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Review

Left ventricular assist devices and their complications: A review for emergency clinicians

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ABSTRACT

Introduction: End stage heart failure is associated with high mortality. However, recent developments such as the ventricular assist device (VAD) have improved patient outcomes, with left ventricular assist devices (LVAD) most commonly implanted.
Objective: This narrative review evaluates LVAD epidemiology, indications, normal function and components, and the assessment and management of complications in the emergency department (ED).
Discussion: The LVAD is a life-saving device in patients with severe heart failure. While first generation devices provided pulsatile flow, current LVAD devices produce continuous flow. Normal components include the pump, inflow and outflow cannulas, driveline, and external controller. Complications related to the LVAD can be divided into those that are LVAD-specific and LVAD-associated, and many of these complications can result in severe patient morbidity and mortality. LVAD-specific complications include device malfunction/failure, pump thrombosis, and suction event, while LVAD-associated complications include bleeding, cerebrovascular event, infection, right ventricular failure, dysrhythmia, and aortic regurgitation. Assessment of LVAD function, patient perfusion, and mean arterial pressure is needed upon presentation. Electrocardiogram and bedside ultrasound are key evaluations in the ED. LVAD evaluation and management require a team-based approach, and consultation with the LVAD specialist is recommended.
Conclusion: Emergency clinician knowledge of LVAD function, components, and complications is integral in optimizing care of these patients.

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1. Introduction

Heart failure is increasingly common in the United States, affecting over 6.5 million patients in the United States [1,2]. This disease is associated with significant morbidity and mortality, with mortality at 5 years approaching over 50% [1-3]. However, recent developments have improved survival, specifically ventricular assist devices (VAD) in patients with advanced heart failure. The number of patients with VAD will continue to increase. Newer FDA approved devices include the axial-flow HeartMate II (HMII) and centrifugal-flow HeartMate III (HMIII) LVADs (Abbott Laboratories, Lake Bluff, IL), and HeartWare (HVAD) (Medtronic, Minneapolis, MN) [4-6]. These devices, as well as improved patient selection, are associated with significantly improved survival, ranging from 76 to 83% at 2 years, compared to a survival rate of 54% with first generation devices [4,5,7].

Most patients with VADs undergo significant education concerning the device, including the components and means of self-management. These patients also receive significant outpatient care and follow up with specialists. However, LVAD patients will likely present to the ED with increasing frequency. Approximately half of patients with a newly placed LVAD present to the ED within the first month after implantation, and patients present on average 7 times to the ED within the first year postimplantation [8,9]. Emergency clinicians must understand the associated anatomy and physiology with the device present, the device components, and complications.

2. Methods

Authors searched PubMed and Google Scholar for articles using the keywords “ventricular assist device” OR “left ventricular assist device” OR “VAD” OR “LVAD” AND “emergency” for production of this narrative review. Authors included case reports and series, retrospective and

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prospective studies, systematic reviews and meta-analyses, clinical guidelines, and other narrative reviews. The literature search was restricted to studies published in English. Initial literature search revealed over 600 articles. Authors reviewed all relevant articles and decided which studies to include for the review by consensus, with focus on emergency medicine-relevant articles, including guidelines. A total of 97 resources were selected for inclusion in this review.

3. Discussion

3.1. VAD function and components

A VAD can be placed into either or both ventricles; in other words, the patient can have a right ventricular assist device, left ventricular assist device, or biventricular assist device. The anatomic location of insertion obviously is heavily dependent upon the patient's clinical syndrome, strategic goals, and body habitus. In particular, the strategic goals are quite important in the placement consideration. These goals include three different strategies: bridge to recovery, bridge to transplantation, or destination therapy (i.e., the patient is unlikely to recover and not a candidate for cardiac transplant) [10-14]. Contraindications to placement include metastatic cancer, irreversible renal/hepatic failure, and cerebral accident with severe neurologic deficits [12-14].

The most commonly placed device is the left ventricular assist device, or LVAD. The emergency physician is most likely to see patients with an LVAD, and therefore, this review will discuss LVAD structure and placement.

The LVAD has two basic designs which produce different patterns of perfusion, including the pulsatile and continuous-flow devices. The pulsatile pump, found in <10% of patients with LVADs, attempts to mimic natural perfusion [9]. First generation devices include pulsatile positive displacement pumps, but these devices have significant limitations including limited durability, extensive surgery for implantation, large external lead, and audible pump [4-6,9]. Second generation devices include continuous flow devices, such as the HMII. Third generation devices use centrifugal pumps, with longer lifespans. These include the HVAD and HMIII devices. Continuous-flow devices are present in over 90% of patients with an LVAD. These devices provide constant perfusion. This is in contrast to cardiac-mediated perfusion with pulsatile pumps. Continuous flow devices deliver superior organ perfusion compared to the more “natural” pulsatile pump design [4-6,19]. In addition to the more effective perfusion, continuous-flow devices are both more durable and more compact [4-6].

