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REVIEW ARTICLE

Approach to patients with left ventricular assist device and its complications at the emergency department

Atención del paciente con dispositivos de asistencia ventricular izquierda y sus complicaciones en el servicio de urgencias

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Abstract

Advanced heart failure is becoming more frequent and, in this population, left ventricular assist devices have emerged in the last thirty years as bridge-to-transplant or destination therapy for those with a contraindication for transplant. These devices are continuous flow, contrary to the normal hemodynamic physiology, which leads to associated complications with the interaction between the device and the patient, a phenomenon known as "hemocompatibility." One of its implications is the absence of a central pulse and the need to measure blood pressure through uncommon methods such as Doppler. Various complications are frequently associated with this and can be grouped under the mnemonic $A_2B_2C_2D_2$, in Spanish including those specific and related to the device. Each of these must be recognized for early and prompt diagnosis and treatment, including prompt cardiopulmonary resuscitation, if indicated, to impact the survival of these patients.

Keywords: *Ventricular assist device. Heart failure. Complications. Pump. Cardiopulmonary resuscitation.*

Resumen

La falla cardiaca avanzada es cada vez más frecuente. En esta población, los dispositivos de asistencia ventricular izquierda han emergido en los últimos treinta años como una terapia puente al trasplante o definitiva en aquellos con contraindicación para este. Estos dispositivos son de flujo continuo, contrario a la fisiología hemodinámica normal, fenómeno que conlleva una serie de complicaciones asociadas a la interacción entre el dispositivo y el paciente, la cual se conoce como "hemocompatibilidad". Una de sus implicaciones es la ausencia de pulso central y la necesidad de toma de presión arterial a través de métodos poco frecuentes, como el Doppler. Las diferentes complicaciones asociadas a este son frecuentes y pueden reunirse bajo la mnemotecnia $A_2B_2C_2D_2$, incluyendo aquellas específicas y relacionadas con el dispositivo. Es necesario reconocer cada una de estas para el diagnóstico y tratamiento tempranos y oportunos, incluyendo el inicio precoz de maniobras de reanimación en caso de estar indicado, para impactar la supervivencia de estos pacientes.

Palabras clave: *Dispositivo de asistencia ventricular. Falla cardiaca. Complicaciones. Bomba. Reanimación cardiopulmonar.*

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Introduction

The prevalence of advanced heart failure has been growing and accounts for up to 10% of all patients with heart failure[1]. Previously, the only definitive treatment for these patients was heart transplantation but left ventricular assist devices (better known as LVADs) have emerged in the last 30 years as an alternative for patients waiting for transplant, and even as definitive or palliative treatment in patients for whom transplantation is contraindicated[2]. However, this is not an innocuous treatment, and it is estimated that approximately 70% will require hospital readmission within a year of implantation[3]. These devices are a reality in Colombia, and based on the premise of more than 40,000 people having advanced heart failure in the country, the number of possible candidates is not negligible and will continue to grow, as will the frequency of their visits to the emergency room[4]. For all of these reasons, emergency room staff must be trained on these devices and be familiar with the diagnosis and treatment of their main complications. In response to this, we present below a description of LVADs, their complications, handling and treatment in this setting.

The history of left ventricular assist devices

In 1953, John Gibbon began the era of mechanical circulatory support by finishing the first open heart surgery successfully using a cardiopulmonary bypass machine in an 18-year-old woman[5]. In 1963, Michael DeBakey and Domingo Liotta implanted a paracorporeal device for the first time in a patient by the name of George Washington, after aortic valve replacement; however, this patient died four days later[6,7]. The advent of these techniques brought along the challenge of supporting patients who could not be taken off the pump after cardiac surgeries. This is where the idea of providing prolonged ventricular assistance was born, leading to the establishment of a United States National Institutes of Health program in 1964 to create a total artificial heart (TAH)[8]. During this same decade, DeBakey and Liotta were able to successfully implant a paracorporeal device in a patient who could not be taken off the pump after aortic and mitral valve replacement, and after 10 days the device was able to be removed and the patient was discharged, constituting the first successful case of temporary support up to discharge. On the other hand, in 1969, the first version of a TAH was implanted in a 47-year-old man who survived for 64 hours before receiving a heart transplant, but later died from infectious complications[7,8].

In 1972, the LVAD development program was established in the United States, and formal research was begun, as TAH development improved. During the 80s, patients on transplant waiting lists were able to be sustained with a TAH for up to 620 days; however, hemodynamic, infectious and hemorrhagic complications, as well as death, were the common denominator[8]. In 1984, the first LVAD was implanted, and, in this same year, studies showed their usefulness as inpatient bridge-to-transplant therapy. However, the possibility of a patient being discharged with one of these devices was still a utopian dream[8]. It was in 1994, 30 years after their invention, that a patient was able to be discharged with an implanted, portable LVAD. These devices, known as first generation devices, had a pulsatile flow that mimicked the native heart's systolic and diastolic flow[2,8].

