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Clinical Review

APPROACH TO COMPLICATIONS OF VENTRICULAR ASSIST DEVICES: A CLINICAL REVIEW FOR THE EMERGENCY PROVIDER

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☐ **Abstract—Background: Heart failure is a major public health problem in the United States. Increasingly, patients with advanced heart failure that fail medical therapy are being treated with implanted ventricular assist devices (VADs). Objective: This review provides an evidence-based summary of the current data for the evaluation and management of implanted VAD complications in an emergency department context. Discussion: With a prevalence of >5.8 million individuals and >550,000 new cases diagnosed each year, heart failure is a major public health problem in the United States. Increasingly, patients with advanced heart failure that fail medical therapy are being treated with implanted VADs. As the prevalence of patients with VADs continues to grow, they will sporadically present to the emergency department, regardless of whether the facility is a designated VAD center. As a result, all emergency physicians must be familiar with the basic principles of VAD function, as well as the diagnosis and initial management of VAD-related complications. In this review, we address these topics, with a focus on contemporary third-generation continuous flow VADs. This review will help supplement the critical care skills of emergency physicians in managing this complex patient population. Conclusions: The cornerstone of managing the unstable VAD patient is rapid initiation of high-quality supportive care and recognition of device-related complications, as well as the identification and use of specialist VAD teams and other resources for support. Emergency physicians must understand VADs so that they may optimally manage these complex patients.** © **2019 Elsevier Inc. All rights reserved.**

☐ **Keywords—complications; heart failure; VAD; ventricular assist devices**

INTRODUCTION

With a prevalence of >5.8 million and 550,000 new cases diagnosed each year, heart failure is a major public health problem in the United States; in addition, advanced heart failure has a 5-year survival rate of 50% (1). In an effort to improve this dismal statistic and improve quality of life, patients with advanced heart failure who fail medical therapy are increasingly being treated with implanted ventricular assist devices (VADs).

As the prevalence of patients with VADs continues to grow, they will increasingly present to emergency departments (EDs), regardless of whether the facility is a designated VAD center. As a result, it is important for all emergency providers to become familiar with the different types of VAD devices and to be aware of common

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complications associated with VADS. The ability for an emergency provider to recognize and stabilize the common VAD complications is critical and potentially life-saving (2).

Advanced heart failure is defined as an ejection fraction <25% and by the New York Heart Association symptoms class IIIB and IV, includes shortness of breath, and evident limitation in physical activity (3–5). Management options for advanced heart failure include medical therapy, automatic implantable cardioverter defibrillator implantation, heart transplant, and the use of mechanical circulatory systems, such as VADs. Cardiac transplant is considered the criterion standard treatment for refractory end stage heart failure; however, it is fraught with issues of patient eligibility, limited availability of organs for transplant, and morbidity caused by chronic immunosuppression and graft rejection (6). In part, these limitations have driven recent progress in VAD technology and the rapid increase in the number of patients treated with VADs.

Patients with VADs are generally divided into 2 groups—those for whom a VAD is intended to be a temporary measure and those for whom a VAD is definitive therapy. The first group includes patients where a VAD is intended to be a temporary therapy while awaiting a transplant, known as "bridge-to-transplant" (BTT), or those with acute heart failure that may be reversible, known as "bridge-to-recovery" (BTR). As of 2013, the most recent year for which data are available, 42% of heart transplant patients were receiving BTT VAD therapy before transplant, an increase from 20% in 2010, and a number that is likely even higher now (7,8).

In select patients with acute heart failure caused by potentially reversible conditions, such as dilated cardiomyopathy and myocarditis, BTR VAD therapy can induce beneficial structural and molecular remodeling. VAD therapy causes considerable changes in the atrial diameter and volume index and can induce electrical remodeling that improves cardiac function in patients with varying conditions (9). While VADs inserted for this reason currently comprise a minority of total devices implanted, a better understanding of the molecular remodeling that occurs in patients on VAD therapy as a BTR may lead to VADs being a useful tool in the treatment for a variety of cardiac conditions (10).

