

Temporary Ventricular Assist Devices in the Intensive Care Unit as a Bridge to Decision

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ABSTRACT

Morbidity and mortality in patients with cardiogenic shock remain high despite the recent advances in therapy. New temporary ventricular assist devices (VADs) that are rapidly applied to normalize cardiac output in patients with severe heart failure are being used more frequently. Bridge to decision describes the temporary use of a VAD to stabilize critically ill patients until complete diagnostic tests are performed and decisions about more definitive therapy are made. The CentriMag, Tandem-Heart, and Impella VADs offer versatility for use in many patients and in multiple hospital set-

tings. These VADs provide continuous blood flow, altering the usual assessment of arterial blood pressure. Patients are usually immobilized during support to prevent dislodgement of cannulas. Anticoagulation therapy is commonly required, and bleeding is a frequent complication. Infection prevention measures must be used to avoid septic complications. In the past 10 years, clinical experience with these devices has expanded, but they remain underused.

Keywords: bridge to decision, cardiogenic shock, ventricular assist device

Cardiovascular care has advanced greatly in recent years because of the evolution of pharmacological agents, interventional procedures, cardiovascular devices, surgical procedures, and management protocols. Modern therapy allows more patients to survive acute cardiac events, but the number of patients who are unresponsive to therapy and progress to cardiogenic shock is substantial. The mortality rate for this population ranges from 50% to 70%.^{1,2} Cardiogenic shock refractory to maximal medical treatment is the leading cause of death for patients admitted to the hospital following myocardial infarction.³⁻⁵ Patients with acute decompensated chronic heart failure treated with conventional medical therapy are also at high risk of in-hospital death.⁶ Postcardiotomy shock occurs in up to 6% of all cardiac surgery cases and carries a very poor survival rate even with the most advanced level of circulatory assistance.⁷⁻⁹ Unlike most other

cardiovascular disorders, the incidence and mortality of acute heart failure with cardiogenic shock have not improved for at least the past 3 decades.¹⁰ Consequently, ventricular assist devices (VADs), which can provide high levels of cardiac output support, are being used with increasing frequency. The expanded use of VADs is driving the introduction of this technology to more institutions that provide cardiovascular care, even those that do not offer cardiac transplantation.

Survival following cardiogenic shock requires restoration of cardiac output to a normal range before hypoperfusion causes irreversible end-organ failure.^{11,12} The intra-aortic

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balloon pump (IABP) offers the advantage of rapid deployment in various hospital settings; however, the maximum amount of support is only 1.5 L/min, and most patients require higher levels of support. Adequate circulatory support may require the use of a VAD that has the capability to provide normal physiological flow rates and, when necessary, can be used for univentricular or biventricular support. The left ventricular assist device (LVAD) is used most often because left ventricular failure is common and results in failure to provide adequate systemic circulation. In many cases of cardiogenic shock, a right ventricular assist device (RVAD) is needed along with an LVAD to provide adequate systemic perfusion.¹³

Rapid hemodynamic stabilization with a VAD allows time for complete assessment of a patient's recoverability or for planning definitive therapy. The proper deployment of a temporary VAD in patients with cardiogenic shock may reduce the mortality rate for this critically ill population. In this review, an overview of this technology is provided as VADs are being used with increased frequency in the intensive care unit. Emphasis is placed on management issues that can guide staff caring for this complex patient population.

Bridge to Decision

In recent years, terminology that describes paradigms of care for patients receiving VAD support has evolved. Bridge to decision (BTD) describes the temporary use of a VAD until decisions about more definitive therapy are made or until a thorough assessment of cardiac recovery is made. A somewhat overlapping term is bridge to recovery, which is usually the immediate goal of VAD support. Patients who do not have recovery of cardiac function and have good neurological function may eventually undergo heart transplant, which results in a transition from BTD to bridge to transplant (BTT). Few patients undergo heart transplant while being supported by a short-term VAD because of the lengthy wait time for a suitable donor. Therefore, one outcome of BTD is to transition the patient to a long-term LVAD to wait for a transplant as an outpatient. The use of a short-term VAD followed by implantation of a long-term LVAD for BTT or destination therapy (DT) is referred to as bridge to bridge.

The use of temporary VADs for BTD is not new, but with the advent of newer and more reliable VAD technology, these therapeutic

approaches are becoming more common. Contemporary VAD systems are designed to be biocompatible and reliable and to provide physiological flow rates. The new VAD systems can be applied in a timely manner with the availability of properly trained personnel. The temporary use of VADs for BTD allows recovery of organs damaged by low cardiac output and thorough assessment of neurological function.

After the need for VAD support has been identified, the optimal therapeutic strategy may be uncertain, and it may take several days to complete thorough multisystem assessments. Some patients have the potential to recover cardiac function after varying durations of myocardial unloading. For those who do not have signs of cardiac recovery, long-term VAD support or heart transplantation may be the only option for extended survival. Coronary revascularization or valvular repair may provide sufficient circulatory recovery and allow removal of VAD support. Stabilization of hemodynamics and maintenance of systemic perfusion with VAD support provide the time needed to optimize the patient's condition and to plan therapy.

Indications for Short-term VAD Support

Acute heart failure with cardiogenic shock has many clinical presentations and causes that determine the course of therapy. Upon initial presentation of cardiogenic shock, it is usually not clear whether patients will recover cardiac function or whether they will require long-term LVAD support, transplant, revascularization, or other surgical procedures. The most common causes of cardiogenic shock are acute myocardial infarction, postcardiotomy failure, and decompensated chronic heart failure. Other less common causes include acute myocarditis, posttransplant rejection, peripartum cardiomyopathy, valve abnormalities, and congenital cardiac defects.

After stabilization, the ideal treatment of acute myocardial infarction is prompt coronary revascularization, which can be done surgically or by percutaneous techniques.¹⁴ Patients who do not complete cardiopulmonary bypass weaning and remain in a low cardiac output state may require time to recover from the surgery, or when there is a lack of recovery, they may become candidates for transplant or long-term VAD support. Patients with decompensated

chronic heart failure may respond to medical therapy, but long-term LVAD support as DT or as BTT is a likely approach. Acute myocarditis, posttransplant rejection, and peripartum cardiomyopathy can resolve with a return of normal cardiac function following a few months of VAD support.^{15–18}

Prompt restoration of adequate systemic perfusion is critical for survival following the onset of cardiogenic shock.^{19,20} Cardiogenic shock occurs when the arterial blood pressure is below 90 mm Hg, the cardiac index is less than 2.2 L/min/m², the pulmonary capillary wedge pressure is greater than 15 mm Hg, and these conditions are sustained for more than 30 minutes.²¹ Some institutions further qualify this definition by requiring 2 or more high-dose inotropes with or without IABP support.²² The immediate goal of therapy is to restore cardiac output to avoid multiple-organ failure and reduce ventricular work by unloading the ventricle(s). Initial therapy usually includes inotropes, vasopressors, volume correction, diuretics, anticoagulants, and IABP support. Once clinicians determine that a patient is not responding to this therapy, more aggressive therapy with VAD support is necessary for survival.^{23,24}

The optimal outcome of temporary VAD support is recovery and successful device removal. Assessment of cardiac recovery is not standardized, and protocols vary among institutions. Weaning from VAD support can take place when renal, hepatic, pulmonary, and neurological functions are adequate, with minimal inotropic and ventilatory support and discontinuation of dialysis. Ventricular assist device support is gradually decreased, while hemodynamics, arterial blood gas levels, renal function, and neurological status are carefully monitored. Increasing anticoagulation therapy during VAD weaning is essential to avoid device thrombosis. Once patients are able to maintain acceptable hemodynamics and other organ function while on minimal VAD support, devices can be removed. Following VAD removal, meticulous monitoring must be continued until the patient remains stable for many hours.

