ABSTRACT: Cardiac arrest in patients on mechanical support is a new phenomenon brought about by the increased use of this therapy in patients with end-stage heart failure. This American Heart Association scientific statement highlights the recognition and treatment of cardiovascular collapse or cardiopulmonary arrest in an adult or pediatric patient who has a ventricular assist device or total artificial heart. Specific, expert consensus recommendations are provided for the role of external chest compressions in such patients.

Mechanical circulatory support (MCS) has evolved from a rarely used therapy reserved for the most critically ill hospitalized patients to an accepted long-term outpatient therapy for treating patients with advanced heart failure. This growth is attributable to improved technology, improved survival, reduced adverse event profiles, greater reliability and mechanical durability, and limited numbers of organs available for donation. With the number of patients supported by durable MCS systems increasing in the community, so too is the need for emergency care providers to receive specific guidance on how to assess and treat a patient with MCS who is unresponsive or hypotensive.

No evidence-based or consensus recommendations currently exist for the evaluation and treatment of cardiovascular emergencies in patients with MCS. Because of the unique characteristics of mechanical support, these patients have physical findings that cannot be interpreted the same as for patients without MCS. For example, stable patients supported by a durable, continuous-flow ventricular assist device (VAD) often do not have a palpable pulse. Unfortunately, different and sometimes conflicting instructions are given by hospital providers and emergency medical services (EMS) directors to EMS and other healthcare personnel on core resuscitative practices such as the role of external chest compressions in such a patient who suddenly becomes or is found unresponsive.

PURPOSE

The purpose of this scientific statement is to describe the common types of MCS devices that emergency healthcare providers may encounter and to present expert, consensus-based recommendations for the evaluation and resuscitation of adult and pediatric patients with MCS with suspected cardiovascular collapse or cardiac arrest. These recommendations focus initially on emergency first-response providers, whether outside or inside the hospital, with additional sections on advanced care that may be provided in the emergency department or in-hospital settings.
CONSENSUS PROCESS

The need for standardized recommendations for the emergency treatment of acutely unstable patients with MCS was identified during the 2014 meeting of the American Heart Association (AHA) Science Subcommittee. A writing group was commissioned to review the current literature and to develop consensus-derived recommendations for the initial treatment of these patients. Members of the writing group were chosen for their combined expertise in MCS, cardiopulmonary resuscitation (CPR), emergency care, and circulatory support nursing. The writing group held a series of teleconferences and webinars from October 2014 to December 2015. An extensive literature search was performed and reviewed on the initial phone conference, and a manuscript outline was developed. Sections of the outline were assigned to group members, and the first draft of each section was sent to the chair to be compiled into a working document. The document was available to all writing group members in Dropbox, and all authors had continuous access to the documents to provide input. Revised versions of the manuscript were sent to all writing group members until consensus was achieved.

CONFLICT OF INTEREST

The AHA has a strict conflict-of-interest policy for all writing groups; however, the organization understands that experts who have an actual or perceived conflict of interest can strengthen the process as long as these conflicts are transparent. Each writing group member is required to declare all relevant, current conflicts, and >50% of the writing group must be free of relevant conflicts. The chair is not permitted to have any relevant industry-related conflicts. The writing group members must update an electronic file of conflict-of-interest data from the beginning of the work until the article has been published, and each member is asked to report any new relevant conflicts at the beginning of each teleconference. See the Writing Group Disclosures table for details on individual conflict-of-interest reporting.

INDICATIONS FOR MCS

Nearly all patients who receive MCS have end-stage systolic heart failure. Although there are only a limited number of approved devices, they differ in the indications for their placement, their configuration, and the means by which they pump blood. Long-term MCS devices suitable for nonhospitalized patients are placed largely for 1 of 2 indications:

1. Bridge to transplantation refers to patients awaiting heart transplantation whose heart failure progresses despite medical therapy. In such cases, MCS is used as a bridge until a donor organ becomes available.
2. Destination therapy refers to patients with advanced systolic heart failure who are not candidates for transplantation because of factors such as comorbidities or advanced age. In this population, MCS is the destination itself, as opposed to transplantation, and patients will typically live the remainder of their lives on mechanical support. This population is growing rapidly, with some patients being supported for many years.

These 2 designations are fluid. Patients can have their status changed from bridge to transplantation to destination therapy or vice versa on the basis of clinical changes.

MCS CONFIGURATION

MCS can support function of the left ventricle (LV) with a left VAD (LVAD), the right ventricle (RV) with a right VAD (RVAD), or both ventricles with a biventricular assist device. A total artificial heart (TAH) replaces the heart itself. Most patients who are discharged home with MCS currently have a durable LVAD. Thus, LVADs serve as the basis for most of these guidelines, although all current devices that may be encountered in the community are discussed.

Most devices are implanted inside the thoracic/abdominal cavity (intracorporeal). These devices pump blood from the weakened ventricle back into the circulation. With an LVAD, blood enters the device from the LV and is pumped to the central aortic circulation, "assisting" the heart. The outflow cannula is typically anastomosed to the ascending aorta, just above the aortic valve (Figure 1A).

With an RVAD, the inflow is the RV or atrium, and the outflow is the main pulmonary artery, just distal to the pulmonic valve (Figure 1B). When an LVAD and an RVAD are used in the same patient simultaneously, the patient is referred to as having biventricular support or a biventricular assist device, indicating that both ventricles are supported mechanically (Figure 1C).

Intracorporeal devices are connected to a controller that controls and powers the pump via a subcutaneously tunneled driveline that exits the skin, typically in the abdomen. The controller houses the electronics that run the device and monitor its function. The controller is connected to rechargeable batteries that provide its power. The device can also receive power from a base unit, typically located at the patient's home, or via adapters to other power sources such as a grounded electric outlet or car battery. These VAD components are depicted in Figure 2 for the most commonly placed VADs.
LVADs can have 2 distinctly different mechanisms of blood flow; therefore, they are different physiologically. There are older and rarely used pulsatile-flow LVADs and the current generation of continuous-flow LVADs. Because palpable pulses are often absent in patients with continuous-flow LVADs, it is important to understand the differences in the physical examination and in methods that can help rescuers determine if an unresponsive or mentally altered patient is, in fact, in cardiac arrest or circulatory collapse.
PULSATILE-FLOW LVADS

Most early generations of LVADs had pulsatile pumps, which filled and emptied a volume-displacement chamber sequentially, generating pulsatile flow similar to the native heart. Given the advantages of continuous-flow devices, intracorporeal pulsatile devices are no longer available commercially. However, paracorporeal (the pump sits outside of the patient’s body) pulsatile devices are still in use (Figure 3). Although not commonly used in adults, pulsatile LVADs remain the most commonly used LVAD in children but are typically not seen in the outpatient setting in the United States.

