An Emergency-Medicine Focused Summary of the HFSA/SAEM/ISHLT Clinical Consensus Document on the Emergency Management of Patients with Ventricular Assist Devices

Chad E. Darling, MD,[1] Jennifer L. Martindale, MD,[2] Brian C. Hiestand, MD,[3] Mark Bonnell, MD,[4] Monica Colvin, MD,[5] Ersilia M. DeFilippis, MD,[6] Tonya Elliott, RN, MSN,[7] Eman Hamad, MD,[8] Sean P. Pinney, MD,[9] Keyur B. Shah, MD,[10] Juliane Vierecke, MD,[11] and Michael M. Givertz, MD[6]

From the [1] UMass Memorial Medical Center, UMass Medical School, Worcester, MA; [2]Icahn School of Medicine at Mount Sinai New York, NY; [3]Wake Forest School of Medicine, Winston-Salem, NC; [4]Mclaren Greater Lansing, Lansing, MI; [5]University of Michigan Medical Center, Ann Arbor, MI; [6]Brigham and Women's Hospital, Harvard Medical School, Boston, MA; [7]MedStar Washington Hospital Center, Washington, DC; [8]Temple University Hospital, Philadelphia, PA; [9]Mount Sinai Medical Center, New York, NY; [10]VCU Pauley Heart Center, Richmond, VA; [11]University of Cincinnati College of Medicine, Cincinnati, OH.

Address correspondence to:
Michael M. Givertz, MD, Cardiovascular Division, Brigham and Women's Hospital, 75 Francis Street, Boston, MA 02115, Tel: 617-525-7052, Fax: 617-264-5265, E-mail: mgivertz@bwh.harvard.edu

This is the author manuscript accepted for publication and has undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the Version of Record. Please cite this article as doi: 10.1111/ACEM.13964

Introduction

Mechanical circulatory support is a viable long-term treatment option for patients with end-stage heart failure. As the range of indications for implantation of ventricular assist devices grows, so does the number of patients who live with durable support. These patients are at high risk for medical urgencies and emergencies (**Table 1**). This manuscript is an emergency medicine-focused summary of a consensus document written collaboratively by the Heart Failure Society of America (HFSA), Society for Academic Emergency Medicine (SAEM) and International Society for Heart and Lung Transplantation (ISHLT).[1] It aims to educate emergency medicine and prehospital providers managing patients with left ventricular assist devices (LVAD).

I. Overview of Ventricular Assist Devices

Mechanical circulatory support is a major advance in the treatment of patients with end-stage heart failure.[2] Currently, there are three recognized indications for the use of LVADs: 1) bridge to transplantation (BTT); 2) destination therapy (DT) for patients ineligible for heart transplant; and 3) bridge to myocardial recovery.[3]

Volumes and Survival

According to the eighth annual Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) report, there were a total of 22,866 VAD implants from June 2006 to December 2016. Of these, 18,987 were primary implants for left ventricular support.[4]

LVADs offer superior survival when compared to optimal medical management in patients who are ineligible for cardiac transplantation. For continuous flow devices implanted since 2008, the overall 1-year survival is 81% and 2-year survival is 70%.[5]

LVAD 'Anatomy'

Contemporary LVADs consist of an "inflow" cannula that drains the left ventricle and an "outflow" graft to a central artery – usually the ascending or descending aorta. Other internal components consist of the pump and part (20-30 cm) of the driveline, containing wires that power and control the pump. The driveline typically exits through the upper abdominal wall. External components are a controller and an external power source (**Figure 1**). Most current

LVADs provide continuous flow (CF) through a rotary pump, resulting in blood flow with reduced or no pulsatility.[6] However, the HeartMate 3 (Abbott) is a fully magnetically levitated pump that provides an artificial pulse (once every 2 seconds, the pump modifies its speed) that was approved for BTT in 2017.[7,8]

II. Clinical Aspects Specific to LVAD Patients

Assessing Equipment

While evaluating a patient with an LVAD, one should determine the make and model of the pump. All LVADs have an external driveline connected to a control device and power source. Locate the driveline, which allows the pump to receive information and power to run the LVAD system. This cable should be attached at one end to the implanted pump and connected to the controller.

The dressing on the abdominal wall where the driveline exits the skin should be dry and intact. Patients will either wear an abdominal binder or an anchor device to secure the driveline. Look under binders and dressings to inspect the entirety of the line. The anchor or binder should be kept in place during transport and treatment.

The size and configuration of controllers differ by manufacturer. The controller communicates with the pump, displays pump parameters (e.g., speed, flow, power) and alarms for both advisory and hazardous conditions. The controller accommodates two power sources – either dual batteries or 1 battery plus an AC/DC adapter. Dual batteries can provide anywhere from 8-12 hours of support while patients are active. The percent of charge remaining in each battery can be determined by pushing the button on the top of the battery.

All patients should have an extra controller and batteries as backup equipment. Exchange the primary controller (attached to the patient) for the backup controller only when indicated by controller alarms, such as "Controller Fault, Change Controller" and with the guidance of a provider trained to perform this exchange. Pre-hospital personnel are advised to transport a patient's backup LVAD equipment.

LVAD Parameters

Pump function and flow, displayed on the controller, are the "vital signs" of the LVAD. A typical display shows blood flow (liters per minute), pump rotary speed (rotations per minute),

and power consumption (watts). LVAD flow is calculated based on rotary speed and power consumption and approximates cardiac output.

Device Alarms

The controller has visual and auditory alarms that indicate problems with the pump, controller, connections, or power supply. Address alarms by first looking at the controller and reading the condition. Contacting the implant center or the manufacturer's clinical specialist and/or accessing on-line support are critical for managing alarms.

Anti-thrombotic Therapy

Anti-thrombotic therapy is necessary for all patients with VADs. Typically, patients are maintained on warfarin with an international normalized ratio (INR) target of 2.0-3.0 and aspirin 81-325 mg daily. Some VAD programs use dipyridamole or clopidogrel as additional antiplatelet therapy (for example, in patients with a history of threatened pump thrombosis or transient ischemic attack), but there is no supportive data. Reversal of warfarin with vitamin K, fresh frozen plasma or prothrombin concentrate complex may be considered when treating life threatening bleeding events or preparing for emergent surgery.