The continuous-flow LVAD has several basic parts, including the internal pump, an external power source, and a control unit; specific components of the LVAD include the inflow cannula, pumping chamber, outflow cannula, percutaneous driveline, controller, and power source. The inflow cannula, usually placed in the apex of the left ventricle (LV), provides the route for blood flow from the native LV cavity to the LVAD pumping chamber. The pumping chamber, the component of the circuit which provides perfusion, is located in different positions depending upon the LVAD model type: the LV apex for the HMIII and HVAD devices and the subdiaphragmatic space adjacent to the heart for the HMII device. The pumping chamber contains the impeller, a near-friction-less rotor with rotation speeds ranging from 2500 to 9800 rpm; these types of impeller designs can generate blood flow up to 10 L per minute. The outflow cannula provides the conduit back to the patient's native cardiovascular system and connects the pumping chamber to the ascending aorta [4-6,9]. Fig. 1 details LVAD components, Fig. 2 depicts an LVAD appearance on a patient, and Table 1 presents normal LVAD parameters. Fig 3 demonstrates the controller panel of the HMIII device.

The percutaneous driveline provides a conduit for the electrical wiring, connecting the pump to the system controller. These wires not only connect the power source to the pump, but they also provide controlling and sensing functions for the LVAD. The driveline is tunneled subcutaneously from the pump and exits the skin in the anterior abdominal area to connect to the controller. Thus, it is a frequent source of infection in the LVAD patient. The controller performs multiple functions and contains several important components. It controls LVAD functioning, including power source monitoring and regulation, overall system monitoring, data collection, and alarm system function. The controller has a panel which demonstrates important functions, status, and warnings regarding system operation. Lastly, the power source is usually composed of rechargeable batteries. Most systems have the capability to have two batteries.

3.2. History and examination for the LVAD patient

Initial evaluation of patients with an LVAD is similar to most critical patients, though it differs in several aspects. Patients who are hemodynamically stable with an LVAD should be evaluated in the same manner as other patients, with the exception that the LVAD team or coordinator should be consulted. History, examination, and device evaluation are recommended for hemodynamically stable patients. Heart rate is dependent on the patient's rate and rhythm, though many patients with an LVAD may have a pacemaker or implantable cardioverter defibrillator (ICD). Blood pressure measurement depends on whether a palpable pulse is present [4-6,9,13]. A continuous flow LVAD will not typically produce a palpable pulse on its own, but patients may have enough ventricular function to produce pulsatile flow and a pulse [4-6,9,13]. A palpable pressure may be due to pump thrombosis, and thus, it is important to determine if the patient has a palpable pulse at baseline [4,9]. If a pulse is palpable, a standard sphygmomanometer may detect a blood pressure, which reflects a systolic blood pressure, rather than mean arterial pressure (MAP) [15,16]. However, a continuous flow LVAD with low pulse pressures may not allow measurement of blood pressure by this method. If the pulse is not palpable, a pencil Doppler probe should be placed over the radial or brachial artery. The point at which Doppler signal returns corresponds to the MAP for continuous flow devices [4-6,9]. If this is unobtainable, an arterial line may be required, which is the most accurate device for monitoring MAP. Invasive arterial monitoring will demonstrate minimal pulse pressure or flat arterial waveform [4-6,9,13,14]. Caution is recommended in using pulse oximetry, as a low reading commonly reflects a lack of pulsatile flow. However, a normal value may be accurate [17].

LVADs, especially those with continuous-flow, are sensitive to afterload and preload. Guidelines recommend maintaining a MAP of 70–90 mm Hg [18,19]. Elevated MAPs, corresponding to increased afterload, may cause decreased pump flow, which may result in worsening symptoms of heart failure. This may also cause subendocardial ischemia and ventricular dysrhythmias. Acute hypertensive adverse event is associated with MAP >110 mm Hg in patients with continuous flow pumps [6,18,19].

Physical examination should otherwise evaluate the primary systems involved with the chief complaint, as well as inspection of the device components [4-6,9,13,14]. Heart sounds are typically difficult to ascertain in patients with an LVAD due to the device's mechanical hum that often obscures other sounds. The mechanical hum indicates device power and function. Signs of volume overload (extremity edema, ascites, elevated jugular venous pressure) can be due to subacute or chronic right ventricular failure. However, acute dyspnea, pulmonary edema, or hypotension are more commonly due to acute malfunction of the device, such as cannula obstruction or pump thrombosis [4-6]. Clinicians must also assess the extremities for capillary refill and temperature. Altered mental status, focal neurologic deficit, or new headache are concerning for neurologic pathology such as intracranial hemorrhage. The device exit site, which is normally covered with a sterile dressing, and line should be examined with sterile gloves and mask for warmth, erythema, and discharge, which suggest infection. Clinicians should evaluate the controller, current settings, pump parameters,

