exacerbations while taking diuretic therapy. Aldosterone receptor antagonists such as spironolactone and eplerenone may be used to limit the need for potassium repletion in patients with adequate renal function and for potential antifibrotic effects on the myocardium (Class I, Level of evidence: C).[7] Digoxin may be used to control ventricular response in the patient with atrial fibrillation (Class II, Level of evidence: C).[7]

Antithrombotic therapy

Anticoagulation and antiplatelet therapy are started once hemostasis is achieved following implantation of the LVAD to reduce the risk for thrombosis, thromboembolic stroke, and peripheral thromboembolism.[33-35] With blood returning to the ascending aorta through the device outflow cannula, the pressure within the aortic root may maintain the competent AV in a closed position, promoting stasis of blood at the LV outflow tract and increasing the risk for intraventricular thrombus formation.[36,37] Thrombosis can also occur in the aortic root, as well as on the cusps of AV due to the positioning of the inflow cannula.[38,39] There are two types of pump thrombosis – subacute hard white thrombi (secondary to platelet aggregation activated by shear stress) and acute soft red thrombi (precipitated by stasis, activation of the coagulation cascade, and trapping of red blood cells within a fibrin mesh).[40] Thrombus in the left ventricle and subaortic region can be sucked into the VAD and cause the pump to malfunction. LVAD malfunction results in reduced systemic flows, life-threatening hemodynamic instability, cardiogenic shock, and death.[41,42] These effects may partly be ameliorated by decreasing pump speed to reduce flow velocity, allowing the AV to open for at least a quarter of the cardiac cycle (Class IIb, Level of evidence: B).[7]

Currently, warfarin remains the anticoagulant of choice for patients with LVAD (Class 1, Level of evidence: B).[7,43] Depending on the device, the target International Normalized Ratio (INR) may range from 2.0 to 3.5, with pulsatile devices requiring a higher maintenance INR.[7] The recommended range of maintenance INR for HMII and HVAD is 2.0–3.0. INR <2.0 increased the rate of thrombotic events and approximately 40% of ischemic strokes occurred in patients with INRs <1.5.[44] Conversely, 33% of hemorrhagic strokes occurred in patients with INRs >3.0.[44,45] Warfarin is a pregnancy category X drug. Women of childbearing age are advised against pregnancy after implantation of LVAD and the use of contraception is recommended (Class I, Level of evidence: C).[7,46] Aspirin (81–325 mg daily) is added to decrease the risk of platelet activation and aggregation (Class I, Level of evidence: C).[7]

Gastrointestinal bleeding

An undesirable outcome of antithrombotic therapy while on a CF-LVAD is bleeding episodes. Such events are associated with INR >2.5.[47] Over 50% of patients with CF-LVAD have bleeding from gastrointestinal tract (BGIT), with a readmission rate ranging between 1.6 and 2.5 admissions per patient year.[13,20,48,49] The most common sites of BGIT were the stomach (40%), duodenum (25%), and jejunum (15%).[49] Patients with BGIT may have had previous BGIT from the same site, from the upper GIT, and be of an older age.[44] Melena is a common presentation.[50]

Due to the altered flow dynamics, patients with CF-LVADs develop type IIa acquired von Willebrand disease (avWD), which is determined by an acquired loss of large von Willebrand factor (vWF) monomers.[51-53] Explantation of LVAD has been associated with reversal of the avWD state.[54] The pathogenesis of avWD involves high shear stress-induced amplified ADAMTS-13 (also known as vWF-cleaving protease) mediated proteolysis of the highest molecular weight multimer of vWF.[55] Proteolysis of vWF prevents the binding of collagen and platelets at sites of vascular injury. Approximately 50% of patients with confirmed avWD had BGIT.[49,56]

Gastrointestinal arteriovenous malformation (AVM) or angiodysplasia accounted for 55% of BGIT, 37% of which occurred in the duodenum.[49] The development of AVM in patients with CF-LVAD is likely a result of nonpulsatility of blood flow and gradual mural smooth muscle relaxation. Gradual distension of the submucosal vessels results in intestinal mucosal hypoperfusion and AVM.[57]

