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Complications Following Left Ventricular Assist Device Implantation: Diagnosis and Management of Common Adverse Events

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Abstract Increasing use of left ventricular assist devices (LVADs) has led to improved survival in patients with chronic end-stage systolic heart failure. More than 2,000 devices are implanted each year in the United States. Newer generation continuous-flow LVADs have improved safety profiles over the previous pulsatile-flow devices. Post-implantation complications are still common, though, and lead to significant morbidity and mortality. Adverse events, such as right ventricular failure, bleeding, pump thrombosis, and cardiac arrest, require special consideration in the setting of an assist device. The intensivist should be familiar with the diagnosis and management of complications following LVAD placement.

Keywords Left ventricular assist device · LVAD complications · Right ventricular failure · Mechanical circulatory support · Chronic systolic heart failure · Acquired von Willebrand syndrome · Pump thrombosis · Cardiovascular accident (CVA) · Cardiac arrest · Device failure

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Introduction

More than 650,000 people are diagnosed with heart failure (HF) annually in the United States [1]. Most can be managed medically, but patients with end-stage disease require more advanced therapies. Heart transplant is the definitive treatment for end-stage HF, but this therapy is limited by the scarce number of organs available for transplantation [2]. In recent years, mechanical circulatory support (MCS), in the form of durable left ventricular assist devices (LVADs), has become standard therapy for patients who will not receive an imminent heart transplant.

Several indications for LVAD exist: bridge-to-transplant (BTT) for patients who are transplant-eligible, bridge-to-candidacy (BTC) for those who may become eligible with continued therapy, and destination therapy (DT) for patients who are not transplant candidates. Regardless of the indication, the goal of durable LVAD therapy is the same: to mechanically decompress the failing left heart by pumping blood out of the left ventricle (LV) and into the aorta, improving cardiac output and end-organ perfusion. Offloading the LV may also lead to a reduction in pulmonary venous congestion, pulmonary arterial pressures (PAPs), and tricuspid regurgitation (TR), with subsequent improved right ventricular (RV) function. Over time, remodeling of the pulmonary vasculature and improvement in pulmonary arterial hypertension (PAH) may occur. Because PAH is a relative contraindication for heart transplant, LVAD therapy may allow some patients to move from the transplant-ineligible groups (DT and BTC) to the BTT group [3].

The most common continuous-flow devices implanted in the United States today are the axial-flow Thoratec® HeartMate II® (HMII) and the centrifugal-flow HeartWare® HVAD®. Both systems consist of an inflow

cannula sewn to the LV apex, a pump, and an outflow cannula situated in the ascending aorta. The HMII pump is implanted into the preperitoneal space or into an abdominal pocket, while the HVAD pump sits inside the pericardium. The HMII is FDA-approved as BTT [4] and DT [5] therapy, while the HVAD is approved for BTT therapy [6]. These devices show improved survival and less frequent adverse events when compared to earlier generation pulsatile-flow VADs. Current overall 1-year survival after durable LVAD placement is 80 %; 2-year survival is 70 % [7••]. In addition to increased survival, patients enjoy significant improvements in quality-of-life measures and functional status [8]. From 2006 to 2013, more than 10,000 durable VADs were implanted and registered with the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS). Currently, more than 2,000 devices are implanted annually. Rates continue to rise, particularly due to the increase in DT LVADs, which now account for 40 % of implants [7].

VAD Parameters

The only adjustable parameter on the device is the pump speed, set in rotations per minute (RPMs). The VAD monitor will display the chosen speed, a measured pump power in watts, a calculated flow rate in liters/minute, and a dimensionless representation of pulsatility. Power represents the work of the pump at the set speed. Flow is a surrogate for cardiac output; it is determined by the pump speed and the pressure gradient between the LV and the aorta. Pulsatility reflects change in flow through pump over time. All three variables are affected by preload to the pump, native cardiac contractility, LVAD speed, systemic afterload, and by one another. In general, when other variables are held constant, an increase in speed will lead to increased flow, increased power, decreased pulsatility, and greater offloading of the LV. General hemodynamic goals for managing patients with LVADs are to maintain preload while minimizing afterload, thereby promoting forward flow through the VAD. The initial speed is chosen under echocardiographic visualization to optimize LV decompression and maintain right ventricular (RV) function. The speed is adjusted as needed in the postoperative period in response to changing clinical conditions.