The volume-displacement chamber in a paracorporeal device is a clear, thermoplastic polymer chamber divided by a flexible membrane. One side of the membrane is in contact with blood, and the other is in contact with air. The device is powered by compressed air, which is shuttled into and out of the air side of the air chamber, causing the flexible membrane to move back and forth. When air enters the air chamber, the membrane displaces the blood, emptying the blood-filled chamber. When air is withdrawn, the membrane shifts the opposite direction, allowing blood to fill the blood-filled chamber. The device sequentially fills, and unidirectional blood flow is maintained by mechanical valves on the inflow and outflow cannulas.

These pumps typically sense when the chamber is full, which then triggers ejection. The blood-filled chamber fills and empties at a rate dependent on the pump preload. The pumps maintain decompression of the LV by pumping more quickly when preload increases and pumping less quickly when preload decreases. The native heart continues to contract in patients with a continuous-flow device, but the filling and emptying of the device is not synchronous with the heart. Because the flow from pulsatile-flow LVADs mimics that of the native heart, these patients have a detectable pulse on physical examination that reflects forward cardiac output and perfusion. However, because a pulsatile-flow device fills and empties asynchronously with the native heart, the pulse generated by the device may be different from the heart rate of the native heart obtained by electrocardiographic leads and can cause an irregular palpable pulse.

CONTINUOUS-FLOW LVADS

Continuous-flow devices account for the majority of durable LVADs implanted today. These devices contain a motor that turns an impeller rotating at high speed inside its housing. The impeller accelerates blood either forward with axial-flow pumps, in which the spin is in the same axis as blood flow (Figure 4A), or outward with centrifugal pumps, in which the spin is perpendicular to outflow (Figure 4B). The ability of the pump to generate flow is directly dependent on its mechanics and pump speed, typically a fixed number of revolutions per minute that is adjusted and set by the VAD team before discharge, although some pumps might have patient variability options. Pump flow is heavily dependent on preload, native contractility, and afterload.

Conditions that alter preload in the heart affect the loading of the pump and can influence device flow. For example, hypovolemia, tension pneumothorax, or peri-
Cardiac tamponade decreases preload and can cause circulatory failure. When the VAD is on the left side, its preload is also dependent on the filling from the RV. In such cases, RV contractility influences flow and can be impaired by RV failure or arrhythmia. An increase in pulmonary vascular resistance by hypoxia, hypercarbia, or obstruction of pulmonary blood flow (ie, pulmonary embolism) will decrease LV preload and LVAD flow.

Afterload in the clinical setting is essentially the blood pressure (BP). Pulmonary hypertension can result in right-sided heart failure, and systemic hypertension can affect left-sided pump function. Patients with VAD are typically not able to increase their cardiac output adequately when systemic vascular resistance is low in conditions such as vasodilatory septic shock, postsedation, or anaphylaxis. This can result in inadequate tissue perfusion despite normal device function. VAD-supported patients are at higher risk for complications during anesthesia and sedation.3–5

Continuous-flow LVADs result in an unconventional, unique physiologic state of hemodynamically stable pulseless electric activity (PEA), which we refer to in this population as pseudo-PEA. Patients supported by continuous-flow MCS devices may not have a palpable pulse, and their normal heart sounds will be replaced with an audible “VAD hum.” Vital signs such as noninvasive BP (NIBP) or oxygen saturation may be difficult to obtain. These factors can easily confuse healthcare providers rendering care to these patients. In a patient without MCS, cardiac arrest and resuscitation decision algorithms rely on the presence or absence of a pulse to identify complete cardiovascular collapse and cardiac arrest. Lack of a pulse alone in a patient with a continuous-flow LVAD is common and cannot be used as a means to determine whether a patient is in cardiac arrest or a low-flow, low-perfusion state.

BP can be obtained noninvasively with a manual sphygmomanometer and a Doppler ultrasound probe.6 Doppler BP measurements have excellent success rates of ≈95%, correlating accurately with invasively measured mean arterial pressure (MAP) and systolic arterial pressure.6 One potential pitfall in using Doppler to assess BP in unresponsive patients with continuous-flow LVADs is that the urgency of the situation may lead providers to inadvertently exert too much pressure on the Doppler probe itself, leading to the erroneous conclusion that the BP is nondetectable.

Automated BP monitors are accurate, but their success rate in obtaining a BP is only ≈50% because of reduced pulse pressure in patients with a continuous-flow LVAD.6,7 Unfortunately, most EMS systems currently lack Doppler equipment. In such cases, an NIBP can be used with the above limitations.7 Similarly, in patients with significantly reduced pulse pressure, pulse oximetry may be inaccurate.

Medical simulation can be used to teach emergency healthcare providers how to assess both normal and abnormal findings in patients with a VAD and allow them to safely practice interventions that might be needed in an emergency.8

**COMMON COMPLICATIONS RESULTING IN INSTABILITY**

The 2 most common causes of pump failure are disconnection of the power and failure of the driveline. Therefore, the first step in assessing an unresponsive, mentally altered, or hypotensive patient with a VAD is to ensure that all connections are secure and an adequate power source is connected. Although the current generation of continuous-flow LVADs is quite reliable and the incidence of pump dysfunction/stoppage is low, an interruption of power from the batteries or through the controller or a driveline malfunction may lead to pump stoppage.

Patients with a current LVAD system typically have 2 batteries connected to power the device. If both batteries are disconnected or completely drained of charge, pump dysfunction/stoppage can result (Figure 2). Controller malfunction, damage, or disconnection can also lead to pump dysfunction/stoppage. All patients should have a backup controller with them, as well as backup batteries for emergency replacement in case of damage or malfunction. EMS providers must keep patients and
their backup equipment together at all times because replacement equipment may be limited or nonexistent at receiving hospitals, particularly at non-VAD centers. To reiterate, when a patient with MCS is transported by EMS, all of the patient’s VAD equipment must accompany him or her to the hospital to ensure continued mechanical support.