Patient Assessment

Vital Signs

The ability to obtain vital signs in LVAD recipients depends on the extent that remaining native ventricular function can generate pulsatile blood flow. For most patients, the absence of a pulse is a normal finding and differences in systolic and diastolic blood pressure are undetectable by automatic and manual sphygmomanometers.[9] A vascular Doppler can be used to assess blood pressure - it is commonly assumed that the first sound heard approximates the mean arterial pressure (MAP), yet studies show this may be closer to the systolic pressure.[10] Regardless, we suggest using the Doppler opening pressure as a surrogate for MAP, with current ISHLT guidelines recommending a mean blood pressure goal of ≤80 mm Hg.[11] Oxygen saturation is another vital sign that might be inaccurate, as pulse oximetry depends on pulsatile flow.[12] Absent a typical pulse oximetry waveform, the result is likely inaccurate. Clinicians must rely on direct

assessments of mental status, perfusion, and general appearance to gauge the clinical condition of LVAD recipients.

Clinical assessment

Important elements of patient history include preceding symptoms (e.g. shortness of breath, chest pain, headache, blood in the urine or stool), the model of their LVAD, and alarm history. Patients may be able to identify their own equipment malfunctions or driveline concerns.

Assessment of airway patency, work of breathing, and adequacy of perfusion are paramount. Clinicians should be able to auscultate a mechanical hum and feel vibrations generated by the LVAD. Beyond initial assessment, clinicians should specifically look for physical signs of heart failure, decreased peripheral perfusion, infection, and blood loss.

III. Emergency Management System Challenges

Field Assessment and Transport

Sending patients that are pulseless and device-dependent into the community requires coordination of the community and pre-hospital providers with the implanting center.[13] As part of the certification process, LVAD centers are required to provide first responder education and coordination plans.[14] Current versions of the field guides for LVADs can be accessed online at https://www.mylvad.com/medical-professionals/resource-library/ems-field-guides.

When LVAD patients are transported to community hospitals, the on-site team should contact the LVAD center as soon as clinically possible. A collaborative course of action can then be implemented that best serves the patient by determining whether the specific problem can be dealt with in a community hospital or requires transfer.

IV. Medical Emergencies in Patients with LVADs

Cardiac Arrest

In patients with continuous flow LVADs, cardiac arrest may be difficult to ascertain clinically, or to differentiate from other conditions resulting in syncope or impaired consciousness. As a result, cardiac arrest resuscitation is often delayed in patients with LVADs compared to other medical patients.[15] In the unconscious patient, the absence of mechanical hum on precordial auscultation, inability to obtain a Doppler signal on manual blood pressure measurement, and cardiac standstill on echocardiography are diagnostic of cardiac arrest. In the

prehospital setting, a patient who is unresponsive, apneic, and in whom a mechanical hum cannot be auscultated, should be assumed to be in cardiac arrest and receive ACLS. Assessment of cardiac rhythm with a portable monitor or ECG is indicated in all patients, although the LVAD may cause significant artifact/interference.

There are varying opinions regarding both efficacy and safety of chest compressions in patients with LVADs, with the debate informed by case series[16] and retrospective cohort studies.[15,17] Primary safety concerns include damage to or dislodgement of the cannula or displacement of the pump, resulting in catastrophic failure and potential intrathoracic exsanguination. Neurologically intact survival without device damage is possible following cardiac arrest in LVAD recipients,[16] and the AHA recommends bystander CPR for LVAD recipients.[18] However, based on the lack of evidence of efficacy and equipment concerns, we do not recommend routine use of mechanical CPR devices.

Unstable Arrhythmias

Arrhythmias, both atrial and ventricular, are common and seldom life-threatening in patients with an LVAD. Ventricular tachycardia (VT) is common and results from underlying cardiomyopathy, RV failure, or mechanical compression of the ventricle by inflow cannula. Because of the degree of physiologic support afforded by the LVAD, patients may complain only of fatigue, nausea, or lightheadedness caused by arrhythmias that would be otherwise fatal.[19] Therefore ECGs should be obtained, even when the patient's chief complaint is not overtly cardiac in nature.

Clinicians treating VT or VF in the LVAD recipient are typically afforded more time to pursue an underlying cause and consider different treatment options. If point of care echocardiography is available, examination of the inflow cannula position within the ventricle may demonstrate the presence of an overly decompressed LV with collapse and marked septal shift, which may trigger arrhythmias due to myocardial irritation. This collapse may respond to decreasing LVAD speed, allowing for increased ventricular filling and migration of the septum away from the inflow cannula. An intravenous fluid bolus can augment preload and limit systolic ventricular collapse against the cannula during systole as a temporizing measure in hypovolemic patients regardless of the degree of their right ventricular dysfunction. **Table 2** summarizes abnormal echocardiographic findings in LVAD emergencies.

Arrhythmias resulting in severe hemodynamic instability should be treated according to standard ACLS protocols. While many patients with LVADs will have an implantable cardioverter-defibrillator (ICD), for those without ICDs, standard cardioversion and defibrillation are not contraindicated and may be performed without disconnection from the device. If possible, the pads should not be placed directly over the device itself – anterior / posterior placement is preferred. Anti-arrhythmic agents such as amiodarone should be considered in the absence of a mechanical cause of the arrhythmia if the patient is hemodynamically stable. However, in refractory or hemodynamically significant ventricular arrhythmias, cardioversion/defibrillation will often be required.[19]

Myocardial Infarction

Acute myocardial infarction (MI) can occur in patients with LVADs due to plaque rupture from underlying coronary artery disease or coronary embolism from ventricular or aortic root thrombus. Aortic root thrombus tends to occur early post-implant often in the setting of a subtherapeutic INR[20] and can be visualized by transesophageal echocardiography (TEE) or CT angiography.[21] Depending on the cause of acute MI, management may include percutaneous coronary intervention, intensification of anticoagulation or aortic root thrombectomy.[22] Since patients are typically fully supported by the LVAD, chest pain may be the only presenting symptom with heart failure being less common.

Unexplained Hypotension

LVAD systems do not have a direct way to measure the amount of blood in the left ventricle, making patients vulnerable to low flow complications and hypotension (defined as a MAP or Doppler opening pressure < 60 mmHg for a CF device). Factors resulting in preload reduction (e.g., hypovolemia, RV failure) lead to suboptimal LV filling, which in turn can cause suboptimal flow or suction in the inflow cannula. The pump will continue to spin with a minimal ability to reduce speed to compensate for decreased volume, and instability can ensue. Multiple conditions, including sepsis, arrhythmia, pulmonary embolism, and hypovolemia can reduce LVAD preload leading to a low-flow alarm (**Table 3**). In addition, pump thrombosis or cannula obstruction can also impair device flow.[14]

Low flow in the device accompanied by increasing central venous pressure can suggest right ventricular failure.[23] In the subacute and chronic settings, RV dysfunction can be due to

ventricular arrhythmias, volume overload, pulmonary embolism (if INR is subtherapeutic), persistent pulmonary hypertension or tricuspid regurgitation.[24] Excessive pump speed and flow can also overwhelm an already compromised right ventricle at any time following LVAD implant. In addition, use of anesthetic agents with negative inotropic properties (e.g., propofol) should be avoided in patients with marginal or reduced RV function. RV failure can lead to hemodynamic deterioration, ICD shocks, and even cardiac arrest with VT/VF caused by impaired filling and inadequate LVAD flow. In all low-flow cases, an echocardiogram should be urgently obtained to assess RV and LV dimensions and filling and rule out tamponade.[14] If the pump stops (see below) and the patient is in cardiogenic shock, vasopressors along with inotropes may be needed to support diminished heart function.