For those patients who do not exhibit recovery of cardiac function, other methods for prolonged circulatory support are necessary. Although heart transplantation is a potential option, the wait time is lengthy because of the severe shortage of donor organs. The majority of transplants after VAD support occur when patients have undergone substantial recovery

and rehabilitation. Viable patients with adequate end-organ function and poor cardiac function should be considered for a durable long-term LVAD implant for either BTT or DT. Because of the expense and extensive surgery associated with LVAD implantation, the risk/benefit analysis must be considered carefully. Financial and social resources play a key role in the decision to proceed with BTT or DT. The need for and the availability of a caregiver, such as a family member, are crucial aspects of long-term LVAD support.

Right-sided heart failure necessitating RVAD support often follows elevated pulmonary vascular resistance, which is reversible with pulmonary vasodilators and mechanical support. In these cases, the goal is to provide support for only a few days until right ventricular function improves and pulmonary vascular resistance returns to an acceptable level.

Complications of Temporary VAD Support

Patients requiring temporary VAD support are susceptible to serious complications due to existing comorbidities and cardiogenic shock. Patients with decompensated chronic heart failure often have 1 or more comorbidities, such as hypertension, diabetes, vascular disease, and previous surgery. Hypoperfusion associated with cardiogenic shock results in multiple-organ dysfunction, which contributes greatly to the development of other serious complications such as bleeding and infection.

Most VAD-related complications are preventable with careful attention by the critical care team. The most common complications during temporary VAD support are excessive bleeding, infection, right-sided heart failure, arrhythmias, device thrombosis, and hemolysis.^{20,25–27} Small patients or those with peripheral vascular disease are susceptible to the development of leg ischemia when femoral cannulation is used. Mechanical failure of the VAD is rare, but complications may occur if devices are not properly assembled and operated.

Bleeding

Bleeding is a serious complication of temporary VAD support because it is common and can precipitate other complications.²⁷ Patient-related comorbid conditions such as hepatic dysfunction, malnutrition, thrombocytopenia, and pre-existing antiplatelet or anticoagulant therapy predispose patients to postoperative bleeding.

Patients who have undergone cardiothoracic surgical procedures with cardiopulmonary bypass have the additional risk of postoperative bleeding complications. The bleeding risk is increased throughout VAD support because of the requirement for continuous anticoagulation therapy to prevent device thrombosis. Bleeding risk is also increased by exposure of the blood to the artificial surfaces of the VAD system.^{28,29} In most cases, a continuous heparin infusion is used to maintain clotting times at least twice the normal rate. Alternative anticoagulants may be necessary when heparin-induced thrombocytopenia develops.

Bleeding related to VADs is avoidable by careful attention to the setup of the device and during the insertion of cannulas. All connections between the blood pump and cannula or connecting tubing must be meticulously made and then checked often to avoid any accidental disconnection. Peripheral cannulation sites are common sites of bleeding and should be regularly inspected, especially when bulky dressings cover the area. For VAD systems that have bedside extracorporeal pumps, careful attention must be given at all times to avoid accidental disconnection of cannulas, tubing, and junctions at the blood pump.

Bleeding potential must be frequently assessed during VAD support by performing coagulograms (prothrombin time, activated partial thromboplastin time, international normalized ratio, platelet count), and/or thromboelastogram (TEG). The TEG provides information on the overall ability of blood to maintain hemostasis. Thromboelastogram assesses the strength and elasticity of clots. Results of the TEG include the reaction time (*R* value) for the start of clot formation, the time from *R* to when the clot is 20 mm (*K* value), the angle of *K*, and the maximum amplitude. These 4 parameters are then used to calculate a coagulation index, which provides an assessment of blood coagulability. Many experienced VAD centers use the TEG as the primary guide for anticoagulant and antiplatelet therapy. Blood product administration is important to avoid loss of volume and critical blood components. Packed red blood cells, fresh-frozen plasma, and platelet transfusions are necessary in patients when significant bleeding develops.

Infection

Infectious complications are very common in patients receiving temporary VAD support. Multiorgan dysfunction, malnutrition, surgery

and reoperations, and the presence of multiple intravenous or intra-arterial catheters put these patients at very high risk of localized and septicemic infections. Endotracheal intubation, chest tube drains, urinary catheters, and dialysis cannulas are common and add further to the infection risk.

Ventricular assist device cannulas or drive-lines that exit the chest wall or abdomen are a frequent site of infection and are the leading cause of morbidity in long-term LVAD support.^{30,31} Strict adherence to infection-prevention measures is vital in this population.³²

Right-sided Heart Failure

Development of right-sided heart failure during LVAD support is a great concern because of the associated high mortality rate.³³⁻³⁵ Worsening right-sided heart function is identified when LVAD flow decreases, the central venous and pulmonary artery pressures increase, and laboratory values indicate deteriorating renal and hepatic function. Common causes of right-sided heart failure are fluid overload, increased pulmonary vascular resistance, arrhythmias, and ischemia.³⁶ Right-sided heart failure may develop when the LVAD is unloading the left ventricle, completely causing a shift of the intraventricular septum to the left, thereby decreasing the pumping effectiveness of the right ventricle. In addition, higher arterial flow from the LVAD increases blood return to the right side of the heart, causing further contractile dysfunction due to distention.

The first-line therapy for right-sided heart failure, or as prophylaxis in those patients at high risk, consists of pulmonary vasodilators by intravenous administration or inhalation, such as prostacyclin or nitric oxide, and inotropic medications, such as dobutamine and milrinone. Maintaining adequate ventilation is also carefully assessed to maintain normal gas exchange. Hypoxemia, hypercarbia, and acidosis can increase pulmonary vascular resistance and increase the workload on the right side of the heart. In patients who do not respond adequately to first-line therapy, implantation of an RVAD is necessary.

Hemodynamic Changes

The temporary VADs discussed later provide circulatory assistance by pumping blood continuously throughout the cardiac cycle. An understanding of the altered physiology of continuous-flow VADs is important for avoiding and

identifying complications during support.³⁷ Unlike the natural heart or pulsatile VADs in which blood flow is intermittent, continuous-flow VADs pump blood during cardiac diastole.³⁸ The most obvious hemodynamic effect is decreased arterial pulse pressure, which may be low enough to have a flat pressure waveform. Appropriate interpretation of this altered arterial blood pressure is essential for management of vasodilator therapy. For example, sudden changes in the arterial pressure waveform may indicate improvements in cardiac function (ie, increased pulse pressure) or acute worsening of left ventricular function (decreased pulse pressure). These observations may vary with the type of VAD and are described later.

Another important hemodynamic difference associated with continuous-flow VADs is the effect of preload and afterload on the amount of circulatory support. Unlike the pulsatile VADs, the amount of blood flow through a continuous-flow pump depends on the differential pressure across the pump. For LVAD support, the differential pressure equates to left-sided heart pressure and aortic pressure, and for an RVAD, it equates to right-sided heart and pulmonary artery pressures. Because of the continuous action of the rotating pump impeller, a constant volume of blood is necessary to avoid negative pressure and atrial or ventricular collapse. Collapse of either chamber may result in total occlusion of the inflow cannula with loss of support. Arrhythmias (tachycardia, premature ventricular contractions, fibrillation) are common with left ventricle collapse.

