Chapter

Anesthesia for Non-Cardiac Surgery for the LVAD Patient

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Abstract

Heart failure is poorly tolerated and end stage heart failure (classified as New York Heart Association (NYHA) class IV) has a two-year survival with medical therapy that approaches 0%. Innovation in this sphere has yielded mechanical therapies, principally the left ventricular assist device (LVAD). In the last decade one-year survival rates of Left ventricular assist device patients have increased from 52–83%. As this therapy is more commonly used to treat advanced heart failure, coupled with the increase in patient survival after implantation, patients are increasingly encountered in the peri-operative arena requiring anesthesia for non-cardiac surgeries. The goal of this chapter is to provide the non-cardiac trained anesthesia provider a primer on what an LVAD is, how it functions, the physiological changes that occur with implantation, and considerations for administering anesthesia to patients with LVADs for non-cardiac surgery. Review of articles from 2018 to 2022 found from a search on PubMed and Google Scholar using the keywords: "Left Ventricular Assist Device", "LVAD", "anesthesia", "non-cardiac surgery", "Doppler blood pressure measurement", "VAD coordinator". Non-cardiac trained anesthesia providers can safely administer the anesthetics to LVAD patients undergoing non-cardiac surgery as long as appropriate considerations are taken.

Keywords: anesthesia, left ventricular assist device, LVAD, non cardiac surgery, blood pressure measurement

1. Introduction

Cardiovascular disease continues to be the leading cause of death in America. Around six million Americans are diagnosed with heart failure of varying degrees each year [1–5]. Left Ventricular Assist Devices are indicated for patients with advanced stage heart failure. Medical optimization can include renin angiotensin-aldosterone system antagonists, sympathetic nervous system antagonists, beta blockers [1, 4–8]. Cardiac resynchronization therapy is often used in chronic heart failure candidates to stave off final implantation [1, 4–8]. In end-stage heart failure, conventional medical therapies have a mortality at 2 years of almost 100% [9]. These patients are classified NYHA class IV or Stage D by the American College of Cardiology Foundation/American Heart Association. Clinically, they have shortness of breath at rest and their echo shows an ejection fraction (EF) of 40% or less. While cardiac transplant is a definitive treatment for severe advanced heart failure and may be the preferred treatment, this solution is limited by donor availability. The failure

Bridge to Recovery (BTR)	VAD is used to support heart until recovered function from shock via ventricular remodeling
Bridge to transplant (BTT)	VAD is used while patient awaits heart transplant
Destination Therapy (DT)	This is definite treatment for patients who are not transplant eligible
Bridge to decision (BTD)/ Bridge to candidacy (BTC)	Used in patients who are not currently but may become eligible for heart transplant related to recent malignancy or potential for improved end organ function with VAD assistance.

Table 1.
Indications of LVAD therapy [4, 5, 8, 11, 14, 15].

of medical management, the shortage of heart donors, and the realization that these patients were not suitable transplant candidates led to the development of the LVAD [2, 4, 5, 10–12].

The first LVADs were used in the 1960s as bridge to therapy (BTT), or as a bridge to recovery (BTR) [2, 4, 5, 8]. Initially, they were designed to emulate the pulsatile action of the heart, had many moving parts and membranes, and were prone to frequent failure. After improvements in portability and mechanical design, the latest generation of devices are all continuous. By 2010, LVADs were approved for destination therapy (DT). As of 2020 destination therapy accounts for 78% of LVAD implantations in the US [13]. **Table 1** describes indications for LVAD therapy.

The evolution of the LVAD devices is a nascent, growing field and improvement to its models was rapid: In 2009, the one-year survival rate of an LVAD patient was 52%. By 2018, the one-year survival rate after an LVAD implant increased to over 80%; the two-year survival was above 70%; some patients lived 4 years or more [1–5, 7, 16–19]. As patients with these devices live longer, they may experience complications or other medical conditions that require surgical intervention and therefore anesthesia. One early review of Medicare patients found that in 2012, 64 LVAD patients had non-cardiac surgery (NCS) and that number increased in 2017 to 304 LVAD patients [7]. No doubt the number now is far higher in the 2020s. As more patients with these devices present for non-cardiac surgery, it is important for anesthesia providers to understand the hemodynamic and physiologic changes that result from LVAD placement and how our anesthetic techniques and medications affect its function [6, 20].

2. The LVAD

The current LVAD is a rotary continuous pump system that is implanted at the apex of the left ventricle and propels blood into the aorta via an outflow graft, typically to the ascending aorta. **Figure 1** shows a basic silhouette of the Heart Mate III, now the most implanted device of the 2020s [1].

The pump receives energy from a driveline that connects extracorporeally to a battery system through a controller when on battery power [1, 3, 11]. This device is so compact, the functional parts fit completely inside the thoracic cavity. The drive train can also be attached to a wall unit for power and with a larger display screen.

2.1 First generation devices

The first generation of LVADs were pulsatile and large [4, 16]. The Thoracec PVAD was the first LVAD to be approved by the Food and Drug Administration. It was used

Figure 1.
The silhouette of the heart mate III components, the most implanted device of the 2020s. (a) Centrifugal pump (b) driveline cable (c) controller panel (d) portable battery [1].

in more than four thousand patients as a bridge to transplant [4]. Its pump was not implanted but had to be carried extracorporeally [16].

The Novacor and HeartMate I (HM I) became the first implantable LVADs. **Figure 2** shows an image of a HeartMate I device and its parts. The pumps of these devices were implanted in a preperitoneal pocket under the abdominal muscles. The Novacor had great durability, lasting for 5–6 years. However, the rate of stroke among its users was near 50% [1]. The HM1 device attempted to recreate physiologic pulsatile flow and was used as a BTT and BTR for more than two decades [4, 16]. The HM I had a 52% one-year survival rate, 48% better results than medical management at those times. Unfortunately, it was mechanically complex, was prone to malfunction, and also had high rates of severe adverse events and infection. The device was trialed for Destination Therapy (DT) but ultimately failed, as it was deemed not ideal and its parts wore out at about 18 months [1, 4].

2.2 Second generation devices

The HeartMate II (HM2) device was introduced in 2008 (**Figure 3**) [4]. It is considered a second-generation device because it delivers continuous flow (CF)

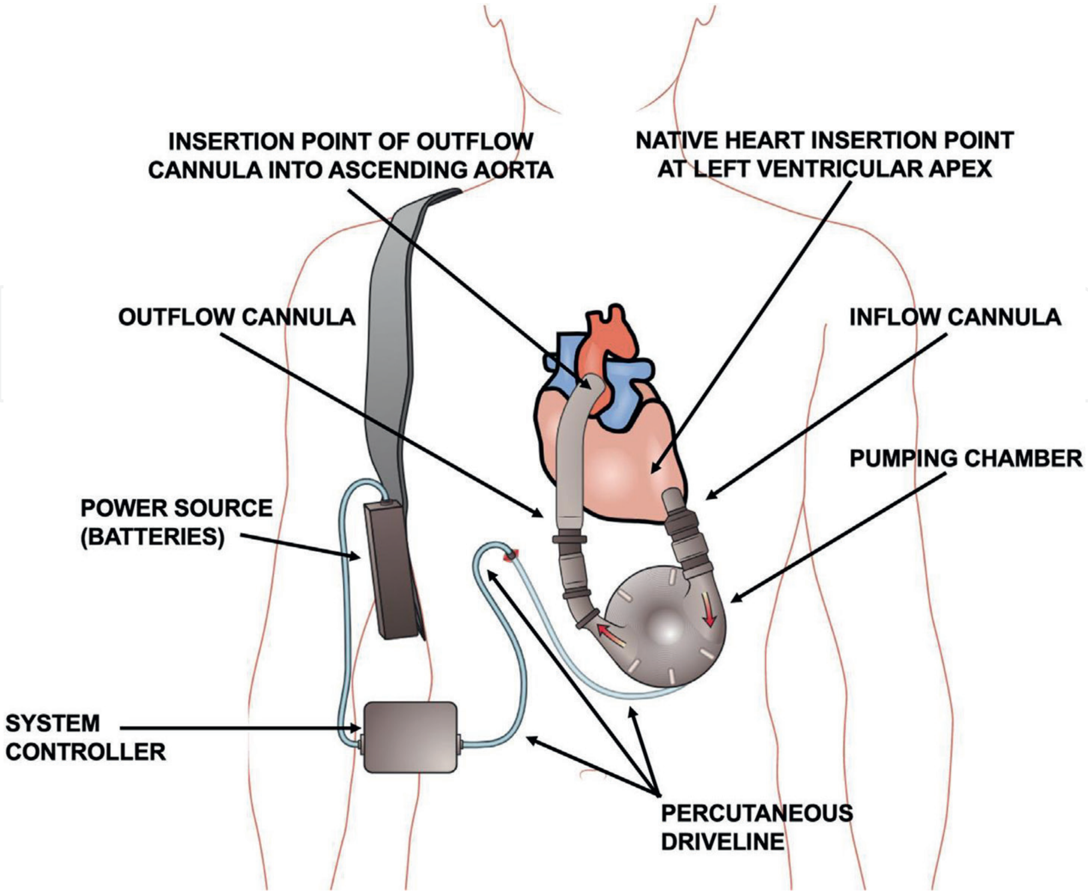


Figure 2.
The HeartMate 1 [3].

powered by an axial flow rotor propeller. The pump of the HM2 is 1/7th the size of the HM I [21], about the size of a D battery but is similarly implanted in a preperitoneal pocket [1, 16]. The HM2 boasted a 68% one-year survival rate with improved quality of life and physical activity noted at 3 months post implantation [4]. It achieved a 58% two-year survival rate compared to the HeartMate I (24%) in the 2010 REMATCH trial.