In 2001, the REMATCH study demonstrated their benefit in patients with advanced heart failure, even as long-term treatment, reducing the risk of all-cause mortality by 48% and showing a 52% one-year survival rate[9]. Despite this advance, these LVADs had limitations: they did not last long, they were heavy and noisy, and they required valves and multiple components to function[8].

At the beginning of the millennium, research migrated to continuous flow devices with the advent of second generation and axial flow devices like HeartMate II in 2008[2,8]. Two years later, their superiority compared with pulsatile flow LVADs was proven, increasing survival from 24 to 58%[10]. Although promising, they were still very large until the appearance, in the last decade, of centrifugal continuous flow devices. These are smaller, implanted in the apex, and the motor is intrapericardial. The first of these was HeartWare, which was removed from the market in 2021 because it increased the incidence of cerebrovascular accidents (CVAs)[2].

Finally, in the last five years, the first third-generation device appeared with continuous, centrifugal flow, and with a completely magnetic levitation motor, which reduces the risk of thrombosis. This device, HeartMate III, showed a 92 and 58% reduction in the risk of LVAD thromboses and CVAs, respectively[11]. From then on, it was the device of choice.

Basic components of left ventricular assist devices and the impact of continuous flow

The three LVADs used over the last two decades have been HeartWare, HeartMate II and HeartMate III[12].

They all have six components in common: 1) an apically-implanted inflow cannula, 2) a pump that transfers the blood to an outflow graft (3) which takes the blood to where it connects with the ascending aorta. A percutaneous lead (4) projects through the abdominal wall and connects the pump directly to an external controller (5) which monitors the device function parameters and is connected to a power source, which can be a portable or fixed battery (6).[12] (Fig. 1)

They all have four basic parameters reported by the controller: a) the pump speed reported in revolutions per minute, b) the power, measured in Watts, which indicates the current applied to the pump, c) the flow, analogous to cardiac output and expressed in L/min, which is actually an estimate based on the power and speed of the pump and, finally, d) the pulsatility index (PI), an indirect measure of the magnitude of the changes in flow and speed as the native left ventricle contracts[12,13].

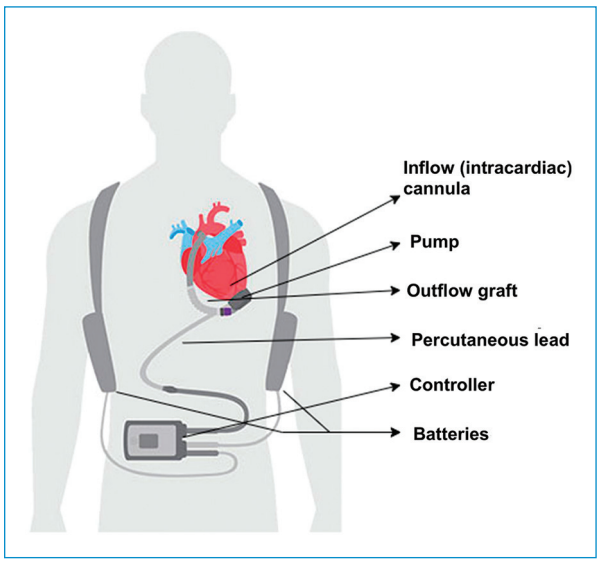

Figure 1. Components of left ventricular assist devices *(adapted from https://www.cardiovascular.abbott/us/en/patients/treatments-therapies/heart-failure/understanding-lvad-therapy.html).*

The physiological impact of continuous flow

The continuous flow of LVADs provides a non-physiological circulation with negative physiological impacts. It is illogical to think that adding a device that functions differently from the heart, a pulsatile pump that has evolved over 750 million years, will have no repercussions on a pulsatile circulation[14]. This close relationship between the LVAD and the heart is responsible for unique complications in this population. This interaction is described under the term "hemocompatibility"[15].

The areas most affected by the continuous flow are precisely those with greater pulsatility. In these sites, pulsatile circulation exerts an antithrombotic effect, as these are areas with greater recirculation and stasis, like the ventricles and aortic arch. This explains the risk of thromboses in these sites in LVAD patients[14].

All of the devices lead to more stress than red blood cells can tolerate, and therefore continuous low-grade hemolysis is common[14]. Other implications include microvascular dysfunction, angiogenic factor release, greater vascular stiffness, sympathetic tone, the risk of aortic regurgitation and proinflammatory cytokine release[15,16]. Von Willebrand factor polymers are also destroyed, leading to acquired von Willebrand syndrome[15].

