Mechanical Circulatory Support

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INTRODUCTION

Mechanical circulatory support, a term that most emergency physicians did not concern themselves with 10 years ago, is now an area of patient care that emergency physicians must understand. The role of mechanical support for both acute and chronic heart failure is rapidly growing. For example, as of April 2012 more than 10,000 HeartMate (Pleasanton, CA, USA) left ventricular assist devices (LVADs) had been implanted, yet only a handful of the patients who received these devices will go on to receive a heart transplant.\textsuperscript{1} Most of these patients will never receive a transplant and will spend most of their time receiving outpatient care. The use of extracorporeal membrane oxygenation (ECMO) for refractory cardiogenic shock and cardiac arrest from a reversible cause.

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KEYWORDS

- Mechanical circulatory support
- Intra-aortic balloon pump
- Ventricular assist device
- Extracorporeal membrane oxygenation (ECMO)
- Cardiogenic shock

KEY POINTS

- The routine use of an intra-aortic balloon pump is no longer recommended for patients with refractory cardiogenic shock.
- Common ventricular assist device (VAD) complications include thrombosis, bleeding, right-sided heart failure, infection, cerebral vascular accidents, arrhythmias, and device failure.
- Driveline infection is the most common infectious complication for the patient with a VAD.
- Ventricular arrhythmias are most common within the first 3 months after VAD placement and should be cardioverted in a timely fashion.
- Venoarterial ECMO is a treatment modality that can be considered in the patient with refractory cardiogenic shock or cardiac arrest from a reversible cause.
arrest is also rapidly growing. As of January 2013, more than 200 centers provide some type of extracorporeal support and more than 4200 cardiac patients have been treated by extracorporeal cardiopulmonary resuscitation (CPR).^2

This review has several purposes: (1) to discuss the use of the intra-aortic balloon pump (IABP) in patients with cardiogenic shock, (2) to describe the complications and management strategies for the critically ill patient with an LVAD, and (3) to explore the emerging role of ECMO in the emergency department for patients presenting in cardiac arrest.

IABP

The IABP has classically been regarded as the mainstay of mechanical support for patients in cardiogenic shock (CS). This device provides hemodynamic support by augmenting diastolic arterial blood pressure during the normal cardiac cycle. By increasing diastolic blood pressure, IABP counterpulsation is believed to improve coronary perfusion, reduce myocardial oxygen demand, and reduce left ventricular afterload during systole.^3 IABP therapy is the most widely used form of mechanical hemodynamic support in patients with CS secondary to acute myocardial infarction.\(^4\) Historically, American and European guidelines have recommended the use of IABP therapy, giving it a class IB and class IC recommendation, respectively.\(^5,6\) However, an emerging body of literature questions the outcome and mortality benefits of IABP therapy for CS associated with acute myocardial infarction (AMI).\(^7–9\) As a result, American and European guidelines have downgraded the use of IABP therapy to class IIa and IIb recommendations, respectively.\(^10–13\)

Despite the recent controversy surrounding the routine use of IABP therapy, it is important for the emergency physician to be aware of the traditional indications for an IABP, the mechanisms of support, and the contraindications. The traditional indications for IABP insertion include refractory CS caused by AMI, refractory unstable angina, and mechanical causes, such as acute mitral regurgitation, papillary muscle rupture, and ventricular septal defect. Generally accepted contraindications to counterpulsation therapy are aortic insufficiency, aortic dissection, and chronic end-stage heart failure with no anticipation of recovery.

The device consists of a double-lumen 8.0-French to 9.5-French catheter with a 25-mL to 50-mL balloon at the distal end. The IABP catheter is inserted percutaneously using a Seldinger technique through the femoral artery. Traditionally, the IABP balloon is placed after measuring the distance from the insertion point to the manubrium, followed by confirmation with a chest radiograph, or alternatively it can be placed under direct fluoroscopic guidance. Ultrasound is also being used to guide tube placement at some centers.\(^14\) The balloon itself is positioned 2 to 3 cm distal to the left subclavian artery and is inflated during the diastolic portion of the cardiac cycle.