For patients that are transplant ineligible, VADs are used as a definitive "destination therapy," and this group has grown most rapidly. From 2008 to 2013 there was a progressive increase in VADs implanted as destination therapy (11). VADs have progressed from first-generation pulsatile-flow devices to second- and third-generation continuous flow devices. Second- and third-generation devices provide a more compact design and are less susceptible to mechanical wear than the older pulsatile or displacement (pusher-plate pumps) devices they replaced. While pulsatile pumps simulated systole and diastole of the cardiac cycle, the bulkier, more complex machinery led to greater device malfunctions, and continuous flow devices were found to be preferable in optimizing perfusion when compared with pulsatile flow devices. With a simpler design, VADs are associated with fewer complications and improved quality of life in BTT/BTR and destination therapy patients, and have been the standard of care since 2007. Older generation pulsatile models are rarely used and will not be discussed here (12). The most common types of VADs in use today are the HeartMate II and III models (Abbott Laboratories, Lake Bluff, IL) and the HeartWare HVAD model (Medtronic, Minneapolis, MN). These devices differ slightly in their components and alarm mechanisms, which are detailed in Table 1. In this review, we describe common complications and current approaches to initial management and stabilization in the ED.

METHODS

To complete a review of the literature, we searched PubMed and MEDLINE databases with the following search terms: "ventricular assist device complications" and "emergency department." Two senior authors reviewed articles bibliographies for consideration of additional studies. Exclusionary criteria included studies that were not published in English, abstracts, unpublished data, and duplicate articles across multiple search criteria. A total of 90 articles were found. Guideline statements, device manuals, and nonsystematic reviews were included. A total of 50 peer-reviewed articles were included in this review. Studies identified then underwent a comprehensive review from which a guideline of ED management could be compiled.

DISCUSSION

While patients with VADs can generally expect an improved quality of life, complications are unfortunately common. More than 50% of new VAD patients have an ED visit within the first month after implantation and have an average of 7 visits to the ED in the first year on VAD therapy (22).

VAD patients can present with a variety of symptoms, with some of the most common being bleeding, chest pain, and syncope (22). Presentations can also include non–device-related complaints, such as trauma or dysrhythmias, as well as device-specific issues including device malfunction, thrombosis, infection, and hemorrhage. The management of patients undergoing VAD therapy presents a unique set of challenges to emergency physicians, and this section of our review aims to present the

Table 1. Review of Most Common Complications, and Bedside Assessment

Complication	Timing	Incidence, %	Type of Alarm	Priority Signal	Bedside Assessment	Management
Pump failure	Early*	13 (13)	HM II/III • "Low flow – call hospital contact" HVAD • No message	HM II/III • Red HVAD • Blank display	Assess connection to power source and confirm correct battery insertion	• Manage clinical conditions associated with pump failure • Contact VAD team
Power disruption	Early	HVAD, 44 (13) HM II/III, 23 (13)	HM II/III • Yellow or red battery icon • "Connect power immediately" HVAD • "Controller fault/ failed," "change controller" • No display: loud continuous alarm	HM II/III • Red HVAD • Flashing yellow	Assess external controller-to-driveline, and controller-to-power supply connection; if connections are secure: confirm charged status on VAD display	• If connections are intact and device is charged, controller device exchange may be necessary • Consult the VAD team
Driveline damage	Early	HM II, 21 (13) (over 3 years) HVAD, 0 (13)	HM II/III • "Connect driveline" HVAD • "VAD stopped" – "connect driveline"	HM II/III • Red HVAD • Blank display	Assess driveline for damage and patient for frank cardiogenic shock	• Notify appropriate surgical team and VAD team • Manage patients presenting with frank cardiogenic shock with vasopressors and ionotropic support
Pump thrombosis	Early	3.7 (14)	Low flow/high power HM II/III • "Low flow – call hospital contact" • Flashing red heart HVAD • "Low flow – call"	HM II/III • Red HVAD • Flashing yellow	Evaluate for frank cardiogenic shock or pulmonary edema; confirm thrombosis as cause with bedside sonogram showing both ventricles enlarged and dilated	Activate: • VAD surgical team immediately Check: • Elevated haptoglobin, lactate dehydrogenase, anemia and hematuria Initiate: • Anticoagulation, potentially including tPA, early and on a case-by-case basis • Fluids and diuretics cautiously • Cardiogenic shock management; monitor hemodynamics