The VAD afterload pressures (aorta for LVAD; pulmonary artery for RVAD) are important in determining the amount of support delivered by the device. High afterload pressures will decrease the amount of flow through the VAD; therefore, systemic and pulmonary arterial hypertension must be avoided during support. Vasodilator therapy is common during temporary VAD support.

A complete understanding of the hemodynamics related to continuous-flow VAD support is important for the entire critical care team. A separate article in this issue provides more detailed information on the physiology of continuous-flow VADs.³⁹

VAD Systems for BTD

Several VAD systems have been used for short-term support in patients with cardiogenic shock. Some of these devices, such as the Bio-Pump

(Medtronic Inc, Minneapolis, Minnesota) or the Sarns Centrifugal System (Terumo Cardiovascular Systems Corp, Ann Arbor, Michigan), are intended for use in cardiopulmonary bypass. The advantage of these devices is that they are readily available in many operating rooms and have been used extensively for postcardiotomy failure. An extracorporeal membrane oxygenation (ECMO) system has historically been constructed from equipment and supplies used in cardiopulmonary bypass with a configuration for use outside the operating room. For the past 3 decades, ECMO has been used primarily to support neonates and adults with respiratory distress syndrome and can be used to support patients with cardiogenic shock and severe hypoxemia.

The MAQUET ECMO (MAQUET Inc, Wayne, New Jersey) system is relatively new and has been used for a variety of indications, including temporary support in patients with cardiogenic shock.^{40,41} Pulsatile VADs of older design include the Thoratec VAD (Thoratec Corp, Pleasanton, California) and the Abiomed AB or BVS (Abiomed Inc, Danvers, Massachusetts) system. All 4 of these systems offer the capability of biventricular support, which is often necessary in patients with profound heart failure. The newer VAD systems that have been designed specifically for short-term use in patients with acute heart failure are the CentriMag (Thoratec Corp, Pleasanton, California), the TandemHeart (CardiacAssist Inc, Pittsburgh, Pennsylvania), and the Impella 2.5 and 5.0 (Abiomed Inc) systems, which are the focus of this article.

CentriMag

The CentriMag device is a small centrifugal-flow blood pump that can produce blood flow in the range of 0 to 10 L/min (Figure 1). Blood flow is generated by rotation of a magnetically levitated impeller that is contained within a clear plastic housing. The rotation of the impeller within a magnetic field eliminates contact between components, resulting in frictionless movement with no heat generation or wear of the moving component. The contact-free frictionless impeller movement reduces or eliminates hemolysis and thrombosis. The pump is placed in a motor housing that provides the electromagnetic force for elevating and rotating the impeller. An ultrasonic flow probe is placed on the tubing for a continuous measurement of the blood flow rate. A console



Figure 1: The CentriMag blood pump, the blood pump in the motor housing, and the bedside control console. Reprinted with permission from Thoratec Corporation.

controls the impeller's rotational speed and displays the pump flow rate. The impeller speed may be adjusted from 800 rotations per minute to a maximum of 5500 rotations per minute. The console also provides alarms for low and high flow rates. Battery power is available for patient transport.

The CentriMag system is versatile and can support a variety of patients. The device is usually implanted through a sternotomy incision with or without cardiopulmonary bypass. Cannulation for this device depends on the size of the patient, the need for univentricular or biventricular support, and whether placement is through an open chest or by percutaneous techniques. Smaller cannulas may be used to accommodate this device in small children.^{42,43} The CentriMag has been adapted for use for ECMO as well by incorporating an oxygenator in the circuit. When placed through an open chest, left ventricular support is accomplished by inserting the inflow cannula into the left atrium or left ventricle, and the outflow cannula in the ascending aorta. For right ventricular support, the inflow cannula is placed in the right atrium and the outflow cannula in the main pulmonary artery (Figure 2). Percutaneous cannulation of the femoral vein and artery with incorporation of an oxygenator also has been used for ECMO support (Figure 3).⁴⁴⁻⁴⁶

The CentriMag system has been used extensively in recent years for indications of myocardial infarction with cardiogenic shock, postcardiotomy shock, right-sided heart failure following LVAD implant, rejection following transplant, and acute myocarditis.⁴⁷⁻⁵¹ A recent clinical trial report on use of this device in patients with cardiogenic shock demonstrated low thromboembolic risk, good device reliability, and good hemodynamic support.⁵² The device

has been used for a wide range of durations; average time of support was 13 days in the trial, but there are reports of patients supported for more than 3 months.^{53,54}

Patient Care Implications

Although the CentriMag blood pump is nonthrombogenic, the combined artificial surface area of the pump, cannula, and tubing is susceptible to thrombus formation. Also, the risk of thrombosis is increased in circumstances of low flow, such as hypovolemia, or during weaning of support. Anticoagulation with low-dose heparin is normally given during support to maintain activated clotting time in the range of 160 to 180 seconds. In

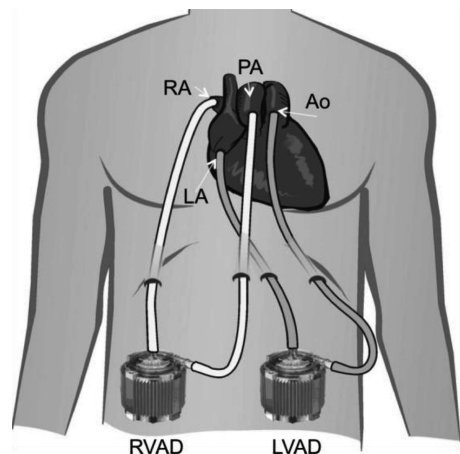


Figure 2: The common configuration of cannulation for univentricular or biventricular support with the CentriMag device.

Abbreviations: Ao, aorta; LA, left atrium; LVAD, left ventricular assist device; PA, pulmonary artery; RA, right atrium; RVAD, right ventricular assist device. Reprinted with permission from Thoratec Corporation.



Figure 3: Cannulas placed percutaneously into the femoral vein and femoral artery.

cases of heparin-induced thrombocytopenia, an alternative anticoagulant may be used, or in some cases, anticoagulation therapy may be withheld.

As is the case with all continuous-flow VADs, the patient’s arterial blood pressure is very different from that generated by the natural heart or pulsatile VADs. Patients with poor ventricular contractility and maximal ventricular unloading by the continuous-flow VAD will have a low pulse pressure (Figure 4). As ventricular function improves and the ventricle generates more cardiac output, pulse pressure will increase. Monitoring the pulse pressure during the weaning process is a valuable tool in the assessment of the patient’s ability to maintain cardiac output.