The HM II was the first device approved for destination therapy (DT) [2, 4, 15, 16, 18]. Rates of stroke, bleeding, infection, and device malfunction were less than its predecessors [1, 4]. With advances in implantation technique, design, and RV support devices, DT patients were approaching a 70% two-year survival rate, with one patient documented to have their HM2 for greater than 8 years [1, 7, 17]. However, the HM2 had its own unique complications related to its continuous axial flow.

Patients with HM2 unfortunately would present with pump thrombus. As a result, use of systemic anticoagulation, such as with warfarin, became a standard for all LVAD patients in 2011 [1]. Acquired von Willebrand deficiency and arteriovenous malformations (AVM) developed related to altered physiology associated with continuous flow LVADs. When combined with prophylactic anticoagulation, the incidence of gastrointestinal bleeding rose [1, 2, 4, 10, 11, 16].

2.3 Third generation devices

Second generation CF devices improved longevity compared to first generation but had multiple moving parts. Third generation CF devices, such as the Heart Mate

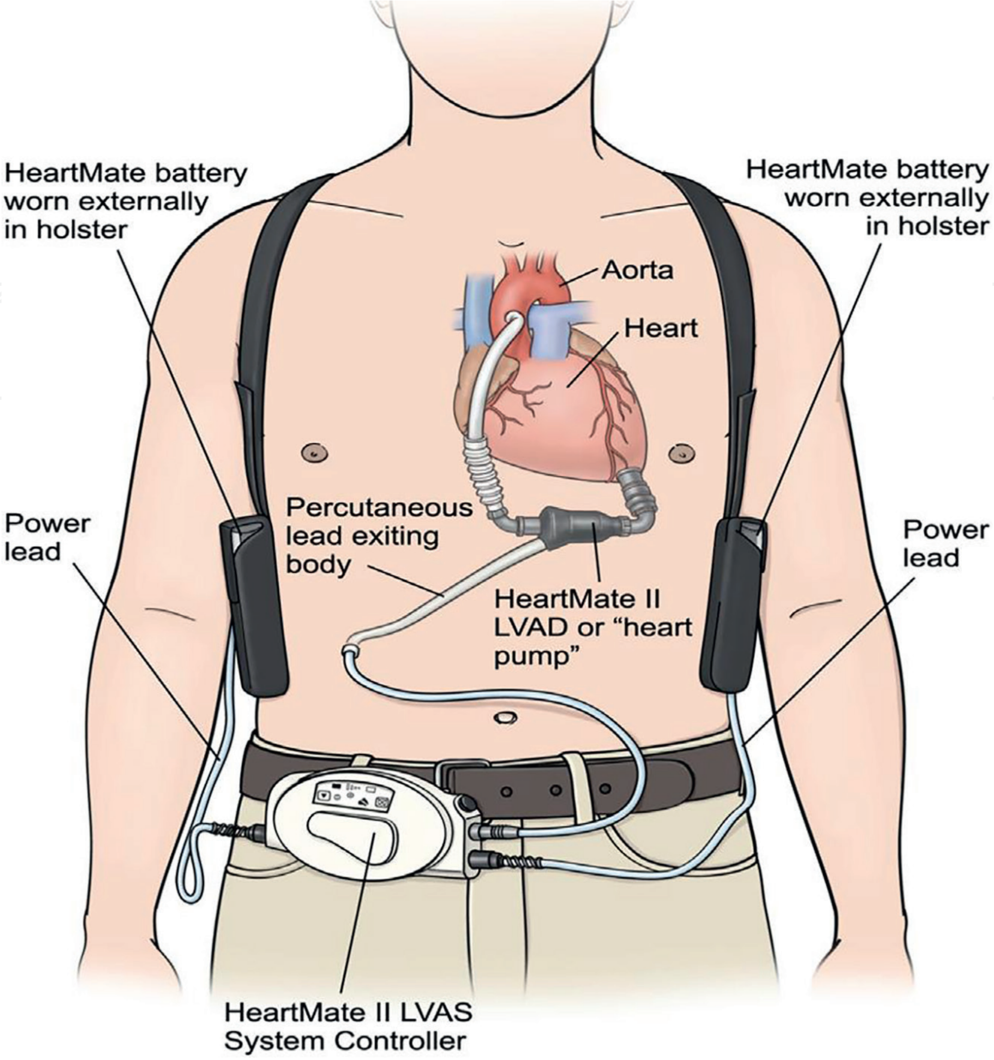


Figure 3.
The heart mate II [4].

III and the HeartWare HVAD, are positioned at the apex of the left ventricle blood pumps blood in a centrifugal manner [1, 4, 16]. Both devices are smaller than their predecessors. They have also shown greater longevity with significantly less need for reimplantation than the HM2 [1, 4, 5].

2.4 HVAD

The Medtronics HeartWare HVAD (**Figure 4**) was approved by FDA in November of 2012 for BTT. It has been implanted in more than 20,000 heart failure patients worldwide, with one HVAD being implanted for greater than 7 years [1, 22]. The HVAD functions via both passive magnetic levitation and a hydrodynamic bearing system [22]. In June of 2021, the sale and implantation of the HVAD was discontinued secondary to technical issues with the device not restarting after planned or accidental power disconnection. The HVAD patients also held a statistically significant incidence of stroke [12, 22]. About 4000 patients worldwide still have HVADs implanted. As a result, Society of Thoracic Surgeons recommends explantation of the HVAD to HM3 only in instances of malfunction, as electively changing devices carries just as much risk as keeping the HVAD [11, 22].

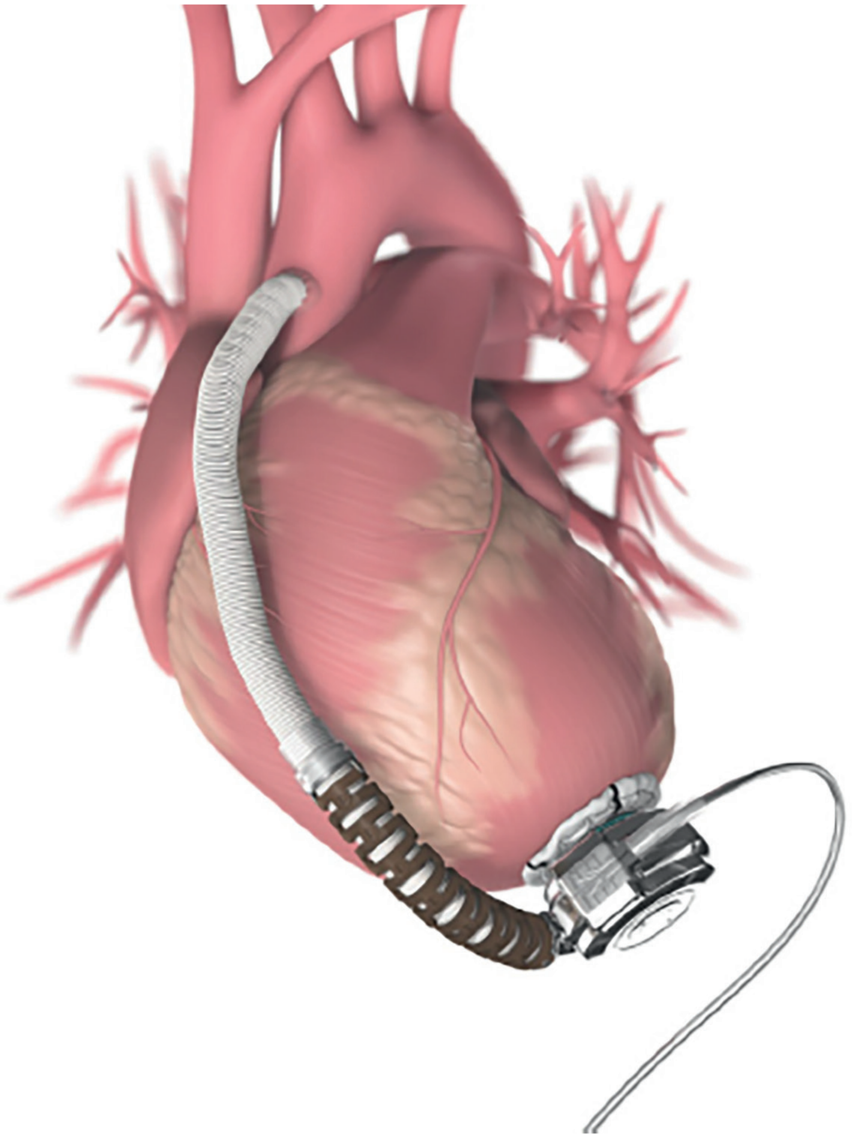


Figure 4.
The internal components of the Heartware HVAD [4].