Initial assessment of patients with left ventricular assist devices

There are unique aspects in the initial assessment of patients with LVADs. When taking the history, it is essential to ask about recent device alarms, heart failure symptoms, changes in urine color suggestive of hemoglobinuria, fever or secretion around the percutaneous lead or other symptoms suggestive of a hemolytic or infectious process.

Since all patients with LVADs should be on anticoagulation with warfarin and platelet antiaggregation with aspirin, tolerance to the medications must be verified as well as the lack of side effects like nosebleeds, melena or hematochezia.

These patients do not have a palpable peripheral pulse, and the different conventional methods for measuring blood pressure are not reliable. Blood pressure measurement will be achieved by palpatory or automatic methods in less than 10 to 50% of cases, respectively. The most reliable method for measuring mean arterial pressure (MAP) in these patients is Doppler[17], which consists of using a regular cuff while the radial artery is insonated. The sound of the radial artery flow is detected with the Doppler, and then the cuff is inflated to 20 mmHg above where this sound disappears. Finally, the cuff is deflated, and the MAP corresponds to the value at which the continuous flow sound reappears[18].

Some useful laboratory tests for assessing these patients include a complete blood count, coagulation times, liver profile and LDH as a hemolytic marker. The electrocardiogram changes after LVAD implantation, with

interference on the tracing; however, it continues to be useful for detecting arrhythmias[19].

All of this helps to suspect one of the various complications associated with LVADs. Thus, the authors propose the mnemonic $A_2B_2C_2D_2$ to remember the potentially fatal or specific complications while caring for these patients (Table 1).

Table 1. LVAD-associated complications

A_2	Arrhythmias
	Cerebrovascular accidents
B_2	Pump thrombosis
	Bleeding
C_2	Altered consciousness and CPR
	Controller alarms
D_2	Right heart failure
	Driveline infection

CPR: cardiopulmonary resuscitation.

Complications associated with left ventricular assist devices

A_2: arrhythmias

At least 30 to 40% of patients will be hospitalized for an arrhythmia within the first two years after implantation[13]. Causes of hospitalization include ventricular scar reentry, the insertion area of the inflow cannula and mechanical irritation around it[12,20,21].

Due to mechanical support, there is a greater tolerance of arrhythmias; there are even reports of cases of recurrent ventricular fibrillation (VF) for days to weeks[13,22]. At least 53% of the patients will be clinically asymptomatic[20]. However, a drop in flow should alert to the possibility of a ventricular arrhythmia, as these can cause flow drops of up to 32% and are not as tolerable as was thought[23].

At least 50% of adults with LVADs have atrial fibrillation (AF) prior to implantation, and some of them improve thanks to lower left atrial pressures[13]. In the event of an AF after implantation, it rarely manifests clinically and will depend on whether right ventricular dysfunction occurs[12,24]. Treatment is similar in patients without LVADs and is guided by the patient's symptoms and stability, but it should be remembered that anticoagulation is indicated in all patients with LVADs[21].

Ventricular arrhythmias are reported in fewer than 20 to 50% of patients and the risk is high mainly in the first weeks after implantation. If the patient had an ICD before, one of the manifestations will be device discharges while awake; otherwise, they will cause few and nonspecific symptoms like dizziness, nausea and fainting[13,20].

The management of these arrhythmias will depend on their clinical presentation. In patients with ICDs, these should be inhibited with a magnet, to prevent further discharges. If the patients are unstable, they should be sedated and cardioverted; if they are stable, the approach will depend on the duration of the arrhythmia. For arrhythmias with a long or unknown duration, right ventricle and aortic valve thrombi should be ruled out. If these are ruled out or the arrhythmia has a short duration, treatment may be started with IV amiodarone, or cardioversion if that fails[12,13,21]. The latter does not interfere with device function, and the LVAD does not need to be turned off or have the batteries disconnected[21].

Finally, if a small, collapsed left ventricle is found on bedside echocardiography, a bolus of IV fluids is indicated, and, if there are findings suggestive of a suction or hypovolemic event, the pump speed should be reduced[13].

A_2: cerebrovascular accident (CVA)

Along with right heart failure, CVAs continue to be the main cause of death in the first six months after implantation and, after this period, they become by far the main cause of death[25]. An estimated 10% of patients will have at least one CVA during follow up, with similar proportions of ischemic and hemorrhagic events[26]. Their main risk factors include female sex, advanced age, arterial hypertension, active infection, time on LVAD support and type of device implanted, among others[26].

HeartMate III is associated with a 58% reduction in the risk of CVAs compared with axial flow devices (HeartMate II); however, despite these advances, the rate of CVAs continues to be at least 9.9% during the first two years[11]. These cases appear to have a J-curve behavior, with the greatest risk during the first three months after implantation and after the first year of follow up[27].

