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REVIEW ARTICLE

Review article: Ventricular assist devices in the emergency department

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Abstract

Ventricular assist devices (VADs) have become an indispensable tool in the management of end-stage cardiac failure, both as a means of bridging to cardiac transplantation and as destination therapy for long-term quality of life improvement. Although the technology continues to advance and these devices continue to be refined, they are still associated with significant complications. This article reviews the basics of VAD function and physiology, as well as the myriad to complications that follow their implantation. This review aims to provide a systematic approach to the troubleshooting, diagnosis and management of both VAD-associated complications and the resuscitation of the decompensated VAD patient presenting to the ED.

Key words: *complication, emergency, resuscitation, ventricular assist device.*

Key findings

- The systematic assessment of patients with ventricular assist devices is crucial.
- Always obtain a 12-lead ECG.
- Consider sepsis and haemorrhage as a cause for hypotension.
- Consult your nearest VAD-coordinating service as soon as possible.

Introduction

The artificial heart programme was first conceived over four decades ago.[1] The first successful implantation of a left ventricular assist device (LVAD) was performed in 1984.[2] In 2001, the REMATCH trial[3] demonstrated that the use of ventricular assist devices (VADs) leads to a significant increase in survival (52% *vs* 25% 1 year survival; $P = 0.002$) and an improved quality of life. In the current era, approximately 91% of LVAD-supported patients will have survived to 1 year post-implantation.[4] With a growing population of LVAD-implanted patients in the community, it stands that they will present more frequently to local EDs, many without cardiothoracic surgeons and VAD-trained teams. Emergency clinicians should have an adequate understanding not only of the basic mechanics of a VAD and their physiologic implications, but also be aware of their common complications and an approach to the VAD patient in extremis.

Methodology

MEDLINE (1946 to March 2013) was searched using the strategy [(ventricular assist devices or left ventricular assist device).mp. or exp Heart-Assist Devices/] AND [complications.mp. or exp Postoperative Complications/] OR [Emergency.mp. or exp Emergencies/]. Embase (1974 to March 2013) was searched using the strategy [exp ventricular assist device/ or ventricular assist device.mp. or exp heart assist device/] AND [exp emergency care/ or exp emergency medicine/ or emergency.mp. or exp emergency/] OR [exp catheter complication/ or exp postoperative complication/ or complications.mp. or exp infectious complication/ or exp lung complication/ or exp medical device complication/ or exp infection complication/ or exp complication/ or exp posttraumatic complication/ or exp neurological complication/]. The Cochrane library was searched for [ventricular assist device]. Each search was limited to humans, English language and full text.

The aforementioned search strategies yielded the following results: MEDLINE, 906 citations; Embase, 1207 citations; and Cochrane library, only two citations. The titles and abstracts were screened by the authors and the relevant articles were retrieved. Reference lists within these articles were searched manually in an attempt to identify any further relevant papers. At total of 59 papers were identified as relevant and were included in this review.

Indications for ventricular assist device insertion

Progressive advances in design and patient management have made VAD therapy an indispensable tool in the management of advanced heart failure.[5–8] One-quarter of all US heart transplant recipients are supported with these devices as a bridge to transplant.[8] In Australia, the majority of VADs are implanted as a bridge to

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transplant. They are also used as a bridge to recovery (for those with reversible myocardial pathology)[9] and destination therapy (long-term assistance for patients ineligible for transplant).[7]

Devices

VADs typically comprise of an inflow cannula (from left ventricle), the pump with its associated mechanical and electrical components, an outflow conduit (anastomosed to ascending aorta), a percutaneous lead (driveline), and the external system components (a controller, monitors and a power source with battery components).[10] Alternatively, these devices can be implanted to support right ventricular function (right ventricular assist device [RVAD]) or simultaneously to provide biventricular support (bi-ventricular assist device [BiVAD]).