(*Continued*)

Table 1. Continued

Complication	Timing	Incidence, %	Type of Alarm	Priority Signal	Bedside Assessment	Management
Non–pump device malfunction	Early	80 (within 3 years of device implantation)	HM II/III • "Call hospital contact" • "Replace controller" HVAD • No message	HM II/III • Yellow HVAD • Blank display	Assess controller and display for functionality	
Restarting a VAD	Early		HM II/III • "Low flow – call hospital contact" • Flashing red heart HVAD • "VAD stopped" – "connect driveline"/ "change controller" OR • No message	HM II/III • Red HVAD • Flashing red • Blank display	Assess thromboembolic risk and determine downtime; verify cause of device cessation	Stable patient • Short downtime (minutes): restart device, considering initial cessation cause • Long downtime (≥1 h): do not restart device, transport patient to nearest VAD center Unstable patient • Attempt device restart • Transport patient to nearest VAD center • Continuous anticoagulation therapy if necessary
Cardiac arrest	Sudden		Literature did not specify how device would alarm		Assess patient stability: • Utilize Doppler to assess brachial artery for MAP • Auscultate heart to listen for "whirring" of functional device	• Use clinical judgment to decide whether to perform chest compressions • In stable patients, weigh risk of device dislodgement vs. need for chest compressions • Fluids and vasopressors should be administered to patients presenting with shock

(*Continued*)

Table 1. Continued

Complication	Timing	Incidence, %	Type of Alarm	Priority Signal	Bedside Assessment	Management
Hemorrhage	Early (first month after implantation)	14	Literature did not specify how device would alarm		Determine: • Date of implantation • Device model • Source of the bleed Assess volume status: • Use bedside ultrasound to confirm hypovolemia as cause of low-flow alarm	• Stabilize hemodynamics • Determine the source and gain control of the bleed; consult GI team as necessary • For refractory GI bleeds, decrease device speed • Stop anticoagulation, resuscitate with fluids or blood products. • Order coagulation panel and type and screen • Target INR for a VAD patient is 2–3
Anticoagulation reversal	Early	2 (14)	Literature did not specify how device would alarm		Determine risk of thrombosis	• Consult VAD team to make anticoagulation reversal decision
Ventricular dysrhythmia	Early	33 (15)	Low flow Low power Low pulsatility index Steady pump speed HM II/III • "Low flow – call hospital contact" • Flashing red heart HVAD • "Low flow – call"	HM II/III • Red HVAD • Flashing yellow	• Obtain ECG • Obtain vitals and MAP • Place patient on cardiac monitor	• Treat ventricular tachycardia/dysrhythmia rapidly pharmacologically and electrically • Unresponsive patient: follow ACLS protocol • Conscious patient: • Lower device speed • Begin intravascular volume repletion • Manage dysrhythmia
Atrial dysrhythmia	Early	10 (16)	HM II/III • "Low flow – call hospital contact" • Flashing red heart HVAD • "Low flow – call"	HM II/III • Red HVAD • Flashing yellow	• Obtain ECG • Obtain vitals and MAP • Place patient on cardiac monitor	• Manage atrial dysrhythmia as would be done for general population

(*Continued*)

Table 1. Continued

Complication	Timing	Incidence, %	Type of Alarm	Priority Signal	Bedside Assessment	Management
Infection	Early	37 (17)	High flow	No indications in literature for how high flow would alarm	• Assess for infection • If pocket infection is suspected, early consultation with a VAD surgeon is critical	• Activate VAD surgical team • Broad-spectrum antibiotics, antifungals when appropriate • Monitor INR carefully for anti-coagulated patients • Culture samples from the driveline and cannula for gram stain, KOH, bacterial and fungal cultures
Stroke	Late† (≥6 months after implantation)	9.8 (18)	Literature did not specify how device would alarm.		• Assess for stroke	• CT angiogram of the head/neck • Basic laboratory assessments including anticoagulation • MRI is contraindicated due to possible device damage. • Carefully consider tPA administration
Right ventricular failure	Late	13–40 (19)	HM II/III • "Low flow – call hospital contact" • Flashing red heart HVAD • "Low flow – call"	HM II/III • Red HVAD • Flashing yellow	Assess: • Hepatic congestion, tricuspid regurgitation, peripheral edema and elevated central venous pressures	• Administer ionotropic agents to increase right ventricular contractility • Alert VAD coordinator early

