



## Left Ventricular Assist Device Management in the ICU

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**Objectives:** To review left ventricular assist device physiology, initial postoperative management, common complications, trouble shooting and management of hypotension, and other common ICU problems.

**Data Source:** Narrative review of relevant medical literature.

**Data Synthesis:** Left ventricular assist devices prolong the lives of patients with end-stage heart failure, and their use is increasing. Continuous-flow left ventricular assist devices have replaced first-generation pulsatile devices. These patients present unique management concerns. In the immediate postimplant period, care must be taken to support the unassisted right ventricle. Invasive monitors for blood pressure, pulmonary artery catheterization, and echocardiography are essential to optimize left ventricular assist device settings and cardiac performance. Anticoagulation is necessary to prevent devastating thrombotic and embolic complications, but bleeding is a major source of morbidity due to inherent bleeding diatheses and prescribed anticoagulants. Infection of the device can be life threatening, and all infections must be aggressively treated to avoid seeding the device. Patients are at risk of ventricular arrhythmias because of their underlying disease, as well as the placement and position of the inflow cannula. Aortic valve stenosis and insufficiency develop over time and can lead to thrombosis or heart failure. Cardiopulmonary resuscitation with chest compressions must be performed with care or not at all due to risk of dislodging the device.

**Conclusion:** Intensivists are increasingly likely to encounter patients requiring mechanical circulatory support with left ventricular assist devices at various points in the trajectory of their disease, from the immediate postimplant period to subsequent admissions for complications, and at end of life. A basic understanding

of left ventricular assist device physiology is essential to the safe and effective care of these patients. (*Crit Care Med* 2014; 42:158–168)

**Key Words:** continuous-flow left ventricular assist device; HeartMate II; HeartWare; left ventricular assist device

Heart failure develops in one in five Americans over age 40; nearly 50% die 5 years after the diagnosis is made (1, 2). In whom end-stage or advanced heart failure develops, nearly 70% will die or be hospitalized in 1 year (3).

Cardiac transplantation is the definitive long-term treatment for end-stage heart failure (4). However, there are insufficient heart allografts available. In 1994, the U.S. Food and Drug Administration (FDA) approved the first-generation pulsatile left ventricular assist devices (LVAD) as a bridge to transplant (BTT) for patients on waiting list and then later for patients not eligible for heart transplantation as destination therapy (DT). Newer generation continuous-flow LVADs designed for less mechanical wear doubled 2-year survival compared with pulsatile LVADs and improved quality of life for BTT and DT patients (5–7). Currently, 2-year survival at greater than 80% for patients with new generation continuous-flow LVADs rivals heart transplantation (8, 9).

With over 4,000 mechanical circulatory support devices implanted since 2006 and more readmissions after index hospitalization, intensivists will surely encounter these patients in the future (9, 10). Management of these patients requires understanding of the principles, indications, and limitations of this unique technology. This review highlights the physiology of patients with continuous-flow devices, the perioperative course, and management of common complications relevant to the critical care physician. Pulsatile intracorporeal LVADs are rarely implanted and will not be discussed.

### DEVICES

The durable implantable continuous-flow LVADs currently in use share the same basic design and components. Each requires an inlet cannula positioned into the left ventricular (LV) apex, a pump, an outflow graft into the ascending aorta (descending aorta in the Jarvik 2000), and a controller with battery pack.

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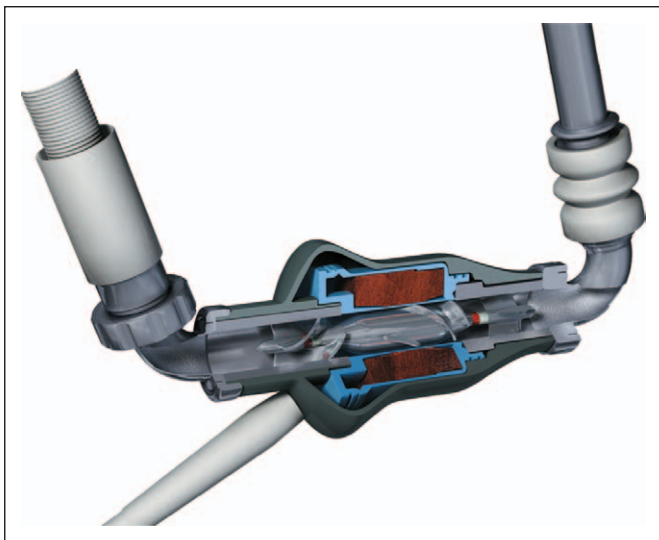
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**Figure 1.** Cutaway image of the HeartMate II left ventricular assist device showing the impeller. Reproduced with the permission from Thoratec Corporation. Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

HeartMate II (HMII) produced by Thoratec is the most used implantable device worldwide and the only device that is currently approved by the FDA for both BTT and DT (11, 12). In this axial flow device, a titanium corkscrew impeller with an internal magnet is supported by bearings at either end (**Fig. 1**). A magnetic field rotates the impeller between 6,000 and 15,000 revolutions/min (RPM) providing blood flow along the axial plane continuously during the cardiac cycle. At 100 mm Hg pressure, it provides up to 10 L/min flow (12–15). Other axial flow devices include Jarvik 2000, and the previously used Incor and MicroMed DeBakey (16–19).

Of the centrifugal flow implantable pumps, HeartWare (HW) is the only FDA-approved device for BTT. Its wide-blade impeller with magnetic elements levitates friction free by passive magnetic force and hydrodynamic thrust (**Fig. 2**). An electromagnetic field spins the impeller from 1,800 to 4,000 RPM and provides up to 10 L/min blood flow. In contrast to HMII, it is small enough to implant in the pericardial space (**Fig. 3**). The driveline is also smaller and more flexible (20–22).