Ischemic CVAs

Ischemic CVAs in patients with LVADs are mostly embolic, due to either passing a thrombus from the left ventricle through the device, or thrombus formation within the system, aortic valve, aortic arch, sinus of Valsalva, etc.

In some cases, they may also be due to a septic embolus within the context of systemic infection or device-related endocarditis[27].

Although LVADs are not an absolute contraindication for systemic thrombolysis, it is clear that most patients will have contraindications to this treatment. Likewise, there is the theoretical consideration that thrombolysis will be less effective since the embolic material in these patients is made up of not only fibrin, but also denatured proteins[28]. It is also unclear if, given their continuous flow, the therapeutic time window is different. Therefore, the current evidence goes back to case reports, and its use is limited to patients without contraindications and suboptimal anticoagulation.

Mechanical thrombectomy has the same indications as in patients without LVADs and is a safe intervention. Despite this, there are certain considerations for its use in this context[27]. Magnetic resonance imaging is prohibited in patients with LVADs, while brain perfusion computed tomography is not available in most of the country's tertiary care centers, and its yield and decision making based on its results are still not clear in patients with continuous flow devices. Thus, mechanical thrombectomy in our setting is relegated to patients in the therapeutic window with computed tomography angiography evidence of a large vessel being affected.

Regarding blood pressure, it has been suggested previously that a MAP of 70 to 130 mmHg be maintained in the first 24 hours[29].

Hemorrhagic CVAs

There are many reasons to believe that intracerebral hemorrhages (ICHs) and subarachnoid hemorrhages (SAHs) are a catastrophe for patients with LVADs. Their onset is a contraindication to heart transplantation. Furthermore, short-term mortality increases significantly and is greater than in patients with ischemic CVAs. According to INTERMACS registry data, survival after a hemorrhagic CVA is 45.3, 34.8 and 30.3% at 1, 6 and 12 months, respectively[26].

Intracerebral hemorrhages and SAHs are multifactorial in patients with LVADs. Anticoagulation is, undoubtedly, one of their main determinants; increased blood vessel fragility has also been described, due to the continuous flow, along with decreased pulse pressure. Finally, the previously described acquired von Willebrand syndrome has been reported due to pump destruction of this factor[28].

Despite understanding their main risk factors and these pathophysiological changes, their onset is still not fully understood. With the latest devices, the risk has diminished, but has not been eliminated. Likewise, evidence of ICHs has been found regardless of the INR level, so the explanation is more than overanticoagulation[28,29]. The most common characteristic in patients with intracerebral bleeding is acquired von Willebrand syndrome[30].

A simple head computed tomography is the diagnostic method of choice and computed tomography angiography is useful for evaluating vascular anatomy and determining the source of the bleeding. There is no contraindication to arteriography but do remember that magnetic resonance imaging is contraindicated.

Treatment for ICHs and SAHs in patients with LVADs is still not clear and, for now, the recommendations for patients without these devices are used for both medical and surgical management. There is a consensus among experts and scientific societies in recommending a MAP between 70 and 90 mmHg in these patients[12,13].

While the suspension of anticoagulation and its reversal with prothrombin complex or fresh frozen plasma is indicated, there is a risk of device-associated thrombotic events[12,28]. Therefore, more conservative management is suggested, reversing anticoagulation only for small ICHs with no major neurological deficit, and even maintaining the INR within the therapeutic range. Meanwhile, in the rest of the cases, anticoagulation may be reversed until an INR less than 1.4 is achieved[30]. Although the outlook is promising, its safety has not been validated.

B_2: bleeding

Non-intracerebral hemorrhages are common in patients with LVADs. They are represented by surgical bleeding immediately after implantation and episodes of gastrointestinal bleeding after three months. An estimated 22 to 32% of patients will have significant bleeding episodes and, together with infection, this is the second most common cause of hospital readmissions and accounts for 2% of deaths in this population[3,28].

The pathophysiology of hemorrhages in this population is multifactorial and includes coumarin anticoagulation and antiplatelet aggregation with aspirin, acquired von Willebrand syndrome, and changes in the vascular wall anatomy, including angiogenesis secondary to continuous flow with the subsequent appearance of arteriovenous malformations, mainly in the gastrointestinal, nasopharyngeal and intracerebral areas[15,28].

Gastrointestinal bleeding has a variable clinical presentation but is usually in the upper tract and rarely

leads to hemodynamic instability. The diagnostic approach is similar to that of patients without LVADs. The choice of endoscopic study depends on the anatomical region in which the source is suspected, in which case upper GI endoscopy is the most used. If the source is not found or there is hemodynamic instability, arteriography could be considered, which could be diagnostic and therapeutic[28].