Blunt and Penetrating Trauma

Patients with LVADs should receive the same initial trauma evaluation as any patient[25,26] in concert with VAD system trouble-shooting and cardiac surgery consultation as needed. While current ATLS protocols do not account for mechanical circulatory support, a proposed accessory algorithm is presented (**Figure 2**). There is ample evidence that patients who are anticoagulated are at higher risk following trauma, but there is no consensus regarding the management of anticoagulation in trauma, let alone in patients with LVADs. Invasive blood pressure monitoring should be considered early in the clinical course. If available, the LVAD team should be consulted to ensure proper device function. A standard chest x-ray helps to verify pump position and basic integrity of the driveline, but a targeted x-ray of the driveline should be performed to ensure there has been no break in the wires. If so, the manufacturer representative should be notified immediately as most extracorporeal wire fractures can be safely repaired at bedside.[27]

Bedside echocardiography can evaluate for pericardial effusion in the trauma patient, but formal echocardiography is recommended to identify proper pump placement, disturbances in the blood flow pathway, and abnormal right ventricular (RV) function. In addition to standard laboratory tests, lactate dehydrogenase (LDH) or plasma-free hemoglobin levels should be obtained to assess for hemolysis, which may be clues to more subtle disturbances in the blood flow pathway. Decisions regarding imaging and surgical management should be based on both hemodynamic stability and functional status of the LVAD. If the patient is hemodynamically

stable, they can be closely monitored in a LVAD-capable ICU or step-down unit. If the patient is hemodynamically unstable, one should proceed to surgery and the pump should be assessed for salvageability.

Abdominal Pain

A patient with an LVAD presenting with abdominal pain presents unique challenges. Patients may have tenderness around the driveline or the pump pocket that may mask or mimic other intra-abdominal processes. The presentation of a driveline or pump pocket infection (discussed below) may be mistaken for other diagnoses and must be considered in the differential. Radiologic examination should be guided by clinical judgment. CT maintains its broad utility for abdominal pathology, but artifact from the pump obscures some windows.

Bleeding Complications

Non-surgical bleeding is a common cause of morbidity in patients supported with LVADs. The most common sources of bleeding include gastrointestinal and epistaxis, although intracranial and intrathoracic bleeding may also occur.[28–30] Platelet dysfunction, lysis of the von Willebrand polymer, and RV dysfunction with hepatic congestion all contribute to bleeding complications during VAD support.[31–33] The event rate of bleeding in these patients far exceeds those observed in patients anticoagulated for other reasons.[29]

Gastrointestinal (GI) bleeding occurs in approximately 27% of patients with CF LVADs.[34,35] The most common etiology in these patients is arterio-venous malformations in either the stomach or duodenum. The diagnostic yield of typical endoscopic procedures may be lower since many patients have a small bowel source of bleeding.[36] If bleeding is not identified, push enteroscopy or other methods of evaluating the small bowel are recommended.[34,36] A suggested algorithm for upper/lower GI bleeding in VAD patients has recently been published.[36]

Management of symptomatic GI bleeding is patients with VADs is challenging. Withholding or reversing anticoagulation drugs should be first discussed with the patient's LVAD team. Transfusion may increase pulmonary artery pressures and worsen RV function. In BTT patients, transfusion of leukocyte depleted blood is preferable to reduce the risk of allosensitization. The added benefit of octreotide and thalidomide in patients with

angiodysplastic lesions has not been demonstrated, although some programs have incorporated these agents into management.[37–41]

Epistaxis is the second most common bleeding complication in VAD patients.[29] Management is the same as with any other anticoagulated patient, including topical vasoconstriction, cautery, nasal backing and embolization as needed. Early involvement of otolaryngology is advisable.

Stroke

Cerebrovascular complications remain one of the more common adverse events experienced by patients with LVADs.[42,43] The incidence of stroke is approximately 10% per year [44,45] with the risk for stroke being highest in the early post-operative period as well 9-12 months after implantation.[45,46]

If an acute neurologic deficit develops in a patient with an LVAD, emergent CT of the head with angiography of the head and neck and neurology consultation should be obtained.[14] LVAD parameters should be reviewed for any signs of device malfunction or thrombosis. Hospitals without VAD programs should urgently discuss the clinical situation with the patient's LVAD specialist(s) to determine if urgent transfer is warranted and in hemorrhagic strokes, to discuss discontinuation or reversal of anticoagulation. Reversal targets an INR < 1.5, and prothrombin complex concentrate may be selected over fresh frozen plasma for more rapid effect and to avoid excess volume.[47]

In patients with ischemic strokes, selective use of systemic or intra-arterial thrombolytic agents or an interventional neuroradiologic procedure (e.g., endovascular thrombectomy) may be considered, but in the absence of prospective data, neither is routinely recommended. Case reports of patients with thromboembolic stroke complicated by LVAD thrombosis have demonstrated safe and successful use of systemic thrombolysis.[48]

Infection/Sepsis

A 2011 ISHLT working group standardized the definitions of LVAD infections and classified them into LVAD-specific, LVAD-related and non-LVAD related.[49] Non-LVAD related infections are those not affected by the presence of the LVAD, but happen to occur in a patient with a LVAD such as urinary tract infection or pneumonia. These infections are treated

in the usual fashion. LVAD-related infections refer to infections not involving the LVAD itself, but that can have different characteristics or implications and management when present in a LVAD patient. These include infective endocarditis, bacteremia and mediastinitis.[50] LVAD-specific infections involve the driveline, pocket, pump and/or cannula.

Approximately 14-35% of LVAD patients develop percutaneous driveline infections.[6,51,52] The probability of developing an infection increases approximately 4% for each additional month of LVAD support.[53] Infection of the percutaneous driveline can range from simple cellulitis to abscess formation and deep soft tissue infection. Pocket infections refer to infection of the space housing the pump. CT scan and/or ultrasound should be used to assess for fluid collections around the device. Needle aspiration can be performed, but would best be performed at the patient's primary LVAD center. The most common pathogens leading to device-related infection include Staphylococcus, Enterococcus and Pseudomonas species. Fungal infections are rare,[54] but may be caused by Candida species, with a few case reports of Aspergillus and other molds.