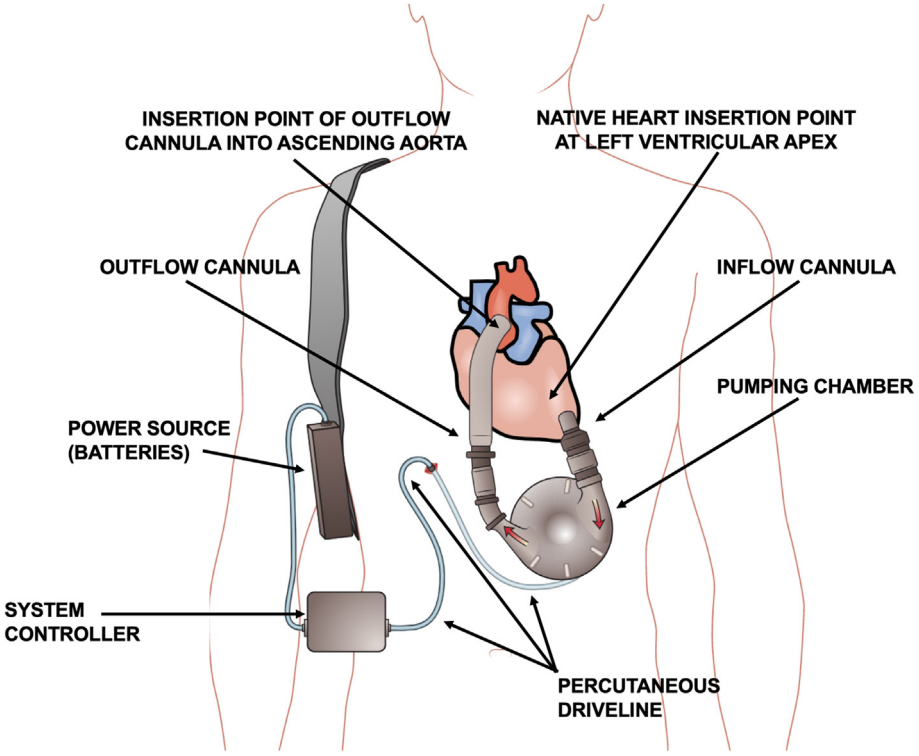

Fig. 1. LVAD components.

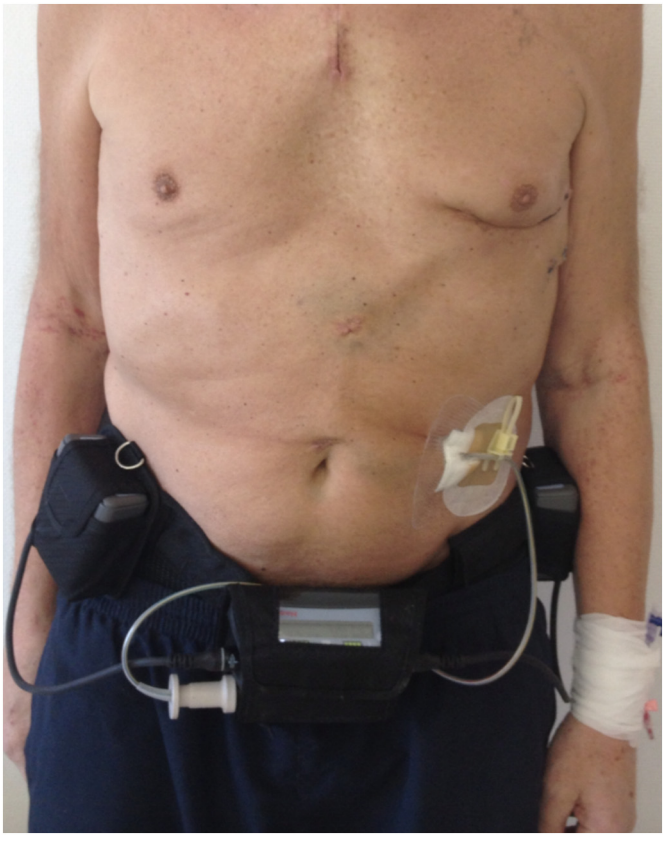

Fig. 2. LVAD example. From https://commons.wikimedia.org/wiki/File:Left_ventricular_assist_device_hannover.PNG

and alarms. Finally, the patient should be asked if he/she brought the back-up battery and back-up controller.

3.3. Recommended emergency department evaluation

Electrocardiogram (ECG) is a vital component of the assessment to evaluate for dysrhythmia. ECG findings are often non-specific but can demonstrate low voltage, electrical artifact, and splintering of the QRS interval [4,20]. Patients with ventricular dysrhythmias most commonly present within the first month of LVAD implantation, but the LVAD may allow for adequate perfusion despite the dysrhythmia. Sustained ventricular dysrhythmias may be due to underlying cardiomyopathy or decompressed left ventricle due to elevated pump speed or right ventricular failure [19,20]. Patients with an LVAD will typically demonstrate normal sinus rhythm.

Chest radiograph provides important diagnostic information including position and the type of LVAD, as well as the presence or absence of an ICD or pacemaker (Fig. 4). Deep space infection of the LVAD components requires assessment with computed tomography (CT). If concern for an acute intracerebral event is present, neuroimaging including head CT without contrast and head and neck CT angiography (CTA) is needed in the evaluation of acute ischemic stroke and intracranial hemorrhage [6,19]. Magnetic resonance imaging (MRI) is contraindicated.

Echocardiogram is the imaging modality of choice for most LVAD patients presenting to the ED. Echocardiogram can evaluate cardiac function and assess for complications such as regurgitation, right ventricular failure, and thrombus formation, though thrombi can be difficult to detect on ultrasound alone [4-6,9,13]. Key components of the assessment include valvular function, inflow/outflow abnormalities, ventricular size and function, and septal position.

Laboratory assessment includes hemoglobin/hematocrit, lactate dehydrogenase (LDH), haptoglobin, free hemoglobin, and coagulation panel. Hemoglobin and hematocrit with type and screen/cross are needed if concern of bleeding is present. Patients with LVADs are anticoagulated with a vitamin K antagonist, with a goal INR of 2–3, as

Table 1
LVAD parameters.