The system controller is the user interface for the LVAD. It controls the LVAD motor power and speed, performs diagnostic assessment, records and stores data, indicates battery fuel levels, and alerts the user to advisory and hazard alarms. The system controller connects the two batteries to the driveline and clips to the patient's belt (**Figs. 3** and **4**). A larger system monitor is used in the inpatient setting to display power, flow, speed, and pulsatility data for clinicians. Two batteries power the device for approximately 3 hours under normal conditions. Patients can be attached to the power base unit rather than batteries for extended periods of time.

As with any percutaneous device, the driveline is at risk of infection and must be handled with sterile technique. Patients and caregivers are trained in maintenance of the driveline and perform dressing changes with sterile supplies.



**Figure 2.** The HeartWare ventricular assist device opened to show the wide-blade impeller. Reproduced with the permission from HeartWare. Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

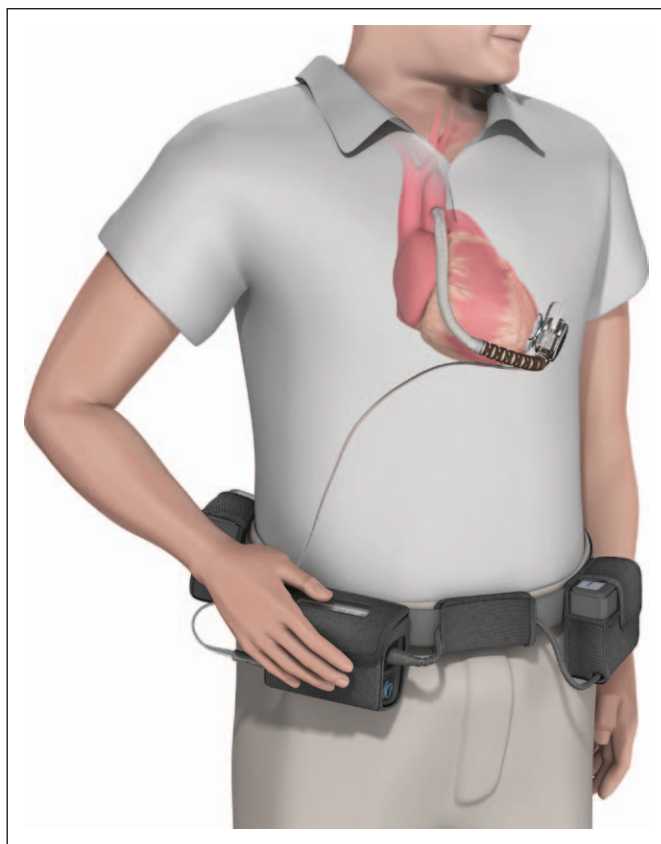
## SURGICAL AND DEVICE IMPLANTATION

Most implantations are performed using a similar technique. The heart is accessed by median sternotomy, although thoracotomy and subcostal approaches are also described (23, 24). Patients with prior sternotomy experience longer cardiopulmonary bypass time and more bleeding (25). Dense adhesions can make dissection more difficult and dangerous for future reentry for device exchange or transplantation (26, 27). A pump pocket for HMII is created in the preperitoneal space of the upper abdomen (**Fig. 4**). The inflow is inserted into the apex of the LV, directed toward the mitral valve. Outflow graft is sutured to the ascending aorta. The drivelines in both HMII and HW are tunneled subcutaneously in the abdomen. Once the system is vented of air, the LVAD is initiated. Optimal pump speed is determined under transesophageal echocardiographic guidance, by septal position, right ventricular (RV) size, and function.

## DEVICE PHYSIOLOGY

Pump speed (as RPM) is the only variable programmed by the operator. The other variables, power, flow, and pulsatility, displayed by the machine depend on the patient's underlying physiology. Pump power is a direct measurement of watts required by the device to pump blood at the set RPM. Pump power correlates well with blood flow, but there are exceptions (20, 28, 29) (**Table 1**).

Blood flow displayed on the LVAD monitor is calculated based on measured pump power and set pump speed. For

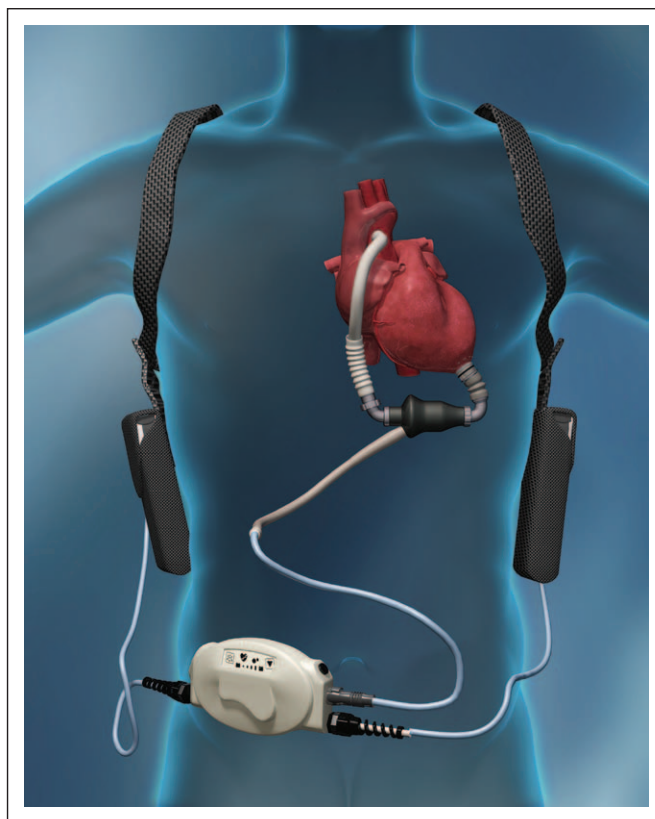


**Figure 3.** The HeartWare device in situ. The centrifugal flow device is small enough to implant in the pericardial space. Reproduced with the permission from HeartWare. Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

HW, blood viscosity has a significant impact on flow, and thus, hematocrit is also integrated into its algorithm (20, 21). Calculated flows have been shown to be inaccurate with the HMII at pump speeds less than or equal to 8,000 RPM (12).