VADs can be classified as pulsatile or continuous flow devices. First-generation devices were volume displacement, pulsatile pumps that emit a wave intended to mimic systole and diastole of the natural heart (e.g. HeartMate XVE [Thoratec Corporation, Pleasanton, CA, USA]) and used an electric motor-driven pusher plate to drive blood flow.[11] They also contained a fail-safe external hand-held pump that could be used to maintain cardiac output in the setting of device or power failure. In contrast to the first-generation devices, the second and third-generation LVADs are continuous flow, rotary pumps, and do not produce a pulsatile wave. They are simpler in design with only a single moving part, an internal rotor used to create flow.[10] These continuous-flow VADs improve the probability of survival free from stroke and device failure at 2 years compared with the pulsatile devices (62% *vs* 7%, $P < 0.001$).[12]

VADs currently used in Australia at present include the Thoratec HeartMate II (Thoratec Corporation) (Fig. 1) and the HeartWare HVAD (HeartWare International, Framingham, MA, USA) (Fig. 2).

The total artificial heart (TAH) is currently undergoing clinical trials and takes a completely new approach than previous devices. The TAH replaces both native ventricles and valves with a complete artificial heart. This still requires an external power source and controller panel, but avoids native valve complications such as aortic and mitral regurgitation, which can complicate VADs.

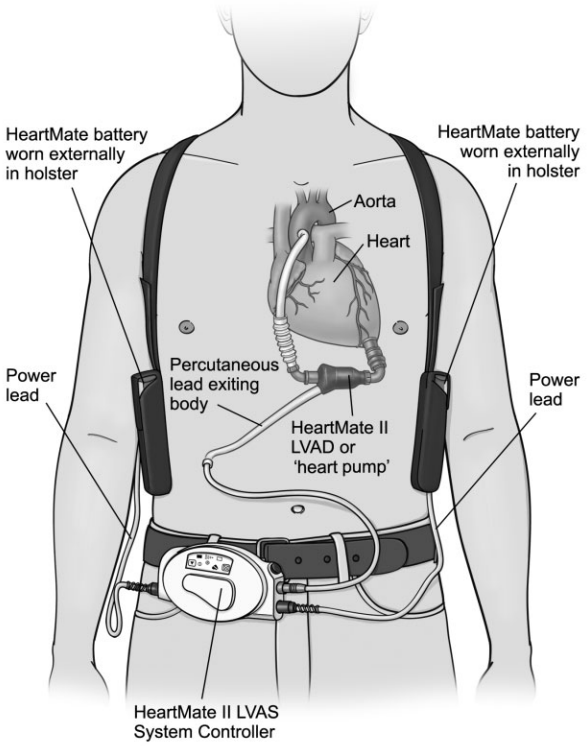

Figure 1. *Thoratec HeartMate II. An axial-flow VAD with only one moving part; a rotor (spinning on bearings) that propels blood from the inflow cannula in the left ventricle to the ascending aorta. It is powered by two lithium-ion batteries for a duration of >10 h (reprinted with permission from Thoratec Corporation).*

Ventricular assist device physiology

Mechanical circulatory support attempts to achieve several goals, including decompression of a hypokinetic ventricle, reduction of myocardial work and oxygen demand, whilst also maintaining adequate systemic perfusion and end-organ function. The VAD achieves this by reducing preload and decreasing myocardial wall tension and oxygen consumption, resulting in decreased left ventricular (LV) end-diastolic pressure and myocardial strain.[13]

Continuous-flow LVADs lead to increases in diastolic pressure and diastolic flow. As these devices pump continuously throughout the entire cardiac cycle, aortic flow is also present during diastole when it would normally be absent.[14] When the pump speed of a continuous-flow LVAD is increased, the diastolic pressure rises, the systolic pressure remains fairly constant,[14] resulting in significantly diminished pulse pressure (average pulse pressure is 5–15 mmHg).[15]

The degree of diminished pulsatility depends on pump speed, native LV contractility and pre/afterload pressures. The effect of a decreased or absent pulse on other organ systems appears to be minimal.[16] Decreased pulsatility is not, however, without complications. It can affect aortic valve opening times, aortic root flow, left atrial and ventricular volumes, right ventricular function and shear stress on blood components.[17] VADs also induce biochemical alterations on the coagulation cascade, immunological system, systemic inflammation and the neuroendocrine axis.[18]