Evidence behind the use of IABP therapy for refractory CS has historically been mixed, especially for CS secondary to myocardial infarction. There is some evidence that IABP therapy reduces preoperative mortality in a subset of patients, including those with CS secondary to acute mitral regurgitation or a ventricular septal defect after myocardial infarction.\(^15\) The routine use of IABP therapy in refractory CS has fallen out of favor. A meta-analysis performed on the use of IABP in ST-elevation myocardial infarction (STEMI) found insufficient evidence to routinely recommend this therapy.\(^16\) These findings were followed by the IABP Shock-II Trial, a multicenter, randomized, prospective study,\(^8,9\) which found no significant reduction in short-term (30-day) or long-term (12-month) all-cause mortality for patients in CS related to AMI undergoing early revascularization. Despite these observations, a small subset of patients,
specifically those with acute mitral regurgitation or ventricular septal defect, could benefit from this intervention. Therefore, it may be considered as a temporizing measure while the patient is being prepared for the operating room.

VENTRICULAR ASSIST DEVICE

Ventricular assist devices (VADs) are being used more commonly today as a durable form of mechanical circulatory support, especially for patients awaiting long-term recovery or transplantation. It is important that the emergency physician be able to recognize the type of device, and become aware of the indications and complications for this type of mechanical circulatory support, as well as how to promptly manage them.

There are 3 major indications for placement of a VAD. Similar to most other mechanical circulatory devices, VADs are used as either a bridge to transplantation, bridge to recovery, or destination therapy. Permanent LVAD implantation (destination therapy) can be performed in certain patients with advanced heart failure and a low chance of transplant. In some patients, a VAD can be considered as a “bridge to decision” when more time is needed to determine if further definitive treatment would benefit the patient. In the most recent American College of Cardiology Foundation/American Heart Association STEMI guidelines, VADs were given a IIa recommendation as an alternative treatment for refractory CS.

There are 2 major types of VADs, which are classified by their mechanism of support. Older, first-generation VADs provide circulatory support through a pulsatile flow pump that is driven by a pneumatic motorized pump. Except for patients with right ventricular failure, these devices are less frequently used today. The older, external VAD pumps are large and bulky, which decreases the patient’s mobility. Pulsatile VADs require frequent pump exchanges, which makes them less desirable as well.

Newer-generation VADs provide circulatory support through continuous-flow pumps that work by axial or centrifugal flow. Continuous-flow pumps have a number of advantages over the older, pulsatile-flow devices. They are often smaller, more durable, and have higher energy efficiency and lower thrombogenicity. These devices are surgically implanted and have minimal external hardware.

VAD commercial brands include the Heart Assist 5 (Houston, TX, USA), HeartWare HVAD (Framingham, MA, USA), Jarvic 2000 (New York, NY, USA), and Thoratec HeartMate II (Pleasanton, CA, USA). The most common LVAD in use today is the axial flow HeartMate II; however, with recent advances in VAD technology, the HeartWare centrifugal flow device is now more commonly inserted. The centrifugal pumps are connected directly to the patient’s left ventricle and lie within the pericardium (above the diaphragm). Blood returns to the ascending aorta by way of a separate return cannula. Axial flow pumps have a left ventricular outflow cannula that is connected in series to an intracorporeal pump located either above or below the diaphragm (Fig. 1).

The evaluation and management of a patient with a VAD can be challenging. It is important that the emergency physician be aware of common complications that can occur with VAD patients, as well as the laboratory and diagnostic tests that should be considered. During the initial evaluation, it is important to ask the patient or the patient’s family how to contact their VAD coordinator and cardiothoracic surgeon. These contacts are often able to provide vital information about the patient and can help coordinate the patient’s care and disposition through the emergency department.

The initial evaluation of any VAD patient should begin with a thorough initial history, physical examination, and laboratory testing. When evaluating the device, the first step should be to auscultate the motor to listen for a characteristic “hum” to ensure the device is running.
Assessing vital signs can be challenging in the patient with a continuous-flow device. These patients often lack central or peripheral pulses if the patient’s intrinsic cardiac contractility is poor. Therefore, their blood pressure should be measured with a manual blood pressure cuff and arterial Doppler. To obtain the mean arterial pressure (MAP), locate either the brachial or radial artery with your Doppler device. Inflate the cuff until you no longer hear auditory flow, then slowly release pressure from the manual cuff. The patient’s MAP is determined at the pressure where arterial flow is heard. The goal MAP is between 70 and 80 mm Hg, with a maximum of 90 mm Hg. Excessive elevations in blood pressure can significantly increase the risk for stroke. Consider arterial line placement early, as this will provide continuous, accurate measurements of the patient’s MAP. Closely examine the VAD driveline for signs of infection, damage, or bleeding (Fig. 2). Last, review the patient’s control box for any alarms and ensure the VAD has adequate battery power (Fig. 3).