Parameter	Device		
	HeartWare	HeartMate II	HeartMate III
Flow (L/min)	4–7	4–8	3–6
Speed (RPM)	2500–3000	8600–9800	5000–6000
Power (W)	3–7	6–7	3–7
Pulsatility	2–4 L/min flow/time waveform pulsatility from peak to trough	4–6	1–4

Abbreviations: L/min – liters per minute, RPM – rotations per minute, W – watts.

well as aspirin [19]. Free hemoglobin and haptoglobin can assess for hemolysis. Elevated LDH >2.5 times the upper limit of normal suggests hemolysis, which is most commonly due to pump thrombosis in an LVAD patient [21-23]. Troponin is recommended in patients with new ECG findings, chest pain, or dyspnea. BNP is a sensitive indicator of volume overload in patients with an LVAD and may be elevated in those with new right heart failure or pump thrombosis or malfunction [4-6,13,14].

Patients should have a controller tag around their waist indicating the type of device, the institution that placed it, and a phone number. Alarms and functional parameters are shown on the external system controller [4-6,9,19]. Pump speed controls flow. Pump power, flow, and speed should be noted, with assessment of alarms and battery. RPMs and pulsatility index must also be evaluated. The pulsatility index depends on the individual patient and device, with an average of flow pulses over 15 s. Hazard alarms indicate poor circulatory support: low flow, pump turn-off or disconnection, low voltage requiring immediate battery replacement or alternate power source, or power loss [9,19].

3.4. Complications

LVAD complications can be categorized via several means. This review will divide complications into those specific to the LVAD and those associated with the LVAD (Table 2).

3.4.1. LVAD-specific complications

LVAD-specific complications center on parameters reported on the controller. These parameters provide important information on the potential underlying conditions, such as a suction event.

A suction event is a common LVAD complication and is associated with low flow events, including dysrhythmia, hemorrhage, and other hypovolemic states such as diarrhea or vomiting [24,25]. Reduced LV preload results in collapse of the LV and decreased inflow into the LVAD. Low flow, speed, and power will be present on the controller [4-6,19]. While bedside US can demonstrate decreased LV volume, this is often difficult in LVAD patients due to poor acoustic windows, and assessment of the LV diameter may assist in evaluating volume status. Treatment requires fluid resuscitation and managing the underlying etiology. With improved preload and intravascular volume, pump speed and flow will improve [19].

Continuous-flow LVADs place patients at high risk of thrombosis, which may originate in the pump or the components such as the inflow or outflow cannula [26-28]. However, current generation continuous flow devices are at much lower risk of thrombus formation [26-28]. Risks of thrombus formation include inadequate anticoagulation, infection, atrial fibrillation, and hypercoagulable states [27,28]. These patients are on chronic anticoagulation, typically with a vitamin K antagonist such as warfarin, and aspirin [9,19]. LVAD thrombosis in the circuit and/or pump may result in increased afterload and low flow and high power alarms on the system controller [4-6]. Types of pump thrombi include acute catastrophic red thrombi entrapped within a fibrin mesh and white thrombi rich in platelets [28]. Red thrombi form at the inlet and outlet areas due to blood stasis, while white thrombi typically form on the pump surface and are associated with turbulent flow [28]. Thrombosis can result in pump dysfunction, hemolysis, emboli, stroke, and death [26-28], but patients with thrombosis present with a variety of symptoms due to these potential complications, ranging from no symptoms to cardiac arrest and death [13,21-23]. Patient

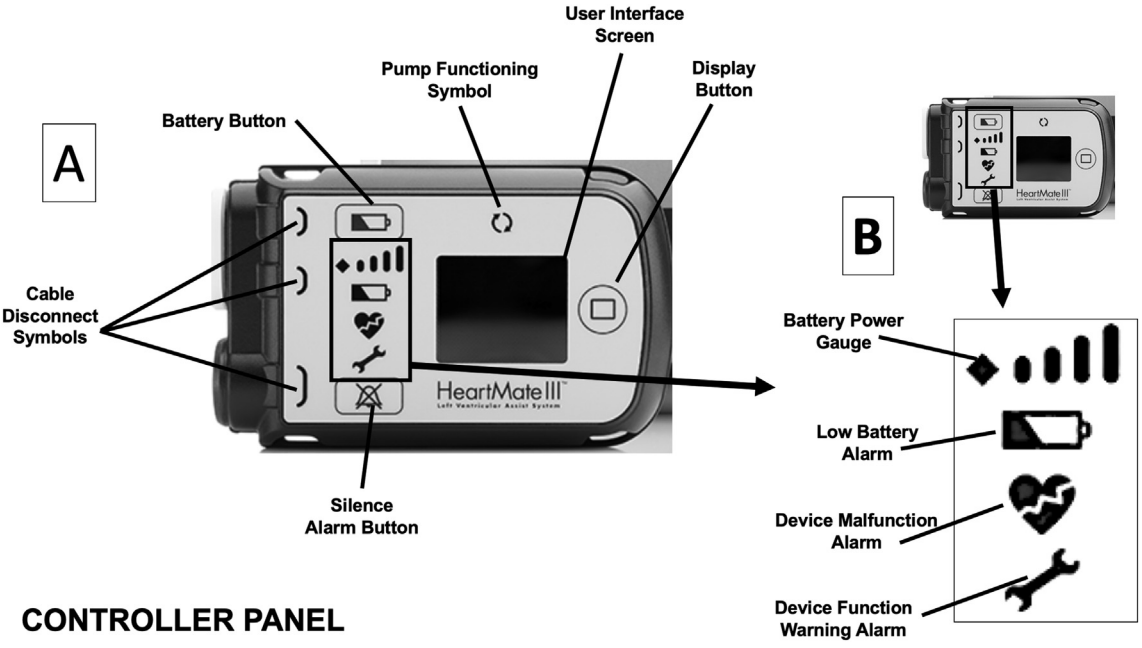

Fig. 3. LVAD controller components (HMIII).