2.5 HeartMate III

The Abbott HeartMate III system is a completely magnetically levitated centrifugal pump (**Figures 1** and **5**) [1, 4, 16, 22]. The centrifugal flow of the HM3 not only improves longevity of the devices but produces less shear on blood components. This has resulted in milder acquired von Willebrand syndrome. Other adverse events such as pump thrombus, stroke, and GI bleeding are decreased in HM3 compared to HM2 [1, 4, 5, 17, 23]. HM3 patients also spend less days in the hospital 2 years post implant [17]. Unfortunately, rates of right heart failure and infection with third generation devices remain similar to previous generations [1, 4, 17].

2.6 External components of the LVAD

Figure 6 shows an example of a Heartmate III controller. The controller contains a screen that displays four values: pump speed (rotations/minute), pump flow (liters/min), pulsatility index (PI), and pump power (watts) [3, 11, 16]. When in the OR

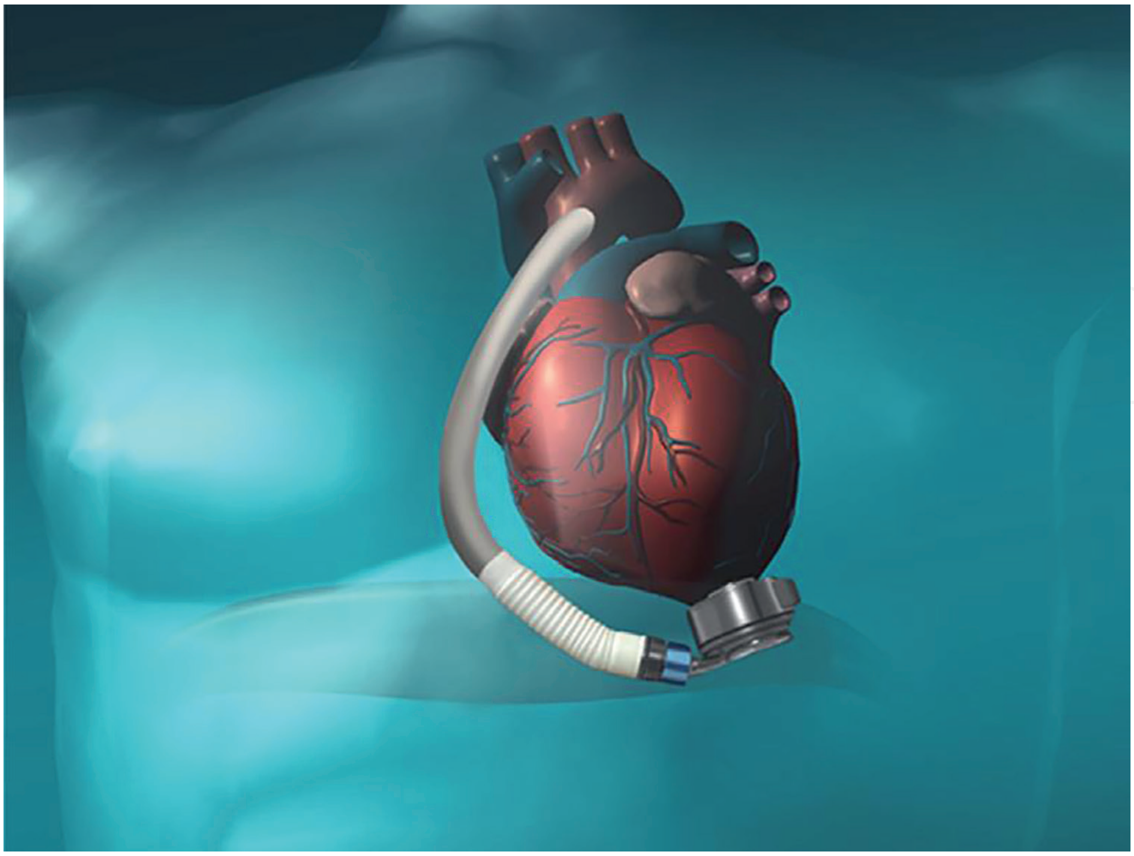


Figure 5.
The internal components of the HeartMate 3 [4].

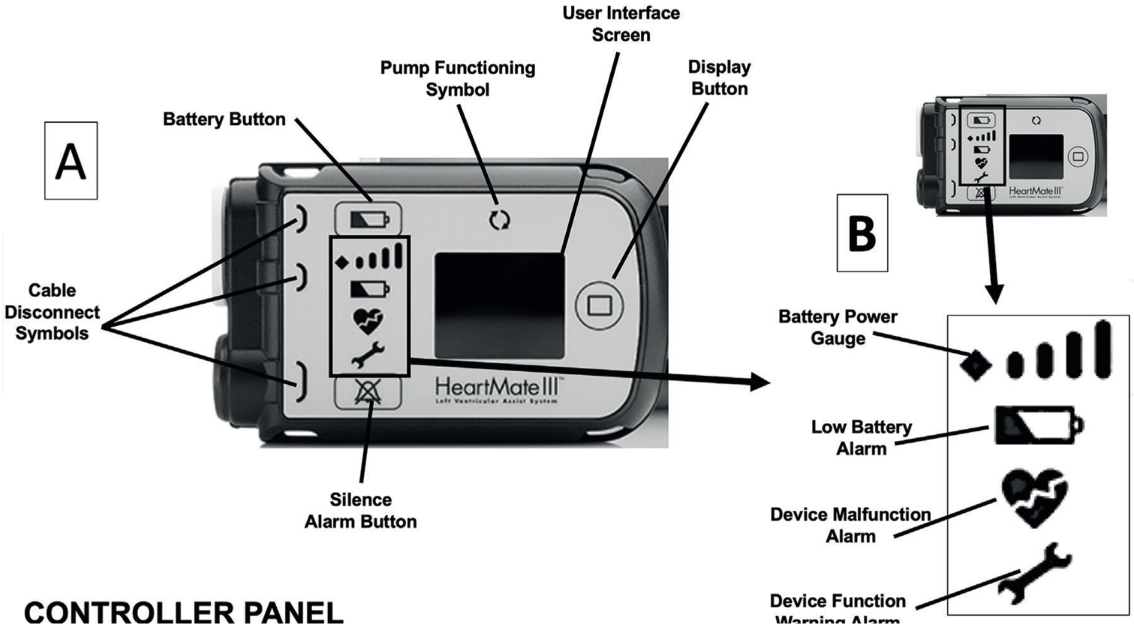


Figure 6.
Controller panel of the HeartMate 3. (A) LVAD controller panel and (B) panel of indicator lights [3].

and connected to wall power, a larger monitor can be fashioned to display all values concurrently.

Pump speed shows how fast the LVAD centrifuge is spinning and is the only directly modifiable value of the LVAD. Pump speed can be adjusted to optimize function under visualizaiton with echocardiogram. Pump flow is analogous to cardiac output and is different for each model; this value is typically a derived number calculated with proprietary formulas [16, 24].

Pump power is indicative of how much power is being required to run the pump at a specific set pump speed. In its normal function, this value varies linearly with systemic vascular resistance.

Pump flow is therefore a calculated value from pump power. Changes and trends of the pump flow value can also indicate complications. For example, any increase in power not related to an increase in actual flow will cause an erroneously high flow to read, such as the presence of a thrombus in the inflow cannula.

The pulsatility index is the difference of systolic and diastolic pressure within the pump system. Its magnitude reflects the amount of assistance provided by the LVAD; when the left ventricle contracts, the PI increases the flow transiently in the LVAD. This value is key to interpreting Doppler blood pressures (DopBP), which will be discussed later in this chapter [16, 19, 25].