The actual blood flow through the pump is dependent on the pump speed and the pressure differential, also known as head pressure, across the pump. For a fixed speed, the difference in mean aortic pressure (i.e., afterload), and LV chamber pressure (i.e., preload), correlates inversely with blood flow (**Fig. 5**) (13, 20, 28). The greatest determinant of the pump differential pressure is LV intracavitary pressure (22). Increased vascular tone (i.e., afterload) can also decrease pump flow (30, 31). In states of increased cardiac activity such as exercise, pump flow increases even at fixed speed due to decreased systemic vascular resistance and decreased pump differential pressure during systole (32, 33). The calculated blood flow may underestimate total cardiac output as it does not account for blood ejected by the native LV across the aortic valve (34, 35).

On physical examination, patients with continuous-flow devices may not have palpable pulses. However, contraction of the native ventricle leads to regular fluctuations in arterial blood flow and pressure (31, 36, 37). During LV systole, the pressure differential decreases and blood flow across the



**Figure 4.** The HeartMate II device in situ. Inflow cannula is inserted into the apex of the left ventricle, the outflow graft suture to the ascending aorta. Reproduced with the permission from Thoratec Corporation. Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

pump increases resulting in oscillations in arterial blood flow even as the aortic valve remains closed. The cyclical flow can produce pulse pressures in the range 5–25 mm Hg (38). A myopathic LV still follows the Frank-Starling principle and increases contractility in response to increased preload. The pulsatility index displayed on HMII models is the mathematical difference in maximal and minimal flows divided by the average flow per cardiac cycle (15). Normal ranges are 3.5–5.5. It should be noted that this calculated value is not reliable (12). In HW models, the pulsatility is not mathematically calculated, but flow and power pulsatility can be visualized graphically on the display. Practically, flow pulsatility represents the equilibrium between native cardiac function and LV preload with ventricular unloading by the LVAD (15, 19, 28). During a change in clinical status, it can aid in diagnosis. Increased pump speed will decrease pulsatility index. For a fixed speed, a drop in pulsatility index suggests either decreased LV preload or contractility.

## INITIAL POSTOPERATIVE MANAGEMENT AND HEMODYNAMICS

Because pulse usually cannot be palpated, measurement of blood pressure is often difficult. Thus, in the immediate

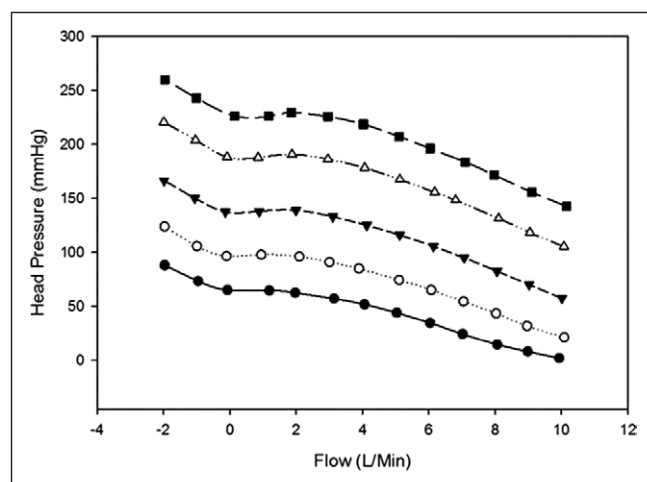


**TABLE 1. Troubleshooting Abnormal Device Conditions**

Device Condition	Causes	Intervention
High flows	Vasodilation	Reduce or hold vasodilators
	Sepsis	Add pressors
		Look for underlying source of sepsis and treat appropriately
Low flows	Hypovolemia	Bolus fluids
	Bleeding	Transfuse and address source of bleeding
	Arrhythmias	Treat arrhythmias
High powers	Pump thrombosis	Add additional antiplatelet and anticoagulants
		Consider thrombolysis (high risk of intracerebral hemorrhage)
		Consider device exchange
High PI	Recovery of left ventricular function	Look for evidence of recovery
	Percutaneous lead damage	Assess ventricular assist device components as appropriate
Low PI	Hypovolemia	Bolus fluids
	Very poor native ventricular function	Add inotropic support
	Excessive pump speed	Adjust pump speed
Suction event: collapse of ventricular cavity around device inflow; usually due to underfilled ventricle; maybe associated with low flows, low PI, or alarms	Hypovolemia	Bolus fluids
	Excessive unloading of ventricle by device	Lower pump speed
	Arrhythmias	Treat arrhythmias

PI = pulsatility index.