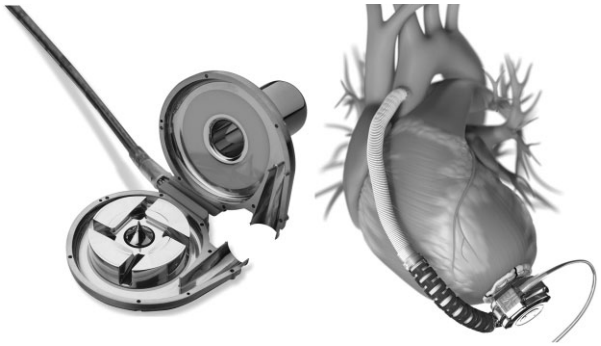

Figure 2. *HeartWare HVAD. A centrifugal, continuous flow device designed to draw blood from the left ventricle and propel it through an outflow graft connected to the patient's ascending aorta. The inflow cannula is surgically implanted into the left ventricle and blood is conveyed through the pump via an impeller (the only moving part) that is hydrodynamic and magnetically levitated, allowing frictionless rotation at operating speeds of 2400–3200* g *(resulting in up to 10 L/min of blood flow). Blood exits the pump into a flexible, gel-impregnated outflow graft that is surgically anastomosed to the ascending aorta. The driveline is tunnelled subcutaneously and carries wires from the pump to an external controller (this regulates power, monitors performance, displays alarm notifications and collects information about operations that can be downloaded for analysis). Two rechargeable lithium ion batteries provide power with the combined capacity of >10 h of use (courtesy of HeartWare International, Framingham, MA, USA).*

Patient and ventricular assist device assessment

The assessment of a patient with a VAD begins with a basic primary survey. If the patient is awake, alert and looks well, then the device must be functioning. Traditional ways of assessing haemodynamic status are difficult in the LVAD patient. Peripheral and central pulses might be diminished or even absent, and non-invasive BP (NIBP) measurements (auscultatory or automated) are often difficult to obtain.

Heart rate is measured as normal via electrocardiography and a 12-lead ECG (which is not directly affected by the LVAD) should be performed on all patients to assess for arrhythmias. NIBP is performed with a manual sphygmomanometer and Doppler ultrasound over brachial or radial artery. A constant sound heard using this method signifies the mean arterial BP (MAP) and should be 70–90 mmHg (Fig. 3). Alternatively, invasive MAP monitoring can be obtained by placement of an ultrasound-guided arterial line. Pulse oximetry, if obtainable, might be unreliable because of diminished pulse pressure.

LVADs are preload dependent and afterload sensitive. They will only pump the volume of blood delivered to it; therefore, maintenance of preload is critically important.[19] Assess for shock in the usual way; check capillary return, peripheral and end-organ perfusion. Hypotension might result from VAD-related and non-VAD-related pathology (Table 1).

Assessing ventricular assist device function

The VAD should initially be checked to verify the pump is running. This can initially be done with auscultation for a continuous or pulsatile noise over the precordium or epigastrium. The device controller will display vital information, including speed (rpm), flow (L/min), power (watts) and pulsatility index, as well as remaining battery life. It is important to note that pump-operating parameters are not surrogates for monitoring the patient's clinical status. Because of marked variability between patients, the parameter trends are often more valuable than the absolute figures presented. Abrupt changes, not accounted for by normal physiologic variation, can be used to identify specific pathology[14] (Table 2).

The VAD controller has the ability to display alarms of varying clinical significance. An approach to these alarms and the associated pathology are found in Table 3.

Ventricular assist device malfunction

Life-threatening VAD failure was not an uncommon occurrence with first-generation VADs (6% at 6 months, 64% at 2 years); however, the incidence of clinically significant device failure has fallen substantially with the use of second and third-generation devices.[20] VAD technology is also subject to technical problems, including driveline fractures, controller software faults and driver defects.[21]

The diagnosis of VAD component malfunction remains a challenge, particularly in the ED. When faced with a patient with a possible VAD-related emergency, contact should be made with your local VAD-coordination team as soon as possible. The patient or their family might also be carrying an information or emergency-responder card to guide your assessment and troubleshooting.