ACLS = Advanced Cardiac Life Support; CT = computed tomography; ECG = electrocardiogram; GI = gastrointestinal; HM II = HeartMate II; HM III = HeartMate III; HVAD = HeartWave HVAD; INR = international normalized ratio; KOH = potassium hydroxide; MAP = mean arterial pressure; MRI = magnetic resonance imaging; tPS = tissue plasminogen activator; VAD = ventricular assist device.

For the HM II/III, red indicates hazard (high priority) and yellow indicates advisory (low priority). For the HVAD, flashing red and blank display indicate a critical priority "hazard" warning, flashing yellow indicates a medium priority warning, and solid yellow indicates a low priority warning.(20,21)

* Less than 3 months after implantation.

† More than 3 months after implantation.

fundamentals of the initial management of VAD complications in order to meet these challenges. After initial assessment of the patient and stabilization of airway, breathing, and circulation (as able), the next step is to determine whether the patient is suffering a device-specific complication, adverse effects of VAD-adjunctive therapies, such as anticoagulation, or from underlying disease processes or comorbidities. (Figure 1).

VAD Complications: Device Failure

Pump failure. Complete pump failure is the most immediately life-threatening device malfunction. Sudden pump failure is most often caused by disconnection from the power source or by incorrectly inserted batteries. Less frequently, sudden pump failure can be caused by pump thrombosis or interruption in the flow of power from the controller to the pump, either by driveline damage or entanglement of external components (13).

Power. VAD function is dependent on connections between the pump, driveline, controller, and power source. Disruption in the continuity of the controller to the power supply/batteries will result in immediate cessation of device function. The first step in evaluating a failing device is to check the external connections between the external controller and the driveline and also between the external controller and power supply. The VAD bag should always be opened to fully expose and inspect the cables of the external controller (23). If all connections are intact and secure, batteries should be checked for proper insertion and adequate charge status. Charge status can be determined by pressing the illuminated button on the VAD display indicating battery level (23).

If pump function does not return after ensuring or restoring the source of power, controller exchange is likely necessary, which will require the assistance of the VAD team.

Driveline damage. With the development of more compact third-generation VAD controllers, the percutaneous driveline that connects the pump to the controller and that contains the power/electronic cables has become slimmer and more flexible. This new design eliminates the need for a peritoneal pocket to be created for implantation but increases the risk of driveline damage. Driveline damage may cause patients with VADs to present to the ED with findings ranging from decreased outflow alarms and no apparent symptoms to frank cardiogenic shock caused by pump shutdown. Drivelines should be assessed for damage in the field by emergency medical services, as