Accordingly, nonsurgical bleeding risk in the first 3 months postimplantation of LVAD has been found to be four times higher in patients with low pulsatility index.[58]

Management of gastrointestinal bleeding

The immediate management of the patient with BGIT includes withholding warfarin and antiplatelet therapy, reducing pump speed, and fluid resuscitation.[7,44] In patients who present with acute BGIT, withholding warfarin therapy was not associated with increased rate of thromboembolic events.[44] Unless the patient has had multiple life-threatening hemorrhages, stopping antithrombotic therapy completely is not recommended. In fact, in a study by Katz *et al*. in 2015, all-type bleeding still recurred in approximately 50% of patients with reduced antithrombotic therapy.[59] Thalidomide, an anti-angiogenic compound, has been used for the management of refractory BGIT in patients with LVAD.[60,61] A summary of recommendations from the 2013 ISHLT for the management of patients with BGIT is listed in Table 1.[7]

Table 1: Summary of 2013 International Society for Heart and Lung Transplantation recommendations for management of gastrointestinal bleeding

Recommendations	Class	Level of evidence
Anticoagulation and antiplatelet therapy should be withheld in the setting of clinically significant bleeding	I	C
Anticoagulation should be reversed in the setting of an elevated INR and clinically significant bleeding	I	C
Anticoagulation and antiplatelet should continue to be withheld until clinically significant bleeding resolves in the absence of evidence of pump dysfunction	I	C
The patient, device parameters, and the pump housing (if applicable) should be carefully monitored while anticoagulation and antiplatelet therapy is being withheld or reduced in dose	I	C
The patient should be comanaged with gastroenterology	I	C
The patient should have colonoscopy and upper endoscopic evaluation	I	C
For first episode of BGIT, once the gastrointestinal bleeding has resolved, anticoagulation and antiplatelet therapy can be reintroduced with careful monitoring	I	C
For recurrent gastrointestinal bleeding with no source or a source that is not amenable to therapy, the use of warfarin (and goal INR) and antiplatelet therapy should be reevaluated	I	C
For the patient with recurrent BGIT due to arteriovenous malformation, reduction of pump speed of the device may be considered	IIb	C

INR: International normalized ratio, BGIT: Bleeding from gastrointestinal tract

Preoperative evaluation

Planning the surgical approach

The surgical team should review the radiographs for the locations of the components of the LVAD, especially for LVADs that are implanted in the preperitoneal region (LVAD pocket, inflow and outflow cannulas) and evaluate the lie of percutaneous driveline cable when planning the surgical approach and optimal positioning of the patient for the surgery. In order to avoid having surgical incisions near the pump pocket or driveline cable and to avoid dislodgement of the inflow and outflow cannula during surgical retraction, laparoscopic approach for abdominal surgery, whenever feasible, may be a better option than laparotomy. However, the presence of preperitoneal-implanted LVAD or the percutaneous driveline cable may not permit adequate access to the abdomen for port placement.[62] With LVADs which are implanted within the left thoracic cavity, satisfactory abdominal port placement for laparoscopic instruments may be possible. When a significant amount of intra-abdominal adhesions from previous abdominal procedures is encountered, the surgeon may still have to convert to laparotomy for easier access.[63] Whatever the surgical decision, the surgical team must be mindful of the potential precarious intraoperative hemodynamic state of the patient and that duration and manipulation of viscera during surgery will affect intraoperative and postoperative cardiopulmonary function and abdominal organ perfusion.

Evaluation of the function of left ventricular assist device

The LVAD pump speed and function should be assessed preoperatively by a specialist from the VAD program. Additional screening for hemolysis should be performed in the setting of an unexpected decrease in hemoglobin concentration or hematocrit level or hemoglobinuria (Class I, Level of evidence: C).[7] These tests include serum lactate dehydrogenase (LDH) concentration and plasma-free hemoglobin concentration (Class IIa, Level of evidence: C).[7]

A VAD specialist from the team should be available to monitor the LVAD function throughout the time that the patient is in the operating room (OR) (Class I, Level of evidence: C).[7] In the OR, the power supply for the LVAD should be transferred to the mobile power unit that is connected to wall power supply. It has been shown that such personnel bring comfort and confidence to the nursing and medical team if they are involved throughout the care of the patient.[25]