Diagnostic pathways for VAD malfunction have not yet been standardised,[22] and there is minimal information in the literature supporting an optimal method for the evaluation of patients presenting with possible LVAD malfunction. Basic investigations such as full blood counts, electrolytes and coagulation studies are essential and should be sought urgently. Echocardiography (ECHO) has been reported to be useful in the non-invasive evaluation of inflow obstruction[22,23] that can be achieved with standard transthoracic windows.

Pump thrombus

Thrombosis can occur within the VAD pump or its conduits, leading to dysfunction of the VAD itself or peripheral embolisation.[24] VAD thrombosis occurs in 1–2% of patients after 2 years of implantation.[5,12] Clinically, it will result in a low cardiac output state associated with falsely high estimates of pump flow by the VAD.[20] ECHO can aid the diagnosis.[20] Cardiac CT

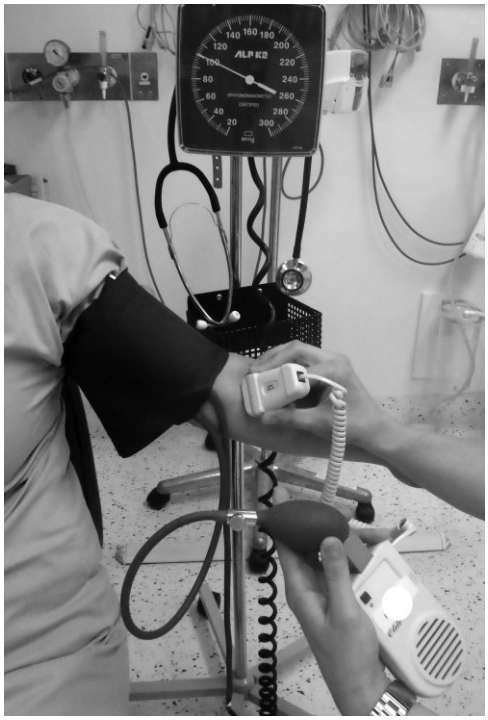

Figure 3. *Determination of mean arterial BP in patients with ventricular assist devices.*

TABLE 1. *Causes of hypotension*

VAD related	Non-VAD related
Pump failure	Hypovolaemia
Pump/cannula obstruction	Dehydration
Thrombus	Haemorrhage
Conduit compression/kinking	Ventricular arrhythmias
Pump speed set too high	Right ventricular failure
	Cardiac tamponade
	Aortic valve regurgitation

VAD, ventricular assist device.

angiography is also capable of diagnosing VAD thrombus.[25]

Pump thrombus is an urgent situation that requires immediate anticoagulation with heparin (plus additional antiplatelet therapy).[11] Life-threatening scenarios require urgent consultation with specialist VAD teams and might require thrombolytic therapy[11,26] or replacement of the VAD.

Suction events

In continuous flow VADs, large negative pressures can be generated within the left heart, causing chamber collapse and marked leftward displacement of the ventricular septum. This is known as a 'suction event'.[20] Suction events might occur in the setting of right ventricular failure, but can also result from hypovolaemia, cardiac tamponade, arrhythmias and malpositioning of the inflow cannulae.[20,24]

Clinically, a suction event results in low cardiac output, low MAP and poor VAD flow. In rotary blood pumps, this can precipitate severe ventricular arrhythmias.[27] Initial treatment involves administration of i.v. fluids and maintenance of preload. Once at the VAD-specialist centre, VAD pump speed might be manipulated (under guidance of transoesophageal ECHO).

Cannulae malposition or obstruction

Occlusion of the inflow or outflow cannula can occur because of sudden or gradual kinking or movement of cannula, thrombosis, endocarditis or anastomotic stenosis.[20,24] Pericardial fibrosis and scarring might cause progressive migration of the inflow cannula, resulting in partial occlusion.[20] Pseudoaneurysms of the outflow grafts have also been reported.[28] These problems can be diagnosed by ECHO, CT and angiography.[23] Management often requires surgical exploration.