Most patients who are being supported by the CentriMag device for short-term BTAD will have cannulas exiting the chest wall, or, in rare cases, there is percutaneous cannulation at the femoral site. In either case, patients must remain immobile to avoid cannula dislodgement or kinking. In cases in which support duration is expected to last more than a few days, secure cannulation that allows safe ambulation may be used.^{55,56}

TandemHeart

The TandemHeart PTVA System (CardiacAssist Inc) uses percutaneous cannulation with an extracorporeal centrifugal-flow blood pump (Figure 5A). This LVAD pumps blood

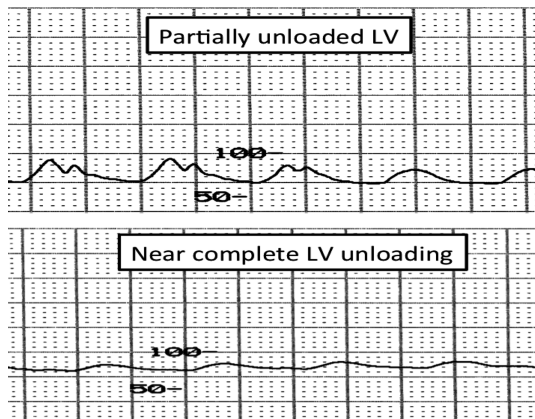


Figure 4: Arterial pressure waveforms during LVAD support with the CentriMag device. The top frame shows partial unloading of the left ventricle with a pulse pressure of 15 to 20 mm Hg and aortic valve opening (dicrotic notch) during 3 of the 5 cardiac cycles. The bottom frame shows increased left ventricular unloading with a pulse pressure of less than 10 mm Hg. No aortic valve opening is available. Abbreviations: LV, left ventricle; LVAD, left ventricular assist device.

continuously from the left atrium to the femoral artery, using a unique cannulation system. Inflow cannula placement is normally performed in the cardiac catheterization laboratory with fluoroscopic guidance or with transesophageal echocardiography by personnel trained in these procedures. One disadvantage of the TandemHeart system is the need for expertise in transeptal procedures, which may not be performed in all cardiac catheterization laboratories. A 21-F polyurethane inflow cannula with an end hole and multiple side holes is passed from the femoral vein to the right atrium and across the atrial septum with the tip in the left atrium for proper support (Figure 5B). The outflow cannula (17 F) is inserted into the contralateral femoral artery, or 2 (15 F) cannulas connected to a Y connector are used, with 1 in each femoral artery. The TandemHeart system also has been adapted for RVAD use with placement of the inflow cannula in the right atrium and the outflow cannula in the pulmonary artery.⁵⁷ After the cannulas are placed, they are connected directly to the pump, which resides on the patient’s anterior thigh during support. Two control consoles are available: one is small and portable, and the other is on a

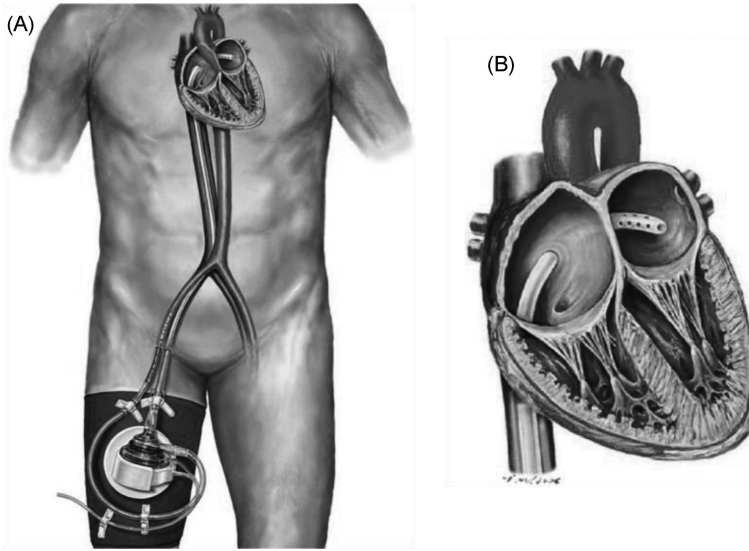


Figure 5: **A,** The TandemHeart system showing cannula placement in the femoral artery and vein with the pump placed on the patient's thigh. **B,** A close-up illustration of the inflow cannula placement across the atrial septum. Used with permission from CardiacAssist Inc.

wheeled cart that is placed at the bedside (Figure 6). The consoles provide power and control to the pump, monitor operation for proper function, and deliver a continuous flow of a heparinized solution to the interior of the pump.

The TandemHeart system uses an ultrasonic flow probe placed on the outflow tubing from the pump to measure device output. The pump flow rate is continuously displayed, and alarms provide audible and visible indicators if the flow falls outside the set range. Also displayed on the console are pump speed and purge fluid pressure. Alerts

for low or high purge pressure are active during operation. Importantly, the system does not monitor cannula position; however, if the inflow cannula tip becomes dislodged and inflow is deoxygenated blood, the blood may change from a bright red to a dark red color. Further assessment of a dislodged cannula is performed with chest radiograph, echocardiogram, and measurement of arterial blood gas levels. Repositioning of the device requires the expertise of a cardiologist trained in transeptal procedures and is performed in the cardiac catheterization laboratory.



Figure 6: The TandemHeart system includes 2 types of control consoles: one is mounted on a large base with wheels (left), and the other is small for portability and may be placed on a bedside table (right). Used with permission from CardiacAssist Inc.

The primary indications for support with the TandemHeart device are cardiogenic shock and high-risk percutaneous coronary intervention.⁵⁸ This device also has been used for support during cardiac operations and postcardiotomy failure.⁵⁹ When used during chest procedures, direct cannulation with cannulas larger in diameter than the percutaneous versions can result in flow rates up to 8 L/min. A recently introduced larger percutaneous cannula is longer and has a greater diameter, which allows blood flow rates of up to 5 L/min when percutaneous cannulation is used for large adults.

Randomized clinical trials that compared circulatory support and outcomes between the TandemHeart LVAD and the IABP demonstrated that hemodynamic parameters are better with TandemHeart support; however, the mortality rate at 30 days was not different.⁶⁰ In the largest single-center experience using the TandemHeart device as a BTD in 117 patients with severe refractory cardiogenic shock or cardiac arrest, the survival rate was 45% at 6 months.⁶¹ This excellent survival rate in a population that has a nearly 100% expected mortality without implantation of a VAD has not been accomplished with IABP support. The likely difference that affected survival between the randomized trial and the single-center experience was the timing of support initiation.

Patient Care Implications

Potential complications related to the TandemHeart device include persistent patent foramen ovale after inflow cannula removal, dislodgement of the inflow cannula, limb ischemia from the femoral cannulation, and thromboembolism. As with all devices that penetrate the skin, infection at the cannula site is a possibility; strict sterile techniques for wound care should always be used. The need for anticoagulation precipitates bleeding at the cannulation site.

The TandemHeart device requires continuous infusion of a heparinized saline solution that functions as a coolant and lubricant for the seal between the rotating impeller and the blood chamber. The purge fluid with heparin prevents clot formation at the seal, and there is minimal systemic anticoagulation. The volume of the purge fluid bag that hangs on the side of the console must be assessed, and it should be replaced when low to avoid running the pump without the infusion.

During TandemHeart support, patients need to be immobilized to prevent damage to

the cannulas at the entry site and to avoid dislodgement of the inflow cannula from the left atrium. Dislodgement of the inflow cannula from the left atrium into the right atrium will result in an immediate loss of oxygen-rich blood flow to the systemic circulation. Instead, when dislodgement occurs, blood will flow from the right atrium to the femoral artery, causing a right-to-left shunt. Inflow cannula dislodgement is apparent when there is a sudden decrease in arterial oxygen saturation, which is usually easily detected by observing a change of the blood in the tubing from bright red to dark red. Other acute changes indicating the lack of support include increased heart rate and decreased blood pressure.

