Ventricular Assist Devices

What Intensive Care Unit Nurses Need to Know About Postoperative Management

Genevieve O’Shea, RN, BN, CCRN-CSC-CMC

ABSTRACT

Patients with advanced heart failure have limited treatment options despite advances in medical management. Ventricular assist devices represent a surgical option that offers improved end-organ function, survival, and quality of life. Postoperative nursing management involves the most complicated aspects of care following cardiac surgery as well as issues unique to advanced heart failure and mechanical circulatory support. Despite growing numbers of ventricular assist device implants, literature about the challenging care of patients following ventricular assist device implantation is limited. This article focuses on the physiological basis for postoperative nursing management strategies and the most important complications of which critical care nurses need to be aware.

Keywords: advanced heart failure, intensive care, mechanical circulatory support, postoperative management, ventricular assist device

Heart failure affects almost 6 million people in the United States, with an additional 500,000 newly diagnosed patients each year.1,2 Despite advances in medical therapies, patients with advanced heart failure continue to have a bleak prognosis, with approximately 50% mortality within 5 years of diagnosis.3 Ventricular assist devices (VADs) offer a therapeutic surgical option that improves end-organ function, survival, and quality of life and that, as a result of improved clinical outcomes and regulatory approvals, is becoming more common.4 Postoperative critical care of patients following implantation of VADs involves the most complicated aspects of post-cardiac surgery care as well as issues unique to advanced heart failure and mechanical circulatory support. This article focuses on the physiological basis for postoperative nursing management strategies and the complications of greatest importance for critical care nurses.

Background

In recent years, the medical management of heart failure has improved, with trials demonstrating clear survival benefits of various medications, such as β-adrenergic blockers, angiotensin-converting enzyme inhibitors, and angiotensin II receptor blockers, as well as technologies, such as pacemakers and defibrillators.4-7 Despite these advances, the prognosis for patients experiencing end-stage heart failure remains grim. Cardiac transplantation remains the definitive therapy for the condition, but so few donor organs are available relative to the affected population that the procedure has minimal impact overall—3175 people currently wait for heart transplantation in the United States, although only 2333 transplants were performed in 2010.8 In addition, thousands more patients with advanced heart failure are ineligible for transplant for clinical or psychosocial reasons.

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Bridge to Where?
In recent years, mechanical circulatory support devices have become a viable option for those who are unable to receive a heart transplant—whether through ineligibility (because of age or comorbid physical or psychosocial conditions), lack of desire, or lack of a donor organ. The landmark Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure trial, completed in 2001, proved conclusively that devices were not just a match for medical treatment of end-stage heart failure but were superior in terms of survival and quality of life. The 1-year survival rate for those with VADs was 52% compared with 25% for those treated medically ($P = .002$).9

Devices originally conceived as an alternative to transplantation (permanent or “destination” therapy) are also used as a “bridge” to enable transplant-listed patients to survive as they wait ever-increasing lengths of time for a donor organ. Beyond preventing patients with end-stage cardiac failure from dying before transplant, VADs permit recipients to complete cardiac rehabilitation and improve their physical condition before transplant.10,11 Indeed, a minority of patients (<10%) recovered sufficient cardiac function while supported by a device to enable explant and avoid the need for transplantation.12,13 The criteria to predict who will recover are unclear and still being studied. At this stage, recovery and device explant rates are specific to the institutions where the surgeries take place.

Ventricular Assist Devices
Ventricular assist devices are categorized by their function and design. Critical care nurses should understand their similarities and differences because they affect the monitoring, troubleshooting, and ultimately the clinical management of patients. All device systems include a blood pump (externally or internally placed) that connects via a cable through the skin to a controller or driver (Figure 1).
controller is the “brains” of the system, controlling power to the pump, managing pump speed, and reporting alarms and other operating parameters to the user. The controller attaches to AC (wall) power or batteries or both. Systems may have a display screen on the controller, a separate hospital monitor, or both. Systems differ in size, weight, and the amount of blood they are able to pump, but most devices intended for durable support provide up to 10 L/min of pump flow or output.

Device Function
Function is categorized as either “pulsatile” or “nonpulsatile.” Pulsatile devices are earlier-generation devices that contain a blood sac, which fills with blood delivered from the left ventricle (LV) and then empties into the aorta. This intermittent ejection of blood resembles the function of the native heart and produces a palpable pulse and blood pressure.

Nonpulsatile devices are newer-generation technologies that have fewer moving components and as a result are more mechanically reliable than older style pumps. The pumps contain a moving blade known as an impeller. The device operating speed varies depending on the pump design. The impeller blades are designed to optimize blood flow paths and to cause less blood cell damage than the mechanical bearings and valves found in older pumps. In addition, blood lubricates the impeller and prevents mechanical wear-out, which limited the long-term application of earlier-generation devices.

Blood circulates through the pump via cannulas attached to the LV and ascending aorta. The cannulas are named for blood flow direction relative to the pump; the inflow cannula delivers blood from the ventricle into the pump, and the outflow cannula carries blood from the pump to the aorta (see Figure 1).