Table 2 displays normal values for the three continuous flow LVADs currently in use or available for implantation [3, 11, 18, 24]. Since the LVAD is essentially a conduit bypassing the left ventricle, it is entirely possible that the aortic valve does not routinely open. Any pulsatility that does occur is not the result of ventricular ejection through the aortic valve, per se. It is actually the result of any residual left ventricular function that with each beat provides an increase in preload to the LVAD, resulting in a transiently higher flow. The newest LVAD devices, including the HM III, routinely cycle their RPMs transiently higher and lower than their set value instead of remaining static, thus creating more pulsatility than their predecessors. This decreases the incidence of AVMs which often are responsible for GI bleeding.

Many factors can affect physiologic LVAD pump function, including hypovolemia, anesthetic agents, surgical positioning and technique. **Figure 7** is a flow diagram of changes in pump values that may indicate different physiologic states when the pump flow is increased. Patients with high pump flows and low PI values may be indicative of vasodilation, aortic valve regurgitation, or high pump speed. Of the three, vasodilation is the most likely cause related to anesthesia and should be treated with titration of vasopressors, inotropes, and intravenous fluids. If the pump flow is high and the PI is increased, then the patient may be hypervolemic or have increased myocardial contractility, such as with inotrope usage [3, 12, 24]. **Figure 8** details a flow diagram to help interpret changes when the pump flow is decreased. Low pump flow with high PI can be caused by hypertension, decreased VAD speed, or partial outflow obstruction from the outflow cannula. Of the three, hypertension is the cause most likely associated with an anesthetic. Titrating antihypertensives, administering pain medication, or increasing depth of anesthesia are ways to address these changes. If pump flow and PI are both low, this may indicate partial inflow obstruction or low

VAD	Pump speed (rpm)	Flow Liters/min	Power, Watts	Pulsitility Index
HeartMate 2	8000-10,000	4–8	4–8	4–6
HeartMate 3	5000-6000	3–6	3–7	2–4
HVAD	2400-3200	4–7	3–7	8 peak, 2 troughs

Table 2.
Normal value ranges of LVAD devices for speed (rotations per minute (rpm)), flow, and power [3, 10, 18, 24].

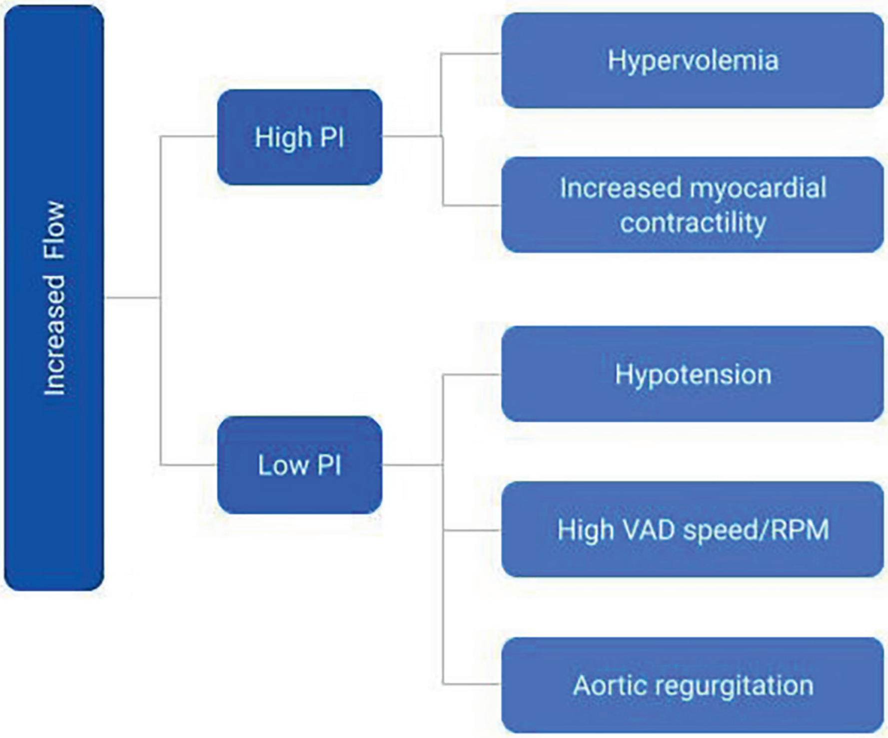


Figure 7.
Flow diagram for interpreting changes in LVAD function with increased flow [12, 24].

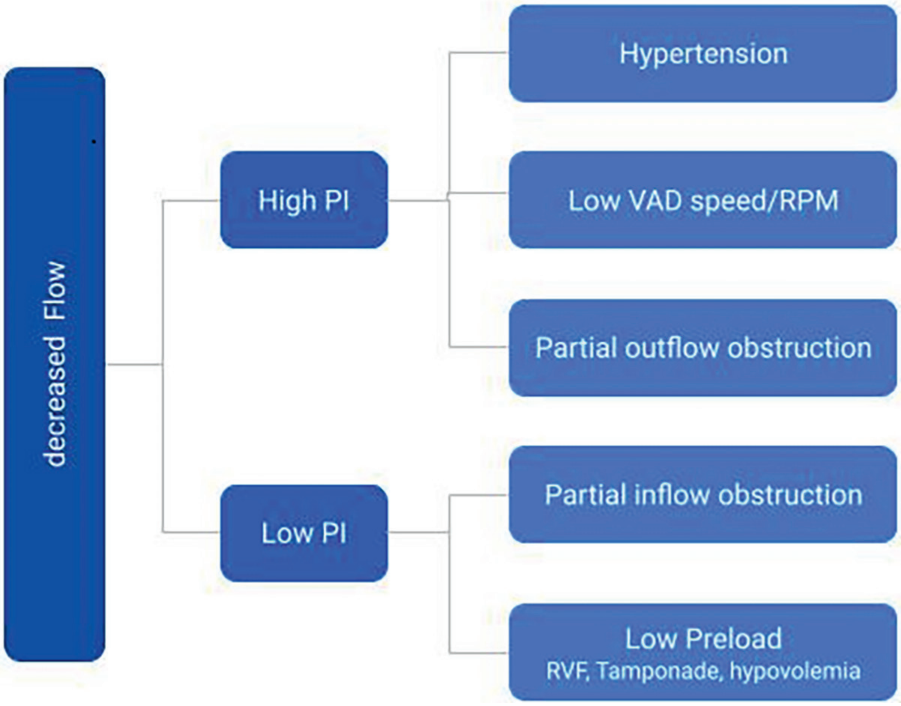


Figure 8.
Flow diagram for interpreting changes in LVAD function decreased flow [12, 24].

preload secondary to hypovolemia, right sided heart failure, or cardiac tamponade. In the setting of an anesthetic, hypovolemia is most likely the cause of this pattern and can be treated with titration of a fluid bolus [3, 12, 24].

3. Complications of LVAD device

The most common complications with continuous flow LVADs are right ventricular (RV) failure, gastrointestinal (GI) bleeding, infection, pump thrombus, stroke, and ventricular arrhythmias [3, 4, 11, 14, 17, 23].

3.1 Right ventricular failure

RV failure is noted in about 35%- 40% of LVAD patients. This may present acutely right after implantation or is a delayed phenomenon attributed to increased preload, septal shift, and less contractility [4, 6, 12, 14, 17]. 10–25% of LVAD patients will require RV support as a poorly functioning RV limits the LVAD system by way of preload [6, 14]. Supportive measures may include a variety of modalities such as lusitropic medications, diuretics, and pulmonary vasodilators [11, 12]. Patients who are refractory to medical management may require a right ventricular assist device (RVAD) or in some cases total artificial heart [4].

3.2 Gastrointestinal bleeding

GI bleeds occur in around 30% of patients with LVADs [2, 8, 16–18]. Upper GI bleeds are more common than lower GI bleeds [3, 11]. The most common source of bleeding is arterial venous malformations, the formation of which is attributed to lack of arterial pulsatility and acquired von.

Willebrand disease [1, 2, 4, 10, 11, 16, 26]. Interestingly, the HM III is thought to partially mitigate this by cycling its RPMs (revolutions per minute), thus creating a partially pulsatile state. Patients with history of gastric ulcers, colon polyps, and hx blood thinner use prior to LVAD placement are at higher risk for developing GI bleeds [4]. The requirement of anti-coagulation, typically with coumadin targeting an INR of 1.5–3, as well as aspirin, also exacerbate the bleeding risk [2, 4, 10, 16]. **Figure 9** provides a visual for how these factors contribute to GI bleeds.