The driveline that connects the controller to the device is a potentially vulnerable component and is subject to wear, damage, or kinking, which can result in device malfunction (Figure 2). Although driveline wiring has built-in redundancy as a safety measure, driveline trauma can cause internal damage and lead to pump failure. Damage can be acute such as a cutting or crush injury or the result of chronic stress/fatigue on the line. In these settings, there will often be alarms preceding or accompanying the pump stoppage, but alarms will cease once the batteries are drained.

There is a risk of de novo thrombus formation within the pump, which can cause hemolysis and a drop in flow. Blood clots can embolize from the LV or left atrial cavities, particularly if the patient develops atrial fibrillation. Pump thrombosis typically develops gradually and is accompanied by altered pump parameters and alarms. There is often physical and laboratory evidence of hemolysis such as jaundice, elevated lactate dehydrogenase, low haptoglobin, and elevated plasma-free hemoglobin. Some patients may experience mild symptoms and be well enough to seek nonemergent medical attention, but others may have profound and acute cardiac insufficiency, leading to syncope, ventricular arrhythmias, or cardiovascular collapse/cardiac arrest. It is important to understand that continuous-flow LVADs display a cardiac output in liters per minute that can be easily seen by EMS and other first responders; however, this number is not an actual cardiac output. This “cardiac output” number is a calculation based on the speed and power consumption of the unit and does not represent true flow. When the LVAD is functioning properly, this output can be used as a surrogate for flow. However, thrombus formation in the device actually decreases flow and can therefore cause hypotension, but because the device has to work harder to try to maintain the set flow, the cardiac output paradoxically reads high. Because of this risk of thrombus formation, all patients on MCS are anticoagulated. EMS and emergency department providers need to be cognizant of this and to consider bleeding, hemolysis, or anemia as the potential reason for hypotension or shock in these patients.

Additional causes of poor pump flow are RV dysfunction, suboptimal pump orientation, or compromise of the inflow/outflow cannula. As noted previously, LVADs are preload dependent and rely on RV function to provide adequate filling to maintain adequate flow. Factors that cause or exacerbate preexisting RV dysfunction can affect pump performance and lead to decompen-sation despite normal pump function. Inotropes can be used to treat RV dysfunction when that is felt to be contributing to hemodynamic compromise.

Preexisting RV dysfunction is common before LVAD implantation because of the effect of the primary cardiomyopathy, pulmonary hypertension, or both. RV dysfunction usually presents in the perioperative setting and, in some cases, requires prolonged inotropic support postoperatively. In contrast, some patients develop late RV dysfunction and require inotropic support. RV dysfunction typically affects functionality, quality of life, and symptoms. However, it can also affect LVAD flow and predispose to LVAD dysfunction. Conditions that decrease RV function (ie, right-sided ischemia, pulmonary embolism, tachyarrhythmias, profound dehydration) need to be considered when there is pump dysfunction and inadequate flow.

Although significant new ischemic events or pulmonary emboli are rare in patients with an LVAD, tachyarrhythmias are common. Many tachyarrhythmias are tolerated well in patients with an LVAD, although they can affect RV filling. Atrial tachyarrhythmias such as atrial fibrillation usually do not affect RV function adversely except when preexisting RV function is truly marginal. However, ventricular tachycardia (VT) or ventricular fibrillation (VF) can be accompanied by a range of hemodynamic presentations. Patients whose systemic flow is coming primarily from the LVAD may be surprisingly stable hemodynamically, whereas some may present with circulatory collapse, usually caused by acute compromise of RV function and decreased LV filling. Most patients have an implantable cardioverter-defibrillator or a cardiac resynchronization device with defibrillation capability implanted before LVAD placement, which can deliver antitachycardia pacing or cardioversion. However, in the setting of electrolyte abnormalities or acute ischemia, VT/VF may persist and compromise pump flow with subsequent clinical decompensation.

Not all patients with an LVAD who are in VT or VF will be unconscious. These devices may be able to provide enough forward flow despite the arrhythmia to maintain consciousness and, in some cases, even adequate perfusion. Similar to the decision made for patients without VADs, the decision to cardiovert (either chemical or electric) or to defibrillate a patient with an LVAD with VT or VF is based on the adequacy of mental status and perfusion. Except for patients who have a TAH, cardioversion and defibrillation should be performed for the same indications as used in the general population, with the understanding that perfusion in these patients does not equate to a palpable pulse.

Although LVADs provide near-complete support of the cardiac output, most patients still have residual native heart function. With LVAD pump dysfunction/stoppage, the native heart fills and may provide some forward flow to support the circulation for a period of time, the dura-
tion of which differs from patient to patient, depending on the severity of underlying cardiac dysfunction. Severe aortic insufficiency can further exacerbate LV failure and decrease the contribution of the native LV to forward flow. Occasionally, patients have their aortic valve oversewn during LVAD implantation because of underlying aortic insufficiency. These patients cannot generate any forward blood flow on their own and are completely dependent on flow from the MCS device.

COMMON NONCARDIOVASCULAR PROBLEMS IN PATIENTS WITH MCS

The most common adverse events during long-term mechanical support are infection (particularly in the driveline), bleeding, and stroke. Meticulous care is needed to keep the driveline free of infection, but despite intensive care, the rate of driveline infections is 7% to 18% in bridge-to-transplantation populations and up to 32% in destination-therapy populations. Device infection or other infectious sources may lead to sepsis. Pumps are afterload sensitive and will increase flow in the setting of reduced afterload up to a point, but patients can still develop septic shock once the ability of the pump to augment forward flow is overcome by the reduction in systemic vascular resistance. The immediate, prehospital treatment of suspected septic shock should be the same as for patients without an LVAD, with fluid resuscitation and vasopressors as needed to maintain an MAP of 65 to 80 mmHg. With hypovolemia and diminished preload, pump flows are low, resulting in hypotension or shock. Figure 5 provides a general approach to the hypotensive patient with an LVAD.

Patients with MCS are usually anticoagulated with antiplatelet agents and warfarin. When these agents are combined with continuous-flow physiology, there is significant bleeding risk, particularly in mucosal surfaces such as the intestine. Continuous-flow MCS devices are associated with an acquired von Willebrand syndrome, which also makes patients more likely to bleed spontaneously. Arteriovenous malformations in the gut are more likely to form in patients with continuous-flow devices and reduced pulsations, and these are a common source of gastrointestinal bleeding. Rates of gastrointestinal bleeding with continuous-flow devices range from 9% to 22% in various series and are a common cause of non–pump-related hemodynamic instability.