Guidelines for the management of LVAD-associated infections have been proposed, and are based on the type and extent of the infection.[54] Patients with documented device-related infection should be treated with targeted antibiotic therapy for 4-6 weeks and surgical intervention/debridement as needed. Central lines and ICD generators and leads should be removed according to current guidelines.[55,56] LVAD removal or exchange is associated with significant morbidity in addition to the difficulty of removing all infected parts and having a device-free period to allow the antibiotics to take effect before re-implant.

V. LVAD-Specific Emergencies

Pump Thrombosis

Pump thrombosis can occur for a number of reasons including inadequate anticoagulation, poorly controlled blood pressure and inappropriately low pump flow.[57] Lasting increases in pump power by greater than 50% from baseline may indicate thrombosis. Fibrin deposition can create drag, necessitating power increases to maintain pump speed. Transient power spikes or sustained increases in pump power accompanied by inaccurate, elevated flow estimates may be observed. Internal pump thrombosis may produce hemolysis and darkened or bloody urine. Hemolysis may be detected by elevations in serum LDH (> 600 mg/dl or 2.5 times

baseline), elevated free hemoglobin (> 40mg/dl) or reduced haptoglobin levels.[58] Heart failure symptoms are often present, although non-occlusive thrombi can result in significant hemolysis without causing hemodynamic instability.

Pump thrombosis is a catastrophic LVAD emergency. Patients should be transported as soon as possible to the nearest LVAD center where pump exchange or lysis can be performed. Guidelines for the detection and management of suspected thrombosis have been published.[59] Initial management begins by providing adequate levels of systemic anticoagulation, usually with unfractionated heparin or a direct thrombin inhibitor. Published reports of successful treatment with heparin or bivalirudin have led some to consider these interventions before considering surgery.[60] Tissue plasminogen activator (TPA) use has been reported with mixed results,[61,62] and we recommend against routine use. Pathologic studies have demonstrated that the clot is highly organized and unlikely to respond to thrombin breakdown, and therefore TPA should only be considered after contacting the implanting center. Although medical treatment with anticoagulant agents or fibrinolytic therapy can lead to clot resolution, the rate of recurrence is high.[63]

Pump Stoppage or Failure, Driveline Trauma

A little more than 3.5% of all deaths on LVAD support can be attributed to device malfunction.[64] Failures can occur in the internal or external portion of the driveline, patient cable, pump controller or with external power.

Pump stoppage occurs when there is a complete loss of power to the pump. This can arise due to depletion of battery power, disconnection of both power leads, or disconnection of the percutaneous lead from the controller. A constant, high-pitched alarm sounds and a "red heart" accompanies it on the pocket controller. This is an unstable situation that leads to severe regurgitation of blood from the aorta into the LV, as the pump does not contain valves to prevent retrograde flow. Patients will present with symptoms of acute heart failure. The pump will remain silent upon chest auscultation. Treatment requires restoring power to the pump even though doing so in patients with subtherapeutic anticoagulation risks thromboembolism and stroke.

Driveline trauma may result in pump stoppages. Repetitive flexing and bending of the percutaneous lead may result in short circuits that may temporarily or permanently stop the pump

from rotating, accompanied by a constant alarm.[65] Temporary or more durable repairs can sometimes be performed by an industry engineer specific to each device by soldering together disrupted wires depending on the location of the fracture (internal vs. external).[27] Complete transection of the driveline will require urgent surgical pump replacement.

Heart Failure with a Left Ventricular Assist Device

Patients implanted with LVADs may present for emergency care of acute heart failure. Typically they present with sub-acute progression of symptoms or, less frequently, with sudden decompensation requiring emergent intervention. Several potential factors can reduce LVAD flow leading to congestion and a low cardiac output state.[24]

Inadequate Left Ventricular Decompression

Inadequate decompression of the LV can lead to heart failure in LVAD patients. Causative factors include: (1) suboptimal pump speed setting; (2) increased afterload; (3) obstruction to blood flow; (4) thrombosis of the pump impeller; or (5) aortic insufficiency. When the LVAD fails to adequately decrease LV volumes, imaging may reveal LV dilation, functional mitral valve regurgitation and frequent aortic valve opening. Patients often experience clinical symptoms of fatigue and dyspnea, and may have signs of congestion on exam. Patients with subacute symptoms presenting to non-LVAD centers may require transfer for speed adjustments, diuresis and antihypertensive therapies.

Increased afterload is most frequently caused by poorly controlled blood pressure, which results in reduced pump output. The monitor will display decreased power consumption and low flow estimates. Extreme blood pressure increases can obliterate flow through the device, increasing the risk of cerebrovascular accidents and pump thrombosis.

Kinks or obstruction to inflow (sudden or gradual) or outflow cannula (gradual) can lead to heart failure with low power consumption and estimated flow displayed on the LVAD controller or monitor. Inflow cannula obstruction reduces preload to the LVAD and may occur gradually due to pannus formation or misalignment of the cannula due to LV remodeling or orientation of the pump pocket (i.e., changing abdominal girth). Abrupt cannula obstruction may present as a medical emergency with sudden heart failure, syncope or shock.[66,67]

Summary and Future Directions

A growing number of adults with end-stage heart failure are living in the community with mechanical circulatory support. Like all community dwellers, patients with LVADs may have urgent or emergent medical needs requiring hospital level care. This consensus document by HFSA, SAEM and ISHLT aims to provide emergency and prehospital providers with information needed to understand the basics of continuous flow pumps and how to handle patients and their equipment in emergency situations. More detailed information on specific devices is available online at manufacturers' websites or by contacting their clinical specialists. A list of LVAD implanting centers in the US and Canada can also be found on line at www.uab.edu/medicine/intermacs/enroll/currently-enrolled. Future studies will focus on management of device settings, fluid resuscitation, and anticoagulation around non-cardiac surgery and trauma. The extrapolation of these emergency management guidelines to a small, but emerging population of pediatric patients living at home with LVADs will also need to be explored.

References

1. Givertz MM, DeFilippis EM, Colvin M, et al. HFSA/SAEM/ISHLT Clinical Expert Consensus Document on the Emergency Management of Patients with Ventricular Assist Devices. J Card Fail 2019;25(7):494–515.

2. Stewart GC, Givertz MM. Mechanical circulatory support for advanced heart failure: patients and technology in evolution. Circulation 2012;125(10):1304–15.