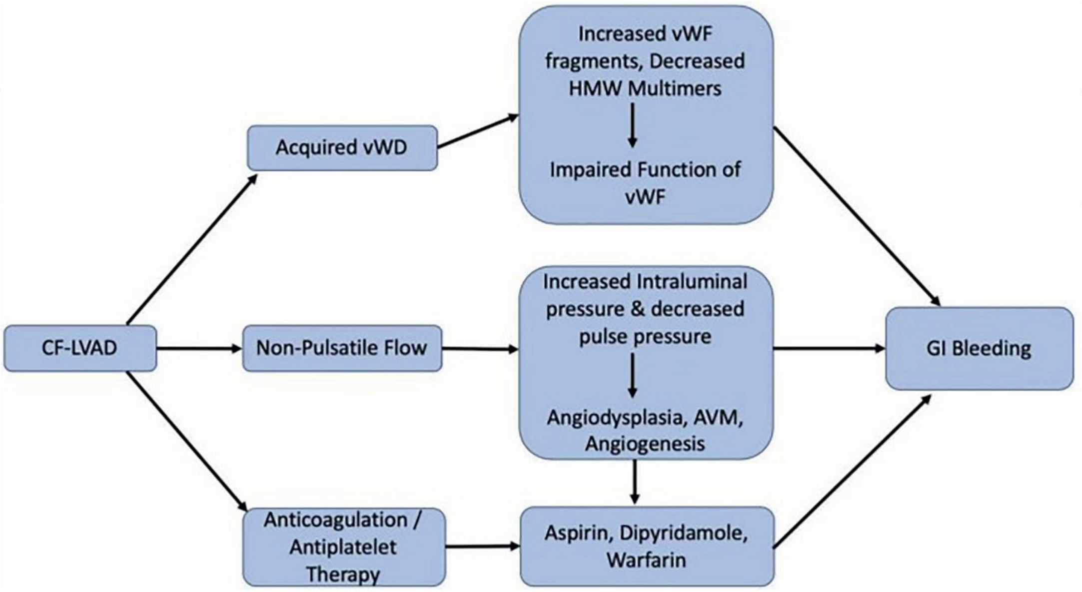


Figure 9.
Factors of continuous flow (CF) LVADs that contribute to GI bleeding [2].

Acute management of GI bleeding will likely include a combination of holding anticoagulation/antiplatelet medications, providing octreotide, and performing an endoscopy exam [2, 11, 12]. Holding anticoagulation has shown to be safe for short periods of time and should be restarted slowly with a lowered INR goal after signs of bleeding have stopped [2, 4, 15, 16]. Octreotide is a somatostatin analog that functions by decreasing gastric secretions that prevent clot formation [12]. Endoscopies require anesthesia and diagnose the source of bleeding in 1/3 of patients. In cases of severe GI bleed, reversal of anticoagulation with vitamin K or fresh frozen plasma may be used. Von Willebrand factor may also be administered [2, 12]. If the source of bleeding is not identified and bleeding continues, angiography may be attempted to identify and embolize source vessels [11]. Maintaining lower doses of anticoagulants may be used as long-term treatment and prevention of GI bleeding [12].

3.3 Infection

Infection can occur at surgical incisions or anywhere along the device system. Driveline infections are the most common, comprising 80% of LVAD associated infections [4, 12]. Any infection in the LVAD patient will be treated with hospital admission and intravenous antibiotics to prevent sepsis [12]. Driveline infections may also require surgical debridement in the operating room [12]. Explantation of the LVAD device with re-implantation is the final treatment if any components of the internal device become infected [4, 10]. Self-care education of LVAD users is key to prevention of infection. Lifelong antibiotic suppressive therapy is an alternative for those who are too high a surgical risk. Using techniques such as anchoring the driveline near the skin and using a silver dressing have shown to decrease infections in a small study [12].

3.4 Pump thrombus

Pump thrombus is a complication unique to continuous flow LVADs. HM3 has the least incidence of suspected pump thrombus; less than 3% at the two-year post implantation mark [4, 12, 17]. Thrombus can occur anywhere within the pump. A thrombus in the inflow and outflow cannula may be seen on CT scan with contrast. Visualization of thrombus anywhere else within the system can only occur with explantation [12]. An elevated pump power with decreased pulsatility index will be noted on the controller screen of the LVAD [4]. Transthoracic or transesophageal echocardiogram may show a dilated left ventricle, mitral regurgitation, and aortic valve opening with systole [12]. Clinically, a palpable pulse may be felt secondary to aortic valve opening [2, 6]. Labs will show an increased lactic dehydrogenase and decreased hemoglobin when suspecting LVAD pump thrombus [12]. In 50% of less severe cases, the patient will successfully be treated with heparin and inotropes [14]. In severe cases where pump thrombus treatment is refractory to medical management, the pump is exchanged as definitive treatment [4, 10]. Strategies such as maintaining INR 2–2.5 with warfarin, daily aspirin, and mid-range pump speeds decrease thrombus rates significantly. The PREVENT study saw a decrease in thrombus rates from 8.9 to 1.9% by implementing the following strategies: coumadin to keep INR 2–2.5, daily aspirin, and pump speeds greater than 9000 RPMs (for HM II) [3, 4, 14].

3.5 Stroke

There is an increased incidence of stroke associated with pump thrombus and mean arterial pressure (MAP) greater than 90 mmHg. For LVAD patients, a MAP greater than 90 mmHg is considered HTN [3, 4, 11]. Data shows that about 9% of patients with an LVAD have a stroke within 34 months of implantation [12]. Favored treatment for ischemic strokes in the LVAD population is endovascular thrombectomy. Intravenous thrombolysis has not yet been tested sufficiently [3, 12]. The histology of clots is different in LVAD patients and clot retrieval devices require more passes of devices are typically required to alleviate ischemic strokes in LVAD patients vs. non-LVAD patients [27]. A decrease in stroke rates by two thirds was noted with adherence to the same regimen that decreased pump thrombus. The protocol includes maintaining mid-range pump speed, anticoagulation with warfarin to keep INR values 2–2.5, and daily aspirin [3, 4]. The newer generation HM3 has less incidence of stroke compared to the HMII [3, 4, 12].

3.6 Ventricular arrhythmias

Ventricular arrhythmias occur in about 15–34% of LVAD patients, with the highest incidence in the first 30 days post implantation. An average of 34% of LVAD patients have an episode of ventricular tachycardia within 1 year of implantation [4, 6, 16]. Many have an ICD implanted prior to LVAD implantation [4, 6, 10, 14, 16]. Ventricular arrhythmias may be caused by so-called “suck down” events: when the left ventricle has a low volume and collapses on itself [4, 6, 11, 16, 28].

Treatment is to slow the VAD speed to allow increased filling of the ventricle, and support with vasopressors [4, 6]. Management of ventricular arrhythmias and suction events will be discussed further in the Intraoperative management section of this chapter.

4. Perioperative considerations for LVAD patients undergoing non-cardiac surgery

As people are living longer with LVADs, other health issues may arise that require surgical intervention and therefore the need for anesthesia [15, 20]. 15–20% of LVAD patients present for non-cardiac surgery (NCS), whether elective or urgent/emergent [7, 18]. In one study held in Europe from 2012 to 2019, within 60% of LVAD patients who had surgical interventions, 39% of procedures were unplanned and 61% were elective [18]. Over half of the patients required general anesthesia, whereas 5% of cases were performed under local [18]. A review of Medicare patients with LVADs in the United states within the same time period shows close to 75% of the non-cardiac surgery cases were unplanned and around 25% reported as elective [7]. Common procedures LVAD patients may undergo include treatment of GI bleeding, surgical debridement of skin infections, ICD generator changes, and emergent orthopedic and cystoscopy cases [7, 18]. Another report described the care of morbidly obese LVAD patients for laparoscopic sleeve gastrectomies, to improve transplant candidacy [29].

Ideally, surgical procedures involving an LVAD patient should take place at a medical center that implants LVADs. It is suggested that for any complex patients or larger surgeries that a cardiac anesthesiologist be the primary anesthetic provider. Many smaller procedures, sedation cases, and well-maintained patients may not necessitate the need for cardiac trained anesthesia providers [15, 16].

4.1 Surgical optimization during the pre admission testing and preoperative period

The typical patient living with an LVAD may be in better physiological condition when compared with patients with severe heart failure not on LVAD therapy. Studies show that around 30–70% of non-cardiac surgery events in LVAD patients are electively scheduled [7, 11, 15, 18]. Efforts should be made to have an LVAD coordinator plan and organize care for these patients [30]. Duties include communicating and coordinating needs of the planned procedure, providing patient education, including anticoagulation management, organizing availability of LVAD staff and anesthesia, and setting up goals for postoperative care [16, 20].