VAD Flow Physiology

Pulsatile Pumps
Pulsatile devices sequentially fill and empty, mostly during native cardiac systole, as the ventricle empties into the blood sac. The pump cycle is unrelated to the native electrical or mechanical cardiac activity. Patients have palpable pulses as the pump fully fills and then ejects blood in a bolus, resembling native cardiac ejection. Note that the pump-produced pulse is not synchronous with native electrical systole. Indeed, even if the patient has a dysrhythmia, such as ventricular fibrillation or asystole on cardiac monitor, he or she will still have a palpable pulse because of ongoing pump ejection, although it is likely to be at a reduced rate, as the pump may fill more slowly because of reduced delivery from the right side of the heart. Pulsatile pumps are preload dependent, and the rate of ejection depends on the speed at which the blood sac can fill. Pump output is estimated by multiplying known blood sac volume by pump rate.

Continuous-Flow Pumps
Continuous-flow (nonpulsatile) devices transmit flow throughout the cardiac cycle during both systole and diastole, but the greatest augmentation is during diastole when there is usually no native flow. Most blood flows into the pump during systole, as this is when the LV ejects into the pump and the pressure difference between the ventricle and aorta is smallest. Compared with patients who do not have VADs, recipients of continuous-flow devices have lower systolic pressures and elevated diastolic pressures, resulting in significantly diminished pulse pressures. An average pulse pressure width in recipients of continuous-flow VAD is approximately 5 to 15 mmHg. Clinically, at usual pump speed settings, patients may not have aortic valve opening or palpable pulses (see Figure 2).

The higher the pump speed is set, the less pulsatility exists, as the pump effectively empties the LV before it can generate much systolic pressure. If the speed is set too high or delivery of blood to the LV is reduced, the pump may decompress the LV too much and effectively collapse the LV walls together, which is known as a suction event. Conditions that may result in reduced blood delivery to the LV (and therefore to the pump) include those that result in either absolute or relative hypovolemia: bleeding, right ventricular dysfunction (RVD), pulmonary hypertension, cardiac dysrhythmias, pericardial tamponade, systemic vasodilation, and hypotension.

Postoperative Nursing Management
Early postoperative management after VAD implantation focuses on avoiding the known potential complications of both cardiac surgery in general and VAD implantation specifically. Later, management goals shift to optimizing the cardiovascular status of the stable device.
on the monitor are independent events—one is the electrical activity of the native heart, and the other is the mechanical activity of the VAD pump. The VAD-generated pulse is either set by the user (fixed-rate mode) or determined by how quickly the pump can fill and empty (volume or automatic mode).

Recipients of continuous flow devices usually have minimal pulsatility, so pulse oximetry and automated blood pressure cuffs generally work as usual. The blood pressure target in the patient with a pulsatile pump is as usual. Hypertension can impede pump ejection because of elevated afterload and requires management of either the patient or the pump parameters (and generally both). Note that the VAD-generated pulse and the heart rate seen on the monitor are independent events—one is the electrical activity of the native heart, and the other is the mechanical activity of the VAD pump. The VAD-generated pulse is either set by the user (fixed-rate mode) or determined by how quickly the pump can fill and empty (volume or automatic mode).

Recipients of continuous flow devices usually have minimal pulsatility, so pulse oximetry and automated blood pressure cuffs tend to work intermittently, unreliably, or not at all. These patients require the use of invasive arterial pressure monitoring in the postoperative period, and blood pressures must be determined via the use of Doppler ultrasonography once the arterial catheter is removed. Target blood pressure with a continuous flow device is based on the device operating characteristics as well as the perfusion pressure required for end-organ function.

Figure 2: Continuous-flow VAD hemodynamics. This bedside monitor display of a postoperative patient illustrates the typical hemodynamics seen in the continuous-flow VAD patient: narrowed arterial pulse pressure at 12 mm Hg, CVP maintained moderately high at 15 mm Hg, and the low arterial pulsatility leading to failure of the oxygen plethysmograph probe to register a reading.

Abbreviations: CVP, central venous pressure; VAD, ventricular assist device.
Target mean arterial blood pressure (or Doppler-derived arterial pressure) in recipients of continuous flow devices is 65 to 80 mm Hg, with a recent article defining hypertension in the continuous flow VAD patient as mean pressure more than 90 mm Hg.\textsuperscript{15,16} 

**Low Pump Flow/Output**
In the patient who is hemodynamically VAD-dependent (ie, no aortic valve opening and, therefore, no native cardiac output), low pump flow/output is a hemodynamic emergency. Pump flow index (pump output/body surface area) should be maintained at more than 2.2 L/min/m\(^2\) to preserve end-organ perfusion.\textsuperscript{19} Note that devices generally have no direct measure of true output. The pump flow seen on the screen is an estimate based on pump speed and power required to maintain that set speed and, therefore, must be considered within the entire clinical context.\textsuperscript{17} Under normal operating conditions, flow estimates are relatively accurate and as a trend are extremely useful to track pump function and patient perfusion. In the early postoperative period, continuous cardiac output data are useful to compare to the VAD flow estimate, and any large discrepancy in measurement should be investigated. No published data are available regarding the accuracy of continuous cardiac output readings in recipients of continuous-flow pumps, but the additional information gained may be useful for comparison with pump parameters.