Complications

Despite their ability to improve survival, modern LVADs carry a substantial risk of associated complications that lead to considerable morbidity and mortality [9]. The incidence of significant adverse events in the first 60 days after implantation approaches a staggering 90 % [10]. Managing complications such as RV failure, bleeding, thrombosis, stroke, and arrhythmias in the setting of an assist device requires special consideration. As increasing numbers of patients receive LVADs, a working knowledge of how to diagnose and treat associated complications is essential for the intensivist. See Table 1 for a summary of diagnosis and management of complications after LVAD implantation.

Right Ventricular Failure

Right ventricular dysfunction is common in end-stage HF, usually secondary to severe left heart failure. Following LV decompression by the LVAD, RV function gradually improves, as evidenced by a decrease in filling pressures, systolic PAP, pulmonary capillary wedge pressure (PCWP), and severity of TR, with a concomitant increase in cardiac output (CO) [11]. However, in the early postoperative period, the vulnerable, thin-walled RV is at risk of failure due to hemodynamic and mechanical changes brought on by LVAD flow. As cardiac output increases, venous return is increased to the RV, which may not be able to accommodate larger volumes. Frequent transfusions in the perioperative period contribute to the volume load. In addition, over-decompression of the LV with high LVAD speeds can shift the interventricular septum toward the LV, resulting in a change in RV geometry, worsened TR, and subsequent RV dysfunction. RV function can be further compromised by intraoperative injury from air embolism down the right coronary artery, poor cardioprotection during cardiac arrest, long cardiopulmonary bypass (CPB) time, or protamine-induced pulmonary hypertension.

The reported incidence of RV failure (RVF) in the post-LVAD period ranges from 19–40 % [5, 11–18]. RVF is often defined as prolonged inotrope requirement greater than 14 days, or need for temporary right ventricular assist device (RVAD) support. Most patients can be managed with inotropic therapy alone; 3–14 % require RVAD. RVF is a significant cause of mortality, accounting for up to 5 % of all deaths in the post-implantation period [5]. Patients requiring RVAD have a 1-month survival of 60 %, while only 30 % are alive at 6 months, compared to 98 and 87 % of patients without RVF [11]. RVF is suspected in patients with unstable hemodynamics (rising CVP, PAP, and PVR, with decreasing CO and LVAD flows), end-organ failure, or symptoms of venous congestion. Echocardiography can confirm the diagnosis.

The incidence of postoperative RVF may be reduced by optimizing volume status with aggressive diuresis or ultrafiltration in the preoperative period [11]. Intraoperative strategies to lower the risk of RVF include limiting CPB time and avoiding cardiac arrest with cardioplegia. In

Table 1 Summary of diagnosis and management of complications after LVAD implantation