The incidence of persistent patent foramen ovale has been low and is usually not a serious problem.⁶² However, after removal of the device, patients should be carefully monitored for respiratory distress, and arterial blood gas levels should be checked to ensure adequate oxygenation.

The arterial blood pressure should be monitored for changes in cardiac function over the duration of support. When myocardial recovery occurs, the pulse pressure may increase, especially when VAD support is decreased. A test of recovery is to reduce the pump speed and observe for maintenance of adequate blood pressure and for changes in pulse pressure while carefully assessing the patient's ability to maintain cardiac output without an increased need for inotropic support. Weaning should take place when the patient is alert and support with mechanical ventilation and dialysis are no longer needed.

Impella LP and LD Devices

The Impella Recover support system (Abiomed Inc) uses an axial-flow blood pump attached to a flexible cannula with a pigtail catheter tip (Figure 7). When in proper position, the pump portion of the device is in the ascending aorta with the cannula passed across the aortic valve, and the tip is within the left ventricle. Support is provided by the continuous aspiration of blood from the left ventricle into the aorta.

The following 3 versions of the Impella device are used for left ventricular support: Impella LP 2.5, Impella LP 5.0, and Impella LD. The LP 2.5 device is intended for percutaneous insertion via the femoral artery. The 12-F pump and cannula are passed retrograde through the aorta until the cannula tip reaches

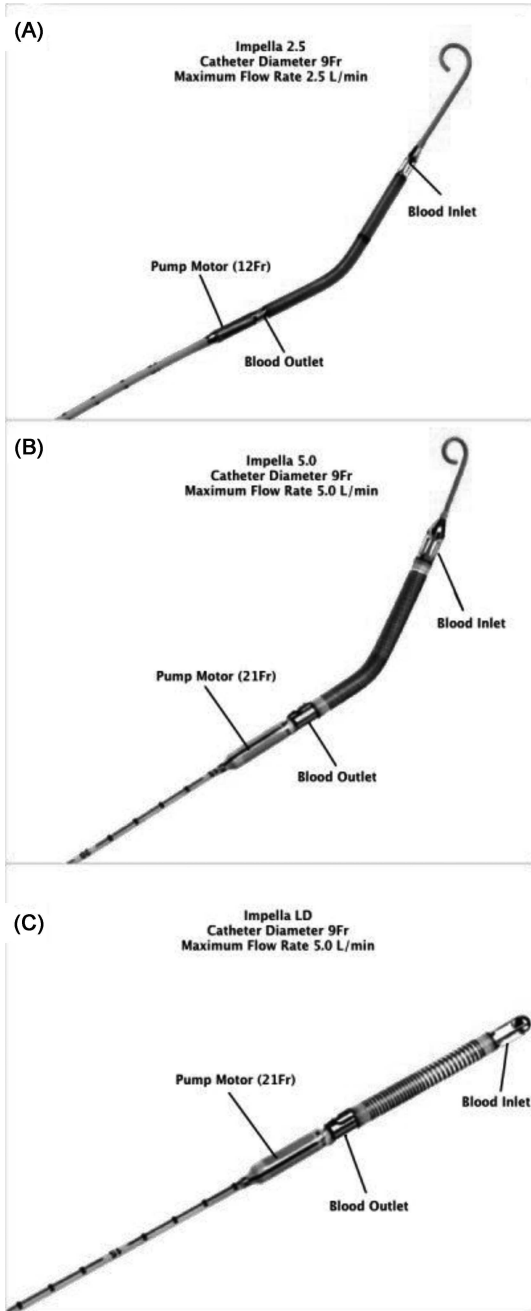


Figure 7: The 3 variations of the Impella device: **A**, 2.5 LP; **B**, 5.0 LP; and **C**, 5.0 LD. The catheter size and maximum flow rate are listed for each device. Used with permission from Abiomed.

the left ventricle (Figure 8). The maximum level of cardiac output support from this device is 2.5 L/min. The LP 5.0 version is larger (21 F) than the LP 2.5 device and can

pump up to 5 L/min. Because of the large size of the LP 5.0, surgical cut down on the femoral artery is required for insertion. Insertion of the pump from the femoral artery requires fluoroscopic guidance. The Impella LD pump is inserted directly into the left ventricle through a graft that is sewn onto the ascending aorta. The pump catheter is connected to a bedside control console that provides power and control for the implanted pump. The console monitors device function and provides alerts for abnormal conditions. Pressure transducers within the cannula allow continuous monitoring of pressure on both sides of the aortic valve, which is used to verify the pump/cannula position. A continuous infusion of a heparinized 20% dextrose solution is necessary to prevent blood from entering the motor. Systemic anticoagulation is not usually necessary. An internal battery provides power to the console for at least 1 hour for patient transport.

The Impella 2.5 device is intended for short-term support of patients with acute heart failure and during high-risk percutaneous coronary interventions. The most frequent indications for use have been postcardiotomy failure,

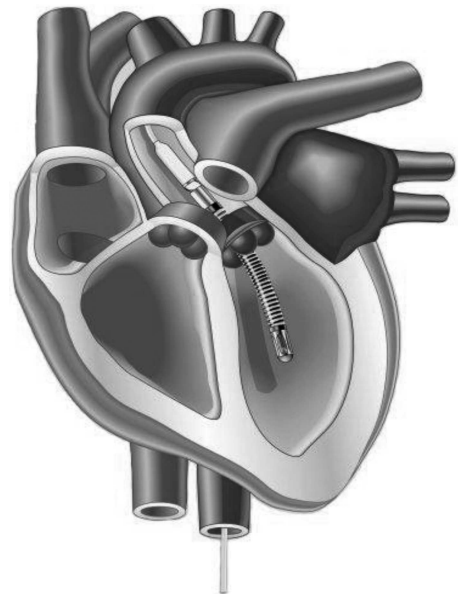


Figure 8: The Impella 2.5 LP device inserted from the femoral artery and passed retrograde through the aorta and across the aortic valve. The blood inlet at the cannula tip is within the left ventricle, and the blood outlet is above the aortic valve in the ascending aorta. Used with permission from Abiomed.

myocardial infarction with cardiogenic shock, acute myocarditis, posttransplant rejection, and decompensated chronic heart failure.^{63–67} Large, multicenter, randomized, controlled clinical trials evaluating the Impella devices for the various indications have not been completed. A meta-analysis of studies in which the Impella devices are compared with the IABP for indication of cardiogenic shock showed that hemodynamics are better with the Impella, but 30-day mortality is not better.⁶⁸ Also, studies comparing Impella and IABP support for high-risk interventions have not demonstrated superiority of Impella support, although the device was safe and hemodynamics were adequate.⁶⁹ A study of postcardiotomy support showed that patients with a native cardiac output of at least 1 L/min had a survival rate of 90%, whereas patients with less than 1 L/min of cardiac output had a survival rate of only 11%.⁷⁰ Because of the limited amount of cardiac output support provided by the Impella devices, patients with severe cardiogenic shock and biventricular failure are better supported by devices that can provide higher levels of support.⁷¹

Contraindications for the use of LP devices include prosthetic aortic valve, calcified aortic valve, and peripheral vascular disease.