Pulse oximetry also can be difficult to obtain, and may be unreliable in patients with a subtle pulse or no pulse. It is important to confirm low values with a rapid arterial blood gas sample.

VADs are preload dependent. For a VAD to run efficiently, the patient’s intravascular volume must be filled appropriately. Hypovolemia can lead to a reduction in VAD power secondary to reduced blood flow.
These devices are also afterload sensitive. Uncontrolled increases in MAP higher than 90 mm Hg will often reduce LVAD power, flow, cardiac output, and distal perfusion. Patients with a MAP greater than 80 are at increased risk of cerebral vascular events (ie, hemorrhage, ischemia) and should be treated promptly with afterload-reducing medications. A flow diagram for the differential diagnosis based on common VAD alarms is presented in Fig. 4.

**Common VAD Complications**

Common complications associated with VADs include thrombosis, bleeding, rightsided heart failure, infection, cerebral vascular accidents, arrhythmias, and device failure. Patients with VADs are at increased risk for bleeding complications, as they are usually on chronic systemic anticoagulation (ie, Coumadin [warfarin]) and antiplatelet therapy (ie, aspirin, dipyridamole). The most common international normalized ratio (INR) target for patients with LVADs is 2.0 to 3.0; however, patients with a HeartWare device target a slightly higher INR of 2.5 to 3.5. The most commonly reported types of bleeding that VAD patients encounter are epistaxis, gastrointestinal, mediastinal, and intracranial hemorrhage. Hemorrhagic events are commonly caused by a supratherapeutic INR, intestinal arteriovenous malformations, or bleeding dyscrasias, such as acquired von Willebrand disease.

**Fig. 2.** Abdominal driveline location with surrounding erythema concerning for cellulitis.

**Fig. 3.** HeatWare control box and battery packs. (A) Battery/power bar. (B) Alarm notification light. (C) Extra battery packs.
Treatment of the bleeding VAD patient should begin with standard therapy to reverse an elevated INR with fresh frozen plasma or prothrombin complex concentrate, if available. Platelet dysfunction due to acquired von Willebrand disease is common and is believed to be the result of platelet exposure to the mechanical sheer stress that occurs with continuous-flow devices. Treatment with desmopressin or cryoprecipitate may be effective. Platelet transfusions may also be necessary if the patient is taking antiplatelet medications. Rapid thrombelastography may assist in targeted blood component therapy to reverse coagulopathy.

VAD pump thrombosis should be considered in any patient presenting with cardiac arrest, decreased pump flow, pump power spikes, recurrent heart failure, or echocardiographic findings that raise concern for abnormal left ventricular unloading. Thrombosis is a worrisome long-term complication, as it can result in stroke, peripheral embolism, heart failure, and death. Thromboembolic events have been reported in 2.7% to 35.0% of all patients. HeartMate devices were once thought to have a lower incidence of thrombosis of approximately 3%, because of their specific mechanical design. However, a recent study detected a rapid increase in HeartMate II VAD thrombosis: up to 8.4% at 3 months for devices implanted after 2011. The reason for this increase is unclear.

Pump thrombus should be suspected if hemolysis is detected by laboratory studies, if the lactate dehydrogenase is greater than 1500 mg/dL or 2.5 to 3.0 times the upper limit of normal, if the patient has hemoglobinuria, or if the plasma-free hemoglobin level is elevated. Systemic anticoagulation with a continuous heparin infusion should be initiated if pump thrombosis is suspected. The VAD team should be consulted quickly to discuss emergent treatment options, including thrombolytic agents, antiplatelet therapy, and the potential need for emergent surgical pump replacement or explantation of the device.

Suction (or “suck-down”) events can occur in patients with decreased left ventricular filling caused by right ventricular failure, restrictive or hypertrophic cardiomyopathies, arrhythmias, or hypovolemia. These patients often present with hypotension. Suck-down events can be visualized under bedside echocardiography as the left ventricular chamber being equivalent in size to the VAD cannula. Prompt recognition and treatment with an intravenous fluid bolus can reverse this problem rapidly.