Most elective and many unplanned procedures may not require a cardiac trained anesthesia provider [15, 16, 18]. However, it is suggested that both a CT surgeon and a cardiac anesthesiologist are aware of the patient having a procedure and be available for consultation. For LVAD patients that present with hemodynamic instability, it is recommended that a cardiac anesthesia team be present for surgery [16].

The type of anesthesia required is case dependent. However, ideally and whenever possible, cases should be performed under local, regional, or MAC [16]. Neuraxial anesthesia is not typically thought of an ideal modality for LVAD patients due in part to their requirement of systemic anticoagulation, but most troublesome is the profound vasodilation and subsequent abatement of preload that can rapidly lead to ventricular "suck down" phenomenon if not appropriately anticipated. In normalization of practice, it has been found that an epidural may even be provided to laboring women with LVADs [14]. General anesthesia can safely be administered in a patient with an LVAD, provided once again, one accounts for the frequent shifts in hemodynamics [20]. Discussing the type of anesthesia and the expected patient experience is important [16].

LVAD patients may need to be admitted 24–72 hours prior to scheduled procedure for heparin bridging and fluid optimization [16, 18, 20]. Management of anticoagulants will depend largely on the type of surgery and anticipated blood loss. Warfarin is most commonly stopped and bridged with heparin in hospital. Aspirin may be continued as the antiplatelet therapy has proven to be beneficial perioperatively [18, 20]. Fresh frozen plasma, prothrombin complex concentrate (PCC), and vitamin K can be used in emergent situations to reverse warfarin [3, 16, 18, 20]. In small cases with little to no anticipated blood loss, patients may be instructed to stop or decrease anticoagulation doses within a few days of scheduled procedure. Studies have shown that stopping or decreasing the dose of anticoagulation does not increase risk of adverse events [4, 15, 16].

Also understand that all LVAD patients have acquired von Willebrand's disease related to blood shearing forces that flow through the pump [4, 16]. Perioperative DDAVP may be indicated depending on the type of surgery. Actual use is quite low at 0.3% [26].

Fluid status is important given the dependency of LVAD to function well with adequate preload. A pre-op echocardiogram can be performed to ensure the most complete assessment of the patient's cardiac function, including RV function and to provide opportunities for fluid optimization.

Should a patient have an ICD, it is recommended that the device be interrogated and/or reprogrammed to accommodate surgery, especially in cases where electrocautery will be used [16]. The anesthesia provider should also assess the driveline location and be familiar with individual patients' pump and baseline parameters [16]. In regards to physical examination, LVAD patients should not have a palpable pulse and heart sounds may not be elicited well upon auscultation, secondary to the hum of the.

LVAD device [3, 10, 11, 25]. In fact, a palpable pulse may indicate pump thrombus [3]. Setup and teamwork among the operating room staff members is important for an LVAD patient. The intended procedure should be reviewed, and all positioning and equipment needs considered and verified. Communication among all staff should be emphasized and the surgical team reminded of the sensitive hemodynamic state of an LVAD patient, for instance, during types of positioning or when viscera is manipulated [18]. Some positions may affect positioning of the inflow cannula and impede pump flow. Improper drive line cushioning could lead to pressure injury and tissue necrosis. For laparoscopic procedures, insufflation of abdomen should be increased in a stepwise fashion and need not exceed 10–12 mmhg until hemodynamic stability is assured [16]. Rapid escalation or high insufflation pressures can impede preload and affect flow. LVAD monitoring equipment such as Near Infrared Spectroscopy (NIRS) and Doppler supplies should be present and properly functioning.

The patient's advance directives, such as goals for CPR, should be discussed during the pre op assessment. Two lines of thought arise with the need for cardiac compressions and whether it is safe for the LVAD device. On one spectrum, compressions should never be performed as components of the LVAD may be dislodged. On the other end, no incidences of this have ever been reported in a case study [3, 16]. It is a consensus of many that early defibrillation and optimization of LVAD dynamics should be the preferred method to achieve return of spontaneous circulation (ROSC). It is not uncommon for patients who are in ventricular fibrillation to be conversant and alert.

4.2 Intraoperative phase

Upon entering the OR, it is prudent to connect the drivetrain to wall power using a red outlet (one that would still work with emergency power) [16]. Do keep extra batteries available in case of power failure [26]. The battery life of HM3 is 10–12 hours. When plugged into wall power the monitor is large enough to display all VAD parameters at once [12]. It is very important to remind the staff that the LVAD will be plugged into the wall and to not unplug it or trip over the cord.

When applying standard vital sign monitors, be aware that traditional NIBP may not be obtainable because of the lack of pulsatile blood flow [16]. The pulse oximeter may periodically work due to intermittent pulsatility. In lieu of standard pulse oximetry, NIRS could be used to monitor cerebral blood oxygen content. Cerebral oximeters work by trending venous weighted oxyhemoglobin saturation [26].

Blood pressure and mean arterial pressure (MAP) can be monitored by an arterial line or Doppler device [3, 4, 8, 11, 16, 25]. Arterial lines are the gold standard for an LVAD patient undergoing general anesthesia [16, 19]. The waveform will have a somewhat flat appearance related to low pulse pressure [3]. The use of Doppler devices for blood pressure monitoring are recommended for smaller cases that involve local anesthesia or IV sedation/MAC. It is often necessary to have a dedicated person to measure this as it is a relatively time intensive process. In many institutions, an LVAD nurse accompanies the patient and is charged with this task. The technique uses a Doppler ultrasound at the brachial artery. The cuff is placed on the upper arm and inflated until loss of pulse. The cuff is then slowly deflated and the pressure at the return of signal noted. If the patient has a palpable pulse, the Doppler pressure is associated with systolic pressure. In the absence of a palpable pulse the noted pressure is associated with the mean arterial pressure [19, 25]. The Doppler pressure has been shown to correlate with arterial lines 88% of the time [14]. MAPs should be kept around 70–80 mmHg to prevent pump malfunction while ensuring end organ perfusion [4, 8, 11, 14, 16, 19, 20, 25, 26, 31]. Slow cuff method is another effective technique, but it is not widely available [16, 19]. The slow cuff system deflates more slowly than common non-invasive blood pressure devices (**Figure 10**) [19].

4.3 Induction, maintenance, and emergence phases of anesthesia

Almost any method of induction can be chosen provided hemodynamic purtubations are anticipated. Propofol induction, for example, is not contraindicated but conservative doses are less likely to cause significant hemodynamic swings. An inhalational induction, total, or in part, is also an option.

Using midazolam and ketamine can decrease the amount of other anesthetics. Opioids can decrease sympathetic tone and should be given judiciously. This author uses a balanced technique incorporating small titrated doses of midazolam, ketamine, and propofol to achieve unconsciousness using the minimum dose required. Often, a pre induction fluid bolus is given and a phenylephrine infusion is titrated to the desired mean arterial blood pressure.

Intubation and ventilation can potentially cause changes to VAD function. Airway manipulation can cause sympathetic stimulation and hemodynamic shifts. Although some sources say placement of a double lumen tube should be avoided in favor of a bronchial blocker for thoracic procedures, both have been placed successfully [20]. Positive pressure ventilation and PEEP can affect preload, as can hypercarbia, hypoxia, and acidosis [16, 20]. Valsalva maneuvers may also impede venous return [20, 24].

Generally, the axial flow of an LVAD depends exquisitely on preload and afterload. It pumps the delivered volume and ejects it systemically. The main objectives are therefore to avoid decreased preload, maintenance of afterload, and avoid