Other measures used to assess adequate systemic perfusion, such as mixed venous oxygen saturations or lactate levels, may be used in combination with clinical assessments, such as normal mentation, peripheral warmth, brisk capillary refill, and adequate urine output. Monitoring parameters—whether directly or indirectly measured—cannot substitute for the
clinical assessment of patient status and symptoms. In most cases, the pump can be set at a fixed speed (within a known range based on experience), and the patient hemodynamics can be managed clinically with medications and volume status management rather than “technically” via pump adjustments.

Pump output is a function of preload and afterload. Treatment of low device output must focus on improving preload, or blood entering the pump, or reducing blood pressure, or afterload. In the absence of right-sided heart failure, initial treatment of low flows involves intravenous delivery of fluids—colloid, crystalloid, or blood products, depending on patient laboratory parameters and clinician preference. Recommended volume status in these patients with severe heart failure is central venous or pulmonary capillary wedge pressure of 10 to 15 mm Hg to generate sufficient pump flows. Filling pressures will fluctuate significantly in the postoperative period with post–bypass diuresis, bleeding, extravascular fluid volume shifts, and vasodilation, and they must be closely monitored. Optimal filling pressures will vary by patient and should be decided by correlation with pump output, blood pressure, and perfusion measures such as mixed venous oxygen saturation and serum lactate level. Heart rate may be optimized with cardiac pacing. Cardiac output increases with heart rate to a certain limit, beyond which further increases are counterproductive as diastolic filling time and thus stroke volumes are reduced. Mean arterial pressures should be maintained between 65 and 90 mm Hg, and most patients will require the support of vasoactive medications to achieve these pressures.

Pump output in recipients of continuous flow devices may also be reduced in the case of elevated afterload related to arterial vasosonstriction. In this case, warming, intravenous fluid infusion, and/or pharmacological vasodilators may be required. Hemodynamic parameters such as blood pressure, pump and cardiac outputs, and pulmonary artery catheter–derived information should be considered in combination to decide on the required treatment option. Preload, afterload, and contractility must be addressed to optimize the patient’s hemodynamic status, generally in that order.

Right Ventricular Dysfunction
Right ventricular dysfunction is a significant risk following left VAD implantation and is associated with increased postoperative morbidity and mortality. Many candidates for a VAD have valvular dysfunction and RVD preoperatively, which is exacerbated by surgery and extended cardiopulmonary bypass. The effective unloading of the LV by the VAD and subsequent increase in venous return to the right ventricle (RV) may also overwhelm the compensatory capacity of the RV. If the RV usually receives 2 L/min of blood, and this rate is suddenly increased postoperatively to 5 or 6 L/min with VAD assistance, the RV may distend and struggle to eject. Right ventricular function may be impaired further by septal shift from right to left caused by the unloading of the LV by continuous-flow devices. For these reasons, some experienced centers recommend limiting the VAD speed postoperatively to avoid septal shift and overdistention of the RV.

Any increase in RV afterload may worsen RVD and precipitate failure. Conditions that may cause acute elevations in pulmonary vascular resistance include pulmonary hypertension, fluid overload, acidosis, hypoxia, ischemia, atelectasis, or pulmonary embolus. Several authors have looked at preoperative factors that are predictive of post-VAD right-sided heart failure. Need for preoperative mechanical ventilation, extracorporeal membrane oxygenation, or mechanical circulatory support has been implicated as contributing to postoperative RVD. Low preoperative pulmonary artery pressures and right ventricular stroke work index are generally bad signs—especially if central venous pressure is elevated and approaches pulmonary pressures—because this indicates that the right ventricular function is so poor that it cannot generate enough pressure to overcome even low afterload and is likely to be sensitive to even minimal postoperative insult. Low preoperative pulmonary artery pressures and right ventricular dysfunction in the setting of pulmonary hypertension usually responds at least somewhat to the improved unloading by the VAD and pulmonary vasodilator medications postoperatively.

Right ventricular dysfunction may be similar to cardiac tamponade, with low VAD or cardiac outputs despite adequate filling pressures. Development of jugular vein distention or rising central venous pressure despite no additional fluid infusion should cause suspicion for RVD. Pulsatile device pump rates will decline as supply from the failing right side of the heart to the pump falls and impairs device
filling. Continuous flow pumps may induce suction events noted by falling pulsatility indices as right ventricular contractility becomes further impaired and the left VAD preload is reduced, which, in turn, can cause dysrhythmias as the ventricular tissue is irritated. Rhythm disturbances and suction events in the setting of adequate filling pressures should always cause suspicion for RV impairment.