There is still an increased risk of pump thrombosis, thromboembolism, and stroke despite therapeutic anticoagulation. The rate of both hemorrhagic and ischemic stroke is 4% to 7% per year with continuous-flow pumps. A significant neurological event may result in a patient being found unresponsive despite a normally functioning device. This scenario of an unconscious patient without a pulse is perhaps the most confusing to assess because stroke patients may be unconscious with completely adequate flow yet appear as though they are in cardiac arrest because they may have no palpable pulse.

Patients with a VAD can develop non–pump-related medical issues (eg, hypoglycemia, drug-overdose hypoxemia) similar to patients without a VAD, resulting in alterations in mental status and hemodynamics. The clinical scenarios discussed above can either contribute to or mimic a cardiac arrest in a nonpulsatile patient. They can also be the inciting factor for hemodynamic collapse or altered mental status/coma.

For these reasons, these conditions should be considered, identified, and treated promptly in altered or hemodynamically compromised patients.

ASSESSMENT OF FLOW AND PERFUSION IN PATIENTS WITH AN LVAD

It is important to understand the difference between blood flow and perfusion when assessing any patient with suspected cardiovascular hemodynamic instability, especially patients with an LVAD, in whom the peripheral arterial pulse is not a reliable indicator. Flow represents the forward movement of blood through the systemic circulation. It can be either adequate or inadequate to provide sufficient oxygen delivery to sustain tissue perfusion. Assessment of adequate tissue perfusion is the most important factor in determining the need for circulatory assistance such as chest compressions.

In patients without an LVAD, an MAP ≥60 mmHg will usually still provide adequate tissue perfusion. In the noninvasive assessment of the BP of a patient with a continuous-flow LVAD, use of a manual BP cuff and a Doppler is the recommended approach, with NIBP as a secondary option because of the limitations of NIBP assessment in this population. The lack of pulse or BP as obtained by a sphygmomanometer or automatic NIBP monitor is not a reliable indicator of either flow or perfusion in a patient with an LVAD.

Clinical findings such as skin color and capillary refill are reasonable predictors of the presence of adequate flow and perfusion, especially in MCS-supported pulseless patients.

Waveform capnography, which measures and displays the partial pressure of end-tidal carbon dioxide (PetCO₂) in exhaled air, is used frequently to track respiration in patients undergoing mechanical ventilation, but it can also be used to track perfusion in patients in whom more common physical findings used to assess perfusion are not reliable. Ventilation in a healthy person with a normal cardiac output and metabolism/body temperature and no significant ventilation-perfusion gradient typically results in a PetCO₂ of 35 to 40 mmHg. During low-flow states
such as during shock or cardiac arrest with or without chest compressions, the PETCO₂ value reflects primarily systemic perfusion. Cardiac output and systemic venous return decrease substantially in very-low-flow states, causing CO₂ to build up inside cells. With the decrease in systemic venous return, less CO₂ returns to the lungs, causing the PETCO₂ to decrease. A PETCO₂ level of 20 mmHg corresponds to a cardiac output of ≈1.5 L/min in animal models. Within seconds after return of spontaneous circulation, the improved cardiac output delivers large quantities of CO₂-rich venous blood to the lungs, and the PETCO₂ climbs suddenly to normal or above-normal levels. The dramatic change from a low to a high PETCO₂ as a result of venous CO₂ washout is often the first clinical indicator that return of spontaneous circulation has occurred.

The 2015 AHA guidelines update for CPR and emergency cardiovascular care recommends the use of waveform capnography to confirm advanced airway placement and to track the quality of chest compressions during resuscitation. An increasing number of EMS providers and hospitals have upgraded or are upgrading their cardiac monitors to include waveform capnography. The significance of this evolution in clinical care is that the PETCO₂ value can help rescuers determine whether pulseless patients with an LVAD are unconscious because their device has failed and their systemic perfusion is critically low or they are unconscious for an unrelated reason with a functioning LVAD. The former may benefit from chest compressions, whereas chest compressions would not be indicated in the latter group. A PETCO₂ value of <20 mmHg in an unresponsive, correctly intubated, pulseless patient with an LVAD would seem to be a reasonable indicator of poor systemic perfusion and should prompt rescuers to initiate chest compressions. Conversely, a higher PETCO₂ in an unresponsive, correctly intubated, pulseless patient with an LVAD strongly suggests that the patient does not need chest compressions, and rescuers should search for other noncardiovascular, non-LVAD reasons for the patient’s altered mental state.

Pulse oximetry can be used in patients with an LVAD; however, the results may not be accurate because of the lack of pulsatile flow. Peripheral pulse oximetry within normal limits is likely believable. Conversely, a low oximetry reading may not represent true hypoxemia because of the diminished pulsations seen in patients with an LVAD.

**EMS CONSIDERATIONS**

The likelihood of a favorable outcome after cardiac arrest increases substantially if the event is witnessed, the event occurs in a public place, bystanders call 9-1-1 and initiate chest compressions promptly, the initial rhythm is VF, an automated external defibrillator is applied and used, and there is a prompt EMS response. Bystander chest compressions are performed in only a third of cases, but this can be doubled when 9-1-1 dispatchers issue “phone CPR” instructions that are carried out promptly and effectively. In addition, 9-1-1 dispatchers are trained to send fire department first responders to the scene of a suspected cardiac arrest so that they can ini-
tiate CPR and defibrillate, if indicated, with an automated external defibrillator before EMS (ie, paramedic) arrival. Because there are usually more fire engine companies than ambulances in most US cities, fire department first responders arrive on scene before paramedics in approximately half of cases and arrive simultaneously with paramedics in another quarter of cases.

These emergency operations practices are highly relevant to the debate about the role of chest compressions in an unconscious, pulseless patient with an LVAD. In most US locations, a bystander or a first responder will likely already be instructed to perform chest compressions on such a patient before paramedic arrival. To the untrained person, an LVAD may be unrecognizable or incorrectly identified.

Therefore, it is the consensus recommendation that if an LVAD is definitively confirmed by a trained person and there are no signs of life, bystander CPR, including chest compressions, should be recommended by emergency medical dispatchers for cardiac arrest situations.