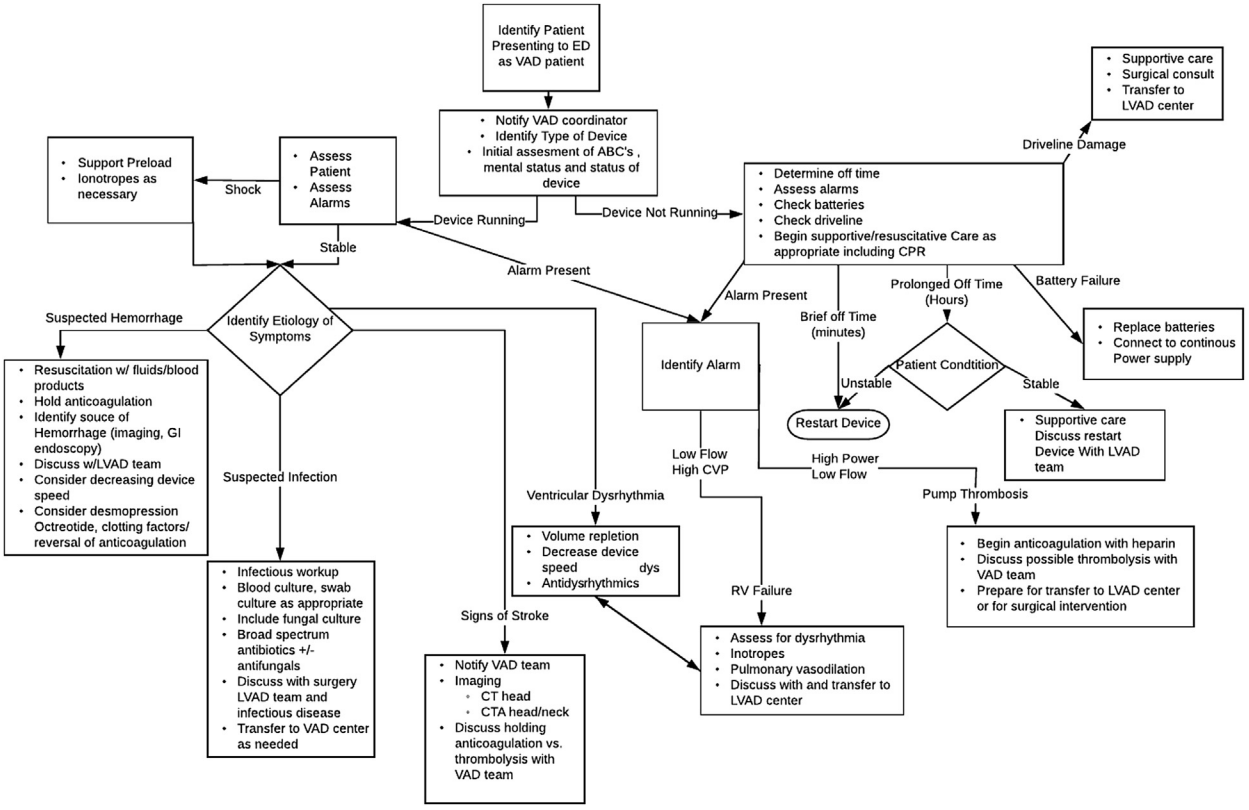

Figure 1. A clinical pathway to approach ventricular assist device complication. ED = emergency department; GI = gastrointestinal; VAD = ventricular assist device; CVP = central venous pressure; LVAD = left ventricular assist device; CPR = cardiopulmonary resuscitation; CT = computed tomography; CVA = cerbrovascular accident.

well as upon arrival in the ED, and the appropriate surgical team should be notified as soon as possible (ideally, prearrival). Patients should be managed similarly to the general population presenting with cardiogenic shock, with vasopressor and inotropic support, until the patient can be brought to the operating room for definitive management.

It is worth noting that there have been reports of emergent reconnection of the driveline at bedside in the ED, where the visible, damaged leads in the driveline were stripped and reconnected by matching colored wires. While this was successful in the case, it in no way replaces surgical intervention (24,25).

Pump thrombosis. Thrombosis can occur within the VAD pump or the internal conduits, even in therapeutically anticoagulated patients. Physiologically, it is analogous to a massive pulmonary embolism, and leads to obstructive shock. Although it is a rare complication, pump thrombosis is extremely dangerous and is associated with significant morbidity and mortality (26). While a confirmed diagnosis of pump thrombosis requires visualization of intraluminal clot in the operating room, suspected pump thrombosis is defined by Interagency Registry for Mechanically Assisted Circulatory Support as the simultaneous presence of hemolysis and new, otherwise unexplained symptoms of heart failure (27). Patients with pump thrombosis may clinically present with pulmonary edema or frank cardiogenic shock (28). The device may be alarming because of low flow and high power consumption as the thrombus causes friction in the pump (23,28). Laboratory evaluation will reveal elevated haptoglobin, significant hemolysis evidenced by elevated lactate dehydrogenase, anemia, and hematuria because of the increased sheer stress on red blood cells created by the thrombus (28).