The treatment for impaired right ventricular function is to reduce the afterload or increase the right ventricular contractility or both. Right ventricular afterload is lowered by reducing pulmonary vascular resistance with pulmonary vasodilator therapy. Inhaled nitric oxide therapy has been used fairly widely following left VAD implantation; however, studies have found conflicting evidence as to the reduction in RVD or need for right VAD. Other pharmacological options include prostaglandins, sildenafil, isoproterenol, or milrinone. Inotropic infusions such as epinephrine may be added to improve right ventricular contractility. Increased filling pressures (such as central venous pressure of 15 to 20 mmHg) may be required to obtain adequate right ventricular output; however, fluid infusion should be used cautiously as overdistention of the RV may worsen the dysfunction. Ventilator settings may be manipulated to address hypoxia, acidosis, pulmonary edema, or atelectasis.

Hemorrhage
Potential Causative Factors. Device recipients generally have multiple significant bleeding risks, including some or all of the following:

- previous cardiac surgery,
- cardiopulmonary bypass,
- prolonged operative time,
- recent anticoagulant or antiplatelet medications,
- renal failure,
- hepatic coagulopathy,
- postoperative anticoagulation,
- device-induced coagulopathies,
- malnutrition, and/or
- advanced age.

Preoperative Factors. The risk of cardioembolic stroke in patients with advanced heart failure, particularly those with arrhythmias preoperatively, often leads to treatment with anticoagulant and/or antiplatelet agents, such as warfarin, aspirin, dipyridamole, or clopidogrel. These agents generally have long half-lives and require cessation at least 3 to 5 days prior to surgery, which is not possible in the case of urgent device implants. Preoperative use of such agents has been associated with postoperative bleeding complications, especially when used in combination.

Severe cardiac insufficiency often leads to congestion-related hepatic dysfunction, which can cause deficiencies of the coagulation factors and predispose patients to severe bleeding if untreated. In some VAD implant centers, patients are treated preoperatively with vitamin K to correct international normalized ratio. Preoperative renal failure is also associated with increased bleeding risk, primarily because of uremia-related platelet dysfunction, anemia, or poor medication clearance.

Intraoperative Factors. Many recipients of a VAD have undergone previous cardiac surgery, with resultant adhesions that may prolong the operative time and increase the number or severity of bypass-related clinical sequelae. Bypass can lead to significant postoperative bleeding by inducing a complex combination of hemostatic defects related to hypothermia, hemodilution, activation of the coagulation cascade, endothelial cell and tissue injury, foreign material contact, coagulation factor consumption, platelet activation and dysfunction, and hyperfibrinolysis.

Postoperative Factors. A frequent cause of post-VAD implant hemorrhage may be premature or overaggressive postoperative anticoagulation. The risk of thrombotic complications in the early postoperative course is generally far outweighed by the bleeding risk, so it is prudent to delay institution of anticoagulation therapy until perioperative bleeding has slowed or stopped (eg, chest tube drainage at the rate of <50 ml/h for 3 consecutive hours). A recent publication suggests that heparin infusion should not be used at all postoperatively but patients should receive warfarin therapy directly when perioperative bleeding has settled.
Device factors may also lead to coagulation abnormalities and bleeding tendency. These factors include interaction of the blood with artificial surfaces, mechanical effects on coagulation factors, and high flow rates through small gaps, such as those within continuous flow devices, that may lead to high shear forces on the blood. Excessive shear can lead to platelet degradation and dysfunction as well as destruction of other blood factors, such as von Willebrand factor. Antiplatelet therapy may be more important to use with these devices than anticoagulation therapy, but it is not yet clear at which point these shear stress-induced abnormalities develop, or what the optimal management strategies should be. The knowledge base regarding the complex interaction between coagulation and devices is still evolving. A recent publication suggests that thromboelastography may be used in addition to routine coagulation parameters postoperatively, although there is insufficient evidence to recommend routine use. (See Kurien and Hughes in this issue for a further description of thromboelastography.)

Postoperative nursing measures to avoid and treat hemorrhage include monitoring and correction of hematological disturbances by administration of blood products or medications as indicated. Patients with a device usually have at least 2 chest drains in place as well as a surgical drain in the pump pocket if present. All drains should be closely monitored for bleeding rates in the postoperative hours. Sudden, intense bleeding via chest or pocket drains may be suggestive of a technical bleed due to dislodged or damaged vessels, which requires operative repair. Bleeding may also be related to coagulopathies, requiring transfusion of blood products. Blood gas analysis may be performed on suspicious bleeds to assess whether the oxygen content is arterial or venous. Strict maintenance of normotension is important, as hypertension can cause additional strain on suture sites, leading to increased bleeding.

Additional measures to avoid hypertension include administering appropriate sedative and analgesic medications. Patients undergoing cardiac surgery may return to the intensive care unit quite cold from the operating room. Hypothermia can impair coagulation; however, slow rewarming is recommended to avoid large changes in bleeding rates or filling pressures as body temperature increases and the vasculature dilates. Positive end-expiratory pressure provided by the ventilator may also be increased to assist with reduction of bleeding, although studies have shown mixed results.