Echocardiographic evaluation

A comprehensive preoperative transesophageal echocardiography (TEE) or transthoracic echocardiography (TTE) should be performed to determine function of the ventricles and valves, estimate pulmonary artery pressure, and exclude the presence of atrial septal defect (ASD), cardiac thrombi, and pericardial effusion.[64] The position of the interventricular septum can serve as a guide to volume status and proper speed of the device.[13] If blood is pumped out of the left ventricle faster than the rate of blood flowing through the mitral valve, the interventricular septum will be "sucked" toward the left ventricle. An unrecognized ASD or patent foramen ovale at the time of implantation of LVAD can promote hypoxemia from right-to-left shunting and increase the risk of paradoxical embolism during Valsalva maneuvers. Dilation of the aortic annulus results in significant aortic regurgitation and recirculation of a portion of blood through the LVAD, reducing effective forward flow and organ perfusion, while increasing LV end-diastolic pressure (LVEDP). Aortic root dilation is likely to be secondary to mural smooth muscle atrophy following prolonged duration of abnormal aortic wall stress from abnormally directed blood into the aorta through the "narrower" outflow cannula.

Management of cardiovascular implantable electronic devices

Transient ventricular arrhythmias (VA) occur in up to 34% of patients after 1 year of LVAD implantation; hence, an implantable cardioverter-defibrillator (ICD) is recommended for patients with LVAD (Class IIa, Level of evidence: B).[7,65] The main concern for the presence of a pacemaker or ICD, collectively known as cardiovascular implantable electronic devices (CIEDs), is the potential for interaction between the CIED and electromagnetic interference (EMI) due to the intraoperative use of diathermy (or electrocautery).[66]

Before surgery, the surgical team and the anesthesiologist should communicate with the electrophysiologist following the CIED to plan perioperative management of the CIED (Class I, Level of evidence: C).[67] The CIED prescription may involve reprogramming a pacemaker or ICD to an asynchronous pacing mode (VOO or DOO), reprogramming an ICD to inactivate tachytherapy, or applying a magnet over the CIED. The OR team should be familiar with the type of CIED (pacemaker versus ICD) and the response of the CIED to magnet application and review the patient's underlying cardiac rhythm. If the procedure involves only bipolar diathermy or harmonic scalpel, interaction with the CIED is unlikely. With monopolar diathermy, the EMI may cause transient inhibition of pacing in pacemaker-dependent patients (usually those with complete atrioventricular block) and may trigger inappropriate shocks in patients with ICDs.

Evaluation by anesthesiologist and preparation of the patient

Besides the routine considerations for general anesthesia (GA) (review of airway, cardiopulmonary function, control and severity of comorbidities, pharmacotherapy and presence or absence of drug allergy, hematology, coagulation profile and biochemistry studies, sequelae of previous surgical interventions), the anesthesiologist should enquire the type and history of the LVAD (indication, duration, settings, and complications), be aware of the exit site and direction of driveline cable, and arrange for reprogramming of the CIED to asynchronous pacing mode and inactivation of tachytherapy function of the ICD. Patients who are educated about their device would likely be able to provide the relevant information. If the patient had surgical AV replacement before this admission for NCS, it may likely have been performed for aortic incompetence. The anesthesiologist should review the preoperative echocardiography findings as the findings will be helpful in planning the conduct of anesthesia to optimize pump flow and cardiac output.

On the day of surgery, except for patients with diabetes mellitus, patients should continue taking essential medications preoperatively. Do note that preoperative administration of ACE inhibitor or ARB increases the tendency for intraoperative hypotension, but the long-term clinical consequences of continuing versus withholding preoperative ACE inhibitor or ARB is unknown.[68] If ACE inhibitors or ARBs are withheld before surgery, it is reasonable to restart as soon as clinically feasible postoperatively (Class IIb, Level of evidence: C).[67]