While there is no universal standard protocol, the cornerstone of medical management of these patients is early initiation of anticoagulation, possibly also with administration of intravenous tissue plasminogen activator (27–29). However, because of the unique thrombolytic threshold of each VAD patient, coagulation management must be contextual and individualized to the patient on a case-by-case basis (27). These cases necessitate early involvement of a VAD surgical team, since for many patients' medical therapy alone is insufficient, and surgical management to facilitate pump exchange is required (27,28,30,31). For these patients, it is crucial to administrate diuretics and fluids with caution, to monitor hemodynamics closely, and to execute resuscitation as would be done with other congestive heart failure patients, with inotropes as necessary (28).

Non–pump device malfunctions. Device malfunctions unrelated to pump failure include controller failure and failure of peripheral components such as the driveline (13). Controller malfunctions have varying degrees of severity from display failure to complete controller shutdown. These non-pump device malfunctions are common; 80% of patients present with some type of device malfunction within three years of device implantation (13).

Restarting a VAD. Other than the obvious hemodynamic concerns with a nonfunctioning VAD, there are also concerns regarding the safety of restarting a stopped device. When flow ceases across a VAD, the risk of clot formation increases significantly. Before restarting a stopped device, the risk of a thromboembolic event must be assessed, factoring the length of malfunctioning VAD downtime as well as the patient's clinical status.

If the device has been off for a short time (i.e., minutes) in a stable patient, the device should be restarted immediately because the risk of a thromboembolic event is low. When the device has been off for longer (i.e., hours) in a stable patient, the device should not be restarted and the patient should be transported to the nearest VAD center. In this situation, the risk of a thromboembolic event outweighs the benefit of restarting the device (32).

If the patient is unstable, a device restart should be attempted regardless of the duration of device failure and, if necessary, continuous anticoagulation can be administered. After restarting the VAD the patient may stabilize but should still be rapidly transferred to the nearest VAD center (32).

Chest compressions. Chest compressions in patients with failing VADs that are not perfusing is an area of controversy. Many VAD manufacturers advise against performing chest compressions because of the risk of dislodging or damaging internal components; however, recent studies have shown that patients with VADs who received chest compressions had increased cardiac perfusion without evidence of an increased risk of device dislodgement (33–35).

Currently there is no clear consensus on whether or not chest compressions are recommended in patients with VADs. Clinical judgment should be used when deciding to perform chest compressions, with the risk of dislodgement and potentially fatal hemorrhage weighed against the immediate necessity of chest compressions. In all patients presenting in shock, fluids and vasopressor medications should be administered, keeping in mind that these devices are preload-dependent.

Medical complications of VAD therapy. While device malfunctions may be more dramatic, they only comprise a minority of total VAD complications. Common

non–device failure complications typically occur within 1 year of implantation and include bleeding, thromboembolic events, infection, dysrhythmias, and a variety of long-term complications (3,36).

Complications that occur outside of VAD malfunction can be divided into immediate complications and long-term complications. It is important for emergency providers to obtain a detailed history, because a thorough knowledge of the duration of implantation and history of prior complications can aid in management.

Immediate Complications

Anticoagulation and bleeding. The management of anticoagulation in patients with VADs is challenging because they are at risk for bleeding and thromboembolic events, especially within the first month after implantation. Compared with older pulsatile models, the overall risk of morbidity from thromboembolic events is lower with continuous flow devices; however, the incidence of post-implantation bleeding has risen (37). Other than pharmacologic anticoagulation, potential biological causes of bleeding in patients with VADs includes impaired platelet aggregation, angiodysplasia, resulting arteriovenous malformations (AVMs) that may bleed, and excessive cleavage of von Willebrand factor because of the pump design, which leads to acquired von Willebrand syndrome (11,38–42).

Management of hemorrhage. As in any major hemorrhage, in a bleeding patient with a VAD the priorities are stabilizing the patient's hemodynamics, determining the source, and gaining control of the bleed. In these patients, any anticoagulation should be stopped, and aggressive resuscitation with fluid or blood products should begin immediately, given the sensitivity to hypovolemia of pump function and cardiac output in these patients. In addition, other modalities that have been proven effective include desmopressin, administered either intranasally (total dose 300 μg) or intravenously (0.3 μg/kg of body weight), and von Willebrand factor/factor VIII concentrate (43).