**Cardiac Tamponade in VAD Patients: Recognizing the Signs**

Cardiac tamponade is a known complication of cardiac surgery and occurs when bleeding or thrombosis compresses the ventricle and impairs ventricular contraction and relaxation. It is one of the most common reasons for hemodynamic instability following VAD implantation. Cardiac tamponade occurs most commonly in the immediate postoperative period; however, late tamponade can be associated with institution of anticoagulation or removal of invasive catheters (such as epicardial pacing wires). Chest drains are usually left in place until pacing wires are removed for this reason. Any leakage at the ventricular apical anastomosis can collect and cause tamponade. Cardiac tamponade sufficient to impair hemodynamic status requires surgical evacuation. Prompt recognition of tamponade is critical because the VAD can maintain flows until the condition has advanced to a life-threatening level. The presenting signs of tamponade are summarized in Table 2. The cardinal signs are hypotension and low cardiac/pump output despite adequate intravascular volume and hemodynamic support.

**Cannula Obstruction**

Cannula obstruction is one potential cause for low pump flow despite adequate volume and can occur because of malpositioning or blockage of the cannula. If the ventricular apex was not cleared of thrombus or cardiac trabeculae during implantation, matter can intermittently or continuously occlude the inflow cannula lumen at the base of the ventricular cavity. In this case, you may be able to see reduced pump power consumption on the VAD monitor (power falls because less blood flow is going through the pump; thus, workload, and power consumption, is reduced). Auscultation over the device may elicit an intermittent chattering sound as the device is unable to obtain fill because of the obstruction. Echocardiography can determine whether the inflow orifice points toward the mitral valve (desirable) or is malpositioned toward the septum or LV free wall and becoming intermittently occluded.
Increased flow velocity or turbulence through the cannula may be suspicious for a reduced lumen diameter.\textsuperscript{15,20} Cannula obstruction must be surgically repaired.

**Cardiac Dysrhythmias**

Patients undergoing any type of cardiac surgery are susceptible to rhythm disturbances for a number of reasons: electrolyte imbalances, mechanical trauma, ischemia, increased sympathetic stimulation, and medications.\textsuperscript{19} In addition, many patients with advanced heart failure have preexisting dysrhythmias, especially atrial fibrillation. Optimization of electrolyte levels (particularly potassium and magnesium), and pacing via epicardial wires, is important in the early postoperative phase to maximize delivery of blood to the device and to prevent areas of stasis within the atria, which may lead to formation of blood clots.

Recipients of a VAD, especially those with continuous flow devices, have the added risk of partial or total decompression of the LV caused by fluid unloading via the device (ventricular collapse or suction events). When this occurs, ventricular tissue becomes irritated and arrhythmias such as ventricular tachycardia may occur.\textsuperscript{15} The VAD will function regardless of dysrhythmias as long as there is blood coming into the pump; however, flows will be compromised as delivery is reduced from the right side of the heart. The VAD monitor may show reduced pump flows, pulsatility indices, and pump speed/rates.\textsuperscript{15,17} Pulsatile pumps in automatic modes will have decreased rates resulting from reduced preload. Continuous flow pumps with suction detection may display pump speeds that intermittently ramp down as they recognize suction events occurring.\textsuperscript{17} Eventually if preload is not restored, more severe or sustained dysrhythmias may be induced as the ventricular walls are further collapsed. Adding intravascular volume or slightly decreasing the pump speed often resolves this complication and reverses dysrhythmias.\textsuperscript{15,17} Optimal pump speed setting to avoid excessive ventricular unloading is usually chosen using echocardiography to assess left ventricular end-diastolic diameter and aortic valve opening.\textsuperscript{15,20}

Ectopic cardiac beats should be considered a warning sign, especially when not present preimplant, and laboratory parameters such as potassium, magnesium, and oxygen levels should be monitored and treated.\textsuperscript{19} Excessive pump speed should also be considered as a causative factor for ectopic beats. Recipients of

**Table 2: Signs of Tamponade in Recipients of Ventricular Assist Devices**

<table>
<thead>
<tr>
<th>Clinical Sign</th>
<th>Rationale</th>
</tr>
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<tbody>
<tr>
<td>Unexplained low VAD flow ($&lt; 3 \text{ L/min}$) or cardiac output (measured via pulmonary artery catheter) despite adequate intravascular volume (CVP or PCWP 10-15 mm Hg)</td>
<td>Heart is unable to fill correctly and, therefore, device filling is impaired</td>
</tr>
<tr>
<td>Narrowed pulse pressure (beyond that expected from a device recipient)</td>
<td>Systolic pressure falls as the pressure increases in the pericardium and device is unable to empty the heart</td>
</tr>
<tr>
<td>Elevated filling pressures (CVP or PCWP) despite inadequate pump or cardiac output</td>
<td>Pressure increases in the pericardium and device is unable to empty the heart</td>
</tr>
<tr>
<td>Reduced mixed venous oxygen saturation</td>
<td>Poor central perfusion</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Compensatory mechanism to adjust for reduced cardiac output</td>
</tr>
<tr>
<td>Reduced urine output</td>
<td>Reduced renal perfusion</td>
</tr>
<tr>
<td>Sudden cessation in chest tube drainage</td>
<td>Pericardial fluid/thrombus collection</td>
</tr>
<tr>
<td>Decreased electrical voltage on electrocardiograph</td>
<td>Pericardial fluid/thrombus collection</td>
</tr>
<tr>
<td>Enlargement of the cardiac silhouette or widened mediastinum on chest x-ray</td>
<td>Pericardial fluid/thrombus collection</td>
</tr>
</tbody>
</table>

Abbreviations: CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; VAD, ventricular assist device.
VADs often have implanted pacemaker defibrillators, which are deactivated prior to implant surgery. These devices should be reactivated postoperatively before the patient is discharged from the intensive care unit. Regular antiarhythmic medications should be recommenced as soon as medically indicated.