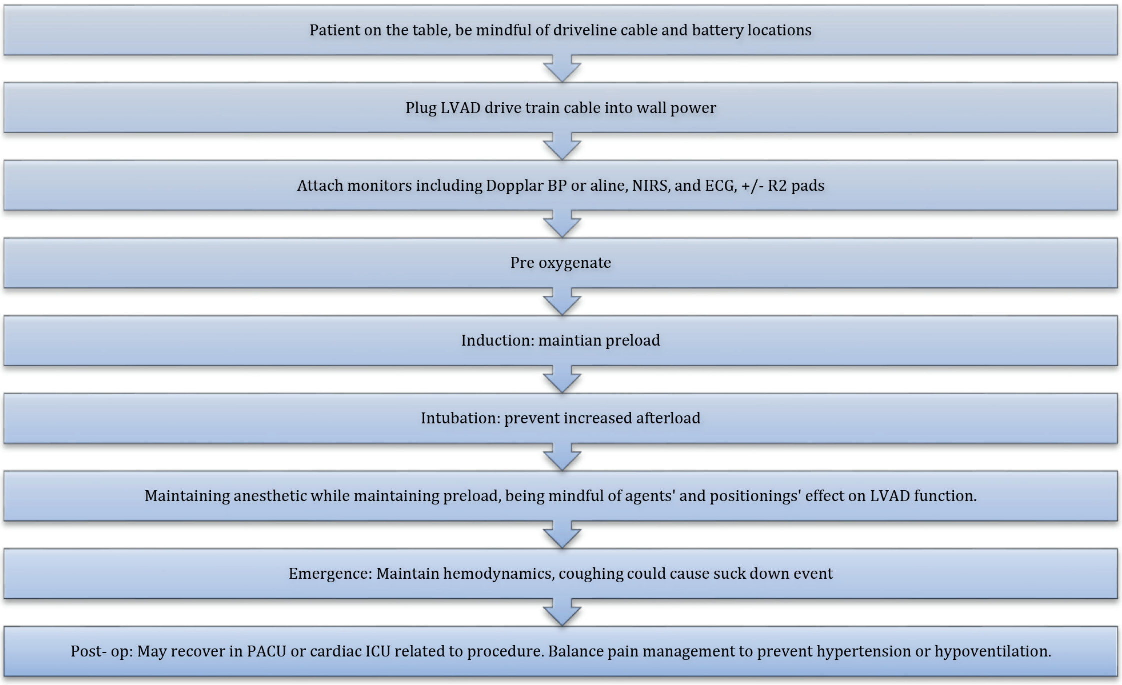


Figure 10.
A flow diagram highlighting key points to consider when managing an LVAD patient in the operating room.

inflow cannula obstruction. Although literature may state to maintain MAPs around 70-80 mmHg to ensure preload and pump function, it is important to pay particularattention to the patient's starting hemodynamics and use them as a target throughout the procedure. Studies have shown that a MAP less than 70 mmHg for greater than 20 minutes is "strongly associated with" acute kidney injury [26]. Avoiding hypovolemia cannot be stressed enough. Often, to optimize preload prior to induction, a judicious amount of fluid is given, and may include 1–2 bottles of 5% albumin [pisanksy]. One must be mindful of blood loss and insensible fluid losses. Arterial blood gas sampling and monitoring of the hematocrit is important. A more liberal transfusion target may be appropriate given ongoing losses. It is important to use irradiated and leukoreduced blood products as many of these patients will be transplant candidates.

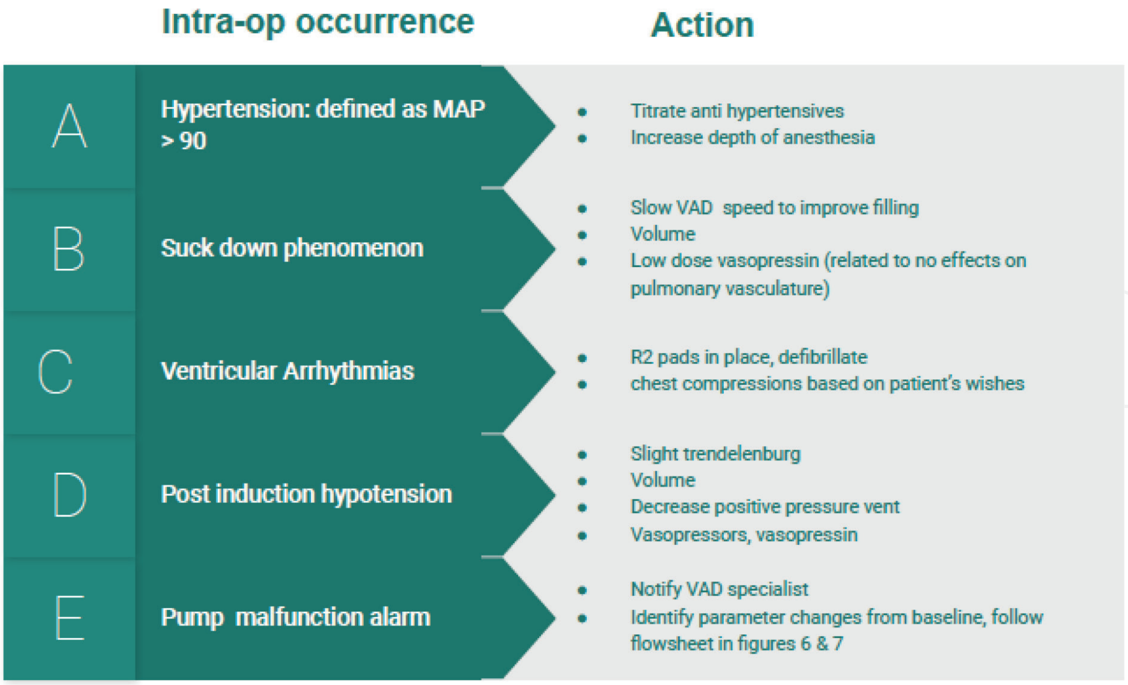
It is also important to use inotropes in addition to vasopressors to maintain right heart function in presence of hypotension, particularly if known right heart dysfunction is already present. Chronic LVAD support changes RV geometry and RV dysfunction might exist but not be clinically apparent. Causes of RV failure intro can be due to multiple factors including the inflammatory cascade, blood product administration, hypoxia, hypercarbia, and acidosis, among many. Vasopressin is cited as a pressor of choice related to its lack of effect on the pulmonary vasculature [20, 28]. Milrinone infusion may also be employed to ensure decreased stress on the right ventricle [3, 11]. Lastly, a TEE machine and appropriately trained personnel can provide additional insights if management is difficult or intraoperative adventures are encountered.

4.4 Positioning

Positioning of the patient with an LVAD requires much attention for multiple reasons. For one, patient positioning can affect a patient's preload, pressure or impingement on the drivetrain, and/or access to the patient to obtain Doppler pressures [11, 20]. Positions such as beach chair, reverse Trendelenburg, prone, and lateral will all decrease preload, and hence, impair proper functioning to the LVAD. As some surgical procedures may necessitate these undesirable positions, a discussion involving potential alternatives with the surgeon or slowly advancing the patient to the desired position as hemodynamically tolerated is imperative [16]. Prone positioning and any position that could affect the positioning of the inflow cannula should be readily reversible. Vigilant attention should also be paid to the driveline cable as it exits the patient typically from the upper abdomen and is the power source to the LVAD. When positioning care should be taken to make sure the cable is not pulled, kinked, or applying excessive pressure to the patient's skin [16].

4.5 Troubleshooting hemodynamic changes with LVAD patients under anesthesia

Figure 11 lists some acute complications that might occur perioperatively while the patient is under anesthesia and some immediate actions to take. Acute hypertension could be caused by sympathetic response to intubation or to surgical stimulation. MAPs above 90 mmHg can affect pump function and should be immediately treated by increasing anesthetic depth or titrating small increments of antihypertensives. Currently there is no standardized management of hypertension in LVAD patients [14]. If hypertension should arise and forward LVAD flow becomes impeded, anesthetic depth can be titrated [18]. Hydralazine is noted to be an appropriate and



	Intra-op occurrence	Action
A	Hypertension: defined as MAP > 90	Titrate anti hypertensives Increase depth of anesthesia
B	Suck down phenomenon	Slow VAD speed to improve filling Volume Low dose vasopressin (related to no effects on pulmonary vasculature)
C	Ventricular Arrhythmias	R2 pads in place, defibrillate chest compressions based on patient's wishes
D	Post induction hypotension	Slight trendelenburg Volume Decrease positive pressure vent Vasopressors, vasopressin
E	Pump malfunction alarm	Notify VAD specialist Identify parameter changes from baseline, follow flowsheet in figures 6 & 7

Figure 11.
Intra operative troubleshooting of an LVAD patient under general anesthesia.

effective choice to treat HTN in LVAD patients. Beta blockers are cautioned because of their negative inotropic effect [4, 11].

Suck down phenomenon can occur with decreased left ventricular filling. The VAD will suction onto the septal wall with low volumes [3, 4, 16]. Suction event may be recognized by speed, flow, and power values all being decreased [3] or low left ventricular volumes on the transesophageal echocardiogram [3, 16]. Treatment for suction events is to slow the VAD speed to allow increased filling of the ventricle, support with vasopressin, and increase preload via fluid bolus [4, 16]. Suction events may trigger ventricular tachycardia.

If an LVAD patient presents with ventricular tachycardia (VT), one should consider placing R2 pads prior to induction. R2 pad placement is the same as for non-LVAD patients and shocking with R2 pads does not disrupt LVAD function. Suction events are the most common cause of VT in LVAD supported patients [4, 16]. It is possible that some patients will be hemodynamically stable during episodes of VT as LVAD will continue to function. Additional treatment for VT includes 300 mg IV boluses of amiodarone [11]. A hemodynamically unstable patient may require advanced cardiac life support measures [3].