The most common bleeding location in patients with VADs is the gastrointestinal tract, with gastrointestinal bleeds comprising 14% of all emergency department visits by patients with VADs (37). In addition to the treatments discussed above, vasopressin has proven effective, and treatment with octreotide has demonstrated promise, particularly in those with arteriovenous malformations (44,45). In the presence of refractory gastrointestinal bleeds, the VAD parameters can be altered, such as reducing device speed (46). Determining the location of the bleed may be difficult in these patients, and a gastroenterologist should be consulted early to help identify the source by endoscopy, angiography, or PillCam device (Medtronic, Minneapolis, MN) (47).

In all patients with VADs who are bleeding, laboratory studies, including a complete blood cell count, coagulation panel, and type and screen should be ordered, as should imaging studies for determining the source (2). Although ED care will generally be limited to resuscitation, and will not involve restarting anticoagulation after a bleed, emergency clinicians should be aware that the target international normalized ratio for patients with a VAD is 2 to 3 (48).

Reversal of anticoagulation. While discontinuing anticoagulation is standard in a bleeding patient with a VAD, there is some concern regarding going one step further and reversing anticoagulation. In these cases, as with non–VAD anticoagulated patients, cautious reversal of anticoagulation should be weighed against the risk of thrombosis. Fresh frozen plasma, prothrombin complex concentrates, and vitamin K can be used to reverse warfarin anticoagulation therapy, which is the standard in patients with a VAD. Recent studies have found that reversal of anticoagulation therapy for acute hemorrhage, urgent surgery, or coagulopathy may in fact be associated with a lower potential risk of thrombosis than previously believed (49). Ultimately, the decision regarding reversal of anticoagulation should be made in consultation with the patient's VAD team when possible, but should be kept in mind as an option to address life-threatening bleeding that is difficult to control.

Ventricular dysrhythmias. Although many patients with a VAD also have implantable cardioverter defibrillators, dysrhythmias may occur. Most ventricular dysrhythmias present in the first month after implantation, and patients with a history of ventricular dysrhythmias before implantation are at the highest risk (50,51). Postoperatively, ventricular dysrhythmia risk may be increased because of the creation of a re-entry circuit near the inflow cannula in the left ventricle (52). Life-threatening ventricular dysrhythmias can be masked by VADs because the continuous flow devices maintain blood flow despite the electrical disturbances; however, the continuous flow does not account for the damage to the right ventricle (RV) by the dysrhythmia (53). The external VAD controller will reflect low flow when presenting with hypotension caused by dysrhythmia. Ventricular tachycardia and ventricular fibrillation should be promptly treated pharmacologically and electrically (54). In patients with VADs, this RV failure is life-threatening because the device will not function without adequate preload (52).

If a patient with a VAD with a ventricular dysrhythmia becomes unresponsive, defibrillation or cardioversion is

indicated as per usual Advanced Cardiac Life Support guidelines (32). If the patient is conscious, the initial step in treating ventricular dysrhythmias is to lower the speed of the VAD and to administer intravascular volume repletion. In patients with ventricular tachycardia, beta-blockers and other antidysrhythmic medications, such as amiodarone or dofetilide, can be used. Mexetiline may be added in refractory cases. There should be no alteration in the patient's baseline anticoagulation regimen (52).

Atrial dysrhythmias. Atrial dysrhythmias are also common in patients with VADs. The treatment of atrial dysrhythmias for post-VAD patients is not different than the algorithm used in the general population (52). Atrial dysrhythmia typically does not impact survival in patients with VADs, but it is associated with a decreased functional status during VAD support (55).

Infectious Complications

While the incidence of infection is lower in patients with continuous flow VADs (cfVADs) compared to the older pulsatile assistive devices, it is still a common complication and major limitation (56). Patients with continuous flow VADs are at risk for major infections, which are classified into the following groups defined by the Interagency Registry for Mechanically Assisted Circulatory Support: 1) localized non–device-related; 2) percutaneous site or pocket infection; 3) pump or outflow tract infection; and 4) sepsis (57). The early recognition of infection is crucial; the external pump controller may indicate "high flow" from loss of tone caused by septic shock (54). A VAD surgeon should be contacted immediately because medical management is initiated when suspected, as replacement of VAD components may be indicated.