**Secondary Organ Dysfunction**

Secondary organ dysfunction may occur postoperatively because of a variety of insults related to surgery, including ischemia related to periods of hypoperfusion. Care providers must maintain adequate systemic perfusion pressure more than 60 to 65 mm Hg at all times to minimize multiorgan failure, which is associated with significantly increased morbidity and mortality. More important than hemodynamic monitoring and VAD flow estimates, clinical measures should be assessed to confirm that adequate perfusion is present. Peripherical pulses may be difficult or impossible to palpate in the recipient of continuous-flow VAD, but peripheries should be pink and warm with brisk capillary refill. If a pulmonary artery catheter is in place, mixed venous oxygen saturation can gauge central perfusion and determine whether the pump flow index is adequate for the patient’s metabolic needs. Nursing assessment of adequate perfusion should include factors such as adequate urine output and mentation.

Hypotension is not acceptable even if VAD flow estimate is within the desired range. Because of the design of continuous flow pumps, outputs will actually increase as the mean arterial pressure falls because there is less resistance to flow, even in the setting of severe organ hypoperfusion. Remember the following: mean arterial pressure is the determinant of the perfusion seen by end organs and should always be maintained at more than 65 mm Hg, regardless of VAD flow estimate.

**Renal and Hepatic Dysfunction.** Renal and hepatic dysfunction negatively affect outcomes after VAD implantation. Renal and hepatic laboratory values should be monitored daily in the early postoperative period. Urine output should be closely monitored via indwelling catheter for both quality and quantity. With failing renal function despite adequate perfusion, early dialysis may be considered. Preoperative renal failure can predict RVD after VAD implantation, possibly because of the cascade of bleeding requiring transfusion, subsequent pulmonary compromise, and RVD. Right ventricular failure may then lead to hepatic congestion, hypoperfusion, and impairment.

**Hemolysis.** Intravascular hemolysis during VAD support may be induced by different mechanisms, including cardiopulmonary bypass, functional blood trauma related to pump design (excess pneumatic or shear forces), or device malfunction (cannula obstruction or device thrombosis). Significant clinical hemolysis is often a sign of device dysfunction requiring intervention. Hemolysis may be detected by derangements in hepatic laboratory parameters such as lactate dehydrogenase, plasma-free hemoglobin, D-dimers, reticulocytes, bilirubin, and or haptoglobin. Abnormal laboratory results, particularly those associated with clinical signs of hemolysis such as weakness, nausea, jaundice, bilirubinuria, hemoglobinuria, or anemia, should be carefully investigated for association with device malfunction (see the “Device Malfunction” section). Remember that indelicate handling of blood samples can lead to falsely elevated hemolysis parameters. Hemolysis in pulsatile pumps may be mitigated by manipulation of parameters such as positive or negative pneumatic pressures or the fill-to-eject ratios to reduce exposure of the blood within the pump and, therefore, reduce exposure to trauma.

**Respiratory Dysfunction.** Atelectasis is a common complication following cardiac surgery, as the lungs are deflated during cardiopulmonary bypass and postoperative pain may limit deep breathing and coughing for airway secretion clearance. Patients with history of obstructive airways disease may suffer more severe complications, requiring prolonged invasive or noninvasive ventilation. Following cardiac surgery, pleural effusions may occur because of increased hydrostatic pressure, increased capillary permeability, reduced oncotic pressure, or negative intrapleural pressure. Pleural effusion is common following device insertion and may require drainage by insertion of a chest tube (depending on the size and severity). If an inadequate pump speed setting is chosen or fluid is rapidly infused (especially postoperative blood product transfusions), pulmonary edema may occur. Chest x-ray films and
can lead to disruption of the ingrowing tissue that protects the site from infection, so immobilization of pump cannulas and leads should be performed using abdominal binders or drain tube holders as a priority.14,15,46 Dressings should be replaced as frequently as necessary to keep them intact and dry. Little data are available to guide VAD wound care practices. However, it makes sense to err on the side of caution and use maximal barrier precautions when performing wound care and catheter insertion or manipulation. Nutrition should be addressed as soon as practical as many recipients of devices are severely malnourished preoperatively as a result of cardiac cachexia, which increases their infection risk.47 Early mobilization is advised to avoid complications of immobility. Aggressive pulmonary management, including early extubation, incentive spirometry, deep breathing, and coughing exercises, should be implemented early to avoid respiratory complications. Invasive catheters should be removed as soon as possible.15,46

Neurological Dysfunction. Adverse neurological events are possible during and after any surgery, particularly those involving ardiopulmonary bypass and aortic cross-clamping. In addition, recipients of a VAD often have existing or new dysrhythmias, which predispose them to thromboembolic events as well as the addition of an artificial surface into the bloodstream. Postoperatively, thorough neurological assessment should be completed and documented as baseline and then reassessed regularly. Any change in motor or sensory function should be promptly reported and investigated. Patients with implanted devices cannot undergo magnetic resonance imaging because of the device components but may undergo computed tomography as required.15,17 Anticoagulation should be instituted per hospital protocol once perioperative bleeding has ceased and checked regularly to ensure that therapeutic limits are achieved and maintained in a stable fashion. Patients should be educated as to the warning signs of neurological impairment and trained to report any symptoms promptly.