Patient Care Implications

Patients are immobilized during support with the Impella devices because of the femoral insertion site. The insertion site should be regularly inspected for bleeding and hematoma, because these patients normally receive a continuous heparin infusion. The head of the bed should not be raised more than 30°, and a knee immobilizer can be used to avoid kinking of the catheter at the insertion site. The catheter and purge tubing from the patient to the console should be carefully positioned, especially during turning and transporting the patient, to avoid dislodgement of the pump/cannula and disconnection from the console.

Because the Impella devices provide only left ventricular assist, worsening right-sided heart function may compromise support. Decreased Impella output and suction alarms may indicate right-sided heart failure or increased pulmonary vascular resistance. Other signs of right-sided heart failure are increased central venous and pulmonary artery pressures and worsening liver enzymes. Severe right-sided heart failure may require biventricular support.

The position of the Impella pump/cannula may change during support and should be frequently assessed. Malposition of the pump/cannula may involve the catheter being too far into the left ventricle or the cannula tip being in the aorta. When a malposition of the pump/cannula occurs, there should be an alarm and a message on the console indicating the problem. Chest radiograph, echocardiogram, cardiac dysrhythmias, and pump performance may be used to assess for proper positioning. The inlet or tip of the cannula should be approximately 4 cm below the aortic valve, and the pump outlet should be well above the aortic valve. When the cannula is too far into the left ventricle, the cannula may be kinked or the inlet area occluded, both of which could cause decreased or absent support. Indications that the cannula is too far into the ventricle are (1) decreased diastolic pressure or widened pulse pressure from the pressure transducers in the cannula as observed on the console, (2) decreased pump flow, (3) premature ventricular contractions, or (4) decreased arterial blood pressure or widened arterial pulse pressure. Ideally, an echocardiogram is used to verify that the pump/cannula is too far into the ventricle, and the position is readjusted with echocardiogram guidance. If an echocardiogram is not readily available and the patient's condition warrants prompt repositioning, the device is gradually pulled back until the diastolic pressure from the cannula transducer increases back to near baseline.

Suction within the left ventricle may occur when the pump speed exceeds the available blood volume. This condition may be detected by alarm messages or premature ventricular contractions. Suction may be caused by low volume in the left side of the heart because of worsening right-sided heart function, increased pulmonary vascular resistance, or an overall decreased blood volume. Also, the pump speed setting may be too high for a patient's normal blood volume, as may occur more commonly in small patients. If the central venous and pulmonary artery pressures and cardiac output are acceptable, the pump speed setting should be adjusted down until the suction events are resolved. Hemolysis may occur if the pump speed is set too high or the patient experiences suction events. Indications of excessive hemolysis include red-tinged urine, low hemoglobin, and elevated lactic dehydrogenase. Measuring

the plasma-free hemoglobin is the best assessment of hemolysis.

The contractile force of the left ventricle may change and alter cannula position. If the ventricular contractility is significantly increased, as may occur with recovery or rapid infusion of inotropic medications, the cannula tip may be ejected out of the ventricle into the aorta. Also, the pump/cannula may be pulled back inadvertently when the catheter is not being properly protected. It is possible that the console will display a message indicating that the position of the cannula is unknown. As mentioned earlier, repositioning should be performed with an echocardiogram or by carefully assessing the cannula pressure waveform. In all circumstances of questionable pump support, the patient's native heart function should be monitored carefully and with appropriate adjustment of inotropic and vasopressor medications.

The purge fluid should be monitored and changed out when necessary. Loss of continuous purge fluid may result in pump failure.

Collaborative Management

The new VAD technology can provide normal levels of cardiac output support soon after the onset of cardiogenic shock, but morbidity and mortality remain high in this population. A multidisciplinary care team is necessary to improve outcomes of VAD support. The refined VAD team includes many professionals who can make important contributions to the care of these challenging patients. Traditionally, VAD programs include a cardiac surgeon, a heart failure cardiologist, and a VAD coordinator or nurse practitioner. Additional medical specialties that can make an important contribution include infectious disease, hematology, pulmonary medicine, nephrology, psychiatry, and palliative care. Hospital staff members who can provide important supportive care include physical therapists, respiratory therapists, pharmacists, and social workers. Hospital administration must be actively involved in the VAD program for appropriate allocation of resources and for managing the financial aspects.

Ventricular assist devices are available in most large academic medical centers that provide comprehensive heart failure care. However, many patients needing advanced care with the most current technology are treated at institutions where VADs are not available. Presently, VAD technology is greatly underused in the

United States. Minimizing the delay in restoring adequate circulation is essential for reducing mortality from cardiogenic shock.⁷² Establishing "hub and spoke" collaborations for the timely transfer of critical patients from smaller community hospitals to academic centers has a positive impact on outcome.⁷³⁻⁷⁵ Accurate communications between referring physicians and heart failure team members are important for the success of the program.

Conclusion

The CentriMag, TandemHeart, and Impella VADs are useful for treating patients facing imminent death from cardiogenic shock or for providing temporary support during high-risk coronary intervention. Like other MCS systems, each device has advantages and disadvantages, and a single ideal system does not exist. These new VAD systems can be applied rapidly, require only moderate-dose to low-dose anticoagulation therapy, provide direct unloading of the ventricles, and deliver physiologically adequate cardiac output.

REFERENCES

1. Goldberg RJ, Spencer FA, Gore JM, Lessard D, Yarzebski J. Thirty-year trends (1975 to 2005) in the magnitude of, management of, and hospital death rates associated with cardiogenic shock in patients with acute myocardial infarction: a population-based perspective. *Circulation*. 2009;119:1211-1219.
2. Barron HV, Every NR, Parsons LS, et al. The use of intra-aortic balloon counterpulsation in patients with cardiogenic shock complicating acute myocardial infarction: data from the National Registry of Myocardial Infarction 2. *Am Heart J*. 2001;141:933-939.
3. Jeger RV, Lowe AM, Buller CE, et al. Hemodynamic parameters are prognostically important in cardiogenic shock but similar following early revascularization or initial medical stabilization: a report from the shock trial. *Chest*. 2007;132:1794-1803.
4. Menon V, Fincke R. Cardiogenic shock: a summary of the randomized shock trial. *Congest Heart Fail*. 2003;9:35-39.
5. Hochman JS, Buller CE, Sleeper LA, et al. Cardiogenic shock complicating acute myocardial infarction-etiology, management and outcome: a report from the Shock Trial Registry. Should we emergently revascularize occluded coronaries for cardiogenic shock? *J Am Coll Cardiol*. 2000;36:1063-1070.
6. Adams KF Jr, Fonarow GC, Emerman CL, et al. Characteristics and outcomes of patients hospitalized for heart failure in the United States: rationale, design, and preliminary observations from the first 100,000 cases in the Acute Decompensated Heart Failure National Registry (ADHERE). *Am Heart J*. 2005;149:209-216.
7. Rastan AJ, Dege A, Mohr M, et al. Early and late outcomes of 517 consecutive adult patients treated with extracorporeal membrane oxygenation for refractory postcardiotomy cardiogenic shock. *J Thorac Cardiovasc Surg*. 2010;139:302-311, 311.e1.
8. Paul S, Leacche M, Unic D, et al. Determinants of outcomes for postcardiotomy VAD placement: an 11-year, two-institution study. *J Card Surg*. 2006;21:234-237.