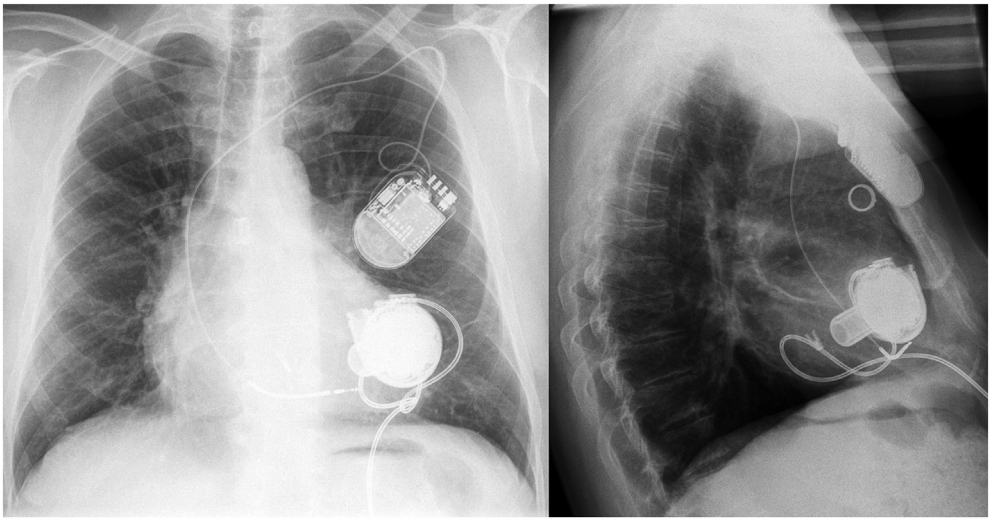

Fig. 4. Chest radiograph of LVAD with ICD. From https://commons.wikimedia.org/wiki/File:Left_ventricular_assist_device_-_56jm_-_Roe_-_001.jpg

presentation may resemble that of massive pulmonary embolism. On examination, evidence of hemolysis may be present with scleral icterus, dark urine, and fatigue. Serum LDH is typically >2.5 times normal. Urinalysis may demonstrate hematuria. Other important laboratory assessments include hemoglobin, free hemoglobin, haptoglobin, and coagulation panel [21-23,29]. Chest radiograph is needed to assess device location and evaluate for any evidence of pump displacement. Other imaging tests include US and computed tomography (CT) in stable patients [30,31]. US may demonstrate inappropriate unloading, while CT may visualize thrombi [6,22]. The controller will demonstrate high power consumption and increased pump speed, but decreased pump flow [6,21-23]. Emergent consultation is recommended with the LVAD specialist, as anticoagulation with heparin and temporary circulatory support should be considered and discussed with the LVAD specialist. Inflow or outflow thrombosis may benefit from endovascular stenting [32-34]. However, severe thrombosis can result in full stoppage of the pump, and stoppage duration that is unknown or lasting over several minutes requires emergent therapy. Full pump stoppage can also occur during a controller exchange or internal wire short. Thrombolysis may be required if patients are hemodynamically unstable [32,35,36]. Emergent surgical pump exchange may be needed if the pump stops, the patient is unstable, or if alarms are present.

Mechanical failure is the second most common cause of death in LVAD patients and may result from several different issues [4,11,32]. Pump failure is the most important life-threatening complication requiring immediate care. The controller may demonstrate low flow, low voltage, and power loss. A low flow alarm should always be evaluated by first checking the power [4-6]. Physicians should auscultate over the LVAD and evaluate for disconnected leads and cannula issues such as kinking or obstruction. A disconnected lead should be reconnected. However, if auscultation reveals no pump activity but all leads are in place, the clinician must assess power and power leads. Complete loss of power should result in the sounding of an alarm, and the physician will be unable to auscultate the motor, blood pressure will be undetectable, and the power light will be absent. If this occurs or if the low voltage alarm is present, a substitute power source or replacement battery should be used. Specific alarms can assist the physician in determining the cause of pump failure. For example, a red heart icon may appear on the HMII/HMIII controller with pump stoppage. If all leads are connected, the pump can be reset. If a power lead is not connected to the batteries or unit cable, the cable disconnect advisory will alarm and demonstrate a flashing symbol [14,37,38]. Power lead connection will result in discontinuation of the alarm. Clinicians must consider that even a short time of noncirculating flow within the LVAD drastically increases the risk of thrombus formation and potential emboli [4-6,14]. If the patient is stable and the device has been off for only several minutes, immediately restarting the device is recommended. However, if the device has been off for over an hour and the patient is stable, consultation with the LVAD specialist is required, as the device should not be immediately restarted due to high risk of thromboembolic evets. In the setting of hemodynamic instability, the device should be restarted immediately no matter the duration of stoppage, with continuous anticoagulation. If the clinician and/or LVAD specialist cannot restart the LVAD, pump exchange is needed, which requires discussion with the LVAD specialist and surgeon. For patients with inadequate perfusion and hemodynamic instability without an alarm activated, resuscitation with IV fluids and standard ACLS protocol is needed.