The initial treatment of gastrointestinal bleeding is similar to that of patients without LVADs, with regard to fluid resuscitation and early endoscopic intervention. Proton pump inhibitors are indicated since the main anatomical bleeding site is the gastric mucosa[28]. If a blood product transfusion is required, irradiated red blood cells should be used for transplant candidates to avoid immunization.

It has been recommended that anticoagulation reversal be limited to patients with major or life-threatening bleeding, understanding the risk of device thrombosis that this entails, especially with the use of prothrombin complex. In the rest of the cases, dual anticoagulant and antiplatelet treatment should only be temporarily suspended or even continued, depending on the severity (if bleeding is minor and not clinically significant)[12].

Successful case series and varied results have been reported with octreotide treatment (a somatostatin analogue that increases vascular resistance, decreases angiogenesis and improves platelet aggregation) as well as studies in which it is applied monthly for prophylaxis[28]. In addition, taking omega 3 has been reported in this scenario; however, the use of either of these depends on each institution's protocol.

B_2: pump thrombosis

Pump or LVAD thrombosis is characterized by the presence of any thrombus within the flow of any of the device's components: the inflow cannula, pump or outflow graft[12]. It is considered a potentially fatal event, setting up a condition similar to left ventricular outflow tract obstruction, and has clinical signs and symptoms ranging from progressive dyspnea to acute heart failure, cardiogenic shock and even death[28]. Its incidence has progressively decreased with the advent of magnetic levitation devices (HeartMate III) that provide a 92% reduction in the risk of pump thrombosis (1.4 vs. 13.9%; $p < 0.001$)[11].

This diagnosis involves two scenarios: a) thrombosis in the internal cannula or inside the pump and b) thrombosis in the outflow graft. The first is characterized by the triad of acute heart failure, hemolysis (LDH more than three times above the upper limit of normal) and rapid flow reduction. Outflow graft thrombosis is not necessarily associated with hemolysis and may be caused by external compression or graft torsion, etc.[12,28]. Table 2 shows the main clinical and imaging differences between the two obstruction sites.

The finding of a rapid change in flow and power, together with elevated LDH, is the classic finding in these cases[12]. Computed tomography angiography is the imaging modality of choice to confirm outflow graft thrombosis[28]. Bedside echocardiography is very useful and can show indirect signs, like left ventricular dilation, aortic valve opening, severe mitral regurgitation and turbulent flow, etc.[31].

There is insufficient evidence to recommend a treatment strategy in patients with device thrombosis. The expert consensus recommends ensuring an INR between 2.5 and 3.5. If, despite this, thrombosis occurs or persists, anticoagulation should be started with unfractionated heparin. In more select cases and as a last resort, the use of systemic thrombolysis with or without glycoprotein IIb/IIIa inhibitors has been described; however, the risk of bleeding is extremely high[11,12,28]. The use of thrombolysis in this scenario is still not clear and has the same considerations presented in the section on ischemic CVAs. Recently, a protocol was described for administering low doses of rt-PA, repeated according to the clinical and paraclinical response, achieving a 69.2% rate of resolution[32]. Nevertheless, other authors have reported lower rates of resolution with high rates of major bleeding using similar dosing schemes, and therefore their use is subject to the institutional protocol[33].

Finally, outflow graft thrombosis could benefit from percutaneous treatment and, if any of the previous treatments fail or the patients are unstable or in cardiogenic shock, extracorporeal support, device replacement or heart transplantation are indicated[12,28,32].

C_2: altered consciousness and CPR

Approximately 13% of patients with LVADs will experience loss of consciousness, corresponding in most cases to orthostatic syncope[12]. The scene is challenging to deal with, since the acceptance of a lack of pulse in these patients may lead to a delay in beginning compressions in the event of a true LVAD failure. There is also controversy regarding the possibility of dislodging the inflow cannula with compressions; however, this has been found to be a rare event and manual compressions are considered to be safe[34].

Table 2. Differences in the array of LVAD thromboses

	Cannula or pump thrombosis	Graft thrombosis
Signs and symptoms	Progressive dyspnea Acute heart failure	Dyspnea Distal embolism
Onset	Short	Progressive
Hemolysis	Yes	No or scant
Device parameter	Low flow and high power	High power
Laboratory findings	Elevated LDH Anemia Tea-colored urine	Absent
Bedside echocardiography	– Dilated LV – Severe mitral regurgitation – Turbulent flow within the internal cannula on Doppler – Visible thrombus within the LV	– Dilated LV – Lack of thrombi in the LV – No turbulent flow within the internal cannula – Turbulence within the graft on Doppler, in a high left parasternal or right parasternal plane
Computed tomography angiography	No obstruction within the graft	Confirms graft obstruction or stenosis and identifies the cause

LDH: lactate dehydrogenase, LV: left ventricle.
Adapted from Ben Gal T et al., *2021*[12].