Complication	Signs and symptoms	LVAD findings	Echo findings	Diagnosis and treatment
RV failure	⇓ MAP ⇑ CVP, PAPs, PAPd ⇓PCWP ⇓ CI End-organ dysfunction Venous congestion	⇓ Flow ⇓ Pulsatility	RV is distended (⇑ RVEDD) Poor RV systolic function (⇓ TAPSE, ⇓ RVSWI) Increased TR	Avoid RV volume overload: diuretics, UF, rapid pacing Increase RV contractility: inotropes Reduce RV afterload: inhaled pulmonary vasodilators, MV to avoid hypercarbia/ hypoxia RVAD
Bleeding/ hypovolemia	⇓ MAP ⇓ CVP, PAPs, PAPd, PCWP ⇓ CI End-organ dysfunction	⇓ Flow ⇓ Pulsatility Suction events	LV is over-decompressed (⇓ LVEDD) RV is decompressed IVS may be shifted toward LV → increased TR	Volume resuscitation with crystalloid or blood Surgical exploration
Tamponade	⇓ MAP, PCWP ⇑ CVP, PAPd ⇓ CI Equalization of pressures End-organ dysfunction	⇓Flow ⇓ Pulsatility	Impaired filling of RV or LV due to external compression Pericardial effusion	Drainage of pericardial fluid: bedside pericardiocentesis vs. return to OR
Pump thrombosis	⇓ MAP Systemic thromboembolism End-organ dysfunction	⇓Flow ⇑ Power (usually gradually over time, but can be abrupt) or power spikes ⇓ Pulsatility	LVAD Ramp study: LV not progressively offloaded with increasing LVAD speeds Aortic valve closes at higher speeds	Check LDH ⇑ Anticoagulation ± thrombolytics Device exchange
Ventricular arrhythmia	⇓ or ⇔ MAP ⇑ CVP, PAPd ⇓ CI End-organ dysfunction Venous congestion	⇓Flow ⇓ Pulsatility Suction events	RV may be distended (⇑ RVEDD) LV decompressed (⇓ LVEDD)	Volume resuscitation Decrease LVAD speed Antiarrhythmics Assess cannula position
Sepsis	⇓ MAP ⇓ CVP, PAPd ⇑ CI End-organ dysfunction	⇑ Flow ⇑ Pulsatility	RV may be hyperdynamic	Volume resuscitation Antibiotics Vasopressors for vasoplegia Identify and treat source of infection

CI cardiac index, *CVP* central venous pressure, *IVS* interventricular septum, *LV* left ventricle, *LVAD* left ventricular assist device, *LVEDD* left ventricular end-diastolic diameter, *MAP* mean arterial pressure, *MV* mechanical ventilation, *PAPd* diastolic pulmonary artery pressure, *PAPs* systolic pulmonary artery pressure, *PCWP* pulmonary capillary wedge pressure, *RV* right ventricle, *RVAD* right ventricular assist device, *RVEDD* right ventricular end-diastolic diameter, *RVSWI* right ventricle stroke work index, *TAPSE* tricuspid annular plane systolic excursion, *TR* tricuspid regurgitation, *UF* ultrafiltration

addition, concomitant tricuspid valve repair or replacement for moderate or severe TR has been shown to reduce postoperative RV dysfunction [11, 19, 20]. In the post-CPB period, RV function can be maximized by maintaining euvolemia, increasing contractility, and reducing afterload. It is critical to avoid volume overload in the early postoperative period by judicious blood product and crystalloid administration. Diuretics are often begun on the first or second postoperative day. Ultrafiltration should be considered in patients resistant to intravenous diuretics. Rapid pacing at a rate of 90–110 bpm will reduce RV diastolic filling time and decrease distension. Hypovolemia

should also be avoided, as the LVAD is preload dependent; rather, euvolemia should be maintained. RV contractility and resultant LV filling can be improved with inotropes such as epinephrine, milrinone, and dobutamine. Early initiation of inotropic therapy during weaning from CPB, as opposed to after the RV shows signs of failure, may prevent progression to RVF [11]. Milrinone and dobutamine have an added vasodilatory effect, which can offload the RV by reducing PAPs. Unfortunately, systemic hypotension can also occur with these agents, and infusions of vasopressors may be required to maintain adequate systemic vascular resistance (SVR) and perfusion to the right coronary artery. Inhaled pulmonary vasodilators can decrease PAPs without significantly reducing SVR. Inhaled nitric oxide (iNO), and nebulized formulations of iloprost [21], epoprostenol [22], and milrinone [23], used alone and in combination, have been shown to reduce PVR, mean PAP, RV systolic pressure, PCWP, and increase LVAD flows [21]. Hypercarbia, hypoxia, acidosis, pain, anxiety, and high dose alpha-adrenergic agents can all result in increased PVR, and should thus be avoided. Invasive or non-invasive mechanical ventilation may be helpful in preventing hypoxia and hypercarbia-induced respiratory acidosis. Finally, LVAD speed can be adjusted under echocardiographic visualization to ensure optimal interventricular septum position and RV geometry.