Table 2
LVAD complications.

LVAD-specific complications	LVAD-associated complications
Suction event	Bleeding
Pump thrombosis	Cerebrovascular pathology: ischemic or hemorrhagic stroke
Pump complications: failure, stoppage, driveline damage	Infection: device-related, device-specific, non-LVAD
	Right ventricular failure
	Dysrhythmia
	Aortic regurgitation

3.4.2. LVAD-associated complications

Patients with LVAD are at elevated bleeding risk, with one study finding rates of bleeding requiring transfusion to be 1.66 and 1.13 events per patient year in early and mid-trial groups, respectively [39]. Bleeding can occur from several sources: pump connections, grafts in the conduits, and most commonly, mucosal surfaces such as the gastrointestinal (GI) tract. GI bleeding affects 15–30% of patients with an LVAD [40,41]. Bleeding in the immediate postoperative period is often due to hepatic congestion associated with severe heart failure and the effects of extracorporeal circulation of the bypass machine [40,41]. After the first post-operative week, several different factors contribute to bleeding. First, patients are on lifelong anticoagulation using a vitamin K antagonist with goal INR of 2.0–3.0 as well as aspirin, though patients with prior thrombosis may have a higher goal INR [19,42,43]. Patients also develop the acquired form of von Willebrand factor (vWF) disease due to the high shear stress associated with LVAD circulation resulting in cleavage and deficiency of vWF [43-50]. Bleeding in elderly patients with acquired vWF is more severe [43,50,51]. The continuous device

Table 3
LVAD conditions and complications

Physiological problem	Considerations and differential	Evaluation	Management
Decreased Preload	Hypovolemia (bleeding, dehydration, septic shock), RV failure, device failure (inflow cannula or mechanical obstruction) - Suction event can occur in this setting	US to evaluate RV function and IVC - Small RV suggests low preload - Small LV suggests suction event - Large RV and small LV suggest RV failure - Large RV and LV suggest pump thrombosis/obstruction	Hypovolemia – Provide IV fluid or blood products if bleeding RV failure – Provide vasopressors/inotropes, pulmonary vasodilators Inflow/mechanical obstruction due to thrombosis - anticoagulate
Increased Afterload	Inability to empty the LVAD chamber: hypertensive event, outflow cannula obstruction (thrombosis/mechanical kink)	Evaluate with US to evaluate ventricular size and IVC MAP >90 mmHg impairs the LVAD's flow ability	Reduce blood pressure with vasodilators to MAP <80 mmHg

VAD complications	Alarm/notification	Considerations	Management
Pump failure	HMII/III: "Low flow – call hospital contact"; Red visual signal HVAD: No auditory or visual alarm	Evaluate connections to battery and the battery itself for appropriate insertion	Emergent LVAD consultation; Reconnect any disconnections and ensure battery is inserted
Power disruption	HMII/III: "Connect power immediately"; Yellow/red battery icon HVAD: "Controller fault/failed", "Change controller", with loud continuous alarm; Yellow flashing signal	Evaluate all connections, including controller-driveline, controller-power supply, and evaluate connections	Emergent LVAD consultation; if connections are intact with charged device, exchange of the device may be required
Driveline damage/electrical fault	HMII/III: "Connect driveline"; Red visual signal HVAD: "VAD stopped", "Connect driveline"; No visual alarm	Driveline contains 6 separate wires with redundancy; evaluate all lines for damage Patient may present in cardiogenic shock	Emergent LVAD specialist consultation; patients with cardiogenic shock require resuscitation with vasopressors and/or inotropes
High flow	High flow alarm	High flow and normal watts suggest infection/sepsis due to peripheral vasodilation	Closely evaluate for sepsis: Treat with antibiotics, may require surgery consultation
Low flow	Low flow alarm: Assess clinical stability and perfusion status Evaluate pump function and components; examine for hypovolemia, thrombosis, dysrhythmia (VT/VF)	Evaluate pump and connections Obtain emergent ECG and US US - Collapsible IVC suggests decreased preload; high RV: LV ratio suggests RV dysfunction Laboratory assessment with hemoglobin, free hemoglobin, haptoglobin, lactate dehydrogenase, urinalysis Obtain IV access bilaterally Thrombus may cause low flow alarm	Treat as pump malfunction if mechanical complication associated with LVAD is found Anticoagulate for thrombosis Vasopressors and/or inotropes may be needed Collapsible IVC on US suggests decreased preload and need for IV fluid resuscitation RV dysfunction may require vasopressors/inotropes, pulmonary vasodilators, and IV fluid resuscitation Cardiovert for unstable dysrhythmia

Table 3 Abbreviations: LVAD – left ventricular assist device, US – ultrasound, LV – left ventricle, RV – right ventricle, IV – intravenous, ECG – electrocardiogram, VT – ventricular tachycardia, VF – ventricular fibrillation, IVC – inferior vena cava, MAP – mean arterial pressure.

flow and decreased pulse pressure also result in formation of arteriovenous malformations, especially in the GI tract [40,41].