In a study over 10 years, Stone *et al*. described a practice of decreasing anticoagulation to the lower limit of therapeutic levels for most elective cases, and only reverse in neurosurgical, ophthalmologic, or emergency cases.[25] Bhat *et al*. also demonstrated that anticoagulation could be withheld preoperatively without thrombotic complications.[69] Thienopyridine antiplatelet agents should be stopped at least 5 days prior to elective surgery (Class IIb, Level of evidence: C).[7] Aspirin and warfarin may be continued perioperatively in nonemergent NCS if the risk of bleeding is low (Class I, Level of evidence: C).[7] However, if the risk of bleeding is high, warfarin may be held off and bridged with heparin infusion, subcutaneous low molecular weight heparin (LMWH), or heparin alternative which should be stopped on the morning of the planned procedure (Class I, Level of evidence: C).[3,7,70-73] For emergency surgery, warfarin effect may be rapidly reversed with fresh frozen plasma or prothrombin complex concentrate. Vitamin K may be administered, but the onset of reversal of anticoagulation profile is slower (Class I, Level of evidence: B).[7] After the procedure, warfarin and antiplatelet therapy may be resumed when the risk of surgical bleeding is acceptable. The patient may be bridged with LMWH while waiting

for the INR to reach the target range (Class I, Level of evidence: B).[7]

The usual fasting guidelines before surgery and anesthesia apply. The patient can have clear fluids (e.g., clear broth, apple juice, or water) up to at least 2 h before anesthesia and surgery.[74] During the period of fasting, adequate intravenous fluid should be administered to the patient to avoid dehydration which will cause a gradual reduction in cardiac output.

Preoperative team briefing

At the earliest opportunity, a preoperative team briefing should take place for all OR personnel to understand the patient's predicament and the surgical plan. During this briefing, the surgeon describes the operative plan and specifies the patient position, prophylactic antibiotics, and surgical instruments that are required for the procedure; the anesthesiologist shares the findings of preoperative evaluation, the anesthetic considerations, and plan(s) for the patient. This will be the best opportunity to highlight to everyone that LVAD is preload dependent for optimal function and that venous return and preload will be reduced if the surgical approach requires any of the following positions: reverse Trendelenburg, beach chair, lateral decubitus, and prone positions.[75-78] If laparoscopic approach is planned, it will be important to limit the intra-abdominal pressure to approximately 10–12 mmHg in order not to have a great impact on venous return during the laparoscopic procedures.[79] Stepwise peritoneal gas insufflations will be helpful as volume therapy can be calibrated. Care should be taken to protect the driveline cable from pressure and bending before placement of sterile surgical drapes.

Having a shared mental model will benefit the patient as procedures and care can be coordinated minimizing unexpected delays in the OR, improving quality of care and possibly reducing health-care cost.[80]

Choice of anesthetic technique

The anesthesia options that can be provided to the patient depend on the type of surgery that the patient is scheduled for. If the surgical procedure can be performed under local anesthesia or peripheral nerve block, MAC would be ideal, as the patient maintains spontaneous respiration and cardiovascular tone. Nearly half of the patients with LVAD require endoscopy and these procedures can frequently be done safely under MAC.[81] If GA with endotracheal intubation is required for the surgical procedure, rapid sequence induction is recommended in patients with early models of LVADs which had preperitoneal placement.[70]

Intraoperative monitoring

Cardiac rhythm

Patients with ICDs who have preoperative reprogramming to inactivate tachytherapy should be monitored with 5-lead electrocardiography continuously during the entire period of inactivation for early detection of myocardial events (Class 1, Level of evidence: C).[67]

Blood pressure and central venous pressure

Patients with CF-LVAD may not have a pulse, especially during anesthesia, making meaningful pulse oximetry and noninvasive arterial BP monitoring impossible. Even with pulsatile flow LVAD, pulsatility of blood flow may be lost if the patient becomes hypovolemic or vasodilated secondary to intraoperative events.[25] For minor or brief procedures (endoscopy for BGIT) suitable for monitored anesthetic care, use of Doppler ultrasound over the brachial artery distal to a sphygmomanometry BP cuff or combining sphygmomanometry with finger pulse oximeter permits assessment of systolic BP, which in these patients is often similar to MAP (Class I, Level of evidence: C).[82-85] For other procedures, intra-arterial monitoring of MAP is advisable (Class I, Level of evidence: C).[7] In the absence of a palpable pulse, use of ultrasound can facilitate placement of these lines.[13] A central venous catheter may be placed for monitoring of central venous pressure (CVP) and administration of vasoactive drugs during surgical procedures of moderate to high risk (Class I, Level of evidence: B).[7]