When RVF persists despite maximal medical therapy, MCS may be necessary. Several devices are available for temporary RV support. The Thoratec® Centrimag® is a centrifugal-flow pump that provides up to 10 L/min of flow; it is approved for use as an RVAD for up to 30 days[1]. The inflow cannula is inserted percutaneously in the femoral vein or centrally into the right atrium (RA); the outflow cannula returns blood via a graft on the PA, bypassing the RV. The axial-flow Abiomed® Impella RP® is approved for 14 days[2] and consists of a single cannula that is inserted percutaneously through the femoral vein. The inflow port is positioned in the IVC, while the outflow port on the tip of the cannula sits in the PA. The ROTAFLOW® Extracorporeal Membrane Oxygenation (ECMO) circuit has also been used to treat RVF; cannulation sites are similar to the Centrimag [24]. If RV function fails to recover with temporary MCS, permanent biventricular support with either total artificial heart or biventricular VADs (BiVADs) [25] may be considered. Outcomes for these patients are poor; 6-month survival is only 56 % in patients with BiVADs, compared to 86 % in patients with LVAD alone. Adverse event rates for infection, bleeding, neurologic events, and device failure are also much higher [26].

[1] http://www.thoratec.com/medical-professionals/vad-product-information/thoratec-centrimag.aspx, accessed 12 June 2015

[2] http://www.abiomed.com/products/impella-rp/, accessed 12 June 2015

Bleeding and Thrombosis

Hematologic Derangements

Both bleeding and thrombosis are common complications that contribute significant morbidity after LVAD implantation. Patients with end-stage HF often have hematologic derangements even prior to LVAD placement, including both platelet dysfunction [27] and a pro-thrombotic state resulting from chronic activation of the extrinsic coagulation pathway [28]. LVAD flow exacerbates endothelial dysfunction and sustains activation of this pathway in the postoperative period. In addition, all patients with MCS develop acquired von Willebrand syndrome (AVWS), due to the shear stress on the high molecular weight multimer of von Willebrand factor. This results in decreased platelet adhesion and aggregation and, subsequently, less effective hemostasis. The onset of AVWS is immediately after LVAD implantation, and resolves after explant of the device [29, 30]. These hematologic abnormalities are exacerbated by chronic antiplatelet and anticoagulation therapy, which are used to prevent pump thrombosis. Achieving the appropriate therapeutic levels for these medications can be challenging, increasing the risk of bleeding and thrombotic complications.

Surgical Bleeding

Post-LVAD bleeding can be categorized into surgical and non-surgical bleeding. Surgical bleeding occurs in the first few postoperative hours to days [31]. Chest tube output, hypotension, low flow rates on VAD monitor and suction events are all clues of ongoing blood loss. Suction events can result from an underfilled LV, due to hypovolemia or tamponade, in which partial ventricular collapse causes obstruction of the inflow cannula. Blood product transfusion is required in up to 80 % of patients, and 15–30 % of patients undergo surgical exploration for bleeding [5, 12] Bleeding and hemorrhage are responsible for 3–6 % of postoperative deaths [5, 14, 16].

Gastrointestinal Bleeding

Non-surgical bleeding presents mostly as gastrointestinal bleeding (GIB), but also as epistaxis, genitourinary bleeding, and intracerebral hemorrhage [32]. Ten to thirty percent of LVAD patients experience GIB at some point [6, 14, 33–35]. Many GIB are caused by arteriovenous malformations, which occur more frequently in the setting of reduced pulsatility and AVWS [36]. Risk factors include prior GIB, elevated international normalized ratio (INR) or

low platelets [34]. Management of GIB in the LVAD patient starts with discontinuing anticoagulation and antiplatelet therapy and transfusing to correct anemia. Leukoreduced blood should be used in BTC or BTT patients, to avoid development of anti-HLA antibodies that could limit potential donor organ matching. It may be appropriate to reverse supratherapeutic anticoagulation levels, but clinical judgment should be used as device thrombosis may result. The source of bleeding should be investigated with endoscopic studies, tagged RBC scan or angiography [37••], and treated with banding or coiling as possible. Anticoagulation and antiplatelet therapy should be restarted as early as is deemed safe. Although morbidity is increased due to hospital readmissions, blood transfusions, and invasive procedures, mortality after GIB is unchanged [38].