3. Miller LW, Guglin M. Patient Selection for Ventricular Assist DevicesA Moving Target. J Am Coll Cardiol 2013;61(12):1209–21.

4. Kirklin JK, Pagani FD, Kormos RL, et al. Eighth annual INTERMACS report: Special focus on framing the impact of adverse events. J Heart Lung Transplant Off Publ Int Soc Heart Transplant 2017;36(10):1080–6.

5. Smith LA, Yarboro LT, Kennedy JLW. Left ventricular assist device implantation strategies and outcomes. J Thorac Dis 2015;7(12):2088–96.

6. Slaughter MS, Rogers JG, Milano CA, et al. Advanced heart failure treated with continuous-flow left ventricular assist device. N Engl J Med 2009;361(23):2241–51.

7. Mehra MR, Naka Y, Uriel N, et al. A Fully Magnetically Levitated Circulatory Pump for Advanced Heart Failure. N Engl J Med 2017;376(5):440–50.

8. Uriel N, Colombo PC, Cleveland JC, et al. Hemocompatibility-Related Outcomes in the MOMENTUM 3 Trial at 6 Months: A Randomized Controlled Study of a Fully Magnetically Levitated Pump in Advanced Heart Failure. Circulation 2017;135(21):2003–12.

9. Myers TJ, Bolmers M, Gregoric ID, Kar B, Frazier OHH. Assessment of arterial blood pressure during support with an axial flow left ventricular assist device. J Heart Lung Transplant Off Publ Int Soc Heart Transplant 2009;28(5):423–7.

10. Lanier GM, Orlanes K, Hayashi Y, et al. Validity and reliability of a novel slow cuff-deflation system for noninvasive blood pressure monitoring in patients with continuous-flow left ventricular assist device. Circ Heart Fail 2013;6(5):1005–12.

11. Feldman D, Pamboukian SV, Teuteberg JJ, et al. The 2013 International Society for Heart and Lung Transplantation Guidelines for mechanical circulatory support: executive summary. J Heart Lung Transplant Off Publ Int Soc Heart Transplant 2013;32(2):157–87.

12. Oleyar M, Stone M, Neustein SM. Perioperative management of a patient with a nonpulsatile left ventricular-assist device presenting for noncardiac surgery. J Cardiothorac Vasc Anesth 2010;24(5):820–3.

13. Cook JL, Colvin M, Francis GS, et al. Recommendations for the Use of Mechanical Circulatory Support: Ambulatory and Community Patient Care: A Scientific Statement From the American Heart Association. Circulation 2017;135(25):e1145–58.

14. Feldman D, Pamboukian SV, Teuteberg JJ, et al. The 2013 International Society for Heart and Lung Transplantation Guidelines for mechanical circulatory support: executive summary. J Heart Lung Transplant Off Publ Int Soc Heart Transplant 2013;32(2):157–87.

15. Garg S, Ayers CR, Fitzsimmons C, et al. In-hospital cardiopulmonary arrests in patients with left ventricular assist devices. J Card Fail 2014;20(12):899–904.

16. Mabvuure NT, Rodrigues JN. External cardiac compression during cardiopulmonary resuscitation of patients with left ventricular assist devices. Interact Cardiovasc Thorac Surg 2014;19(2):286–9.

17. Collins SP, Levy PD, Lindsell CJ, et al. The rationale for an acute heart failure syndromes clinical trials network. J Card Fail 2009;15(6):467–74.

18. Peberdy MA, Gluck JA, Ornato JP, et al. Cardiopulmonary Resuscitation in Adults and Children With Mechanical Circulatory Support: A Scientific Statement From the American Heart Association. Circulation 2017;135(24):e1115–34.

19. Raasch H, Jensen BC, Chang PP, et al. Epidemiology, management, and outcomes of sustained ventricular arrhythmias after continuous-flow left ventricular assist device implantation. Am Heart J 2012;164(3):373–8.

20. Fried J, Garan AR, Shames S, et al. Aortic root thrombosis in patients supported with continuous-flow left ventricular assist devices. J Heart Lung Transplant Off Publ Int Soc Heart Transplant 2018;37(12):1425–32.

21. Tanna MS, Reyentovich A, Balsam LB, et al. Aortic root thrombus complicated by left main coronary artery occlusion visualized by 3D echocardiography in a patient with continuous-flow left ventricular assist device. Echocardiogr Mt Kisco N 2017;34(2):306–10.

22. Spina R, Siriwardena M, Bart N, et al. Primary percutaneous coronary intervention for inferior ST-segment elevation myocardial infarction in a patient supported by the HeartWare left ventricular assist device. Intern Med J 2017;47(9):1068–71.

23. Dang NC, Topkara VK, Mercando M, et al. Right heart failure after left ventricular assist device implantation in patients with chronic congestive heart failure. J Heart Lung Transplant Off Publ Int Soc Heart Transplant 2006;25(1):1–6.

24. Burke MA, Givertz MM. Assessment and management of heart failure after left ventricular assist device implantation. Circulation 2014;129(10):1161–6.

25. ATLS Subcommittee, American College of Surgeons' Committee on Trauma, International ATLS working group. Advanced trauma life support (ATLS®): the ninth edition. J Trauma Acute Care Surg 2013;74(5):1363–6.

453 26. Hussmann B, Lendemans S. Pre-hospital and early in-hospital management of severe injuries:
454 changes and trends. Injury 2014;45 Suppl 3:S39-42.

455 27. Stulak JM, Schettle S, Haglund N, et al. Percutaneous Driveline Fracture Following Implantation
456 of the HeartMate II Left Ventricular Assist Device: How Durable is Driveline Repair? ASAIO J Am Soc Artif
457 Intern Organs 1992 2017;

458 28. Crow S, John R, Boyle A, et al. Gastrointestinal bleeding rates in recipients of nonpulsatile and
459 pulsatile left ventricular assist devices. J Thorac Cardiovasc Surg 2009;137(1):208–15.

460 29. Wever-Pinzon O, Selzman CH, Drakos SG, et al. Pulsatility and the risk of nonsurgical bleeding in
461 patients supported with the continuous-flow left ventricular assist device HeartMate II. Circ Heart Fail
462 2013;6(3):517–26.

463 30. Nascimbene A, Neelamegham S, Frazier OH, Moake JL, Dong J-F. Acquired von Willebrand
464 syndrome associated with left ventricular assist device. Blood 2016;127(25):3133–41.

465 31. Uriel N, Pak S-W, Jorde UP, et al. Acquired von Willebrand syndrome after continuous-flow
466 mechanical device support contributes to a high prevalence of bleeding during long-term support and at
467 the time of transplantation. J Am Coll Cardiol 2010;56(15):1207–13.