Resuscitation of patients with significant hemorrhage with LVAD includes product replacement and reversal agent administration. However, reversing anticoagulation should be weighed with the risk of thrombotic complications, and consultation with the LVAD specialist is recommended. Reversal agents include vitamin K, fresh frozen plasma, and prothrombin complex concentrates. A patient with significant GI bleeding typically requires endoscopy to determine the source and provide control of any lesions, and gastroenterology consultation is recommended [40,41]. Bleeding rates are higher in the upper GI tract, compared to the lower GI tract [51,52]. If upper endoscopy does not reveal a source of hemorrhage, colonoscopy is conducted [40,41]. Lesions are typically treated with coagulation or clips. Due to the risk of sensitization and reducing the success of heart transplant, blood product transfusion should not be reflexive in patients who are hemodynamically stable [40,41,50-52]. Significant transfusion can also increase afterload and exacerbate underlying heart failure. However, hemodynamically unstable patients due to bleeding require resuscitation with blood products. Leukoreduced and irradiated blood products are recommended if available [50]. Octreotide has demonstrated efficacy in LVAD-related GI bleeding in several studies [53-56]. Desmopressin can be provided, which is a synthetic analogue of vasopressin, or infusion of vWF concentrates [50,57]. Discussion of platelet transfusion is needed with the LVAD specialist if the patient is thrombocytopenic and bleeding, as well as those with severe hemorrhage.

Ischemic and hemorrhagic stroke can result in poor outcomes and demonstrate a prevalence of 6.8% and 8.4%, respectively [42,58]. The incidence in the first several months after placement approximates 8–25% [59-61]. While some of these events are clinically silent and discovered on imaging, neurologic events can drastically affect quality of life, with increased debility, loss of independence, further requirements for support, and even compromised candidacy for heart transplantation [61,62]. Stroke is also associated with a 4–18 fold increase in mortality in patients with an LVAD [63]. Patients are at high risk due to thrombus formation with severe heart failure, the greater incidence of atrial

fibrillation, and the presence of a foreign mechanical device [60-63]. High systolic blood pressure, prior stroke, and postoperative infection are the greatest risk factors for a neurologic event [64,65]. A systolic blood pressure >100 mm Hg is associated with over a 2.5-fold higher risk of stroke, with a 19% increase in stroke risk with every 5 mm Hg increase in systolic blood pressure [66]. The ENDURANCE trial found a lower stroke rate with MAP <90 mm Hg, with patients receiving close blood pressure control demonstrating a 24.7% reduction in total neurologic events and 50% decrease in hemorrhagic stroke rate [67]. Acute ischemic stroke more commonly affects the right cerebral hemisphere in patients with an LVAD [64,65]. Stroke risk is decreased by targeting a MAP <90 mm Hg, prophylactic daily aspirin 81 mg, and ensuring INR levels remain within 2.0–3.0 [58,68]. Acute hemorrhagic stroke was the leading cause of death in one study, but this is decreasing [6,7,39]. Cerebral hemorrhage requires immediate blood pressure control with avoidance of hypertension; consultation with the LVAD specialist, neurology, and neurosurgery; and consideration of reversal of anticoagulation. Surgical intervention may be required. For ischemic stroke, thrombolytic therapy has not been widely studied in LVAD patients, and these patients are at high risk for hemorrhagic conversion. A multidisciplinary approach with consideration of endovascular therapy is recommended [69,70].

Patients with an LVAD are at high risk of sepsis, with rates of infection approaching over 42% in the first year post-implant, usually 2 weeks to 2 months [8,11,19,71]. Infection is the third most common cause of death in patients with LVAD within the first year, and infections also increase the risk of thrombosis, stroke, intracerebral hemorrhage, and GI bleeding [42,71]. First generation pulsatile devices had significant rates of bacterial and fungal infection, though continuous flow devices and improved surgical techniques have decreased the infection rate. LVAD infections include those specific to the device, related to the device, and non-LVAD infections [72]. Device-specific infections are those associated with the device components. The driveline and VAD pump pocket are the most common infectious sites, with 80% of driveline infections occurring in the first 30 days of transplant [71-76]. The exit site is an entry site for bacteria. Infection along this site can range from superficial, involving the exit site only, or involve deeper structures within the pump pocket or pump [71,77]. The system controller may demonstrate a high-flow alarm with distributive shock due to loss of vascular tone [78]. LVAD-related infections include those that may occur in patients without an LVAD, but occur with greater frequency in LVAD patients such as mediastinitis, endocarditis, and bacteremia [71]. Non-LVAD infections include pneumonia, *Clostridium difficile* infection, and urinary tract infection (UTI). Within the first 3 months post implantation, the most common sources of infection typically include catheters, pneumonia, and *C. difficile*, while later sources of infection are more commonly related to the device [73,78,79]. Only half of patients will demonstrate fever, leukocytosis, or meet criteria for systemic inflammatory response syndrome [73]. Some patients will demonstrate malaise, pain over the infected site, fever, warmth of the exit site, or drainage from the wound [73,80]. Though a variety of organisms may result in device infection including gram-negative bacteria and *Pseudomonas*, staphylococcal species are the most common organisms found on culture [72,75,80-83]. Candidal infection is associated with mortality reaching 90% [83]. Patients may present with systemic symptoms and severe infection, requiring fluid resuscitation and rapid administration of broad-spectrum antimicrobials [71,72,80-82]. Laboratory assessment includes blood cultures, complete blood cell count, lactic acid, and inflammatory markers, as well as driveline samples (including bacterial and fungal cultures, Gram stain, potassium hydroxide) in those with suspected driveline infection. Chest radiograph is also recommended, but definitive imaging includes CT with contrast to evaluate for deep space infection. Central venous access may be required. Fluid resuscitation is needed in patients with severe toxicity due to sepsis, and except for those with severe right ventricular failure, volume overloading is unlikely. Antibiotics should include coverage for gram-positive and gram-negative species, as well as methicillin-resistant *S. aureus*. Discussion with the LVAD specialist and cardiothoracic surgery is recommended [71-74,80]. Deep infections typically require surgical debridement, while persistent bacteremia may require removal and implantation of a new device [71-74,80].