Pump Thrombosis

Pump thrombosis is a serious complication after LVAD, occurring in 3–13 % of cases [5, 6, 15, 33, 39, 40]. For unclear reasons, the incidence appears to be increasing in recent years, while the time to thrombosis is decreasing. Most recent data suggests that 8 % of patients with VADs experience device thrombosis, at a median time of approximately 3 months following implantation [41•]. Each episode of pump thrombosis results in substantially increased mortality, as well as increased risk of cerebrovascular accident (CVA) and infection [7]. One-year survival for patients with thrombosis is 70 %, versus 85 % in patients without thrombosis [42]. Mean INR in patients who develop thrombosis is not significantly different than those free from thrombosis [33]. However, patients with a history of GIB are at higher risk, possibly due to periods of interruption in therapeutic anticoagulation [43]. Other factors that increase risk for pump thrombosis include younger age, larger BMI, worse renal function, and poor LV function.

Pump thrombosis can be suspected by a combination of laboratory data, LVAD parameters, echo findings, and clinical assessment; however, it can only be conclusively diagnosed after device explant. Historically, elevated plasma-free hemoglobin has been used as a marker for hemolysis or thrombosis, but it has recently been shown that lactate dehydrogenase (LDH) is a more sensitive and specific predictor of pump thrombosis [44]. LDH can be chronically elevated in any patient with an LVAD, but levels >600 units/L (normal range 140–280 units/L) are concerning for thrombosis and warrant further investigation. LDH levels can start to rise several weeks prior to appearance of any signs of pump thrombosis. The LVAD monitor can provide clues to the diagnosis as well. Interrogation of historical pump data may show a gradual increase in power and decrease in flow over time. Abrupt changes in power and flow are also possible but less common. Uriel, et al [45] developed an echocardiography ramp test that can be performed for suspected pump thrombosis in the HMII device. It consists of gradually increasing device speed, while noting LVAD and clinical variables. With device thrombosis, the LV will not progressively decompress with increasing LVAD speeds, and the aortic valve will close at higher speeds than in absence of thrombus. Clinically, pump thrombosis may result in thromboembolism, unstable hemodynamics, and end-organ dysfunction.

Some patients may be successfully managed with an increase in anticoagulation, which includes heparin infusion and glycoprotein IIb/IIIa inhibitors. There are several reports of thrombolytic therapy used to treat pump thrombosis [42, 46, 47], administered either intravenously or directly into the LV via a pigtail catheter. However, medical therapy frequently fails [42] and thrombolytic therapy has resulted in intracerebral hemorrhage [46]. The gold standard for treating pump thrombosis is device exchange.

Neurological Events

A sequela of bleeding and thrombosis, neurological events are a major cause of morbidity and mortality after LVAD implantation [7]. Both ischemic and hemorrhagic CVAs are common, with a combined incidence ranging from 9–44 %. In some studies, ischemic strokes were more likely with a subtherapeutic INR, while hemorrhagic strokes were more likely with a supratherapeutic INR [48]. However, many hemorrhagic CVAs occur in the setting of appropriate anticoagulation [49]. Another major risk factor for CVA is infection, particularly persistent blood stream infections [50]. Neurologic events are a leading cause of death, responsible for 9–24 % of all mortality [5, 14]. The 30-day mortality of CVA in the setting of LVAD is 25 % [48]. Diagnosis and treatment of CVA is similar to patients without LVAD, with several exceptions. LVADs are not MRI-compatible, so diagnosis must rely on less sensitive imaging such as CT scanning. For hemorrhagic stroke, anticoagulation and antiplatelet therapy should be discontinued immediately, but restarted as soon as possible; the risk of extension or recurrence of intracerebral hemorrhage must be weighed against the risk of pump thrombosis.