Other ventricular assist device-related complications

Bleeding

Nonsurgical bleeding is one of the most common adverse events within the first month of VAD implantation[12,29,30] and the most common reason to present to the ED.[31] In the original REMATCH trial, the frequency of bleeding events was 42%.[3] Common sites of bleeding included gastrointestinal tract (GIT), epistaxis, intracerebral haemorrhage and intrathoracic bleeding.

The mechanisms of bleeding are likely to be multifactorial. Firstly, these patients receive both anticoagulation and multiple antiplatelet therapy to attenuate the risk of thromboembolic events.[32] Secondly, an acquired von Willebrand syndrome occurs in patients with continuous-flow assist devices,[32–34] a result of the high shear stresses on blood as it moves through device. Finally, the reduced pulse pressure associated with axial-flow VADs can lead to increases in arteriovenous malformations and angiodysplasia in the GIT.[30,35]

The management of bleeding in a VAD patient is a challenging scenario and poses a difficult therapeutic dilemma. It should include both rapid assessment of anticoagulant therapy (full blood count, coagulation profile) and prompt involvement of the VAD-coordinating team. Referral to endoscopists is also crucial. Anticoagulation reversal might be required and should

TABLE 2. *VAD parameters, normal ranges and clinical significance (adapted from Slaughter et al.[14])*

Parameter	Measurement	Normal values	Important points/Clinical significance
Pump speed	Direct measurement through motor	2200–2800 rpm (HVAD) 8000–10 000 rpm (HeartMate II)	– System operates at a fixed rate – Large speed changes signifies abnormal condition For example, suction event (automatically drops speed) or controller/pump issues
Power	Direct measure of current and voltage applied to motor	4–6 Watts	– Power varies directly with pump speed and flow • Increased flow = increased power • Increased/decreased pump speed = increased/decreased power • Physiologic demand alters power – Inflow obstruction can reduce power – Abrupt changes in power mandates evaluation for ingested thrombus into the pump itself – There might be gradual power increase in cases of deposition/thrombus onto bearings or rotors
Flow	Estimation based on pump speed and power	4–6 L/min	– Calculated value that becomes imprecise at high and low ranges • Estimation assumes normal pump operation • Will vary normally according to patients physiologic requirements – Increased flow occurs with a falling pressure differential across the pump and with increased pump speed – Overestimated high-flow rates occur in settings of high-power (e.g. thrombus or deposition) – Low-flow rates occur with inflow cannula obstruction – Flow estimation helpful as a trend (not an absolute measure of flow)
Pulsatility index (PI)	Calculated from pump power normalised by mean power	1–10	– Indicates the amount of assistance provided by the LVAD – Measures magnitude of flow pulses that occur during native cardiac cycle – Higher values indicate more ventricular filling and higher pulsatility (VAD providing less support) – Lower values indicate less ventricular filling/lower pulsatility (pump providing greater support) – Decreased PI can signify hypovolaemia, inflow/outflow obstruction or abnormal power elevation (thrombus)

HVAD, HeartWare HVAD; VAD, ventricular assist device; LVAD, left ventricular assist device.

be performed for as short time period as possible as these patients are at high risk of thrombosis and stroke. Therapeutic options for life-threatening haemorrhage include tranexamic acid, desmopressin, prothrombin complex concentrates, recombinant factor VII and fresh frozen plasma; however, these should only be used under the direct instruction of the VAD specialist teams.[20,34,36]

Right ventricular failure

Acute right ventricular (RV) dysfunction occurs in up to 20–25% of patients[37] and has a high rate of morbidity and mortality.[38,39] This typically occurs soon after VAD implantation in the intensive care environment. Causes of RV failure in these patients include pulmonary hypertension, RV infarction and pulmonary embolism.