Cerebral perfusion

There is evidence to support the utilization of cerebral near-infrared spectroscopy (NIRS) to monitor oxygen delivery in patients with LVAD for NCS.[86] Significant decrease in regional cerebral oxygen saturation (rSO_2) from baseline values throughout the intraoperative period increases the risk of postoperative cognitive decline, delirium, longer Intensive Care Unit and hospital length of stay, and major organ dysfunction.[87-91] In NCS, decline in rSO_2 is usually associated with blood loss.[92] Early interventions, such as optimizing mean BPs, ensuring adequate alveolar ventilation to correct systemic desaturation and correcting hyperventilation to normalize partial pressure of carbon dioxide (PaCO2), to reverse decreases in rSO_2 and to maintain rSO_2 to within 10%–20% of baseline, will help reduce complications associated with intraoperative oxygen debt.[93]

Transesophageal echocardiography

Intraoperative TEE should be performed by physicians (cardiologists or anesthesiologists) with advanced training in the intraoperative assessment of cardiac structure and function (Class I, Level of evidence: B).[7] Echocardiography during NCS allows the anesthesiologist to monitor patency and position of inflow and outflow cannulas, investigate the source of thromboembolic material, and monitor adequacy of LV filling and unloading, RV function, and effects of volume replacement therapy and pharmacologic support.[94]

Left ventricular assist device function

The system monitor in HMII and HM3 displays speed (in revolutions per minute, RPM), power

(in watts, W), flow (in L/min), and pulsatility index (PI, dimensionless value). The HVAD monitor displays the RPM, power, and the flow rate. The pulsatility is displayed as a waveform in the monitor. The only control one has over the CF-LVAD is the pump speed. Pump flow, a surrogate for cardiac output, is estimated based on power consumption and pump speed.[13] Low pump flow can be caused by low preload or high afterload. The variation in pump flow (Q) is used to derive the PI: 10 × (Qmax−Qmin)/Qavg. A gradual increase in pump power consumption may suggest thrombus formation in the device. Thrombus formation on the impeller of the pump will increase the resistance to the rotation, resulting in increased power consumption, increased calculated value of the pump flow, and the system monitor displaying a higher flow rate than the actual real-time cardiac output. The diagnosis of pump thrombosis is further supported by the following features: TEE confirmation of increased ejection of blood through the AV and decrease in flow through the inflow cannula, accompanying hypotension, red to reddish-brown urine, worsening renal function, decreased serum haptoglobin, increased plasma-free hemoglobin, and elevated serum LDH concentrations.[95] A low value (<5 W) paradoxically suggests occlusion of flow path.[13]

For HMII, typical pump speed ranges above 9000 RPM, with pump power ranging from 5 to 8 W and PI in the range of 3–4. Keeping device speeds >9000 RPM reduces the risk of pump thrombosis.[96] For HVAD, while the operating guidelines indicate that the pump speed can be set between 1800 and 4000 RPM, the clinical operating speed range is 2400–3200 RPM. Speeds <2400 RPM should only be used during the implant procedure when weaning from cardiopulmonary bypass. Speeds >3200 RPM are seldom needed and increase the risk of suction events. HVAD pump power ranges from 2.5 to 8.5 W when operating within the operating speed range.[97] Power values >8.5 W suggest a problem which should be evaluated by log file analysis. For HM3, the typical pump speed range is between 5200 and 5500 RPM.[98]

Maintaining left ventricular assist device function

Afterload to the left ventricular assist device

The output of the CF-LVAD is afterload sensitive. Maintain MAP <80 mmHg with CF-LVAD and systolic BP <130 mmHg and diastolic BP <85 mmHg with pulsatile pumps (Class IIb, Level of evidence: C).[7] Ensure adequate depth of anesthesia and analgesia during laryngoscopy and surgery, as light anesthesia may result in an abrupt increase in SVR during laryngoscopy and precipitously drop cardiac output.[3]