**Infectious Complications**

Infectious complications are a major concern for patients with implanted hardware and can occur anywhere along the device circuit. Driveline, pump pocket, and device infections can be devastating and occur most frequently within the first 3 months after
placement, although they can occur at any time.\textsuperscript{35,36} VAD infections are the most common cause of death among patients who require this device for long-term mechanical circulatory support.\textsuperscript{35}

The most common location of infection is the driveline insertion site. Patients with infected hardware often present with symptoms of malaise, low-grade fever, and mild tenderness around the driveline. The most common infecting organisms are skin and gastrointestinal pathogens: \textit{Staphylococcus aureus}, \textit{Staphylococcus epidermidis}, \textit{Enterococcus}, \textit{Pseudomonas}, \textit{Enterobacter}, and \textit{Klebsiella} species.\textsuperscript{37–40} Fungal infections have been reported and are estimated to occur in approximately 9\% of VAD patients. \textit{Candida} infections are less frequent than bacterial infections but are responsible for the highest mortality rates.\textsuperscript{41–43}

During implantation, the VAD pump is commonly placed within the pericardium, in an anatomic “pocket” within the intra-abdominal cavity, or in the preperitoneal space below the lateral rectus muscle. The “pump pocket” is at risk for infection because it is contiguous with the driveline. Consider a pump pocket infection in VAD patients presenting with fever, leukocytosis, and abdominal pain, especially in the setting of a driveline infection.\textsuperscript{36,44} An abdominal ultrasound or computed tomography scan is highly recommended to identify a VAD-related fluid collection or abscess.

Pump endocarditis can occur when the hardware itself becomes colonized with a bacterial pathogen. The presentation is similar to that of nonmechanical endocarditis. Symptoms include low-grade fever, signs of septic emboli on physical examination, and even pump obstruction. Sources include the driveline infection, transient bacteremia or fungemia, and other health care-associated infections from urinary or pulmonary sources.\textsuperscript{45} Pump endocarditis carries a particularly high mortality, estimated to be as high as 50\% in several case series.\textsuperscript{36,47}

Treatment of any suspected VAD infection should begin with aggressive fluid resuscitation to maintain adequate preload. Early broad-spectrum antibiotic

Fig. 5. A suck-down event visualized by bedside echocardiography.
administration should include treatment for both methicillin-resistant *S aureus* (MRSA) and gram-negative bacterial infections. Empiric antifungal therapy should probably be given as well. Definitive source control with surgical drainage of the localized fluid collection, device exchange, or explantation for transplant is often required.

**Arrhythmias**

Patients with a VAD may present to the emergency department with cardiac arrhythmia, classified as either primary or secondary, based on the underlying cause.

Primary arrhythmias are intrinsic to the electrical conduction pathways of the heart and occur independently of the VAD itself. Ventricular arrhythmias are common and can be caused by cannula migration, malposition over time, electrical remodeling, secondary scarring, or fibrosis. Because the patient’s cardiac output is supported by the VAD, primary arrhythmias, such as a supraventricular tachycardia or ventricular tachycardia, are often tolerated without clinical signs or symptoms.

A persistent primary arrhythmia can eventually cause right ventricular failure and reduced left ventricular filling, ultimately leading to reduced cardiac output. As a result, primary arrhythmias should be managed in a timely fashion with cardioversion or anti-arrhythmic medications. There is limited evidence to guide the choice of antiarrhythmic medication in patients with a primary arrhythmia. Amiodarone is often used as a first-line agent, although the use of β-blockers can also be considered. Mexiletine can be given during in-patient management for refractory ventricular tachycardia.

Secondary causes of cardiac arrhythmias can occur if the left ventricular septum or free wall is sucked into the VAD outflow tract. Hypovolemia and inadequate pulmonary venous return are the most common causes of a secondary arrhythmia.

In the emergency department, it is often difficult to determine whether the patient’s arrhythmia has a primary or secondary cause. The management of any VAD patient presenting with an abnormal rhythm should begin with a prompt fluid challenge followed by an emergent bedside echocardiogram to determine if the patient would benefit from more aggressive volume resuscitation or adjustments of the VAD settings.

**Mechanical Failure**

Pump failure is one of the most feared complications in VAD patients. Signs of pump failure include an absence of detectable blood pressure by Doppler, the absence of a running motor on auscultation, or the absence of power displayed on the VAD’s control box. If pump failure is suspected, it is important to immediately contact the patient’s cardiothoracic surgeon, VAD engineer, and on-call nurse to obtain vital information about the make and model of the hardware.