RV failure is a major cause of morbidity and mortality, occurring in 15–40% of patients [4-6,19]. Late onset right heart failure is increasingly being reported with RV dysfunction, ventricular dysrhythmias, pulmonary hypertension, tricuspid regurgitation, and device thrombosis or malfunction [84]. This can result in reduced preload to the LV, decreasing LVAD flows and triggering a low-flow alarm. RV failure may result in elevated liver function tests, creatinine, and lactic acid. RV failure requires inotropes and/or vasopressors, pulmonary vasodilators, and LVAD specialist consultation [6]. Patients may require careful fluid resuscitation, with 250 mL boluses.

Dysrhythmia can be a significant cause of morbidity and mortality, with 10 episodes for every 100 LVAD patient months in the first 3 months after placement, though this deceases after this period [19]. Patients may tolerate severe ventricular dysrhythmias with minimal symptoms due to the LVAD producing adequate cardiac output to meet end organ perfusion [13,85-90]. Etiologies for dysrhythmia include ischemia, RV failure, electrolyte abnormalities, and electrophysiologic changes after implantation [5,6,85-87]. The greatest risk of ventricular dysrhythmia after LVAD implantation is ventricular dysrhythmia before device placement. Primary dysrhythmias are due to patient intrinsic cardiac physiology, while secondary dysrhythmias are associated with the LVAD [85-87]. The most common secondary causes of dysrhythmia are hypovolemia and poor venous return. Patients often have an ICD prior to LVAD placement. Dysrhythmias may eventually result in compromised blood flow and can also contribute to RV dysfunction, suction events, thrombus formation, and poor perfusion [14,87,91-93]. The controller will demonstrate low flow in patients with hypotension due to the dysrhythmia [14,87,91-93]. If an ICD is not in place, advanced cardiac lifesaving interventions are required with electrical and/or chemical therapy. Consultation with the LVAD specialist is also imperative. With this risk of dysrhythmia, ECG is an integral component of ED evaluation, as well as electrolyte assessment. If hemodynamically unstable, electrical cardioversion is recommended. If the patient is stable with dysrhythmia, fluid resuscitation is recommended with bedside US. Primary dysrhythmias can be managed with antiarrhythmics, though this should be discussed with the LVAD specialist. Patients with an ICD may have received a shock [14,87,91-93]. Physicians should determine if this shock was appropriate or inappropriate [14,87,91]. If the shock was appropriate, evaluation for ischemia and RV dysfunction is needed.

Aortic regurgitation (AR) may develop de novo in up to 25% of patients after LVAD placement [84,94-96]. AR more commonly occurs in patients with a closed aortic valve compared to patients in whom the valve frequently opens [84,94-96]. AR results in decreased LVAD efficacy and may require modifications in pump speed, managed by the LVAD specialist. Patients may require aortic valve replacement.

3.4.3. Resuscitation

Standard procedures for resuscitation are recommended as needed. Hypotension in LVAD patients is defined by MAP <60 mm Hg [4-6,13,14]. Patients who are conscious should be assessed with history and examination, with close assessment of volume and perfusion status. ECG and bedside echocardiogram are vital components of the assessment, with analysis of LVAD components.

Patients who are unresponsive and hypotensive require external chest compressions. Prior recommendations discouraged chest compressions due to fear of dislodging the LVAD. However, literature suggests no cases of dislodgement during cardiopulmonary resuscitation (CPR) [17,18,97]. If the patient has a MAP >50 mm Hg or end tidal CO2 >20 mm Hg with a device possessing an audible hum, perfusion is likely adequate, and compressions are not necessary [17,18,97]. A

MAP <50 mm Hg without an audible hum in the unresponsive patient is associated with compromised perfusion and requires chest compressions at the same depth and frequency as in those without an LVAD [4-6,13,14]. Defibrillation should be performed for unstable ventricular dysrhythmia. The pads should be placed distant from the pump, and if an ICD is present, the pads should not be placed directly over the ICD [4-6,13,14]. In patients with adequate perfusion and respiration but who remain unconscious, evaluate for hypoglycemia, stroke, hypoxia, sedation, and coma.

Chest thoracostomy with chest tube placement in the setting of trauma with pneumothorax and/or hemothorax is recommended, but clinicians must avoid the driveline [4,5]. Arterial line placement can be beneficial, and US guidance is recommended. Pericardiocentesis should be avoided due to risk of serious device complications, but it is recommended in the case of pericardial tamponade with hemodynamic compromise [4-6].

4. Conclusions

An LVAD is a potentially life-saving device in patients with end stage heart failure. Most current LVAD devices provide continuous flow. While LVAD patients may present to the ED with complaints unrelated to their devices, there are many complications that are related to the LVAD, and emergency physicians should be familiar with these complications. These include LVAD-associated complications and LVAD-specific complications. Assessment of LVAD function, patient perfusion, and MAP is needed upon presentation. LVAD evaluation and management require a team-based approach, and consultation with the LVAD specialist is recommended.

Conflicts of interest

None.

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