Outside of the hospital setting, the chain of survival is similar. If untrained people witness a sudden collapse and there is no pulse, chest compressions should be started as soon as possible, and the emergency system should be activated[35]. The latter can instruct the passer-by, by telephone, to begin these maneuvers, if he/she is not trained to identify patients with LVADs. Subsequently, the patient should ideally be transferred to a center with LVAD experience or where the device was implanted[35]. If trained rescuers are available, the two main actions in a patient with altered consciousness are to determine if there are signs of lifelessness or poor perfusion, and confirm if the device is functioning[12].

The absence of signs of life is evaluated by answering the two classic questions: Is the patient unresponsive to stimuli? and Is breathing absent or abnormal? If the answer to both questions is "Yes," the authors recommend beginning compressions, as their benefit outweighs the risk of performing them if there is no cardiac or pump arrest. Also, confirming device functioning can significantly delay beginning resuscitation maneuvers. Other measures for evaluating perfusion include a MAP < 50 mmHg, capnography values falling to less than 20 mmHg and, if available, carotid or femoral arterial Doppler[12,13].

After beginning the maneuvers, LVAD function should be investigated. The controller has a visual signal on its screen indicating its proper functioning. The two main causes of intrinsic device failure are percutaneous lead dysfunction and lack of batteries; therefore, the apex should be auscultated to confirm the device's sound, and each of its components and connections should be verified.

These two assessments will allow an initial conclusion to be reached: in patients with LVADs who are perfused and whose device is functioning properly, it is highly unlikely that their altered consciousness is due to the heart, and thus compressions could be stopped, continuing with ventilatory support and studying other causes like hypoxemia, CVA, hypoglycemia, intoxications, etc.[12].

If the patient has poor perfusion but the device is working, chest compressions should be continued[12,36]. In this scenario, it is important to consider the rapid use of extracorporeal circulation support based on the length of resuscitation, the availability of the resource and the patient's prognosis[35].

During advanced life support, the use of adrenaline, antiarrhythmics and external defibrillation has the same considerations as in patients without LVADs[36]. The limitations of the therapeutic effort and the presence of unequivocal signs of death, like fixed pupils, rigidity or intra-arrest echocardiographic asystole must be kept in mind as elements for considering terminating resuscitation efforts.

If the LVAD is not working, efforts should be made to restart it and change the batteries and the controller, if necessary. If despite checking the entire device and attempting to restart it this is not successful, chest

compressions should continue along with advanced life support measures with the same considerations mentioned above[12]. Echocardiography in critical patients with LVADs is very useful, as this can help evaluate signs of right ventricular dysfunction, look for indirect signs of pump obstruction or volume depletion, confirm if the aortic valve opens during systole, and identify possible causes of cardiac arrest, etc.[31]. Figure 2 presents an algorithm that summarizes the approach to patients with LVADs and altered consciousness, as well as the recommendations for beginning cardiopulmonary resuscitation.

C_2: controller and alarms

Left ventricular assist devices have two alarm levels: warning and critical. The first is yellow, intermittent and requires attention, but not necessarily emergency attention; these alarms include: low battery, low flow, low speed, a disconnected battery cable, etc.[37] The red alarms, on the other hand, are critical, constant and require emergency action; these include: pump arrest, percutaneous lead disconnection, disconnection of both batteries, less than five minutes of battery time, and minimum flow (less than 2.5 L/min)[37].

Pump arrest, although feared, is rare. In these cases, with decreased cardiac output and stasis, ventricular thrombi may form, and therefore care should be taken when deciding to restart the LVAD. If there is hemodynamic instability, the LVAD should be restarted regardless of the time elapsed, as its benefits outweigh the risks; initiating heparin anticoagulation should also be considered[12,37]. If the patient is stable and the LVAD failure only lasts a couple of minutes, it can be restarted; otherwise it should not be restarted, and the patient should be evaluated at his/her LVAD center[12].

The low flow alarm is another important scenario. It has multiple causes including dehydration, right heart failure, cannula obstruction, etc. If the alarm is silenced by assuming the Trendelenburg position, this indicates hypovolemia and, in that case, intravenous fluids would be indicated[13]. Bedside echocardiography can also help narrow down the cause of this alarm[36].

The initial approach to any alarm is to verify each of the controller elements, including the percutaneous lead connection, the two different batteries and their charges, as well as apical auscultation to confirm pump function; management of each alarm will depend on its specific cause. Table 3 shows other possible alarms, their potential causes and their specific approach.