In any patient without evidence of perfusion (eg, MAP <40 with at least one other sign of hypoperfusion, such as loss of consciousness or cyanosis), it is important to promptly begin treatment with volume resuscitation and standard advanced cardiac life support (ACLS) interventions. Strongly consider initiation of a heparin infusion if there is any suspicion for pump thrombosis. An epinephrine infusion should be initiated to maximize intrinsic cardiac output. Rapid initiation of veno-arterial ECMO (VA ECMO) can also be considered in conjunction with the patient’s cardiac surgeon.

First-generation VADs can be pumped manually to provide adequate blood flow. In general, it is recommended that the provider deliver 60 to 90 pumps per minute to provide adequate cardiac output if the VAD has failed. Newer-generation VADs generally do not have manual hand pumps. One of the biggest misperceptions about the patient with a VAD is that you should never perform CPR on a patient with mechanical pump failure. Although CPR may increase the risk of device dislodgement, it is not
contraindicated. If the patient continues to have a MAP of zero after 1 minute, connect the patient’s manual hand pump or initiate chest compressions.

ECMO
Overview

Over the years, major advances in technology have increased the availability and ability to use ECMO as a viable form of prolonged mechanical circulatory support. The historical roller pumps used for cardiopulmonary bypass were associated with higher rates of pump-induced hemolysis and lower flow rates compared with the newer centrifugal pump heads. The transition from older bubble-type oxygenators to newer hollow-membrane silicone oxygenators has also reduced blood trauma significantly, allowing increased duration of extracorporeal support.

Two major forms of ECMO circuits are used today. Venovenous ECMO (VV ECMO) is used primarily for patients in refractory respiratory failure. Currently there are no universally agreed on indications for the initiation of VV ECMO, but general indications include refractory hypoxemia, hypercapnia, and the failure of conventional mechanical ventilatory strategies. In general, initiation of VV ECMO is rare in the emergency department, because this modality is chosen well after more advanced resuscitation measures have been attempted.

VV ECMO can be considered the “ultimate” form of lung rest therapy, as ventilator strategies can be used to minimize barotrauma, volutrauma, and biotrauma. In the patient with refractory respiratory failure on ECMO, it is generally acceptable to follow ARDSnet ventilation strategies (between 4 and 8 mL/kg of ideal body weight, with plateau pressures <30 cm H2O), which can be readily used without worrying about alveolar gas exchange.

The second major form of extracorporeal support is VA ECMO. VA ECMO not only provides assistance with gas exchange, but also provides hemodynamic support by way of venous and arterial cannulation. VA ECMO can be considered for any patient with CS that is refractory to standard medical management, especially if the cause of the patient’s shock is believed to be reversible.

Cardiogenic shock can be defined as heart failure resulting in hypotension (systolic blood pressure <80–90 mm Hg, mean arterial pressure <30 from baseline), end-organ dysfunction, cardiac index less than 1.8 (without support) to 2.2 L/min/m² (with support), or evidence of pulmonary edema (objectively defined with a pulmonary capillary wedge pressure >18 mm Hg). When a patient with CS has failed traditional medical management, VA ECMO can be used as a potential salvage therapy; however, although a number of international organizations recognize the role of extracorporeal support, they have not agreed on strict inclusion criteria. The ultimate goal of VA ECMO is to provide temporary support as a bridge to shock recovery, bridge to a more durable form of cardiovascular support (ie, LVAD or total artificial heart), heart or lung transplantation, or termination of further efforts (if futile).

ECMO Pump Set-Up and Cannulation

Current ECMO circuits are composed of heparin-coated polyvinylchloride tubing running to and from the patient, a centrifugal pump, a membrane oxygenator, a heat exchanger, and a bladder reservoir (Fig. 6). Blood is driven through the oxygenator by the centrifugal pump head, warmed to a prespecified temperature, then returned to the patient by way of continuous, nonpulsatile flow. Systemic anticoagulation with unfractionated heparin is used to prevent thrombosis of the ECMO circuit.
VV ECMO cannulation can be performed by a number of methods under ultrasound or fluoroscopic guidance. Traditionally, two 21-French to 28-French cannulas are used. Deoxygenated blood is removed from the inferior vena cava by way of a multi-port cannula inserted into the right femoral vein and advanced to the level of T11/T12. It is important to avoid advancing this catheter higher than approximately the level of T8, as this can lead to hepatic vein obstruction. Deoxygenated blood is then sent to the pump’s oxygenator and finally returned to the right heart through a cannula inserted into the right internal jugular vein.