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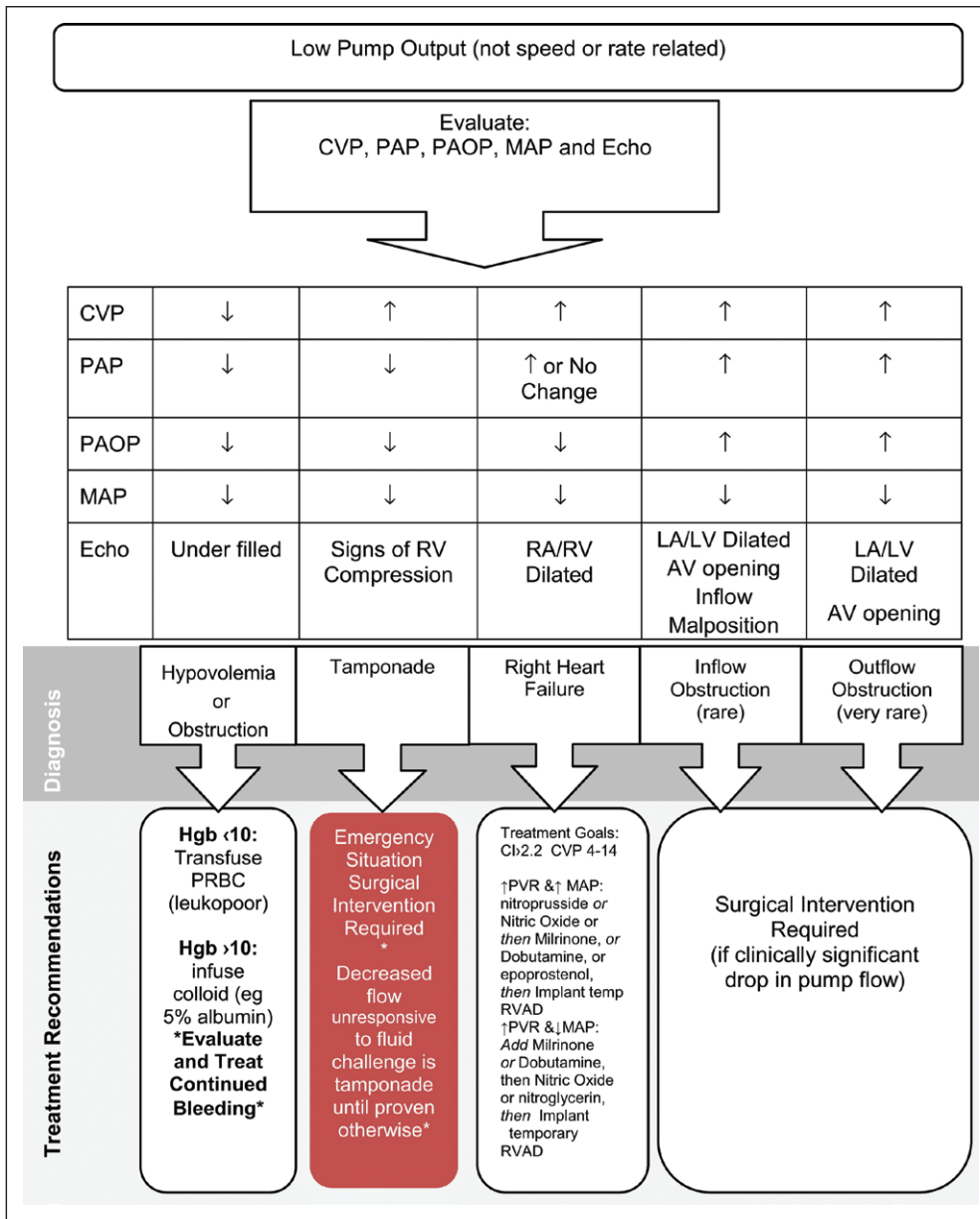


**Figure 5.** Pressure-flow curve for HeartWare pump at a constant blood viscosity. Increase in differential pressure, that is, head pressure leads to a reduction in flow for a given pump speed. This curve is used to estimate blood flow. *Closed circle* = 1,800 repetitions per min (RPM), *open circle* = 2,400 RPM, *closed inverted triangle* = 3,000 RPM, *open triangle* = 3,600 RPM, *closed square* = 4,000 RPM. Reproduced with permission from Larose et al (20). Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

postoperative period, blood pressure is best measured by invasive arterial catheter. Automated cuff or manual auscultation does not yield a measurable value reliably, and values obtained usually underestimate mean arterial pressure (MAP) and systolic pressures (35, 39, 40). Occlusion pressures obtained by Doppler at the brachial artery correlate well with invasive arterial measurement. This measurement reflects MAP but not systolic pressure (39). In general, MAP is maintained at approximately 70–90 mm Hg (19). Assuming there is sufficient intracavitary volume, up titration of pump speed increases MAP and diastolic pressure without change in systolic pressure (40, 41). Excessive blood pressure can lead to neurologic events, bleeding, and reduced flow of LVAD.

Pulse oximetry can be unreliable in setting of little or no pulse. Low values require confirmation by arterial blood gas analysis (14).

Invasive monitoring with a pulmonary artery catheter is helpful to diagnose shock states and device function (13, 42). Echocardiography provides other valuable information to determine preload, ventricular function, and device position and performance (43) (**Fig. 6**). Septal positioning and chamber size provide assessment of cardiac function and guide pump speed adjustments. Assessment of aortic valve opening



**Figure 6.** Algorithm for diagnosis and management of low pump output based on pulmonary artery catheter and echocardiographic findings. CVP = central venous pressure, PAP = pulmonary artery pressure, PAOP = pulmonary artery occlusion pressure, MAP = mean arterial pressure, RV = right ventricular, RA = right atrium, LA = left atrium, LV = left ventricular, AV = arteriovenous, PRBC = packed RBC, PVR = pulmonary vascular resistance, RVAD = right ventricular assist device. Reproduced with permission from Feldman et al (51). Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

is important to appreciate the contribution of native cardiac function to overall output and valvular function (44).