Post induction hypotension is best prevented by slow thoughtful induction, as described earlier. In the event of hypotension placing the patient in slight Trendelenburg and decreasing positive pressure ventilation, for positive pressure ventilation can put strain on the RV and decrease preload [16]. Small fluid bolus and titration of vasopressors should be used to support blood pressure and maintain preload.

Pump malfunction alarms may indicate the pump is not functioning, power is disrupted, or flow rate has changed. Consult the LVAD specialist. Meanwhile check that all connections are intact, from the wall/battery to the control panel to the driveline. Check the driveline for connections, kinks, or damage. If an alarm is sounding because of low or high flow rates use the diagrams in **Figures** 7 and **8** to recognize a pattern of changes and identify a potential cause.

4.6 Post-operative

Patients can be recovered in PACU but may be recovered in the ICU [11, 16, 20]. The AICD should be interrogated as soon as possible in the immediate postoperative period. It is paramount to take steps to prevent increased preload by way of hypoventilation and hypertension related to pain [16]. Opioid sparing techniques are suggested for pain control to prevent hypoventilation and sequela post operatively [16, 20]. Again, the focus is to maintain pump flow and pre-operative physiology [18]. Post operative readmissions to the hospital are common as patients with LVADs undergoing non cardiac surgery have high rates of bleeding and acute kidney injury. The need for transfusion may be delayed several hours post surgery [7].

4.7 Case studies for LVAD patients and non cardiac surgery

A. A 24-year-old parturient with a heartware HVAD requests an epidural placement for induction of labor and subsequent cesarean section. The patient stopped lovenox 24 hours prior to hospital admission.
Once admitted, invasive BP monitoring, central line and Swan Ganz placement were performed.
Subsequently, an epidural catheter was placed prior to induction of labor.
The patient was permitted a patient-controlled epidural device (PCEA) that dispensed a bupivacaine and fentanyl solution. With the decision to perform cesarean section the epidural was dosed with 2% lidocaine in small increments.
Vasopressin and norepinephrine drips were used throughout to maintain MAPs greater than 70 mmHg.
The patient received transverse abdominal plane blocks post operatively for pain control. No complications with the LVAD were noted [28].

B. A 72-year-old female with a HM3 underwent a total knee replacement procedure. Pre operative surgical optimization assessments by orthopedics, cardiology, and anesthesia were performed. Warfarin was discontinued 6 days prior to surgery and was admitted 2 days prior to surgery for heparin bridging.
The patient received a 500 mL intravenous fluid bolus and an abductor canal block for postop pain control in the holding suite. Upon entering the operating room, American society of Anesthesiologists (ASA) standard monitors were placed, as well as an arterial line. The patient then received a spinal of 12 mg of hyperbaric bupivacaine. Her pressures were supported with epinephrine and phenylephrine drips. Sedation was maintained with propofol. The case reported minimal blood loss and no anesthetic complications. The patient was taken to cardiac ICU for recovery on no drips [32].

C. A 68-year-old with a HM3 had a total thyroidectomy procedure under monitored anesthesia care. The patient entered the operating room and after connecting the LVAD to wall power an arterial line was started in addition to ASA standard monitors. The patient received 2 mg of versed and an alfentanil infusion and the surgical team administered a superficial cervical block. This technique was chosen related to minimal potential of shifts in hemodynamics compared to general endotracheal anesthesia. No pressors were required throughout the case and the patient did not have any pain or discomfort throughout. The patient was recovered in the ICU post operatively [31].

D. A 66-year old male with a HMII with prostate cancer present for robotic laparoscopic prostatectomy.

   Prior to the surgical date the patient had a ramp transthoracic echocardiogram to optimize his LVAD function. The patient was admitted the night prior to his procedure for LVAD and ICD interrogation. His warfarin was held the night before surgery. An INR of 3.2 was noted prior to surgical start time, prothrombin complex concentrate was administered; prior to incision INR was 1.4. An arterial line and right internal jugular central venous catheter were placed prior to induction. A rapid sequence induction was performed with 1.5 mcg/kg fentanyl, 1 mg/kg of propofol, and 1.2 mg/kg rocuronium. Anesthesia was maintained with sevoflurane. Post induction the patient became hypotensive and was treated with a 250 mL bolus of albumin. With the start of pneumoperitoneum the patient became hypertensive. This was managed with a bolus of propofol and initiation of dobutamine and nicardipine drips to maintain preload and afterload. As the patient was transitioned into trendelenberg a rise in central venous pressure was noted, but LVAD parameters maintained within the patient's normal range and no action was taken at that time. Approximately 40 minutes after being positioned in trendelenberg the CVP had increased significantly and the PI was decreasing. A cardiac anesthesiologist was consulted for TEE, which showed septal bowing and right ventricular dysfunction. Inhaled epoprostenol was administered as treatment, CVP and PI returned to baseline. During desufflation of the pneumoperitoneum the patient became hypotensive requiring a second albumin bolus and short term epinephrine and phenylephrine drips while the surgical procedure finished. All drips were weaned off; the patient was extubated and taken to the cardiac intermediate care unit to recover. No postoperative complications were noted and the patient was discharged to home 2 days postoperatively [33].

5. Discussion

It is estimated that by 2030, the number of Americans with heart failure will increase to over 8 million [1, 5]. The number of heart transplants per year has been between 2 and 5 thousand and will continue to be limited by the number of donors. Donor availability may soon increase related to hepatitis C no longer disqualifying donation, however, even this breakthrough is not anticipated to fulfill the need for heart transplant patients [5, 23]. Therefore, there is likely to be an increased need for alternative definitive treatment for advanced heart failure, such as LVADs.

There is a campaign to recognize advanced heart failure sooner and to implant an LVAD before patients develop significant end organ disease [4, 23]. The strategy is to standardize criteria across all LVAD centers. One suggestion is to use AI algorithms to evaluate electronic medical records for increased frequency of visits and other criteria indicative of advancing heart failure [23].

Current targets for advancement include development of a completely implantable device and standardization of minimally invasive surgery (MIS) technique for LVAD implantation [23]. MIS technique requires two thoracotomy incisions; one 2 centimeters at the right intercostal space and a second larger (8-10 cm) at the 5-6th intercostal space. These incisions expose the ascending aortic arch and apex of the left ventricle, respectively [34]. In addition to preserving the sternum MIS affords less blood loss, less need for transfusion, and less intrathoracic trauma [22, 23, 34]. One retrospective study also noted patients who underwent MIS placement had significantly shorter time to extubation, less incidence of RV failure, shorter ICU time, and fewer readmissions [34].

The fully implantable device has the potential to significantly decrease incidence of LVAD infections [4]. Three fully implantable devices have been developed and begun trials. The Abicor total artificial heart and the Arrow Lion heart did not achieve long term survival but showed significantly lower infection rates than devices with extracorporeal components [23].

A 2019 paper reported two patients received Jarvik 2000 LVADs designed without any percutaneous parts. The modified devices were produced to trial with a coplanar power system. The coplanar energy transfer system (CETS) wirelessly transfers energy from an external energy source to the internal battery/controller component, which directly powers the LVAD. When fully charged the internal battery system provides up to 6 hours of power. The system also includes a wristwatch monitor to display parameters. Patient A was noted to have an intraoperative neurological complication but pump implantation was successful. This patient developed a pump thrombus, in conjunction with his complicated postoperative course the device was turned off and the patient maintained on inotropes.

Patient B had a successful implantation and was discharged 30 post implantation. No infections or issues with the CETS were noted during either patients' hospital stays. No long term follow up information was available at the time of this publication [35].

A fourth device, the Calon Leviticus fiVAD is under development in Europe. In a preclinical study the fully implantable device completed a promising 6 day trial in sheep. It was paired with the same coplanar power system as the Jarvik trial mentioned above [36].

The technology of the LVAD has improved drastically over the last two to three decades increasing the longevity of these devices and those who benefit from them. Currently 1 year survival is 90% and mid 80% for heart transplant and LVAD respectively [4]. As these devices continue to improve and the common complications are better understood and managed, LVADs have the potential to become the preferred treatment for severe heart failure [4].

6. Conclusion

The LVAD was originally intended to support the patient with heart failure until a donor heart became available. Today they are implanted for a variety of therapeutic intentions and have extended the life span of critical heart failure patients. LVAD implantation changes the physiology of the heart and comes with some related complications. Caring for this patient population takes extra planning, optimization, and coordination. Adjustments to pre op assessment, monitoring devices, and peri operative management will be needed. However, by understanding these devices and related physiological changes, a non-cardiac anesthesia provider can safely administer a variety of anesthetics to a patient with an LVAD presenting for non-cardiac surgery.

Conflict of interest

The authors declare no conflict of interest.

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