EMS providers should be mindful that most community hospitals currently do not have VAD programs or equipment. Therefore, backup/spare equipment, which patients often carry on their person, should be brought with them to the hospital whenever possible. For example, at the scene of a motor vehicle crash in which a driver/passenger is a patient with a VAD, it is prudent to spend a few minutes to retrieve all VAD backup equipment that the patient is carrying in addition to the patient.

There is the very real dilemma of non–medical control interactions with VAD coordinators and physicians. Depending on state rules and regulations, often medical control physicians or nurses provide legally authorized medical direction and orders. LVAD coordinators/team members are often the first line of communication for EMS and family members when caring for patients with MCS in emergency situations. Consideration should be given for EMS to be directed by these specialized teams for this unique patient population, but this should be explicitly reviewed and authorized by each state's office of EMS. Finally, patients with MCS, either VAD or TAH, should be transported to a VAD center whenever possible. If the patient is being transported to a non-VAD/non-TAH center, immediate notification of a potential VAD/TAH center should ensure for medical direction of care and potential prompt transfer when necessary. Many family members of patients with a VAD are trained in emergency procedures, and EMS should consider transporting them in the same vehicle if feasible.

**CONSENSUS-DERIVED GUIDANCE FOR EMS CLINICAL PRACTICE**

Figure 6 outlines consensus-derived recommendations for first-responder assessment of a patient with an LVAD.

Identifying the presence of MCS and code status is of initial importance. Some destination-therapy patients with an LVAD will have a legally executed, valid do-not-attempt-resuscitation status and should be treated as any other patient with such a request. Information obtained from caregivers and medical alert identifications/wallet cards should be used to provide definitive identification. In some locations, patients with MCS are often told to keep their medical information (eg, code status information) in the refrigerator. It seems reasonable for VAD centers to standardize their approach to patient identification. Medical alert bracelets and necklaces can help to identify patients with a VAD and their code/induction status, and they should be kept with the patient during transport to the hospital.

Signs of life and perfusion should be assessed by evaluation of mental status, breathing, skin color and temperature, and capillary refill. If it is unclear whether the patient has an LVAD, care should be established with standard basic cardiovascular life support and advanced cardiovascular life support (ACLS) protocols. Breathing should be supported as needed with supplemental oxygen, airway adjuncts, and intubation as indicated.

Once a patient is identified as having an LVAD, EMS providers must recognize that their patient may be in a state of pseudo-PEA and not have a palpable pulse or recognizable BP yet have adequate perfusion. If there is adequate mental status, a provider should assess the VAD for function by auscultating for a VAD hum over the left chest/left upper abdominal quadrant, looking and listening for VAD alarms, ensuring secure connections to the VAD controller, and ensuring sufficient power for the VAD. Prompt notification of VAD center and its personnel (ie, VAD coordinator) is strongly recommended.

If there is inadequate perfusion, unresponsiveness, or other altered mental state, the VAD should be assessed for function by looking and listening for alarms, listening for a VAD hum over the left chest and left upper abdominal quadrant, ensuring secure connections to the controller, and ensuring adequate power for the VAD. If one checks all the above and the VAD is still not functioning, a system controller change-out should be considered if there is a trained provider or family member available. Family members are trained to make this controller change, and EMS should request the assistance of a trained layperson on site. If the VAD is still not functioning after a controller change, especially if the PETCO2 value remains <20 mmHg in a correctly intubated patient, external chest compressions should be initiated and standard ACLS protocols followed with prompt notification of the VAD center.

If the VAD appears to be functioning, noted by a mechanical hum, BP should be checked. If available, a Doppler BP (MAP) should be obtained. If unavailable, an NIBP may be attempted. However, as noted above, this may not be obtainable. If an obtained MAP is >50 mmHg, ACLS protocols should be followed with the exception of...
Assess and treat non-LVAD causes for altered mental status, such as
- Hypoxia
- Blood glucose
- Overdose
- Stroke

Assist ventilation if necessary and assess perfusion
- Normal skin color and temperature?
- Normal capillary refill?

Adequate perfusion?

Assess LVAD function
- Look/listen for alarms
- Listen for LVAD hum

MAP >50 mm Hg and/or PETCO$_2$ >20 mm Hg?*? Yes

LVAD functioning?

No

Attempt to restart LVAD
- Driveline connected?
- Power source connected?
- Need to replace system controller?

Do not perform external chest compressions

Perform external chest compressions

LVAD restarted?

Yes

Follow local EMS and ACLS protocols

Notify VAD center and/or medical control and transport

*The PETCO$_2$ cutoff of >20 mm Hg should be used only when an ET tube or tracheostomy is used to ventilate the patient. Use of a supraglottic (eg, King) airway results in a falsely elevated PETCO$_2$ value.

Figure 6. Algorithm showing response to a patient with a left ventricular assist device (LVAD) with unresponsiveness or other altered mental status.

ACLS indicates advanced cardiovascular life support; EMS, emergency medical services; ET, endotracheal tube; MAP, mean arterial pressure; PETCO$_2$, partial pressure of end-tidal carbon dioxide; and VAD, ventricular assist device.

chest compressions because the VAD is likely providing adequate forward flow. A detailed search for reversible causes (5 Hs [hypovolemia, hypoxia, hydrogen ion [acidosis], hypo/hyperkalemia, hypothermia] and 5 Ts [toxins, tamponade, tension pneumothorax, thrombosis-heart, thrombosis-lung]) should be undertaken.

If there is no detectable BP in a correctly intubated patient, PETCO$_2$ can be measured. If the PETCO$_2$ is >20 mmHg, similar to a patient with an MAP >50 mmHg, chest compressions should be withheld, and standard ACLS protocols should be followed. If the PETCO$_2$ is <20 mmHg or not available, chest compressions should be initiated if there are no signs of life after standard ACLS protocols. Caution is advised in evaluating PETCO$_2$ in nonintubated patients and when a supraglottic airway is used to ventilate, which may cause a falsely elevated reading because of the increased dead space in the supraglottic space.

When available, additional evaluation tools such as point-of-care echocardiography and Doppler blood flow can be considered. Assessment of flow in the carotid or femoral arteries via Doppler may represent an alternative method to check for blood flow in patients with MCS. If a Doppler signal is absent in these large arteries, the
absence of meaningful forward flow can be inferred, and
chest compressions should be initiated. Acquisition of
additional data should not delay application of appropriate
resuscitative measures in acutely ill/unresponsive
patients with MCS devices.