9. Sylvain EA, Stern DR, Goldstein DJ. Mechanical support for postcardiotomy cardiogenic shock: has progress been made? *J Card Surg.* 2010;25:442–454.
10. Pulido JN, Park SJ, Rihal CS. Percutaneous left ventricular assist devices: clinical uses, future applications, and anesthetic considerations. *J Cardiothorac Vasc Anesth.* 2010;24:478–486.
11. John R, Liao K, Lietz K, et al. Experience with the Levitronix CentriMag circulatory support system as a bridge to decision in patients with refractory acute cardiogenic shock and multisystem organ failure. *J Thorac Cardiovasc Surg.* 2007;134:351–358.
12. Webb JG, Sleeper LA, Buller CE, et al. Implications of the timing of onset of cardiogenic shock after acute myocardial infarction: a report from the Shock Trial Registry. Should we emergently revascularize occluded coronaries for cardiogenic shock? *J Am Coll Cardiol.* 2000;36:1084–1090.
13. Moriguchi J, Davis S, Jocsos R, et al. Successful use of a pneumatic biventricular assist device as a bridge to transplantation in cardiogenic shock. *J Heart Lung Transplant.* 2011;30:1143–1147.
14. Jeger RV, Harkness SM, Ramanathan K, et al. Emergency revascularization in patients with cardiogenic shock on admission: a report from the Shock Trial and Registry. *Eur Heart J.* 2006;27:664–670.
15. Unosawa S, Hata M, Sezai A, et al. Successful management of fulminant myocarditis with left ventricular assist device: report of a severe case. *Ann Thorac Cardiovasc Surg.* 2010;16:48–51.
16. Fayssoil A, Nardi O, Orlikowski D, Combes A, Chastre J, Annane D. Percutaneous extracorporeal membrane oxygenation for cardiogenic shock due to acute fulminant myocarditis. *Ann Thorac Surg.* 2010;89:614–616.
17. Khalife WI, Kar B. The TandemHeart PVA in the treatment of acute fulminant myocarditis. *Tex Heart Inst J.* 2007;34:209–213.
18. Oosterom L, de Jonge N, Kirkels J, Klopping C, Lahpor J. Left ventricular assist device as a bridge to recovery in a young woman admitted with peripartum cardiomyopathy. *Neth Heart J.* 2008;16:426–428.
19. Goldstein D, Neragi-Miandoab S. Mechanical bridge to decision: what are the options for the management of acute refractory cardiogenic shock? *Curr Heart Fail Rep.* 2011;8:51–58.
20. El-Banayosy A, Arusoglu L, Kizner L, Fey O, Minami K, Korfer R. Complications of circulatory assist. *Perfusion.* 2000;15:327–331.
21. Topalian S, Ginsberg F, Parrillo JE. Cardiogenic shock. *Crit Care Med.* 2008;36:S66–S74.
22. Samuels LE, Darze ES. Management of acute cardiogenic shock. *Cardiol Clin.* 2003;21:43–49.
23. Park SJ, Nguyen DQ, Bank AJ, Ormaza S, Bolman RM III. Left ventricular assist device bridge therapy for acute myocardial infarction. *Ann Thorac Surg.* 2000;69:1146–1151.
24. Tayara W, Starling RC, Yamani MH, Wazni O, Jubran F, Smedira N. Improved survival after acute myocardial infarction complicated by cardiogenic shock with circulatory support and transplantation: comparing aggressive intervention with conservative treatment. *J Heart Lung Transplant.* 2006;25:504–509.
25. Myers TJ, Khan T, Frazier OH. Infectious complications associated with ventricular assist systems. *ASAIO J.* 2000;46:S28–S36.
26. Topkara VK, Kondareddy S, Malik F, et al. Infectious complications in patients with left ventricular assist device: etiology and outcomes in the continuous-flow era. *Ann Thorac Surg.* 2010;90:1270–1277.
27. Goldstein DJ, Beauford RB. Left ventricular assist devices and bleeding: adding insult to injury. *Ann Thorac Surg.* 2003;75:S42–S47.
28. Slaughter MS, Sobieski MA, Gallagher C, Graham J, Brandise J, Stein R. Fibrinolytic activation during long-term support with the HeartMate II left ventricular assist device. *ASAIO J.* 2008;54:115–119.
29. Spanier T, Oz M, Levin H, et al. Activation of coagulation and fibrinolytic pathways in patients with left ventricular assist devices. *J Thorac Cardiovasc Surg.* 1996;112:1090–1097.
30. Walker PC, Depestel DD, Miles NA, Malani PN. Surgical infection prophylaxis for left ventricular assist device implantation. *J Card Surg.* 2011;26(4):440–443.
31. Chinn R, Dembitsky W, Eaton L, et al. Multicenter experience: prevention and management of left ventricular assist device infections. *ASAIO J.* 2005;51:461–470.
32. Holman WL, Pamboukian SV, Blood M, Tallaj JA, McGiffin DC, Kirklin JK. Managing device infections: are we progressing or is infection an insurmountable obstacle? *ASAIO J.* 2005;51:452–455.
33. Patel ND, Weiss ES, Schaffer J, et al. Right heart dysfunction after left ventricular assist device implantation: a comparison of the pulsatile HeartMate I and axial-flow HeartMate II devices. *Ann Thorac Surg.* 2008;86:832–840; discussion 832–840.
34. Dang NC, Topkara VK, Mercado M, et al. Right heart failure after left ventricular assist device implantation in patients with chronic congestive heart failure. *J Heart Lung Transplant.* 2006;25:1–6.
35. Cleveland JC Jr, Naftel DC, Reece TB, et al. Survival after biventricular assist device implantation: an analysis of the interagency registry for mechanically assisted circulatory support database. *J Heart Lung Transplant.* 2011;30:862–869.
36. Barnes K. Complications in patients with ventricular assist devices. *Dimens Crit Care Nurs.* 2008;27:233–241; quiz 242–243.
37. Slaughter MS, Pagani FD, Rogers JG, et al. Clinical management of continuous-flow left ventricular assist devices in advanced heart failure. *J Heart Lung Transplant.* 2010;29:S1–S39.
38. Myers TJ, Bolmers M, Gregoric ID, Kar B, Frazier OH. Assessment of arterial blood pressure during support with an axial flow left ventricular assist device. *J Heart Lung Transplant.* 2009;28:423–427.
39. Christensen DM. Physiology of continuous-flow pumps. *AACN Adv Crit Care.* 2012;23:46–54.
40. Arlt M, Philipp A, Voelkel S, et al. Hand-held minimised extracorporeal membrane oxygenation: a new bridge to recovery in patients with out-of-centre cardiogenic shock. *Eur J Cardiothorac Surg.* 2011;40:689–694.
41. Arlt M, Philipp A, Voelkel S, et al. Extracorporeal membrane oxygenation in severe trauma patients with bleeding shock. *Resuscitation.* 2010;81:804–809.
42. Kouretas PC, Kaza AK, Burch PT, et al. Experience with the Levitronix CentriMag in the pediatric population as a bridge to decision and recovery. *Artif Organs.* 2009;33:1002–1004.
43. Hirata Y, Charette K, Mosca RS, Quaegebeur JM, Chen JM. Pediatric application of the Thoratec CentriMag BIVAD as a bridge to heart transplantation. *J Thorac Cardiovasc Surg.* 2008;136:1386–1387.
44. Saeed D, Kizner L, Arusoglu L, et al. Prolonged transcatheter cardiopulmonary support for postcardiotomy cardiogenic shock. *ASAIO J.* 2007;53:e1–e3.
45. Aziz TA, Singh G, Popjes E, et al. Initial experience with CentriMag extracorporeal membrane oxygenation for support of critically ill patients with refractory cardiogenic shock. *J Heart Lung Transplant.* 2010;29:66–71.
46. Fitzgerald D, Ging A, Burton N, Desai S, Elliott T, Edwards L. The use of percutaneous EMO support as a “bridge to bridge” in heart failure patients: a case report. *Perfusion.* 2010;25:321–325, 327.
47. Loforte A, Montalto A, Lilla Della Monica P, Musumeci F. Simultaneous temporary CentriMag right ventricular assist device placement in HeartMate II left ventricular assist system recipients at high risk of right ventricular failure. *Interact Cardiovasc Thorac Surg.* 2010;10:847–850.
48. Jaroszewski DE, Marranca MC, Pierce CN, et al. Successive circulatory support stages: a triple bridge to recovery from fulminant myocarditis. *J Heart Lung Transplant.* 2009;28:984–986.