On the other hand, systemic vasodilatation can cause excessive offloading of the left ventricle, altering the geometry of the right ventricle, precipitating RV dysfunction, and limiting preload to the LVAD. TEE will be able to demonstrate the "suck down" phenomenon. When excessive offloading of the left ventricle is encountered, decreasing the LVAD speed momentarily may allow for improved LV filling, though this requires close coordination with the VAD specialist.[99] The treatment of hypovolemia and bleeding requires infusion of intravenous fluids and blood transfusion, respectively. Vasopressors can be administered in small bolus doses and titrated to effect, taking care to avoid creating an excessive afterload on the right ventricle. Low-dose vasopressin (<2.4 U/h) may be used, due to its minimal effect on the pulmonary vasculature.[100] Delivery of anesthesia guided by BIS (bispectral index) may avert overly deep anesthesia.[101]

Preload to the left ventricular assist device

The LVAD is preload dependent. Preload to the LVAD is influenced by systemic venous return, RV function, and pulmonary vascular tone. In the event of decreased venous return after induction of anesthesia, placing the patient in a slight Trendelenburg position, judicious volume therapy, and adjusting ventilatory settings that will promote venous return are helpful strategies. It is important to maintain adequate myocardial perfusion to optimize RV function.

Positive pressure ventilation, placing the patient other than supine position for surgery as mentioned in a previous section, presence of a gravid uterus, and an intra-abdominal pressure higher than 15 mmHg during laparoscopic procedures are intraoperative factors that decrease venous return.[79,102] Judicious fluid loading is advisable if a nonsupine position of the patient is required for surgery. The hemodynamic instability may be limited by reducing the degree or duration of surgical position that is contributing to the decline in venous return. If reverse Trendelenburg is necessary, flexing the operating table at the level of patient's hip can limit venous pooling in the limbs. The pattern of the CVP trace provides information about the right atrial volume. Patient's urine output and amount of blood loss should be monitored to optimize fluid therapy and blood transfusion, if necessary. The overall effect on venous return during the phase of pneumoperitoneum for laparoscopic procedures depends on whether Trendelenburg or reverse Trendelenburg position is incorporated. Inducing pneumoperitoneum subsequent to placing the patient either in lithotomy or lateral position increased preload, probably as a result of blood shifting from the abdomen to the thorax with institution of pneumoperitoneum.[103,104] When high positive end expiratory pressures and pneumoperitoneum are applied together, preload is significantly decreased.[105] Volume therapy should be restricted if the patient is placed in the steep Trendelenburg position for laparoscopic colorectal surgery or prostate surgery. For adequate surgical exposure at low intra-abdominal pressures of 10–12 mmHg, the abdominal wall muscles should be well relaxed. This can be achieved through maintenance of adequate depth of anesthesia and analgesia as well as regular monitoring of neuromuscular function.

During laparoscopic procedures, there is a direct relationship between arterial partial pressure of carbon dioxide and the amount of carbon dioxide insufflation. It is important to maintain normocarbia as hypercarbia can increase pulmonary vascular resistance which in turn places an additional strain on the right ventricle.[106] Beyond ventilatory measures and ensuring acid–base balance, pharmacotherapy can be instituted to induce pulmonary vasodilation to reduce RV afterload.[4,107]

Management of intraoperative ventricular arrhythmias

VAs are extremely common in these patients, especially if they had preexisting VAs before LVAD implantation. External adhesive defibrillation pads should be applied on the patient preoperatively.[3,106] A magnet should be available for all patients with a CIED who are undergoing a procedure that could involve EMI. External defibrillation equipment with transcutaneous pacing capability should be readily available in the OR. Where diathermy is unavoidable, limit its use to short bursts and ensure that the return electrode for the diathermy is anatomically positioned so that the current pathway between the diathermy electrode and return electrode is as far away from the CIEDs and leads as possible. Patients should be kept in normal sinus rhythm when possible as VA may impair the unassisted right ventricle, leading to RV dysfunction, and decreasing inflow to the LVAD.[72,108]