D_2: right heart failure

Right heart failure (RHF) is common and can occur prior to discharge (early RHF) or after discharge (late RHF). According to the INTERMACS registry, its prevalence in the first month after implantation reaches 24%, then declines and remains chronically at approximately 8 to 10%[38]. This has prognostic implications, since its onset is related to lower survival and a higher risk of all the previously described complications[39].

The pathophysiology of post-LVAD RHF is multifactorial. After implantation, there is an abrupt increase in cardiac output and right ventricular preload; this, together with decreased pressure within the left ventricle, leads to septal movement and increases the right ventricular workload. The right ventricle is unable to respond to this increased workload and this causes dysfunction, either *de novo* or worsening of chronic dysfunction[40].

The risk factors associated with its early onset include clinical and echocardiographic characteristics like prior right ventricular dysfunction, as well as hemodynamic characteristics (CVP greater than 15, pulmonary artery pulsatility index less than 1.85), among others[41]. There are also factors that favor the onset of late RHF, like arrhythmias, valve disease, the progression of right ventricular dysfunction and pulmonary hypertension[40].

Ventricular arrhythmias should always be ruled out in these cases. The ultimate management of right heart failure is similar to that of patients without LVADs. Right ventricular preload should be optimized, and therefore diuretics are indicated if there is congestion; if there is low output, inotropes with a pulmonary vasodilatory effect should be started promptly[42]. Echocardiography is useful for evaluating the position of the interventricular septum in patients with RHF and LVADs. If it is significantly deviated to the left, flow reduction through the LVAD should be considered. Finally, the use of right ventricular assistance and mechanical circulatory support would be indicated for patients who remain in shock despite the mentioned treatments[42,43].

D_2: driveline infections

Infections are the “Achilles’ heel” of LVADs. These can be specific to the LVADs (cannula, pump, outflow graft or lead infections) or related to the LVADs (endocarditis, bacteremia, mediastinitis, etc.)[12]. They represent the main cause of hospitalization in the first year after implantation and approximately 20% of patients will have a device infection during follow up[3,12].