Infection

Infection is one of the most common and devastating complications of VAD therapy and may occur at any site such as VAD lead skin exit site, pump pocket, invasive catheters, lungs, or bloodstream.15,35,46 In-depth discussion of device infections is beyond the scope of this article, but recipients of devices have numerous concomitant conditions that predispose them to infections in both the acute and chronic periods, including malnutrition, obesity, and diabetes.15,35,46,47 Infection control practices for VADs tend to be based on guidance from experts at implant centers. In the immediate postoperative period, care should be taken to maintain asepsis of invasive lines and catheters, administer antimicrobial therapies as ordered, and maintain utmost asepsis and stability of the device cannulas or lead exit site(s).14,15,17,46 Trauma at the lead exit site can lead to disruption of the ingrowing tissue that protects the site from infection, so immobilization of pump cannulas and leads should be performed using abdominal binders or drain tube holders as a priority.14,15,46 Dressings should be replaced as frequently as necessary to keep them intact and dry. Little data are available to guide VAD wound care practices. However, it makes sense to err on the side of caution and use maximal barrier precautions when performing wound care and catheter insertion or manipulation. Nutrition should be addressed as soon as practical as many recipients of devices are severely malnourished preoperatively as a result of cardiac cachexia, which increases their infection risk.47 Early mobilization is advised to avoid complications of immobility. Aggressive pulmonary management, including early extubation, incentive spirometry, deep breathing, and coughing exercises, should be implemented early to avoid respiratory complications. Invasive catheters should be removed as soon as possible.15,46

Device Malfunction

Mechanical Failure

In the immediate postoperative period, device malfunction is unlikely but possible. Patients should always be accompanied by spare power sources such as AC power adapters and batteries, as well as a spare device driver/controller that is programmed to the same settings as the primary driver and labeled as such so that in case of mechanical failure, the backup is ready to use. Pulsatile devices generally have additional hand pumping devices, which allow manual delivery of air and vacuum to the blood pump in case of emergency. These devices should be present in a visible location near the patient at all times (generally in the pocket of the driver case). Note that nonpulsatile devices do not contain valves, so any pump stop (due to mechanical failure or the pump, controller, lead, or power sources) may lead to regurgitant flow of blood from the aorta and back through the pump. It is, therefore, essential to maintain adequate primary and backup power sources to the controller at all times.15,17

Any and all staff who will be assuming responsibility for care of patients with a VAD should be familiar with both the location of the emergency backup equipment and the procedures required to use the equipment. The
primary nurse should ensure that all backup equipment is present during and after any transport off the unit, as it may be inadvertently left behind in other departments or on transport stretchers. Backup cases should be labeled as VAD equipment with contact details for return within the hospital if found.

**Cable Malfunctions**

Device cables, particularly any transcutaneous power leads, should be well protected at all times from both the patient and external forces such as portable x-ray machines and bedrails. External device equipment such as controllers should not be secured to bedrails, which may be moved and subsequently dislodge equipment. Care should be taken to ensure that equipment does not fall from the bed or place tension on any cables. Percutaneous leads should be dressed in a sterile manner, secured to the patient’s abdomen with a catheter securement device, and then additionally protected with an elastic abdominal binder. Prevention of stress on the lead that causes tension or torque at the skin exit site is critically important to maximize skin incorporation at the site and subsequently to reduce infections at the lead exit site that tend to be difficult to eradicate.\(^1\)\(^\text{15,46}\) Cables should be inspected during each shift to ensure their integrity, and any twisting, torsion, or crimping should be corrected. Any break in cable integrity should be reported to the VAD coordinator and biomedical engineering team for assessment and repair.

**Pump Thrombosis**

The other category of device malfunction that is concerning in the postoperative period is that of device thrombosis. Patients may have visible device clot, clinical signs of hemolysis, pump parameter alterations, or laboratory derangements in the absence of clinical compromise. Pump thrombus can occur at any point during device support, from implant to explant. The variety of presentations makes it a challenging condition to diagnose and treat.\(^1\)\(^\text{15,43,45,48}\) Events may be as mild as minor elevations in pump power consumption, which cause no clinical compromise (presumed to be related to cell deposition on the internal impeller of continuous-flow pumps), or as severe as complete occlusion of device flow due to massive thrombosis or devastating neurological injury after dislodgement of loose pump thrombus.\(^1\)\(^\text{14,45,48}\) Pump thrombus may develop in situ or may be ingested from the native heart.