It is reasonable to provide standard postarrest care,
including mild therapeutic hypothermia and early per-
cutaneous coronary intervention when indicated, to
patients with an LVAD who survive a cardiac arrest. It
should be remembered that patients with an LVAD need
adequate anticoagulation, which may be difficult to track
accurately while a patient is hypothermic.

ECHOCARDIOGRAPHY IN THE EVALUATION OF
AN ACUTELY ILL PATIENT WITH AN LVAD

Echocardiography is an invaluable noninvasive tool for
the rapid, real-time evaluation of LVAD performance in a
patient with an LVAD who is critically ill. Because few, if
any, EMS services have an echocardiography device or
are trained in its use, this evaluation is currently almost
always performed in a dedicated echocardiography lab-
atory, intensive care unit, or hospital emergency depart-
ment. We include this detailed section to assist hospital-
based providers who have real-time echocardiography
availability in responding to acutely decompensating
patients with an LVAD. We encourage the use of eco-
cardiography when available to assess unstable patients
with an LVAD because it allows rapid assessment of se-
veral parameters of pump function and hemodynamics.
As point-of-care echocardiography with small handheld
ultrasound devices becomes more widespread, the clini-
cal emergencies discussed in this section may poten-
tially be diagnosed and treated in the field by emergency
personnel specially trained in echocardiographic diagno-
sis of cardiovascular emergencies.

The design and functionality of newer-generation
LVADs differ importantly from earlier devices. Newer
LVADs deliver systemic perfusion by means of an impel-
er that operates at speeds of several thousand rev olu-
tions per minute, resulting in axial aortic blood flow
that is continuous. Because the newer rotary devices
generate lower pressure gradients at the inlet cannula
than earlier “pulsatile” LVADs, native cardiac function is
an important determinant of rotary LVAD performance.
Specifically, optimal LVAD function depends on the right
side of the heart for delivery of blood to the LV and is
influenced by the ability of the LV to augment LVAD filling.

Clinical emergencies in patients with an LVAD or LVAD
alarms such as low flow, power spikes, suction events,
and pulsatility alarms most often occur as a result of
processes that are extrinsic to the LVAD itself. Events
within the LVAD also occur but less frequently. By provid-
ing comprehensive assessment of cardiac anatomy and
function, along with evaluation of LVAD function, echo-
cardiography can provide critical information for physi-
cians caring for patients with an LVAD who are acutely ill.

A systematic approach to such patients can be orga-
nized around the following 2 questions.

Question 1: Are the Clinical Symptoms
Attributable to an Acute Deterioration of Native
Cardiac Function?

Is Cardiac Filling Adequate?
Assessment of inferior vena cava diameter, and its
change during inspiration, will provide an estimate of
central venous pressure. Inadequate filling of the RV
because of volume depletion, hemorrhage, sepsis, arrhyth-
mas, or cardiac tamponade will result in impaired
delivery of blood to the LV, with consequent reduction in
LVAD flow and/or suction events caused by abutment of
the inflow cannula against the LV wall.

Has RV Systolic Function Worsened?
Echocardiography can assess downward trends in right-
sided heart function by demonstrating increased right
atrial pressure, as manifested by increased inferior vena
cava diameter and decreased collapse (<50%) with inspira-
tion or even complete loss of respiratory variation in
inferior vena cava diameter. Echocardiography can show
progressive dilation of the RV or an interim decrease in
RV systolic function as indicated by decreased RV frac-
tional area change and, in some cases, by changes in RV
systolic pressure.

Acute decompensation of RV function may also re-
sult from an acute increase in pulmonary artery pressure
caused by cor pulmonale, pulmonary embolus, or intrin-
sic pulmonary vascular disease.

Echocardiographic findings in such cases resemble
those seen in patients with intrinsic RV failure, except
that RV systolic pressure will be significantly elevated
above baseline values for that patient.

Is There Significant Valve Dysfunction?
Dysfunction of the tricuspid or pulmonic valves can oc-
cur secondary to changes in pulmonary artery pressure
or RV function, can impair delivery of blood to the left
side of the heart, and can cause suboptimal LVAD per-
formance. Aortic regurgitation can significantly impair the
ability of the LVAD to deliver antegrade blood flow to the
systemic circulation. Mitral regurgitation is less often a
problem because the LVAD reduces LV size and filling
pressure and improves mitral leaflet coaptation.

Question 2: Is the LVAD Itself Malfunctioning?

Is the LV Being Unloaded Effectively?
If the LVAD is functioning optimally, LV end-diastolic in-
ternal dimension (LVIDd) should decrease. Comparison
of the parasternal long-axis 2-dimensional preoperative
LVIDd with the postoperative LVIDd is the most important
clinical measure of the degree of LV unloading achieved by the LVAD. If endocardial definition is suboptimal, a microbubble contrast agent should be used for accurate LVIDd measurement. Impaired LVAD performance can be detected echocardiographically by noting an enlarged LVIDd compared with previous values obtained when the patient was stable.23

**Does the Aortic Valve Open During Systole?**

Intermittent, often mild aortic valve opening is considered desirable after LVAD implantation. However, an aortic valve that opens widely during every LV systole signifies inadequate unloading of the LV by the LVAD. In some cases, this can be addressed by adjusting LVAD speed, the effectiveness of which can be confirmed by serial echocardiographic assessments of aortic valve opening, including M-mode measurements. When LVAD speed adjustments fail to improve LV unloading, as determined by serial echocardiography, a search for intrinsic causes of LVAD dysfunction is necessary.

**Is There Obstruction to LVAD Inflow or Outflow?**

Inflow obstruction may occur as a result of thrombus, pannus, disruption of the submural apparatus, or abutment of the cannula against an LV wall. Although the metallic LVAD inflow cannula usually causes significant acoustic artifact, the presence of inflow obstruction can often be identified with a carefully targeted 2-dimensional and color Doppler echocardiographic examination. If there is no LVAD inflow cannula obstruction, the color Doppler should demonstrate laminar, nonturbulent unidirectional flow from the LV into the inflow cannula. Three-dimensional echocardiography may be helpful. In cases when there is suspicion of LVAD inflow obstruction, transesophageal echocardiography can confirm the diagnosis with confidence.