In the immediate postoperative period, hemodynamic management is based on determination of MAP, cardiac index, and ventricular function. A combination of inotropes, vasodilators, and vasopressors are used in addition to augmentation of intravascular volume and speed adjustments. See Table 1 for treatment recommendations in the early postoperative period.

The approach to hypotension is described in **Figure 7**. Vasodilation leads to an increase in flows (assuming adequate

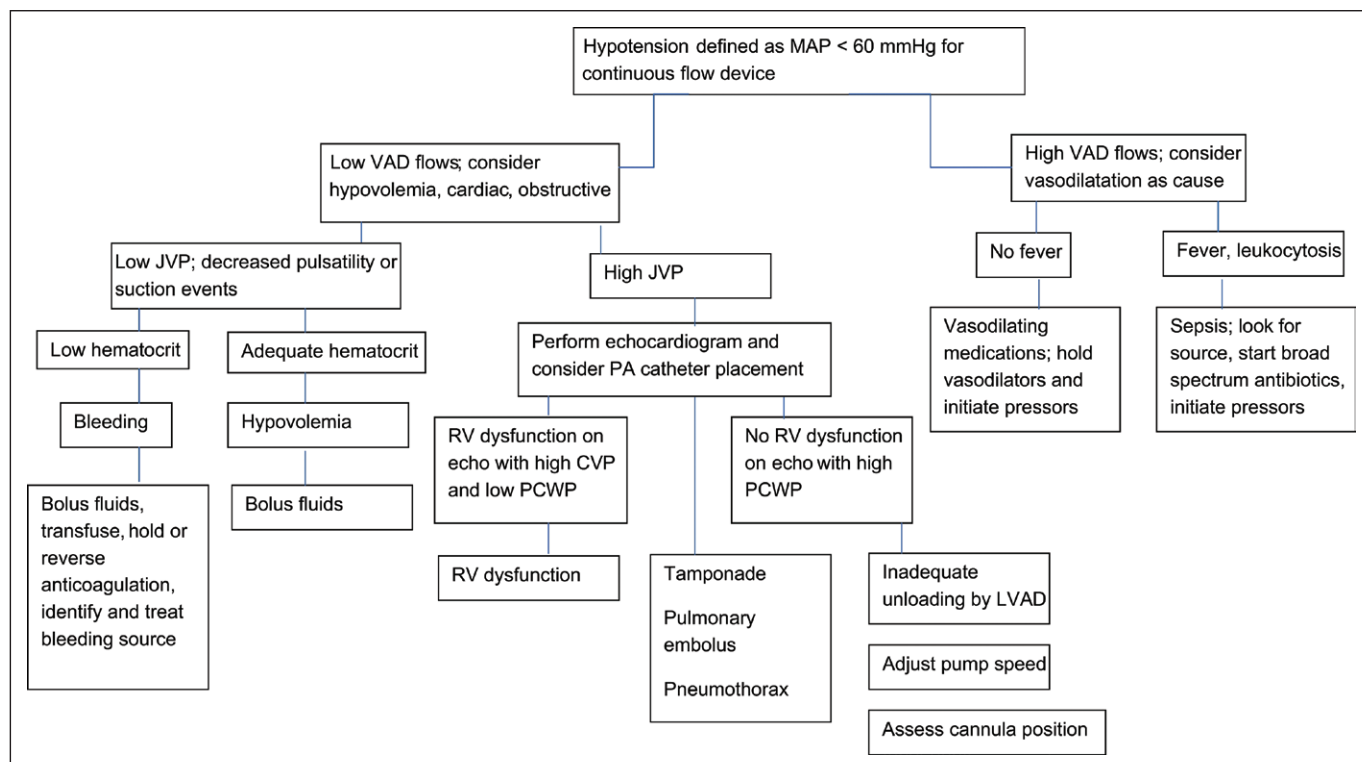
preload) and can be an early sign of sepsis. Hypotension with low LVAD flows requires assessment of cardiac function and filling pressures, as this can be a sign of hypovolemia, RV failure, arrhythmias, or other causes, such as tamponade or device-related complications.

## RV FAILURE AND PULMONARY HYPERTENSION

After implantation, RV geometry changes as the septum shifts to the left with LV unloading, causing an increase in RV compliance but a decrease in contractility. Venous return is increased due to improved cardiac output from the LVAD, but right ventricular afterload remains high due to pulmonary hypertension from chronic heart failure. Although difficult to predict, acute RV failure develops in some patients after LVAD implantation (45–47). This is characterized by elevated central venous pressures, elevated pulmonary vascular resistance, and pulmonary artery pressures with reduced LVAD flows and cardiac output (13). In very severe RV failure, pulmonary artery pressures may be low or normal. In as little as 1 month after implantation, right- and left-sided filling pressures decrease, and the right ventricular stroke work index decreases whereas cardiac output increases (11, 47). The improvement in pulmonary

hemodynamics is sustained throughout the duration of LVAD support and persists after heart transplant as well (11, 48).

Milrinone reduces pulmonary artery pressures and pulmonary vascular resistance (PVR) without inducing excessive hypotension or arrhythmias (15, 49) (Fig. 6). Selective pulmonary vasodilators, such as inhaled nitric oxide or epoprostenol, can also be used. Because RV contractility is impaired by systemic hypotension (50), MAP should be maintained between 70 and 90 mm Hg (51, 52). Phosphodiesterase 5a inhibitors such as sildenafil are used to decrease pulmonary vascular resistance (53). Those failing drug therapy should be considered for



**Figure 7.** Diagnosis and management of hypotension. MAP = mean arterial pressure, VAD = ventricular assist device, JVP = jugular venous pressure, PA = pulmonary artery, RV = right ventricular, CVP = central venous pressure, PCWP = pulmonary artery occlusion pressure, LVAD = left ventricular assist device. Reproduced with permission from Feldman et al (51). Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

temporary RV assist device placement, venoarterial extracorporeal membrane oxygenation (54), or total artificial heart. Some centers have implanted the HW ventricular assist device (VAD) in both the RV and LV for biventricular support (55).