Although the diagnosis of RV failure should be clinically apparent, ECHO can further define RV function.[40] Patients with RV failure are incredibly complex and their management involves inotropic support, volume unloading and use of pulmonary vasodilators. Consider intubation in these patients in extremis to help correct respiratory acidosis (which might worsen pulmonary hypertension). Mechanical RV support by way of extracorporeal membrane oxygenation (ECMO) or RVADs are occasionally required to restore pulmonary and LV blood flow.[38]

Arrhythmias

Patients with VADs are at significant risk for both atrial and ventricular arrhythmias with an incidence as high as 50%.[41–43] These might be because of local trauma (direct contact of the VAD with endocardium), volume status or electrolyte shifts.[27] There is also the background risk factors related to the underlying myocardial disease responsible for the initial VAD requirement.[24]

The treatment algorithm for atrial fibrillation post-LVAD is no different

TABLE 3. *Common alarms, causes and suggested actions used by the HeartWare HVAD*

Alarm type	Alarm display	Meaning	Potential causes	Actions required
Hazard/Critical	Pump off VAD stopped	Pump has stopped Pump is not operating correctly Pump flow <2.5 L/min Percutaneous lead is disconnected	– Driveline disconnected – Driveline fracture – Connector malfunction/ breakage – VAD electrical failure	Check connections from driveline, controller and power source Change controller
	Critical battery Low voltage	<5 min of battery power remaining. Controller receiving inadequate power	– Limited battery time – Battery malfunction	Replace battery Connect to alternate power source • Do not remove both batteries simultaneously – VAD will stop
	Controller failed	Controller component failed	– Controller component failed	Change controller
Advisory/Medium	Controller fault	Controller fault. Alarms disabled	– Controller component malfunction – Electrostatic discharge	Change controller Review alarm thresholds
	High watts	High power condition in running VAD pump	– VAD thrombus – High RPM – High flow – VAD electrical fault	Assess and optimise preload/afterload Assess for thrombus
	Low flow Suction	Pump flow < 2.5 L/ min Average flow dropped below threshold	– Suction event – Poor VAD filling – Elevated BP/ afterload – Outflow graft kink – RPM too high or low	• Ensure adequate preload/filling • Treat arrhythmias • Investigate for VAD occlusion (inflow and outflow tracts) • Consider afterload reduction or decreasing pump speed
Advisory/Low	Low battery Low voltage	<15 min of battery power remaining		Replace battery Connect to alternate power source
	Power cable disconnected	Power cable disconnected		Ensure all connections intact
	Replace system controller	System controller is operating in backup mode		Replace system controller

HVAD, HeartWare HVAD; VAD, ventricular assist device; RPM, revolutions per minute.

than that used for the general population.[43]

Ventricular arrhythmias can be well tolerated in patients with VADs.[44] Presentations of asymptomatic, haemodynamically stable patients in ventricular tachycardia or ventricular fibrillation have been reported.[45,46] Rapid evaluation for the underlying cause of the ventricular arrhythmia should take place, including volume status, electrolytes and assessment for potential myocardial ischaemia.

The management of ventricular arrhythmias might involve volume replacement, reduction in pump speed, and either pharmacological or electrical cardioversion.[43] I.v. amiodarone has been successful, with lignocaine and procainamide used as second-line

agents.[47] Electrical cardioversion or defibrillation is often required as these arrhythmias can be refractory to medical therapy.[47] When cardioversion, external defibrillation or pacing is required, anterior-posterior placement of the pads is preferred. Some VADs such as the HeartWare VAD can also be internally defibrillated.

Infection

Infection can involve any portion of a VAD, including the surgical site, percutaneous driveline, device pocket and the pump itself.[48,49] The REMATCH trial[3] reported that 42% of patients developed sepsis within 1 year of implantation. Infectious complications of VADs are associated with severe morbidity and mortality, and will subsequently preclude future transplantation for some patients.[50] It is important to engage your nearest VAD surgeon early in care of these patients, particularly if a hardware infection is suspected.