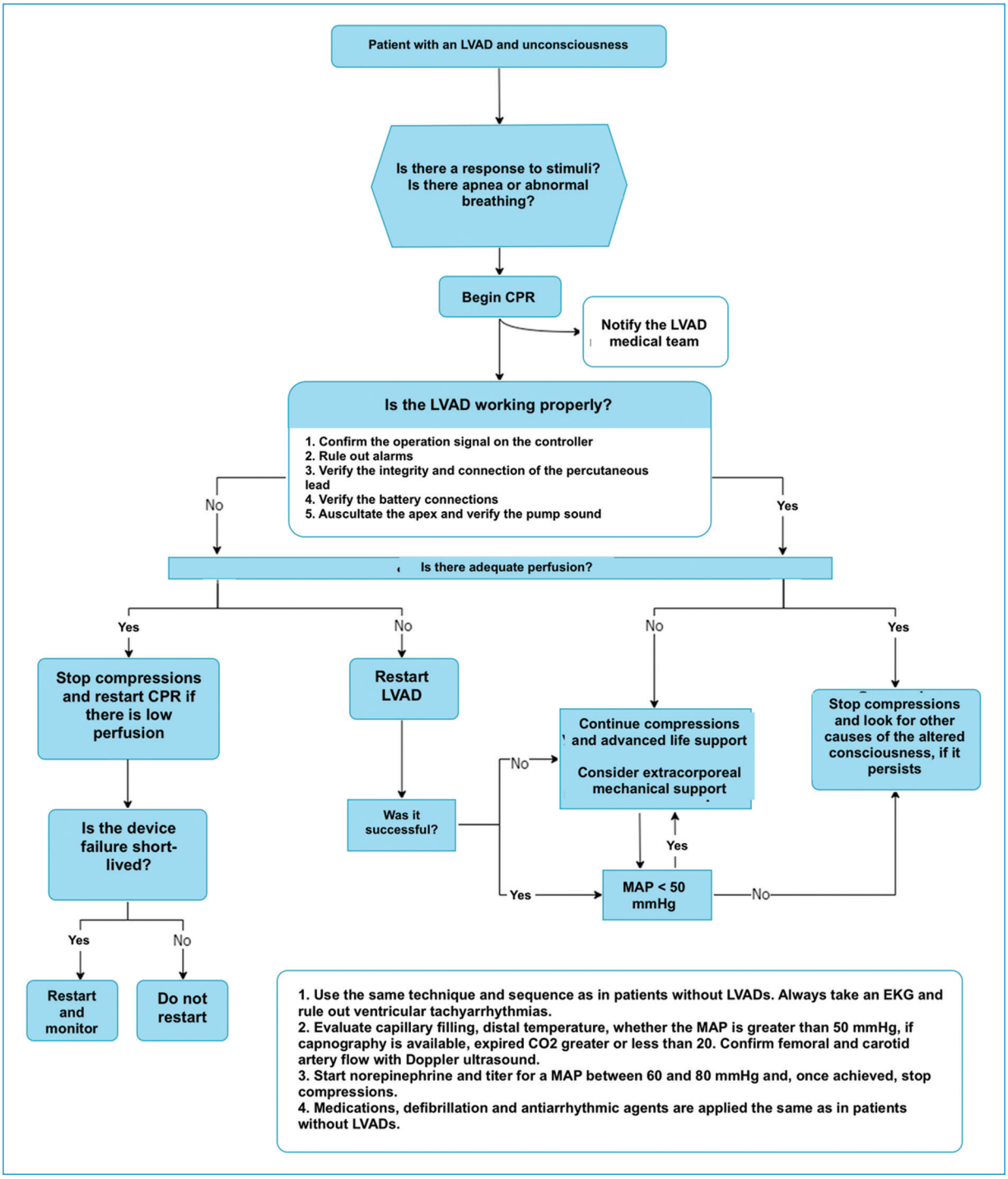

Figure 2. Algorithm of care in patient with LVADs and altered consciousness.

All of the device components are susceptible to infection; the most common infection is of the percutaneous lead (49% of the cases), followed by loco-regional and pump infections with 37 and 14%, respectively[44]. The most frequently isolated germ is *Staphylococcus aureus*, in approximately 56% of cases, but the infection may also be caused by both Gram positive and Gram negative germs[45,46].

It is essential to evaluate the integrity and the exit site of the percutaneous lead in the abdominal wall. When in doubt, a set of blood cultures should be taken, and cultures attempted of the local collections to identify the

Table 3. Complications visible on the controller and their management

Complication	Causes	Considerations	Management
Pump failure	No power source	Check the connections and battery	Emergency cardiology assessment; in the event of shock, restart the pump, give anticoagulation and inotropic support
Low power	– Battery with no charge – Disconnected percutaneous lead		– If it is due to a low charge, connect to a steady power source – In the event of primary failure, establish support measures and consider replacing the device
High power	– Thrombosis – Hypertension – Electrical failure	Bedside echocardiography looking for direct or indirect signs of device thrombosis	– Ensure a MAP of 70-90 mmHg – Actively search for hemolysis, including LDH measurement – Start anticoagulation with UFH in the event of thrombosis – Consider replacing the device in the event of electrical failure
High flow	Infections/sepsis	Evaluate the power; when the power is normal, it is associated with vasodilation and sepsis	Close follow up, consider actively searching for device-specific or associated infections
Low flow	– Suction events – Hypovolemia – Right heart failure – Cannula obstruction – Hypertension – Graft obstruction – Arrhythmias – Very high speed	Bedside echocardiography: – Septal deviation or misalignment of the cannula with the mitral valve in suction events – Turbulent flow or regurgitation on Doppler in the event of cannula obstruction or pump malfunction – Presence of blood clots – Low end-diastolic volume, collapsed vena cava in the event of hypovolemia	– Perform an EKG and echocardiogram – Anticoagulation for thrombosis – Inotropes for shock – Cardioversion for arrhythmias and hemodynamic instability or short-lived VT/VF – Fluid resuscitation for hypovolemia or suction events

EKG: electrocardiogram; VF: ventricular fibrillation; UFH: unfractionated heparin; LDH: lactate dehydrogenase; MAP: mean arterial pressure; VT: ventricular tachycardia. *Adapted from Long B* et al., *2019*[36] *and Cook* et al., *2017*[37].

causal germ. In the event of systemic disease or sepsis, empirical intravenous antibiotics should be started to cover Gram positive cocci and enterobacteria, keeping the local resistance profiles in mind[12].

Conclusions

Left ventricular assist devices are an increasingly common treatment for which emergency staff must be trained. They are the result of more than 50 years of research on mechanical circulatory support and are useful in patients with advanced heart failure as a bridge to transplant or as definitive therapy. They have differential care, including a different method for taking blood pressure. They have frequent complications associated with their functioning, including ischemic and hemorrhagic cerebrovascular accidents, thrombosis, device infections, significant bleeding, RHF and altered consciousness, gathered under the $A_2B_2C_2D_2$ mnemonic. Each has a specific approach, including beginning chest compressions if there is a lack of response to stimuli and absent or abnormal breathing, while the device's function is checked. Finally, each of these interventions should be balanced with the consideration of redirecting the therapeutic efforts, if necessary, in patients whose indication is purely palliative.

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Conflicts of interest

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Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this study.

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