The causes of VA include mechanical irritation from the inflow cannula, excessive ventricular offloading, altered ventricular repolarization, previous history of VA, VAD dysfunction with increased LVEDP, metabolic imbalance, effect of pharmacotherapy, and re-entry (scar related).[95,109] Higher mortality rates have been reported in patients who developed VA, 30% compared to 18% in patients without arrhythmias, and more than 50% if occurring less than a week after LVAD implantation.[110] One of the most common causes of both sustained and nonsustained ventricular tachycardia among LVAD patients is suction events where the inflow cannula is in contact with the septal wall. Suction events are more common when the LV is relatively underfilled and can easily be confirmed by TEE imaging. Immediate management include momentarily decreasing the LVAD speed while administering intravenous volume therapy.[99]

The 2013 ISHLT guidelines recommend the following: if the patient is hemodynamically stable, manage reversible causes such as electrolyte abnormalities (Class I, Level of evidence: C). Cardioversion is recommended for VT that results in poor device flows and/or hemodynamic compromise (Class I, Level of evidence: C). Amiodarone is reasonable and beta-blocker therapy is useful (Class IIa, Level of evidence: C). Cardioversion of AF is recommended in patients with rapid ventricular rates compromising device performance (Class IIa, Level of evidence: C).[7]

Recurrent VAs that are difficult to treat should prompt consideration of ischemia as the driving mechanism, particularly in patients with ischemic cardiomyopathy. Maintain MAP to approximately 80 mmHg to ensure coronary perfusion. Slower VT is well tolerated and may not always require therapy. External chest compression should be avoided during cardiac arrest as there is a risk of cannula dislodgement and life-threatening hemorrhage.[71] If chest compressions have been administered, the position of the inflow cannula must be checked with TEE immediately after the event.[97]

In summary, the immediate management for VA occurring during NCS includes momentarily reducing the LVAD speed while administering volume therapy, performing TEE to exclude mechanical irritation of the ventricular wall as a cause of VA, a point-of-care test to exclude electrolyte abnormalities, and initiating cardioversion.

Postoperative management

Ensure that the ICD is reprogrammed to active therapy (Class I, Level of evidence: C)[67] Patients should be extubated when they meet standard criteria. Care should be taken during extubation to minimize hemodynamic changes such as hypertension and tachycardia, which may negatively affect LVAD output.[23]

The patient can be recovered in the standard postanesthesia care unit unless intensive postoperative care is indicated.[3,111-113] Postoperative care should be well planned and coordinated effort, as residual anesthetic effects and subsequent hypoventilation had resulted in unexpected deaths.[23]

Optimization of RV preload, reduction of RV afterload, and avoidance of excessive LV unloading with destabilization of RV geometry by leftward shift of the ventricular septum remain important in the postoperative period. Common causes of increase in RV afterload such as hypoxia, hypercarbia, and acidosis should be avoided. Ensuring adequate analgesia will help attenuate the sympathetic and hypertensive responses.

Postoperative bleeding is a frequently reported complication following NCS.[114] This is related to the reinstitution of full anticoagulation therapy. In general, warfarin therapy is restarted when the risk of postoperative bleed is low. Meanwhile, heparin is administered to achieve partial thromboplastin time of 60–80 s, until the INR reaches the target range of between 2.0 and 3.0 (Class I, Level of evidence: B).[7,114] If the risk of postoperative bleeding is deemed high, heparin bridging may be omitted, or full resumption of anticoagulation delayed.[4,114]

Other complications that require immediate attention include wound infections, pneumonia, urinary tract infection, acute renal failure, pulmonary embolus or deep vein thrombosis, and sepsis.[20] On multivariate analysis, the requirement of NCS (odds ratio: 1.45, 95% confidence

interval: 0.95–2.20, P = 0.08) was not associated with mortality.[20]

Conclusion

The CF-LVAD is the most common LVAD encountered today. It is essential for the anesthesiologist to know the type of LVAD the patient has and its basic features as a part of preoperative evaluation. With close attention paid to optimizing RV function, facilitating preload to the device, maintaining the pump speed in an optimal range, and managing systemic vascular resistance, NCS in the patients supported by LVAD can be safe and feasible.[115] A multidisciplinary collaborative approach ensures best outcomes for this group of patients.

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Conflicts of interest

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