Access can also be obtained by placing a single, 23-French, dual-lumen catheter (eg, Avalon) in the right internal jugular vein. When using a single-cannula, bicaval approach, the internal jugular ECMO catheter is advanced through the right atrium so that the proximal drainage port rests in the superior vena cava and the distal port rests just beyond the cavoatrial junction in the inferior vena cava. Oxygenated blood is returned through a separate cannula by way of a central port located in the right atrium to provide flow across the tricuspid valve.

VA ECMO cannulation can be performed centrally in the operating room under direct vascular visualization, but is more frequently being performed percutaneously at the patient’s bedside. Using a Seldinger technique with serial dilations, a
21-French venous catheter is inserted into the common femoral vein and a 15-French or 17-French arterial catheter is inserted into the contralateral femoral artery. The arterial catheter is advanced to the distal aorta to deliver retrograde blood flow for perfusion.

The catheter can be directly visualized with fluoroscopy or ultrasound. A great deal of care must be taken when performing the serial dilations to avoid laceration of the femoral vessels. Complications such as acute embolism/thrombosis, common femoral artery dissection, perforation, and compartment syndrome can be devastating. Once the femoral arterial cannula is in place, it is important to monitor for critical limb ischemia. A supplemental backflow cannula can be placed distal to the ascending arterial cannula in the superficial femoral artery to provide blood flow if there is concern for limb hypoperfusion. After cannulation, initial cardiac output targets of 1.5 to 2.0 L/min are acceptable but should be titrated up gradually to 3.0 to 6.0 L/min. Because arterial flow will be continuous, a pulse pressure of approximately 10 mm Hg is considered acceptable.

During VA ECMO, the retrograde aorta-to-left ventricular pressure gradient is very high, but the aortic valve should be able to function normally. A post cannulation transesophageal or transthoracic echocardiogram should be performed to check for proper opening of the aortic valve, aortic regurgitation, left ventricular distention, and tamponade. Although usually unnecessary, vasopressor and inotropic agents are occasionally required to reach target blood pressure and cardiac output, respectively.

Unlike VV ECMO, which allows the left heart to provide pulsatile flow to the systemic vasculature, VA ECMO provides continuous blood flow through the arterial system. After initiation, VA ECMO usually rapidly reduces pulmonary arterial pressures, improves end-organ perfusion, and increases PaO₂. For the postarrest patient, the pump’s heat exchanger can be used to facilitate targeted temperature management.

**Clinical Uses for ECMO**

**Toxic overdoses**

Medical management of most cardiotoxic ingestions remains largely supportive, as the effects of these toxic ingestions are often self-limited. A number of case series and reports have been published about the utility of VA ECMO in the overdose patient presenting with refractory CS. For patients presenting early to medical centers with personnel experienced with extracorporeal support, this technology can be used as a bridge to recovery.

Cardiovascular failure is a leading cause of death in patients with severe, acute drug intoxication. In 2010 alone, cardiovascular medication overdoses accounted for 9.4% of all fatalities. According to the American Association of Poison Control Centers, calcium-channel and beta-blocking medications were involved in 30% of these overdoses and accounted for more than 45% of deaths related to cardiovascular medications.

A growing body of literature supports the use of VA ECMO in the overdose patient, especially when the offending agent leads to refractory CS or arrhythmias. Case series describe overdoses with diltiazem, verapamil, flecainide, acebutolol, and other antidepressants agents. In many of these reports, VA ECMO was instituted early to provide effective perfusion and avoid multiorgan dysfunction. After the initiation of extracorporeal support, the duration of therapy was generally less than 1 week.

**Myocarditis**

One well-described use of ECMO is in fulminant CS secondary to acute myocarditis. Infective myocarditis is an acquired inflammatory muscle disorder that is more
common in the pediatric patient population. It can be caused by a wide spectrum of infectious organisms, as well as toxic and hypersensitivity reactions. Viral pathogens are the most common offending agents, specifically parvovirus B19 and human herpes virus 6.