Inflow obstruction by LV myocardium can develop from substantial changes in LV size and shape as the LV is unloaded by the LVAD. Because the LVAD cannula orifice is not surgically anchored, its spatial relationship with LV walls changes over time and indeed can change during the cardiac cycle. In such cases, LVAD inflow varies more significantly over the cardiac cycle as the obstruction is relieved or exacerbated by LV wall motion. Changes in LVAD inflow over the cardiac cycle can be assessed with pulsed-wave Doppler echocardiography. The degree of variation of instantaneous volumetric flow into the LVAD during the cardiac cycle can be estimated by calculating a pulsatility index as follows: [(maximum LVAD inflow velocity−minimum LVAD inflow velocity)/mean LVAD inflow velocity]×10. A normal pulsatility index is <10. Higher values suggest the possibility of inflow obstruction.

Note that changes in Doppler-measured inflow kinetics with this formula may not necessarily be reflected by the LVAD console because console-reported LVAD flow is derived empirically on the basis of internal character-istics, including the driveline electric power required to maintain a constant impeller rotational rate. The LVAD outflow graft anastomosis to the ascending aorta can be visualized from a high-left parasternal long-axis view. If there is anastomotic site stenosis, the color Doppler signal may show flow turbulence, and pulsed- and continuous-wave Doppler may show abnormally high systolic velocity (>2 m/s).22

**Is There Intrinsic LVAD Dysfunction?**

Intrinsic LVAD dysfunction can occur as a result of thrombosis or pannus within the LVAD system, failure of LVAD bearings or rotor, or electric failure because of driveline malfunction. Because LVAD elements are essentially all sono-opaque, the role of echocardiography in this setting is to exclude other causes in acutely ill patients with signs and symptoms of inadequate cardiac output. Thus, when echocardiography does not show evidence of intravascular volume depletion, progression of native heart disease, or LVAD inflow obstruction and when there is echocardiographic evidence of inadequate unloading of the LV as previously discussed, the findings should direct caregivers to the possibility of intrinsic LVAD dysfunction.

Acquisition of echocardiographic data should not delay application of appropriate resuscitative measures in acutely ill/unresponsive patients with MCS devices.

Arterial line placement can be undertaken in hospitalized patients for accurate BP determination and to identify the presence of flow in unconscious patients with an LVAD without a pulse. Ultrasound guidance can be used for line placement given the lack of pulsatility in some patients. It is important to note that the arterial tracing may look flat or with a very low pulse pressure in patients who are not pulsatile.

Hemodynamic measurements, including cardiac output, peripheral resistance, preload, afterload, and RV function, may provide additional important information and insight into the significance of the echocardiographic data. All this information may lead to medication changes or surgical intervention in these extremely ill individuals.

**TOTAL ARTIFICIAL HEART**

The TAH is also an intracorporeal device, but rather than being attached to the native heart with cannulas, the native ventricles are removed completely when the TAH is implanted. The right side of the pump is anastomosed to the right atrium and pulmonary artery, and the left side of the pump is anastomosed to the left atrium and aorta. The pump is powered pneumatically, with each side being driven by a separate driveline that passes through the skin and is attached to a pneumatic driver.25

Three drivers are in use currently in the United States. Two drivers, the Circulatory Support System
and the Companion 2, require the patient to remain in the hospital until transplantation, and the other driver, Freedom, is more portable, allowing the patient to be discharged home. All patients are placed initially on the large, hospital-based device at the time of surgery. The most immediate cause for perioperative collapse is compression of the left pulmonary veins or the inferior vena cava, resulting in reduction of preload. These are best detected by transesophageal echocardiography before chest closure. Cardiac tamponade can similarly occur by compression of venous return after chest closure resulting from bleeding.\textsuperscript{26,27} These complications are often seen immediately after surgery and require the intervention of a surgeon who can correct the mechanical problem quickly by returning to the operating room. Such complications are very uncommon after discharge home.

The pump parameters are set by the medical team. These include the left drive pressure, the right drive pressure, the beat rate, the percent ejection, and a low level of vacuum on the Circulatory Support System console, which helps with movement of air out of the ventricles. Once patients reach clinical stability, very few changes are needed to the settings on the drive pressures, vacuum, or beat rate. The general drive pressures on the TAH range from 180 to 200 mmHg on the left side and 60 to 100 mmHg on the right side, with a beat rate of 120 to 135 per minute and vacuum of about −10 mmHg (Circulatory Support System only). Selected patients can be transferred to the smaller, portable pneumatic driver for discharge to home (Figure 7). This driver has only 1 adjustable parameter, namely the beat rate. The movement of the internal piston generates the negative pressure, and the drive pressures are set at 200 mmHg on the left and 100 mmHg on the right.

The TAH produces a pulsatile waveform and a palpable pulse. It is important to note that as the ventricles are removed, these patients do not produce an ECG tracing (ie, it is a flat line). This absence of ECG tracing may be interpreted as asystole and lead to inappropriate therapy such as chest compressions (ineffective because the mechanical ventricles are rigid and cannot be compressed) or administration of epinephrine, which can raise afterload and lead to worsening pump function. A dramatic rise in afterload after a vasopressor bolus can lead to incomplete pump ejection, a rapid rise in left atrial pressure, and resultant pulmonary edema.

The pump compensates quite well in vasodilated states such as sepsis and should maintain adequate output for a few hours. This should be ample time for EMS to bring the patient to the hospital and contact the primary
TAH team. If absolutely needed for volume refractory hypotension, an infusion of low-dose norepinephrine is the best choice. It is important to avoid an intravenous push of vasopressor and inotropes because they immediately push afterload up and the portable drive cannot pump against this, resulting in elevated left atrial pressures and pulmonary edema. Once this happens, the right-sided pump develops worsening afterload and its output drops. This rapidly spirals into unmanageable hypotension.

The most common cause of hemodynamic collapse after discharge home is kinking of the drivelines. The next most common cause is an increase in BP as patients improve from their heart failure. The increased afterload created by hypertension can affect device emptying and lead to pulmonary edema and respiratory distress. These are true medical emergencies and require immediate assessment and treatment before transport.

Prehospital and emergency personnel need to understand the difference between a TAH and an LVAD. With a TAH, the heart is excised and the patients are dependent on the TAH, whereas with an LVAD, the native heart remains and usually contributes in some fashion to perfusion. All patients should be sent home with a medic alert bracelet, which includes their living will/advance directives, and the portable driver should be marked clearly.