Infection management. Empiric treatment for VAD infections should include broad-spectrum antibiotics, including coverage for locally prevalent multidrug-resistant organisms and, in select cases, antifungals (58). Some antibiotics may alter the efficacy of anticoagulation regimens, so careful monitoring of the patient's international normalized ratio is necessary. The initial workup should include blood cultures, a complete blood cell count, and a chest radiograph. If driveline or cannula infection is suspected, samples should be taken from the device and sent for Gram stain, potassium hydroxide, and bacterial and fungal cultures. In patients with any suspected pocket infection, early surgical consultation is crucial (59).

Late Complications

While the risk of certain complications, such as infection and dysrhythmias, are greatest in the first 3 months postimplantation, long-term hemodynamic alterations place these patients at risk for a different set of complications, in particular stroke and right ventricular failure.

Stroke. Turbulent blood flow through the VAD pump, coagulation cascade activation on the blood–device interface, and the need for pharmacologic anticoagulation places patients with VADs at high risk for both ischemic and hemorrhagic stroke. From 6 months postimplantation onward, stroke is the most frequent cause of death in patients with continuous flow VADs, implicated in 20% of all deaths in this group and split approximately evenly between embolic and hemorrhagic stroke (11,36,60). Patients with VADs presenting with new neurologic symptoms should undergo a standard stroke workup, including a computed tomography scan of the head and a basic laboratory assessment, with particular attention paid to coagulation parameters. Patients with VADs with suspected stroke should also undergo computed tomography angiography of the head and neck and assessment of the VAD as a possible source of thromboembolic event. Magnetic resonance imaging is not recommended in patients with VADs because it can cause pump failure and injury to the patient (46,61). Patients with hemorrhagic stroke should have all anticoagulation discontinued, and patients without computed tomography evidence of hemorrhage may be given tissue plasminogen activator; however, it is controversial because of the associated bleeding risk. All patients with VADs with suspected stroke should be transferred to a VAD center as soon as feasible for intensive management under the care of both VAD and neurosurgery teams (62).

RV failure. Most patients are supported on VAD therapy only, which may lead to RV failure because of a lack of support. RV failure is associated with typical symptoms of hepatic congestion, tricuspid regurgitation, and peripheral edema. Patients with RV failure typically present with reduced flow detected through the VAD as well as elevated central venous pressure (63). Inotropic agents to aid RV contractility and pulmonary vasodilators to decrease pulmonary pressure should be used in immediate management of patients presenting with RV failure. The VAD coordinator should be contacted and involved early in the management of patients presenting with RV failure (64) (Table 1).

CONCLUSION

The cornerstone of management of VAD failure is high-quality resuscitation, supportive care for heart failure/cardiogenic shock, and early contact with the nearest VAD center. Emergency care of patients with VADs is a

growing area with limited VAD centers available; therefore, all providers should be aware of early complications, including infection, atrial/ventricular dysrhythmias, pump thrombosis, and hemorrhage, as well as late complications, including stroke and RV failure. Although we have described the most common complications, other rare problems may occur. With this critical care foundation in place, emergency providers can begin addressing VAD-specific problems and be comfortable managing this complex patient population.

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ARTICLE SUMMARY

1. Why is this topic important?

With increasing numbers of patients living in the community with ventricular assist devices (VADs), all emergency physicians must be prepared to recognize and initiate management of common complications.

2. What does this review attempt to show?

This review aims to consolidate pertinent key management points and provide useful background information regarding VAD function and complications.

3. What are the key findings?

The cornerstone of managing patients who are crashing and who have VADs is the rapid initiation of high-quality supportive care and the recognition of device-related complications, as well as the identification and utilization of specialist VAD teams and other resources for support.

4. How is patient care impacted?

This review helps emergency physicians better understand VADs and their potential complications to better serve this growing and potentially complex patient population.