468 32. Crow S, Chen D, Milano C, et al. Acquired von Willebrand syndrome in continuous-flow
469 ventricular assist device recipients. Ann Thorac Surg 2010;90(4):1263–9; discussion 1269.

470 33. Sparrow CT, Nassif ME, Raymer DS, Novak E, LaRue SJ, Schilling JD. Pre-Operative Right
471 Ventricular Dysfunction Is Associated With Gastrointestinal Bleeding in Patients Supported With
472 Continuous-Flow Left Ventricular Assist Devices. JACC Heart Fail 2015;3(12):956–64.

473 34. Draper KV, Huang RJ, Gerson LB. GI bleeding in patients with continuous-flow left ventricular
474 assist devices: a systematic review and meta-analysis. Gastrointest Endosc 2014;80(3):435-446.e1.

475 35. Mehra MR, Goldstein DJ, Uriel N, et al. Two-Year Outcomes with a Magnetically Levitated
476 Cardiac Pump in Heart Failure. N Engl J Med 2018;378(15):1386–95.

477 36. Axelrad JE, Pinsino A, Trinh PN, et al. Limited usefulness of endoscopic evaluation in patients
478 with continuous-flow left ventricular assist devices and gastrointestinal bleeding. J Heart Lung
479 Transplant 2018;37(6):723–32.

480 37. Aggarwal A, Pant R, Kumar S, et al. Incidence and management of gastrointestinal bleeding with
481 continuous flow assist devices. Ann Thorac Surg 2012;93(5):1534–40.

482 38. Demirozu ZT, Radovancevic R, Hochman LF, et al. Arteriovenous malformation and
483 gastrointestinal bleeding in patients with the HeartMate II left ventricular assist device. J Heart Lung
484 Transplant Off Publ Int Soc Heart Transplant 2011;30(8):849–53.

485 39. Hayes HM, Dembo LG, Larbalestier R, O'Driscoll G. Management options to treat gastrointestinal
486 bleeding in patients supported on rotary left ventricular assist devices: a single-center experience. Artif
487 Organs 2010;34(9):703–6.

488 40. Draper K, Kale P, Martin B, Cordero K, Ha R, Banerjee D. Thalidomide for treatment of
489 gastrointestinal angiodysplasia in patients with left ventricular assist devices: case series and treatment
490 protocol. J Heart Lung Transplant Off Publ Int Soc Heart Transplant 2015;34(1):132–4.

491 41. Ray R, Kale PP, Ha R, Banerjee D. Treatment of left ventricular assist device-associated
492 arteriovenous malformations with thalidomide. ASAIO J Am Soc Artif Intern Organs 1992
493 2014;60(4):482–3.

494 42. Acharya D, Loyaga-Rendon R, Morgan CJ, et al. INTERMACS Analysis of Stroke During Support
495 With Continuous-Flow Left Ventricular Assist Devices: Risk Factors and Outcomes. JACC Heart Fail
496 2017;5(10):703–11.

497 43. Parikh NS, Cool J, Karas MG, Boehme AK, Kamel H. Stroke Risk and Mortality in Patients With
498 Ventricular Assist Devices. Stroke 2016;47(11):2702–6.

499 44. Harvey L, Holley C, Roy SS, et al. Stroke After Left Ventricular Assist Device Implantation:
500 Outcomes in the Continuous-Flow Era. Ann Thorac Surg 2015;100(2):535–41.

501 45. Kirklin JK, Pagani FD, Kormos RL, et al. Eighth annual INTERMACS report: Special focus on
502 framing the impact of adverse events. J Heart Lung Transplant Off Publ Int Soc Heart Transplant
503 2017;36(10):1080–6.

504 46. Frontera JA, Starling R, Cho S-M, et al. Risk factors, mortality, and timing of ischemic and
505 hemorrhagic stroke with left ventricular assist devices. J Heart Lung Transplant Off Publ Int Soc Heart
506 Transplant 2016;

47. Godoy DA, Piñero GR, Koller P, Masotti L, Di Napoli M. Steps to consider in the approach and management of critically ill patient with spontaneous intracerebral hemorrhage. World J Crit Care Med 2015;4(3):213–29.

48. Krstic A, O'Carroll C, Lanza L, Steidley DE, Staley L, Cartin-Ceba R. 1276: SIMULTANEOUS LVAD THROMBOSIS AND ISCHEMIC STROKE TREATED SUCCESSFULLY WITH ALTEPLASE. Crit Care Med 2015;43:321.

49. Hannan MM, Husain S, Mattner F, et al. Working formulation for the standardization of definitions of infections in patients using ventricular assist devices. J Heart Lung Transplant Off Publ Int Soc Heart Transplant 2011;30(4):375–84.

50. Monkowski DH, Axelrod P, Fekete T, Hollander T, Furukawa S, Samuel R. Infections associated with ventricular assist devices: epidemiology and effect on prognosis after transplantation. Transpl Infect Dis Off J Transplant Soc 2007;9(2):114–20.

51. Miller LW, Pagani FD, Russell SD, et al. Use of a continuous-flow device in patients awaiting heart transplantation. N Engl J Med 2007;357(9):885–96.

52. Pagani FD, Miller LW, Russell SD, et al. Extended mechanical circulatory support with a continuous-flow rotary left ventricular assist device. J Am Coll Cardiol 2009;54(4):312–21.

53. Sharma V, Deo SV, Stulak JM, et al. Driveline infections in left ventricular assist devices: implications for destination therapy. Ann Thorac Surg 2012;94(5):1381–6.

54. Nienaber JJC, Kusne S, Riaz T, et al. Clinical manifestations and management of left ventricular assist device-associated infections. Clin Infect Dis Off Publ Infect Dis Soc Am 2013;57(10):1438–48.

55. Baddour LM, Wilson WR, Bayer AS, et al. Infective endocarditis: diagnosis, antimicrobial therapy, and management of complications: a statement for healthcare professionals from the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease, Council on Cardiovascular Disease in the Young, and the Councils on Clinical Cardiology, Stroke, and Cardiovascular Surgery and Anesthesia, American Heart Association: endorsed by the Infectious Diseases Society of America. Circulation 2005;111(23):e394-434.

56. Sandoe JAT, Barlow G, Chambers JB, et al. Guidelines for the diagnosis, prevention and management of implantable cardiac electronic device infection. Report of a joint Working Party project

on behalf of the British Society for Antimicrobial Chemotherapy (BSAC, host organization), British Heart Rhythm Society (BHRS), British Cardiovascular Society (BCS), British Heart Valve Society (BHVS) and British Society for Echocardiography (BSE). J Antimicrob Chemother 2015;70(2):325–59.