Arrhythmias

Atrial Arrhythmias

Patients with end-stage heart failure, both pre- and post-LVAD implantation, frequently suffer from cardiac arrhythmias. Atrial arrhythmias (AAs) are usually well

tolerated from a hemodynamic standpoint in the presence of an LVAD, unless RV filling is compromised and LVAD preload suffers. AAs are treated similarly to patients without LVAD support, with beta blockers, antiarrhythmics, cardioversion and appropriate anticoagulation [51]. AAs do not lead to increased mortality, but patients tend to have less improvement on quality-of-life and functional assessments [52].

Ventricular Arrhythmias

Preexisting ventricular arrhythmias (VAs) can persist following LVAD implantation, and new VAs may develop from new reentrant circuits or suction events. Reentrant circuits can form after coring of the ventricular apex for inflow cannula placement. Suction events occur when the LV is overly decompressed, due to hypovolemia or high LVAD speeds, and the cannula comes in contact with the septal wall, obstructing flow and potentially inducing VAs. Over time, as the LV remodels, the position of the cannula relative to the septal wall may shift, leading to increased frequency of suction events and VAs. The incidence of post-LVAD VAs ranges from 20–43 %. The major risk factor is preexisting VAs; patients without VAs prior to LVAD placement are less likely to develop VAs post-LVAD. Other risk factors include non-compliance with beta blocker therapy [53], prior implantable cardioverter defibrillator (ICD) placement, and history of ICD discharge [54]. VAs can be well tolerated hemodynamically, but may lead to poor RV and LVAD filling and subsequent HF symptoms, cardiogenic shock, and sudden cardiac death [51]. These patients also have increased rates of readmission and more frequently require external defibrillation [54]. Acute treatment is dictated by hemodynamics; if the arrhythmia leads to hemodynamic instability, chemical or electrical cardioversion is indicated. If cannula position is thought to be triggering the arrhythmia, hypovolemia should be treated and the LVAD speed reduced as tolerated, to reduce the risk of suction events. If VA-inducing suction events persist, repositioning of inflow cannula or device exchange may be warranted. Anti-arrhythmic therapy with beta blockers and amiodarone are mainstays of treatment; mexiletine, dofetilide, sotolol and lidocaine are less frequently used. As with all arrhythmias, electrolytes should be optimized. If not already in situ, ICD placement should be considered, as ICD therapy is associated with reduced mortality [55]. In one study, 1-year survival to transplantation was 91 % in BTT patients with ICD plus LVAD compared to 57 % in patients with LVAD alone [56]. Catheter VT ablation has been described in patients with intractable VAs; it is often initially successful but VAs recur in a large proportion of patients [57, 58]. If VAs are refractory to treatment and continue to cause hemodynamic instability, biventricular support in the form of BiVADs or TAH may be considered.

Infection

Infectious complications after LVAD implantation have been classified by the International Society for Heart and Lung Transplantation (ISHLT) into VAD-specific, VAD-related, and non-VAD infections [59]. VAD-specific infections include infections related to the pump, cannulas, pump pocket, or driveline site. VAD-related infections can occur in the absence of a VAD, but require special consideration in setting of VAD; they include endocarditis and mediastinitis. Non-VAD infections can still cause significant morbidity in VAD patients. Driveline infections are the most common, occurring in 5–18 % of patients, usually weeks to months after implantation [60]; they do not increase mortality. Sepsis occurs in 10–18 % of patients and is associated with significant risk of mortality and CVA [61]. Common organisms isolated from VAD-specific and VAD-related infections include *Staphylococcus aureus, Pseudomonas aeruginosa* and *streptococcus sp.* [50, 61, 62]. The major risk factor for infection is duration of support: the longer the device is in place, the higher the risk of both persistent blood stream infections [50] and driveline infections [60]. Treatment includes antibiotics, surgical debridement, relocation of driveline, and device exchange.