## RESPIRATORY FAILURE AND MECHANICAL VENTILATION

Pulmonary vasoconstriction in regions of underventilated alveoli may elevate PVR and worsen RV dysfunction (56). The impact of mechanical ventilation on LVAD performance or cardiac output has not been studied in vivo, and optimal ventilator settings for LVAD patients are uncertain. Computer models of continuous-flow pumps have demonstrated a decrease in LV efficiency and an increase in RV efficiency with increasing positive intrathoracic pressure. This offsets the increase in RV stroke work created by the continuous-flow pump (57). Therefore, patients may experience worsening of RV failure after extubation. The consequence of airway pressure and the interplay of ventilation and perfusion to RV performance should be considered when managing LVAD patients with respiratory failure.

## ADVERSE EVENTS

Patients with LVADs are frequently readmitted with complications (58), most frequently in the first 6 months. Complications include bleeding, heart failure, neurologic events, arrhythmia, infection, and thrombosis or hemolysis (7, 58, 59).

## HEMATOLOGIC CONCERNS

Management of LVAD patients requires balancing the risks of thrombosis and hemorrhage. Both procoagulant and anticoagulant pathways are activated in patients on LVAD support (60). Contact activation of platelets may not be as great as once thought, and in fact, platelet activity may be significantly impaired, independent of aspirin use (61–63). Large von Willebrand factor monomers are absent, characteristic of acquired type 2 von Willebrand syndrome due to shear stress from the rotor, but return to normal after device explantation (61).

Anticoagulation protocols vary by institution, device (62), and individual patient. Anticoagulation with both antiplatelet agent and warfarin is the norm. However, recent data suggest that hemorrhagic complications far outweigh thrombotic complications (64–66). Furthermore, anticoagulation in the early postoperative period may not be necessary (65). Warfarin is begun after drains are removed, usually on the third postoperative day. The target international normalized ratio (INR) in outpatients ranges from 1.5 to 2.5 (64, 67). Aspirin is often given in doses from 50 to 325 mg. Some institutions dose patients based on tests of platelet function (evidence of acquired von Willebrand syndrome) (61), clinical condition (63), or specific device protocols.

Major thrombotic events in LVAD patients include pump thrombosis and arterial thromboembolism. Thrombi form on the impeller or areas of low flow, such as the aortic valve, atrial appendage, or a dilated LV. Ischemic strokes occur in about

8–10% of LVAD patients (7, 59). Active infection increases risk of stroke, because of increased procoagulant pathway activity (68, 69). Hemolysis is present in about 4% of patients with pulsatile or continuous-flow LVAD (7, 59).

Signs of pump thrombosis include hemolysis, thromboembolism, heart failure, and end-organ hypoperfusion with elevated power and flow readings on LVAD (71) (Fig. 6; Table 1). Pump thrombosis has been managed with thrombolysis (72), heparin (67), or lepirudin (61). Patients failing medical therapy or requiring emergent treatment are offered device exchange (71, 73) or urgent transplantation (61).

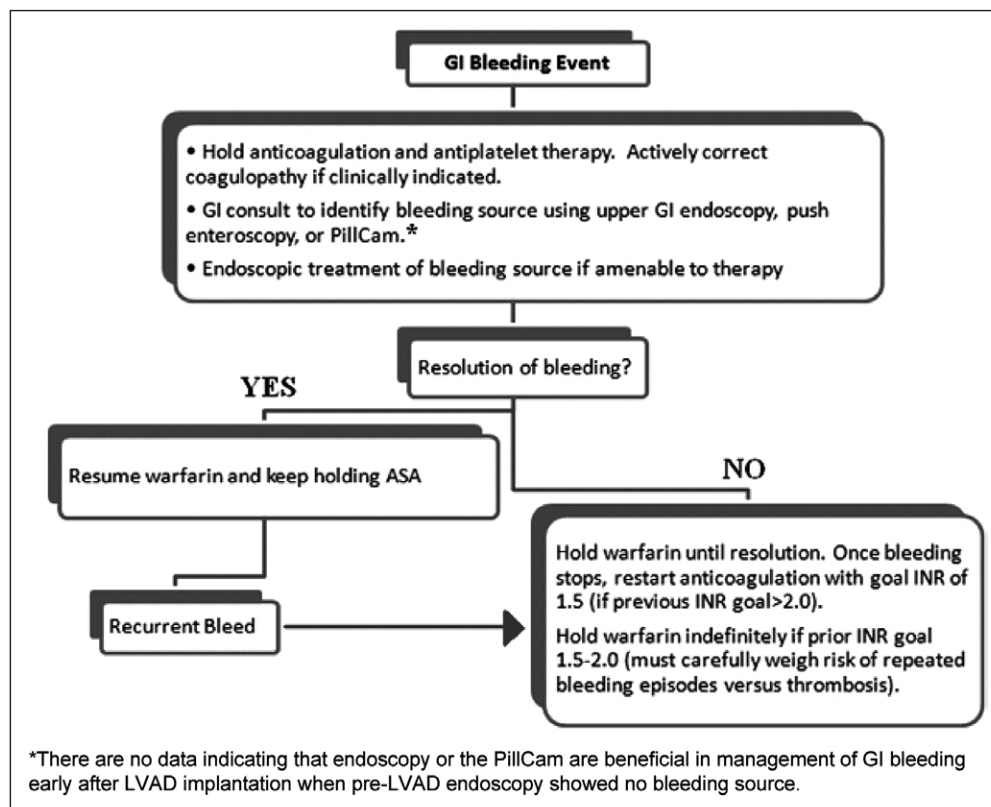
Bleeding is the most frequent adverse event (74). Early bleeding requiring surgery is seen in 26% of patients (70). The most common sites of early bleeding are mediastinal, followed by thoracic pleural space, lower gastrointestinal (GI) tract, chest wall, and upper GI tract (75). Patients who are in consideration for transplantation should receive only leukoreduced, irradiated blood products.