57. Mehra MR, Stewart GC, Uber PA. The vexing problem of thrombosis in long-term mechanical circulatory support. J Heart Lung Transplant Off Publ Int Soc Heart Transplant 2014;33(1):1–11.

58. Tchantchaleishvili V, Sagebin F, Ross RE, Hallinan W, Schwarz KQ, Massey HT. Evaluation and treatment of pump thrombosis and hemolysis. Ann Cardiothorac Surg 2014;3(5):490–5.

59. Goldstein DJ, John R, Salerno C, et al. Algorithm for the diagnosis and management of suspected pump thrombus. J Heart Lung Transplant Off Publ Int Soc Heart Transplant 2013;32(7):667–70.

60. Sylvia LM, Ordway L, Pham DT, DeNofrio D, Kiernan M. Bivalirudin for treatment of LVAD thrombosis: a case series. ASAIO J Am Soc Artif Intern Organs 1992 2014;60(6):744–7.

61. Godown J, Dodd DA, Doyle TP, Smith AH, Janssen D, Mettler BA. Tissue plasminogen activator treatment of bilateral pulmonary emboli in a pediatric patient supported with a ventricular assist device. Pediatr Transplant 2015;19(7):E160-164.

62. Sorensen EN, Carla Williams P, Tabatabai A. Use of tissue plasminogen activator to resolve high purge system pressure in a catheter-based ventricular-assist device. J Heart Lung Transplant Off Publ Int Soc Heart Transplant 2014;33(4):457–8.

63. Upshaw JN, Kiernan MS, Morine KJ, Kapur NK, DeNofrio D. Incidence, Management, and Outcome of Suspected Continuous-Flow Left Ventricular Assist Device Thrombosis. ASAIO J Am Soc Artif Intern Organs 1992 2016;62(1):33–9.

64. Kirklin JK, Naftel DC, Pagani FD, et al. Seventh INTERMACS annual report: 15,000 patients and counting. J Heart Lung Transplant Off Publ Int Soc Heart Transplant 2015;34(12):1495–504.

65. Wever-Pinzon O, Givens RC, Flannery M, Naka Y, Jorde UP. Repetitive HeartMate II pump stoppage induced by transitioning from battery to main power source: the short-to-shield phenomenon. J Heart Lung Transplant Off Publ Int Soc Heart Transplant 2015;34(2):270–1.

66. Kornberger A, Stock UA, Risteski P, Beiras Fernandez A. Left ventricular non-compaction cardiomyopathy and left ventricular assist device: a word of caution. J Cardiothorac Surg 2016;11(1):108.

67. Abraham J, Remick JD, Caulfield T, et al. Left ventricular assist device outflow cannula obstruction treated with percutaneous endovascular stenting. Circ Heart Fail 2015;8(1):229–30.

Figure Legends

Figure 1. Components of a Typical LVAD. A continuous flow left ventricular assist device consists of a pump connected to the cardiac apex and ascending aorta via an inflow cannula and outflow graft, respectively, a percutaneous driveline that exits the skin on the right, and a system controller that is typically worn on a belt. Power to the controller and pump is provided by external batteries or a power-based unit. Adapted from Mehra et al.[7] with permission.

Figure 2. Proposed Algorithm for Management of Trauma in a Patient with an LVAD

VAD Specific Emergencies	VAD Related Emergencies	VAD Unrelated Emergencies
Heart failure	Arrhythmias	Abdominal pain
Left heart failure	Atrial fibrillation	Blunt and penetrating trauma
Right heart failure	Ventricular tachycardia	Burns
Mechanical failure	Ventricular fibrillation	Hypovolemia
Driveline	Bleeding	Infection
Pump stoppage	Epistaxis	Sepsis
Pump thrombosis	Gastrointestinal	
	Cardiac arrest	
	Cardiac tamponade	
	Infection	
	Driveline	
	Pump pocket	
	Stroke	
	Hemorrhagic	
	Ischemic	

	LV Diameter	Aortic Valve Opening	Mitral Regurgitation	IVC diameter	Septal Position	Comments
Inappropriately Low Speed	↑*	-/↑	↑	-/↑	Rightward	Ramping speed leads to LV decompression and AV closure
Pump Thrombosis	↑	↑	↑	-/↑	Rightward	Ramping speed does not result in expected LV decompression or change in AV opening
Pump Stoppage	↑	↑	↑	↑	Rightward	Reversal of flow through inflow and outflow cannulae may be demonstrated
Aortic Insufficiency	↑	-	↑	-/↑	Rightward	"Moderate" AI by color flow mapping can be hemodynamically significant
RV Failure	↓	↓	↓	↑	Leftward	Increased RV dimensions may be associated with moderate-severe TR
Hypovolemia	↓	↓	↓	↓	Leftward	

*Increase or decrease relative to baseline study obtained when patient clinically stable. AI, aortic insufficiency; AV, aortic valve; IVC, inferior vena cava; LV, left ventricular; RV, right ventricular; TR, tricuspid regurgitation

Cause	Signs	Intervention
Bleeding (gastrointestinal, nasal and cerebral hemorrhage)	Low hemoglobin, hematocrit and platelet count ± elevated INR + Stool guaiac Low Flow	Bolus IV fluids, transfusion Hold or reverse anticoagulation Identify and treat bleeding source
Dehydration (infection, vomiting, diuretics, poor oral intake)	Low JVP Low Flow / Low PI ± suction	IV fluids, hold diuretics ± decrease VAD speed temporarily to avoid suction Identify etiology and treat
Right heart failure (tamponade/effusions, PE, cannula position)	High JVP Low Flow	Echo (consider RHC) Initiate inotropic support if RV failure PDE-5 inhibitors may be considered if PH present
Inadequate LVAD speed	High JVP Low Flow	Echo and RHC Inadequate unloading by LVAD: high PCWP, low output Adjust pump speed
Arrhythmia	Obtain rhythm strip immediately Sudden cardiac arrest is difficult to define as VAD patients can be awake while in VF Low Flow ± suction	Use ACLS guidelines to treat arrhythmia
Mechanical obstruction/thrombus	Elevated LDH and plasma free hemoglobin, dark urine Low Flow ± power spike	Echo CTA to evaluate inflow and out flow cannulae

		Optimize anticoagulation
Sepsis (driveline exit site, indwelling catheters or home IVs)	Elevated WBC, fever High Flow due to low SVR	Hold vasodilators Add pressor support Identify source and treat

ACLS, advanced cardiac life support; CTA, computed tomographic angiography; INR, international normalized ratio; IV, intravenous; JVP, jugular venous pressure; LDH, lactate dehydrogenase; LVAD, left ventricular assist device; MAP, mean arterial pressure; PCWP, pulmonary capillary wedge pressure; PH, pulmonary hypertension; RHC, right heart catheterization; SVR, systemic vascular resistance; VF, ventricular fibrillation; WBC, white blood cell count.