49. Bhamra JK, Kormos RL, Toyoda Y, Teuteberg JJ, McCurry KR, Siegenthaler MP. Clinical experience using the Levitronix CentriMag system for temporary right ventricular mechanical circulatory support. *J Heart Lung Transplant*. 2009;28:971–976.
50. Shuhaiber JH, Jenkins D, Berman M, et al. The Papworth experience with the Levitronix CentriMag ventricular assist device. *J Heart Lung Transplant*. 2008;27:158–164.
51. Khan NU, Al-Aloul M, Shah R, Yonan N. Early experience with the Levitronix CentriMag device for extra-corporeal membrane oxygenation following lung transplantation. *Eur J Cardiothorac Surg*. 2008;34:1262–1264.
52. John R, Long JW, Massey HT, et al. Outcomes of a multicenter trial of the Levitronix CentriMag ventricular assist system for short-term circulatory support. *J Thorac Cardiovasc Surg*. 2011;141:932–939.
53. Haj-Yahia S, Birks EJ, Amrani M, et al. Bridging patients after salvage from bridge to decision directly to transplant by means of prolonged support with the CentriMag short-term centrifugal pump. *J Thorac Cardiovasc Surg*. 2009;138:227–230.
54. De Robertis F, Rogers P, Amrani M, et al. Bridge to decision using the Levitronix CentriMag short-term ventricular assist device. *J Heart Lung Transplant*. 2008;27:474–478.
55. Gregoric ID, Cohn WE, Akay MH, La Francesca S, Myers T, Frazier OH. CentriMag left ventricular assist system: cannulation through a right minithoracotomy. *Tex Heart Inst J*. 2008;35:184–185.
56. Takayama H, Chen JM, Jorde UP, Naka Y. Implantation technique of the CentriMag biventricular assist device allowing ambulatory rehabilitation. *Interact Cardiovasc Thorac Surg*. 2011;12:110–111.
57. Kiernan MS, Krishnamurthy B, Kapur NK. Percutaneous right ventricular assist via the internal jugular vein in cardiogenic shock complicating an acute inferior myocardial infarction. *J Invasive Cardiol*. 2010;22:E23–E26.
58. Kar B, Forrester M, Gemmato C, et al. Use of the TandemHeart percutaneous ventricular assist device to support patients undergoing high-risk percutaneous coronary intervention. *J Invasive Cardiol*. 2006;18:93–96.
59. Gregoric ID, Bruckner BA, Jacob L, et al. Techniques and complications of TandemHeart ventricular assist device insertion during cardiac procedures. *ASAIO J*. 2009;55:251–254.
60. Burkhoff D, Cohen H, Brunckhorst C, O'Neill WW; TandemHeart Investigators Group. A randomized multicenter clinical study to evaluate the safety and efficacy of the TandemHeart percutaneous ventricular assist device versus conventional therapy with intraaortic balloon pumping for treatment of cardiogenic shock. *Am Heart J*. 2006;152:469.e1–e8.
61. Kar B, Gregoric ID, Basra SS, Idelchik GM, Loyalka P. The percutaneous ventricular assist device in severe refractory cardiogenic shock. *J Am Coll Cardiol*. 2011;57:688–696.
62. Kar B, Adkins LE, Civitello AB, et al. Clinical experience with the TandemHeart percutaneous ventricular assist device. *Tex Heart Inst J*. 2006;33:111–115.
63. Dhar G, Jolly N. Mechanical versus pharmacologic support for cardiogenic shock. *Catheter Cardiovasc Interv*. 2010;75:626–629.
64. Ferreiro JL, Gomez-Hospital JA, Cequier AR, et al. Use of Impella Recover LP 2.5 in elective high risk percutaneous coronary intervention. *Int J Cardiol*. 2010;1145:235–237.
65. Garatti A, Colombo T, Russo C, et al. Different applications for left ventricular mechanical support with the Impella Recover 100 microaxial blood pump. *J Heart Lung Transplant*. 2005;24:481–485.
66. Granfeldt H, Hellgren L, Dellgren G, et al. Experience with the Impella recovery axial-flow system for acute heart failure at three cardiothoracic centers in Sweden. *Scand Cardiovasc J*. 2009;43:233–239.
67. Iliodromitis KE, Kahler P, Plicht B, et al. High-risk PCI in acute coronary syndromes with Impella LP 2.5 device support. *Int J Cardiol*. 2011;153(1):59–63.
68. Cheng JM, den Uil CA, Hoeks SE, et al. Percutaneous left ventricular assist devices vs. intra-aortic balloon pump counterpulsation for treatment of cardiogenic shock: a meta-analysis of controlled trials. *Eur Heart J*. 2009;30:2102–2108.
69. Dixon SR, Henriques JP, Mauri L, et al. A prospective feasibility trial investigating the use of the Impella 2.5 system in patients undergoing high-risk percutaneous coronary intervention (The PROTECT I Trial): initial U.S. Experience. *JACC Cardiovasc Interv*. 2009;2:91–96.
70. Siegenthaler MP, Brehm K, Strecker T, et al. The Impella Recover microaxial left ventricular assist device reduces mortality for postcardiotomy failure: a three-center experience. *J Thorac Cardiovasc Surg*. 2004;127:812–822.
71. Lamarche Y, Cheung A, Ignaszewski A, et al. Comparative outcomes in cardiogenic shock patients managed with Impella microaxial pump or extracorporeal life support. *J Thorac Cardiovasc Surg*. 2011;142:60–65.
72. Joyce DL, Conte JV, Russell SD, Joyce LD, Chang DC. Disparities in access to left ventricular assist device therapy. *J Surg Res*. 2009;152:111–117.
73. Jaroszewski DE, Kleisli T, Staley L, et al. A traveling team concept to expedite the transfer and management of unstable patients in cardiopulmonary shock. *J Heart Lung Transplant*. 2011;30:618–623.
74. Gonzalez-Stawinski GV, Chang AS, Navia JL, et al. Regional referral system for patients with acute mechanical support: experience at the Cleveland Clinic Foundation. *ASAIO J*. 2006;52:445–449.
75. Helman DN, Oz MC. Developing a comprehensive mechanical support program. *J Card Surg*. 2001;16:203–208.