The microbiology of VAD infections includes a wide range of organisms; however, Gram-positive organisms predominate, especially coagulase-negative staphylococci and *Staphylococcus aureus*.[48,49] Gram-negative organisms and fungi have also been implicated in VAD-related infections.[51]

The presence of infection might be clinically obvious; however, one should maintain a high index of suspicion of occult sepsis in the VAD patient presenting with hypotension or fever.[24,49] The use of cultures, exit site swabs, diagnostic aspirations, US, CT and targeted nuclear medicine studies can assist in localising the source of infection.[52]

Empiric treatment for VAD-related infections should include broad-spectrum antibiotics as well as antifungal coverage and consideration of multi-drug resistant organisms. Local resistant patterns and institutional preference will influence actual therapy.[51]

Neurological complications and stroke

As discussed previously, the use of anticoagulation and antiplatelet therapy is designed to mitigate the well-documented thromboembolic risks associated with VAD implantation. These patients also carry a higher risk of haemorrhagic stroke. Whilst stroke and transient ischaemic symptoms combined can occur in up to 27% of patients with VADs,[20] tight BP control (targeting MAP less than 85 mmHg) and an INR of 2–3 help lower this risk. There is consistent right-hemispheric dominance of stroke in patients with VADs.[53]

Cardiac arrest

If a patient is unconscious, not breathing and has no signs of LVAD function, assume that he/she is in cardiac arrest. While concurrently administering ALS, the priority is to look for patient and VAD factors that might have led to the arrest. The patient should be intubated and given i.v. fluid boluses. Bedside ECHO can provide information on RV and LV size and function. Allocate a member of the resuscitation team to assessing the VAD and all its components, including controller, connections and battery function. If spare batteries are not available, then the patient should be connected to AC power.

If the patient has an older (first-generation) VAD (e.g. Heartmate XVE), then an external 'hand-pump' can be used to provide cardiac output, otherwise usual ALS algorithms apply and all the usual ALS drugs can be given at standard doses.

It is important to note that most VAD manufacturer guidelines state to perform CPR 'only if absolutely necessary'. This is because chest compressions might lead to damage to the VAD itself or physical dislodgment of the outflow graft on the aorta.[54] If chest compressions have been administered, function and positioning of VAD must be confirmed once the patient is stable.

Pre-hospital challenges and further education

The management of patients with VADs does not stop after implantation, and the lack of training can lead to pre-hospital and emergency care personnel being caught unprepared when responding to a patient with a VAD emergency.[55] There is very little literature on out-of-hospital emergency care for patients with LVADs. Schweiger *et al*.[56] make several suggestions towards improving VAD-related out-of-hospital emergencies, including caregiver education, ambulance team notification of local VAD patients, intensive paramedic training on VAD emergencies and having emergency VAD reference cards present on ambulances.

Geidl *et al*.[57] have demonstrated that instituting basic medical training via reference cards and simulated VAD-emergency scenarios leads to an improvement in accurate diagnostics and management of device emergencies.

Interfacility transportation of critically ill VAD patients represents a complex scenario.[58] It requires an organised training programme, experienced clinicians, detailed protocols and vigilant quality assurance.[59] If a patient is transferred from your ED to the local VAD centre, ensure all of the patients' equipment, including extra batteries, are sent with them and consider including the patient's carer in the transport as they might be the best expert on the device in case the patient becomes unconscious en route.

Conclusion

Patients with VADs pose both diagnostic and management challenges to the emergency physician. This review is intended to arm emergency medicine clinicians with an understanding of ventricular assist devices and an approach to their complications.

1. Primary survey should always come first.
2. Make early contact with your local (or nearest) VAD coordinator or cardiac transplant team.
3. Check the device function early and address the alarms systematically.
4. Utilise the patients' family or carer; they might be able to assist in device troubleshooting and maintenance.
5. Hypotension can be a non-specific marker of pathology. It is important to be systematic in your assessment and consider the common diagnoses of sepsis and haemorrhage.

6. The VAD is preload dependent, but afterload sensitive.
7. Check an ECG on all VAD-implanted patients.
8. Consider both VAD-induced complications plus general medical and surgical pathology when developing your differential diagnoses.

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Author contributions

CP carried out literature review and wrote the first draft. BT did the revision and final approval of the article.

Competing interests

None declared.

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