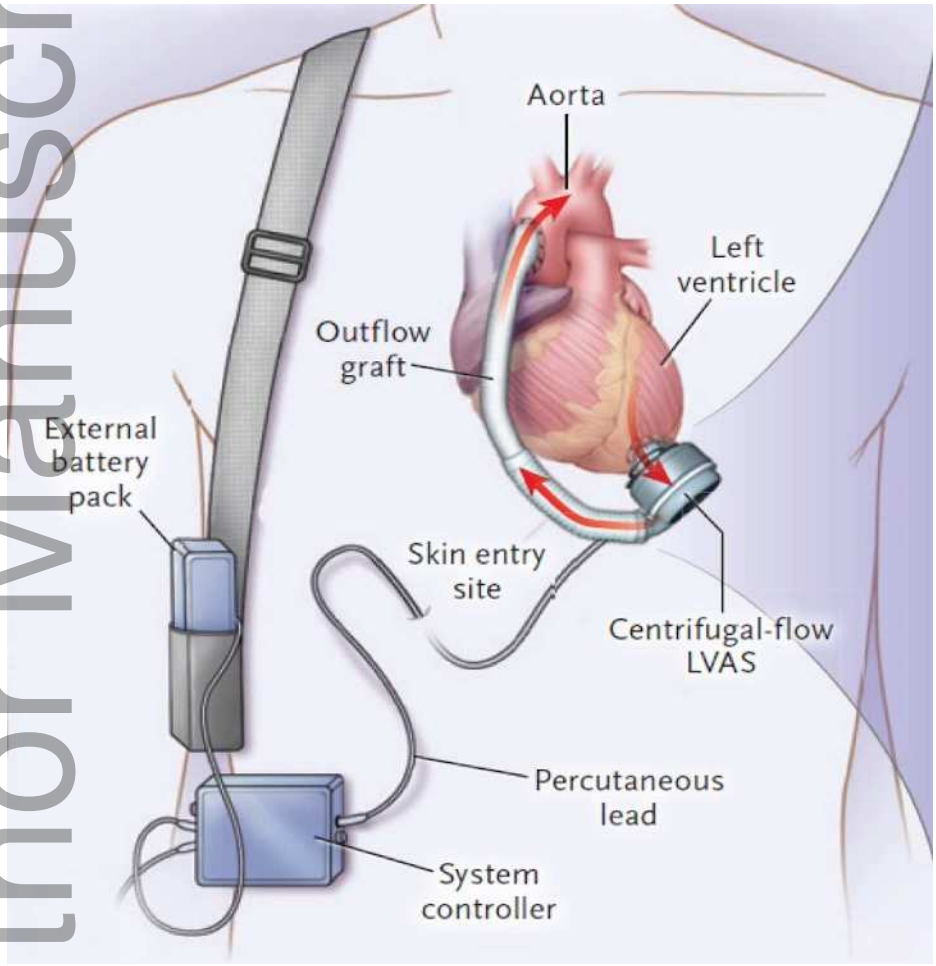

Figure 1

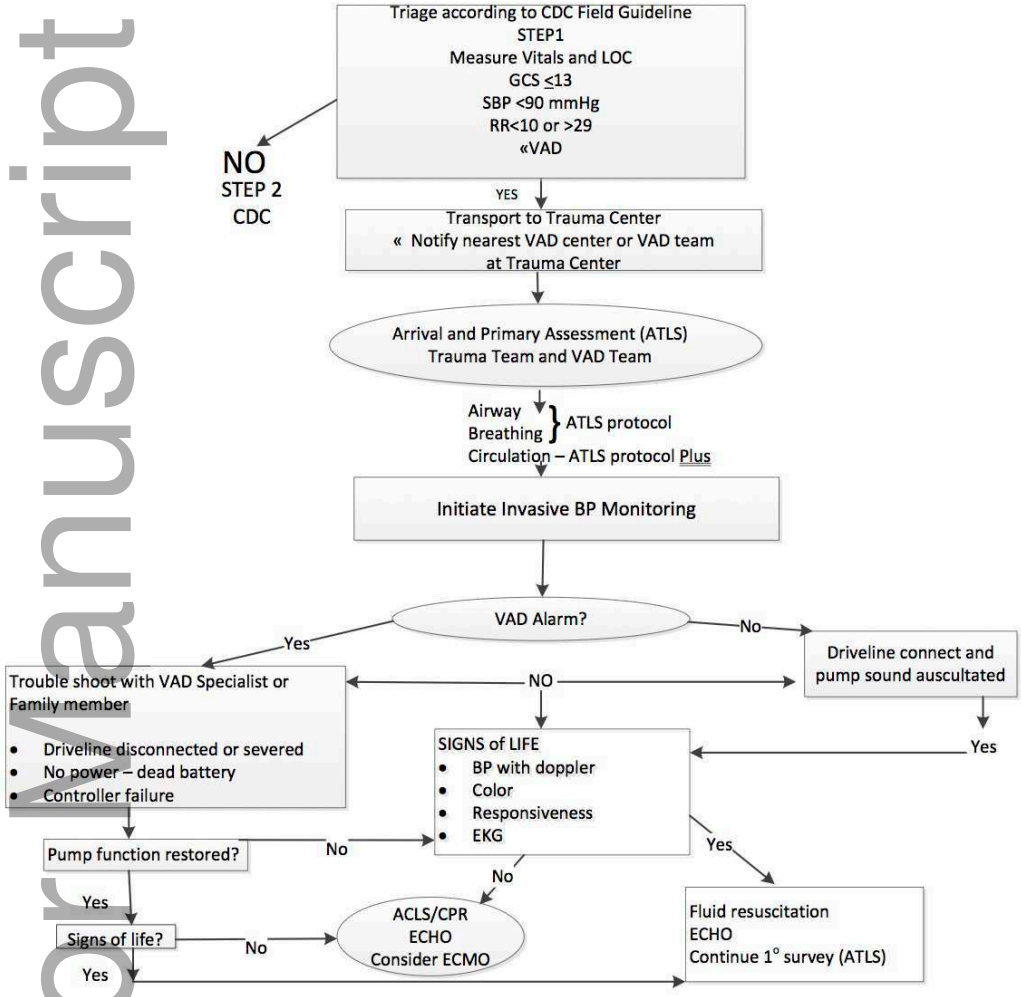

Figure 2