Common bleeding events more than 30 days after implant are epistaxis, GI bleeding, mediastinal and thoracic bleeding, and intracranial hemorrhage (reviewed in [66]) (7, 70, 76). Average INR is not higher in GI bleeding patients than in non-bleeding patients (77). Causes of GI bleeding include most commonly arteriovenous malformations in addition to bleeding from polyps, gastric feeding tubes, and mucosal erosions from gastroesophageal reflux.

Intracerebral hemorrhage can occur as a result of hemorrhagic conversion of ischemic strokes, traumatic subdural and subarachnoid hemorrhage, and spontaneous intracranial hemorrhage. Intracranial hemorrhage is a common neurologic complication in LVADs. Many patients are not excessively anticoagulated at the time of their hemorrhage (78, 79), and postoperative INR or anticoagulation regimen were not associated with neurologic complications (80). We and others have observed excessively high LVAD flows and hypertension (MAP > 90 mmHg) to predispose patients to spontaneous intracranial hemorrhage. Intraparenchymal hemorrhage carries an especially poor prognosis, with a 30-day mortality rate of 59% (78).

An approach to GI bleeding by many programs (Fig. 8) includes holding anticoagulation temporarily. If bleeding stops, goal INR can be slowly increased while continuing to withhold aspirin. Patients have managed without anticoagulation for more than a year without thrombotic complications (62, 67, 81, 82). After an intracerebral hemorrhage event, retrospective analysis indicates that aspirin can be resumed after 1 week and warfarin after 10 days without expansion of the hemorrhage or thrombotic events (77).

In patients with nonsurgical life-threatening hemorrhage unresponsive to standard measures, recombinant activated factor VIIa can be used. Dramatically higher thromboembolic events have been experienced with higher doses of recombinant factor VIIa (84).



**Figure 8.** Management of gastrointestinal (GI) bleeding. ASA = aspirin, INR = international normalized ratio, LVAD = left ventricular assist device. Reproduced with permission from Suarez et al (66). Adaptations are themselves works protected by copyright. So in order to publish this adaptation, authorization must be obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation.

## INFECTION

Infection is the second most common cause of death after cardiac failure (84). The International Society of Heart and Lung Transplantation (ISHLT) classifies infectious complications based on their relationship to the device (51). However, infection of any kind is associated with increased hospital stay and increased mortality (85). Device-associated infections often occur after hospital discharge (86). In addition, an association has been noted between postoperative infections and neurologic complications (69, 79, 80).

LVAD-specific infections are driveline or pump pocket infections. These are characterized by localized warmth and erythema, cellulitis, fever, and leukocytosis. These infections can ascend into pump pocket or descend from the pump. Pump-pocket infection may also exhibit drainage



from the driveline exit site and abdominal tenderness. CT, leukocyte-tagged planar scintigraphy, or hybrid single-photon emission-CT have limited utility in diagnosis (36, 86). Ultrasound can identify fluid collections and guide needle aspiration. Deep tissue swabs or biopsies from devices should be obtained where feasible and appropriate.

Most LVAD-specific infections are caused by Gram-positive organisms, *Staphylococcus aureus* most commonly, but *Enterococcus* and other *Staphylococcus* species are also frequently isolated. The Gram-negative pathogen most frequently isolated in LVAD-specific infections is *Pseudomonas aeruginosa* (87).

Infections of all types need aggressive treatment due to the risk of seeding the device. Localized infection of driveline or surgical site can be empirically treated with Gram-positive coverage alone, with coverage of resistant organisms as dictated by institutional prevalence and resistance profile as well as the patient's history of infection and prior antimicrobial treatment. Deeper wound infections or pump pocket infections are often empirically treated with Gram-negative and Gram-positive coverage. Although the driveline itself cannot be replaced without replacing the pump as well, surgical revision to move the driveline away from an infected tract is sometimes performed. Deeper tissue infections are often treated with surgical debridement (88), and pump pocket infections are sometimes treated with antibiotic impregnated beads (89), omental or muscle flap, or vacuum-assisted closure devices (90–92). In severe or refractory cases, the device is explanted and patients are subsequently transplanted (85) or given extracorporeal support until transplant can occur.

There have not been any trials addressing the need for secondary prophylaxis prior to surgical/dental procedures. The ISHLT guidelines suggest that secondary prophylaxis would be appropriate, but it remains at the discretion of the treating physician (51).

## ARRHYTHMIAS

Although not necessary for LVAD function, loss of atrial kick can still lead to decompensation due to diminished RV cardiac output and function, and patients may therefore require rate or rhythm control. In the absence of a contraindication, the target INR is raised to 2–2.5.

Ventricular arrhythmias are common post-LVAD (93). Reentrant circuits can be created by placement of the LVAD cannula (6, 15). Arrhythmias can also be triggered by contact between the cannula and the ventricular septum during suction events caused by hypovolemia, RV failure, or small ventricular size. Cannula malposition can occur months after implantation due to migration of the device from significant changes in weight or scar tissue (94), and cause recurrent ventricular arrhythmias.