Fulminant myocarditis is characterized by the presence of hypotension, respiratory failure, and signs of end-organ hypoperfusion. For patients presenting in shock, initial management often requires mechanical ventilation, inotropic agents, and vasopressors. If the patient is unresponsive to initial medical management, mechanical circulatory support might be required. Extracorporeal membrane oxygenation appears to be an effective therapy for these patients and can be used as a bridge to recovery.

The decision to initiate mechanical circulatory support can be difficult, because there are no absolute indications. Most experts agree that the decision to initiate ECMO should be strongly considered in the patient presenting with acute end-organ dysfunction, a refractory ventricular arrhythmia, or cardiac arrest.

**ECMO for out-of-hospital cardiac arrest and ECMO-assisted CPR (eCPR)**

The utility of ECMO in managing out-of-hospital cardiac arrest is perhaps one of the most exciting developments in the expanded role of extracorporeal support. Historically, out-of-hospital cardiac arrest outcomes remained poor despite advances in ACLS protocols and the use of conventional CPR. Return of spontaneous circulation (ROSC) rates are regularly reported to be less than 40%. Additionally, survival to hospital discharge rates range from 7% to 11%, and for those who do leave the hospital, favorable neurologic outcome is achieved in only 3% to 5%. Outcomes have improved with post-ROSC therapeutic hypothermia, rapid defibrillation, cardiocerebral resuscitation, and rapid revascularization.

Initiating ECMO for a patient in cardiac arrest remains a heroic procedure, with a small body of growing literature to support its use. In general, there is a lack of large randomized trials evaluating the efficacy of eCPR. Most of the published reports supporting its use are case reports and small observational studies with heterogeneous patient populations.

Appropriate patient selection is critical, as the use of ECMO should be only a temporizing, not definitive, treatment strategy. Accepted indications include a brief arrest period, a condition believed to be reversible (such as coronary occlusion, refractory arrhythmia, or toxin-induced arrest), or a condition that is amenable to transplantation or rapid revascularization. There are a number of suggested contraindications to eCPR, which are listed in Box 1. With respect to the duration of CPR, most published reports have included only those who had not achieved ROSC within 10 to 30 minutes of conventional CPR.

**Box 1**

**Common exclusion criteria for initiation of ECMO-assisted CPR**

- History of severe neurologic damage
- Intracranial hemorrhage
- Terminal malignancy
- Traumatic arrest with uncontrolled bleeding
- Unwitnessed arrest or prolonged arrest
- Aortic dissection
- Severe peripheral arterial disease
A number of studies have reported improved survival and neurologic outcomes with the use of eCPR for patients with in-hospital cardiac arrest.\textsuperscript{89,92,93} When used as a bridge to revascularization in patients who experienced cardiac arrest due to ST-elevation myocardial infarction, a significant 30-day mortality benefit has been achieved.\textsuperscript{92} More importantly, for those with a primary cardiac cause of arrest or shock, an improved 6-month survival with minimal neurologic impairment has been reported.\textsuperscript{93}

The ability to initiate ECMO for patients presenting to the emergency department with out-of-hospital cardiac arrest (OHCA) has become possible with advances in ECMO technology. In 2012, Bellezzo and colleagues\textsuperscript{94} published a staged approach for the initiation of ECMO for OHCA in the emergency department. This small study demonstrated that ECMO can be initiated in the emergency department and, if done carefully, can significantly improve neurologic recovery. Larger reports have shown a significant 30-day survival and improvement in neurologic outcomes as well.\textsuperscript{95–97}

The use of ECMO is extremely resource intensive and the cost alone could prohibit its widespread adoption. The amount of data for the use of VA ECMO in patients with OHCA remains limited, and it is still unclear which patients will benefit most from ECMO therapy. But as the body of literature continues to grow, it appears that rapid initiation of ECMO in the emergency department for OHCA is possible. It is important for the emergency physician to keep in mind that ECMO can be used as a temporary bridge to a more definitive procedure, such as revascularization, the use of a VAD, or transplantation.

**SUMMARY**

Patients who require mechanical circulatory support are some of the most critically ill patients one can encounter in the emergency department. As the use of these technologies continues to grow, emergency physicians have increasing opportunities to participate in the advancement of these potentially life-saving technologies. It is imperative that emergency physicians not only understand the complexities of these patients, but also be well-prepared to handle the many complications that can occur with these emerging therapies.

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**REFERENCES**