**Pulsatile Pump Thrombosis.** Pulsatile pumps, which are externally placed, are the simplest to manage with regard to device clots, because they can be visually inspected. Stasis of blood remaining in the pump sac is a risk factor for device thrombus. Incomplete emptying may occur as a result of subtherapeutic device settings or patient parameters such as blood pressure or volume status. Hypovolemia in a pump set to respond to volume state (automatic mode) will lead to a reduction in the pump rate, which can increase the duration of each pump cycle and, therefore, the residence time of the blood within the pump. Device settings should be adjusted so that an alarm will sound when the pump rate drops below the desired level to alert the patient to contact his or her medical team.

External pumps should be assessed for complete emptying each pump cycle via pump parameters (such as flow waveforms or fill/empty signals) or via the use of a flashlight shone through the device blood sac. Complete emptying will result in no blood remaining in the sac at end systole, and the white light beam will shine through the empty sac and be seen on the other side. Incomplete emptying will result in blood remaining in the blood sac at end systole, and thus the light shining through the blood in the sac will be pink (“poor,” “absent,” or “negative” flash test). Complete ejection of the blood sac should be documented as “positive flash test.” Incomplete ejection should be documented as seen (eg, “pink, quarter-sized flash”) and treated promptly by increasing either the blood sac eject duration or pressure settings to attain complete ejection. These pump settings may be temporarily increased while patient parameters such as blood pressure are controlled, although settings should be minimized wherever possible to avoid blood damage. Significant pump thrombus in a pulsatile pump, particularly with clinical signs of hemolysis or hemodynamic compromise, may be treated with pump exchange.

**Continuous-Flow Pump Thrombus.** Implanted continuous-flow devices are more difficult to assess for thrombus. Pumps should be regularly auscultated so that nurses are aware of the “normal” sound of a running pump. Devices that are running with thrombus within...
the impeller tend to sound “scratchy,” “grating,” or “rough.” This subjective assessment may strengthen suspicion of pump thrombus but cannot be quantified for diagnostic purposes. Pump parameters such as motor current or voltage may be increased if there is material causing the impeller to drag and therefore increasing pump workload.15,17,45 It is critical, then, to consider that pump flow estimates are based on a few parameters, and one of these is pump power, so anything such as device thrombosis or deposition, that deranges pump power consumption can lead to an inaccurate estimation of pump flow being displayed on the system monitor.51,17,45,48

Clinicians must not be lulled into a false sense of security by a robust flow estimate displayed on the system monitor—the patient may, in fact, be severely hypoperfused with minimal flow through a pump occluded by thrombus.48 Clinical signs of recurring heart failure (tachycardia, dyspnea), increased native pulsatility (such as new aortic valve opening or significantly increased pulse pressure), and hemolysis should cause a high index of suspicion for pump thrombus, even if the displayed pump flow appears normal. Complete occlusion of the pump by thrombus leads to reduced flow through the pump and thus a theoretical reduction in workload and power consumption.17 However, the reduction in blood flow through the pump in combination with increased drag on the rotor to maintain pump rotation at the set speed can lead to a “normal” flow estimate being displayed on the display monitor even in the setting of significant pump thrombus and impending circulatory collapse. A recent case report highlights this problem, including the fact that because of the balance of these pump power consumption factors, no system alarms were activated.48

In the case of either sudden spikes or gradual upward trends of pump power consumption, clinicians can download log files from the monitor and submit them to the company to determine whether parameters are outside normal operating ranges. If pump thrombus or deposition is suspected, treatment options include intensive anticoagulant or antiplatelet infusion, local thrombolytic infusion into the device, and reoperation and pump exchange.15,44,45,48 The treatment decision rests with the medical team; however, patient condition, complications, severity of the associated symptoms, and risks of the procedure must be considered. Early deposition of thrombus may be eradicated by thrombolytic or anticoagulant infusion. In the case of significant thrombus, pump exchange is probably required because of the dual risks of recurrent thrombus and hemorrhage related to the required dose of medications.15,44,45,48

Cardiopulmonary Resuscitation
Chest compressions are generally not recommended by device manufacturers because of the risk of dislodgement of cannulas and laceration of the heart by the implanted hardware.15,17 However, in the setting of cardiac arrest with no output, the options are few, so clinical judgment must determine which advanced cardiac life support measures are instituted. This scenario should be discussed with the attending physician in advance, and clear institutional guidelines should be established to guide treatment in the event of emergency. In the postoperative VAD implant period, reoperation will generally be required for definitive treatment. Current devices do not need to be disconnected from power sources or the patient lead prior to external defibrillation. In the case of internal cardiac defibrillation, the pump lead should be disconnected from the controller.17

Conclusion
Postoperative critical care of recipients of a VAD requires complex physiological knowledge and advanced critical thinking by intensive care nurses. The evidence shows that VADs improve quality of life and survival compared with medical therapies for heart failure; however, they are associated with significant risks and complications, particularly in the peri-implant period. A thorough understanding of potential postoperative complications and management strategies to detect and treat them is vital for critical care nurses.

REFERENCES