Ventricular arrhythmias are often treated in stepwise fashion. First, the LVAD speed is decreased to allow for increased ventricular filling, which may shift the cannula away from the septal wall. Hypovolemia is treated with fluid boluses.  $\beta$ -blockers and amiodarone are first-line antiarrhythmics, plus

mexiletine in refractory ventricular tachycardia. Other antiarrhythmics (sotalol, lidocaine) can be used as needed. No change in INR target is indicated (94). In many cases, antiarrhythmic therapy can be titrated off over time since the prevalence is highest in the first month after implantation (94, 95). Those with refractory ventricular tachycardia may be eligible for catheter ablation, cannula repositioning, or device exchange.

## AORTIC INSUFFICIENCY

The prevalence and severity of aortic insufficiency tends to increase with the duration of LVAD support (16). At high LVAD speeds, the native heart works in series with LVAD pump, and the aortic valve does not open. At lower speeds, the LV works in parallel with the LVAD, and LV contraction ejects blood across the aortic valve. However, maximum opening area and time are greatly reduced, resulting in a functional aortic stenosis. Higher transvalvular pressure and increased strain on valve leaflets leads to fusion or incompetence (5, 43) which can lead to regurgitation (95). Aortic regurgitation creates a continuous loop of oxygenated blood from the aorta to the LVAD that does not reach the systemic circulation, reducing the effectiveness of the pump, leading to pulmonary edema and increased shear stress on RBCs (5). When the aortic valve does not open normally, the patient is at risk for thrombosis on the aortic valve (18), posing a threat of embolism to the brain or other organs. Serial echocardiograms are used to determine aortic valve opening and make speed adjustments to balance optimal valve function with LV unloading.

## NONCARDIAC SURGERY

Noncardiac surgery can be performed but presents significant risk of morbidity due to bleeding. A VAD coordinator or nurse familiar with the management of the device should accompany patients in the operating room to manage the console, monitor LVAD flow, and address any alarms. If emergency surgery at a center without VAD support is necessary, care providers should call the manufacturer or nearest hospital with a VAD program to obtain advice and recommendations on management of the device. General anesthesia can be administered safely. Arterial cannulation should be used to monitor MAP. Intra-abdominal procedures must proceed with extreme caution to avoid encountering the subcutaneously tunneled driveline. Ultrasound can be used to mark the driveline's location. Electrocautery is safe with LVAD, but implanted defibrillators should be deactivated prior to surgery.

No protocols exist for managing anticoagulation in the perioperative period, and a variety of approaches have been used (96). Given the low long-term risk of minimal anticoagulation and high risk of perioperative bleeding in anticoagulated patients (64, 67), it seems prudent to discontinue warfarin 5 days prior to elective surgery (96). Antiplatelet therapy with low-dose aspirin can be continued, and patients with additional reasons for anticoagulation (atrial fibrillation, acute thromboembolism) may be bridged with IV unfractionated heparin (96).



## DEVICE STOPPAGE AND CARDIAC ARREST

LVAD patients in cardiac arrest should be managed with the Advanced Cardiac Life Support (ACLS) algorithm for cardiac arrest with a few caveats. Patients with device stoppage can present in severe cardiogenic shock or full arrest and should be managed appropriately while searching for the cause of malfunction. Once restarted, a major concern is thromboembolism from the device. Device exchange can be performed emergently if necessary and feasible. Pulses are not a reliable metric for resuscitation. Electrical activity without cardiac contractility should prompt a search for underlying causes, such as tension pneumothorax and electrolyte derangements. In case of cardiac arrest not due to device malfunction, patients may or may not have adequate flow through their device. When patients in terminal rhythms have power outputs indicating flow through the device, a “chemical code” using only electrical cardioversion/defibrillation, epinephrine, and atropine according to ACLS guidelines may be sufficient to terminate the arrhythmia and restart the heart. When power output is extremely low, chest compressions may be necessary to maintain perfusion. The major risk factor to chest compressions during cardiopulmonary resuscitation is dislodgement of the device or its outflow cannula, located directly beneath the sternum. This is mainly of concern with the larger preperitoneal devices, such as HMII. A potential alternative is abdominal compressions, given 1–2 inches left of midline. Abdominal compressions in an LVAD patient maintained a coronary perfusion pressure of 15 mm Hg (97), the minimum required to achieve return of spontaneous circulation and better than the perfusion pressure usually achieved with chest compressions (98). At return of spontaneous circulation, care should be taken to support the ischemic RV.

## END OF LIFE

Stopping the device at the request of a patient or surrogate is not considered physician-assisted death but rather allowing natural death to occur (30). This may be due to a process unrelated to the device that will inevitably lead to the patient's demise despite LVAD support. More complex still is the patient who has experienced complications and thus suffers from a new set of comorbidities, the burdens of which exceed the benefits of living with LVAD support (99). In this situation, the patient or surrogate may request the device be turned off in compliance with the patient's advanced directives. Unfortunately, most patients' advance directives do not mention the LVAD or withdrawal of LVAD support (100). Device deactivation results in death of the patient, usually within minutes (37).

## CONCLUSION

As the demand for advanced heart failure treatment continues to grow and technology improves, intensivists are increasingly likely to encounter patients supported with LVADs either during their initial hospitalization or on subsequent admissions for complications. Management of these unique patients in the ICU is best accomplished with a multidisciplinary team

that includes specialists in advanced heart failure, LVAD nurse coordinators, and intensivists.

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