Cardiac Arrest and Device Stoppage

Cardiac Arrest

Cardiac arrest in patients with VADs is not an uncommon phenomenon; one institution reported that patients with VADs comprised 4 % of all in-hospital cardiac arrests [63]. However, there are several factors that make the traditional Advanced Cardiac Life Support (ACLS) algorithm inappropriate for these patients. First, ACLS is based on the presence or absence of a pulse to guide decision making; patients with LVADs may not have a palpable pulse even in states of hemodynamic stability. Ideally, perfusion should be assessed by Doppler blood pressures, with a goal MAP between 70 and 90 mm Hg.

Little is known about the physiology of chest compressions in patients with LVADs. The manufacturers of both the HMII and HVAD devices caution that performing chest compressions may result in dislodgment of the underlying inflow cannula or aortic anastomosis (HeartMate II® LVAS Operating Manual, HeartWare® Ventricular Assist System Instructions for Use). This could be potentially catastrophic, leading to device malfunction, and swift exsanguination. There have been several case reports

of both successful chest [63–65] and abdominal-only compressions [66], but there is insufficient data to support recommendation of this therapy. Clinical judgment should be used. If compressions are performed, imaging should be obtained post-code to confirm appropriate cannula position. If feasible, intra-cardiac massage by an experienced provider may be beneficial.

Finally, ACLS guidelines recommend aggressive medical, electrical and mechanical treatment pathway for ventricular tachycardia (VT) or ventricular fibrillation (VF). Ventricular arrhythmias may be very well tolerated in patients with LVADs; if end-organ perfusion is intact with stable hemodynamics, emergency treatment is unnecessary.

In the presence of hemodynamic collapse from VAs or another cause, both pharmacologic and electrical treatments are safe. Because the LVAD is preload dependent, volume resuscitation is often indicated. Epinephrine, vasopressin, and antiarrhythmics can all be given for lack of Dopplerable pulse or malignant arrhythmias, with the consideration that high doses of vasopressors may increase afterload such that LVAD flow is impeded. Defibrillation by external pads or by internal ICD is safe in setting of VAD. Advanced airway management should be performed per ACLS guidelines. Identifying cardiac arrest can be challenging in this population given the lack of pulse and frequent tolerance of VAs; other clues include ECG rhythm, low flow alarms on the VAD monitor, and clinical signs such as end-organ perfusion, mental status, and skin temperature [64]. The chest should be auscultated; in the absence of a VAD hum, device stoppage should be suspected and investigated quickly. Stat echocardiography can help determine the etiology of arrest.

Device Stoppage

Device stoppage is a potentially catastrophic event. Because the LVAD cannulas and pump are valveless, direction of blood flow can be anterograde or retrograde, depending on the pressure differential between aorta and LV. Retrograde flow upon device stoppage has been demonstrated in both computer simulation and bovine models [67]. In this situation, all connections should be checked quickly and battery assessed. If appropriate, arrangements should be made for urgent return to the OR for device exchange or ECMO cannulation [37]. Again, chest compressions carry the risk of cannula dislodgment. Some patients may have adequate native cardiac function to survive until device exchange; others who are more reliant on LVAD flow for systemic perfusion may expire quickly. Patients who have had their aortic valve oversewn due to severe aortic insufficiency at time of LVAD placement are at greatest risk of mortality in setting of device stoppage.

Conclusion

While LVADs have significantly improved survival in end-stage heart failure patients, increased hospital lengths of stay, frequent readmissions [68], diagnostic tests, blood transfusions, and surgical interventions are common. The complications discussed in this review contribute significantly to increased morbidity and mortality in patients with LVADs. As the number of patients with VADs continues to rise, intensivists will be increasingly faced with caring for these patients, both for conditions related to their heart disease and VAD, as well as unrelated medical and surgical conditions seen in the rest of the population. Familiarity with the altered physiology of LVAD patients, as well as being able to recognize and manage LVAD-related complications quickly and appropriately, is necessary to care for these complex patients.

Compliance with Ethics Guidelines

Conflict of Interest Eleanor Anne Vega declares that she has no conflict of interest. T. Miko Enomoto has received support through a research grant from Actelion Pharmaceuticals, Inc.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- • Of importance
- •• Of major importance

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