The most dangerous situation occurs when a patient is hypertensive and in mild respiratory distress and EMS arrives and finds “asystole.” With an LVAD, the native ECG remains. However, with TAH, there is no electric depolarization and therefore no detectable ECG tracing. Thus, ECG assessment is not recommended for patients with a TAH. Epinephrine should not be given for this asystole because it can lead to a further increase in afterload and complete hemodynamic collapse with pulmonary edema. Figure 8 provides an algorithm for evaluation and treatment of a patient with a TAH who is altered mentally, unresponsive, or in respiratory distress.

EMS providers should assess first for responsiveness and then for the presence or absence of a pulse. If a patient is responsive with a pulse, he or she should be transported to the nearest TAH center if possible.

If a patient has a pulse and is unresponsive, ventilations should be assisted as required, and as with patients with an LVAD, noncardiac causes of altered mental status (eg, hypoglycemia, hypoxia, stroke, or overdose) should be considered. BP should be assessed rapidly because systolic hypertension increases afterload and therefore decreases forward blood flow from the device. When this occurs, patients can present with fulminant pulmonary edema because of backflow of blood into the lungs, which is a true medical emergency.

For systolic BPs >150 mmHg, treatment should be given to maintain systolic BP <130 but >90 mmHg. This can be attempted in the prehospital setting with the use of sublingual nitroglycerin or administration of 40 mg furosemide intravenously if the drug and intravenous access are available. Many patients with a TAH are discharged from the hospital with oral hydralazine that they and their caregivers are instructed to use in the setting of hypertension. Advanced life support EMS providers can assist the patient with self-administration of hydralazine to further decrease BP. The patient should then be transported to the hospital. If a patient is hypotensive with a systolic BP <90 mmHg, 1 L normal saline solution should be administered during transport.

The portable TAH is pumped mechanically. As long as it is functioning properly, there should be a palpable pulse. When no pulse is present with a TAH, there is likely a catastrophic mechanical malfunction resulting in no blood flow or perfusion. Because chest compressions are futile and ACLS drugs are contraindicated or ineffective given the presence of the TAH, the only therapeutic option is to try to restore mechanical function of the device. The driveline should be checked immediately for kinks and straightened if needed. If there is still no pulse after ensuring that the driveline appears unkinked, then the battery should be checked for correct positioning and power status and replaced if necessary. Trained personnel should switch to the backup controller if one is available. Finally, 1 L normal saline solution should be administered intravenously to treat for possible hypovolemia. Assisted ventilation should be performed as needed, and the patient should be transported to the hospital as soon as possible.

At no time should manual or mechanical chest compressions be performed in a patient with a TAH. Chest compressions are futile because mechanical chambers have replaced the ventricles. There would be no contribution to forward flow from the cardiac pump mechanism of blood flow during CPR because the “heart” is not affected by intrathoracic pressure changes. There are no data to suggest that adequate blood flow occurs in patients with a TAH as a result of changes in intrathoracic pressure; therefore, there are no data to support the use of chest compressions in these patients because their heart has been completely replaced by a mechanical heart.

An electrocardiograph, a telemetry monitor, and an automated external defibrillator are not useful and should not be placed. Antiarrhythmic drugs are also of no use because the heart has been removed and therefore there are no meaningful arrhythmias. Electric therapy (eg, pacing, defibrillation/cardioversion) should not be used for similar reasons. Standard vasopressor drugs used in ACLS such as epinephrine or vasopressin are contraindicated because they increase afterload and worsen TAH function.

**RESUSCITATION OF INFANTS AND CHILDREN WITH THE USE OF DURABLE MCS DEVICES**

The use of durable MCS devices as a bridge to transplantation in the pediatric population is increasing.31-33
During 2008 to 2010, 18% of 1440 patients listed for transplantation were bridged with a VAD. The Berlin Heart EXCOR VAD accounted for 57% (n=60) of devices used for children.\textsuperscript{33} The Berlin Heart EXCOR Pediatric VAD is the only US Food and Drug Administration–approved device to provide mechanical circulatory assistance as a bridge to transplantation in children (Figure 3).\textsuperscript{34} The type of VAD used in a pediatric patient is determined largely by patient size.\textsuperscript{33} The Berlin Heart Device is an extracorporeal, pneumatically driven pulsatile VAD that can be used in both univentricular and biventricular support configurations, providing support
to the LV, the RV, or both ventricles. Different pump sizes (10, 25, 30, and 50 mL) are available to tailor to the pediatric size ranges. The Berlin Heart EXCOR is currently not used in out-of-hospital settings, and patients supported with the Berlin Heart Device remain hospitalized until transplantation.

Older children approaching adult size (eg, body surface area >1.3 m²) have the option of being supported with adult devices, and a number of adult devices, including implantable VADs, have been used in children. Adult-sized children supported with implantable devices may be discharged home to await transplantation. An estimate of the number of children supported with VAD in out-of-hospital settings is not available, but it is likely a small number. If an adult pump is chosen, resuscitation should follow that of the adult algorithm outlined above.

RESUSCITATION OF CHILDREN SUPPORTED WITH A VAD

There is little published information on the management of cardiac arrest in children supported with a VAD. Resuscitative care for children supported with an adult implantable VAD should be based on algorithms outlined for adults in this document. In younger children, principles for the management of resuscitation are similar to those for the management of adults. Diagnosis of circulatory arrest requires prompt evaluation of the patient and the VAD. Clinical signs of circulatory arrest may include acute change in the level of consciousness and the absence of respiratory effort. Severe bradycardia, asystole, VT, or VF may be present and may be a clue to the presence of cardiopulmonary arrest. Causes of loss of circulation in children supported with VADs include primary device failure (driveline breakage, disconnection, driver failure, and loss of power to the driver) and patient-related factors (hypovolemia, tension pneumothorax, pericardial tamponade, pulmonary hypertension, vasodilatory shock, and apnea related to administration of sedation).

CIRCULATORY ARREST IN THE BERLIN HEART EXCOR VAD

For an unresponsive patient on the Berlin EXCOR Pediatric VAD, the initial patient survey should include a pulse and device check (<10 seconds). A palpable peripheral pulse is expected in this population and should be used as an assessment of flow and perfusion. Device function should then be determined by observing movement of the device membrane. If the device membrane is not moving or not providing adequate flow, the drivelines should be assessed to ensure that they are not kinked or disconnected from the driver and that the IKUS driver is powered and functioning. Chest compressions should be started or the hand pump should be used to provide flow from the device if it is not functioning. Ventilation should be supported as for pediatric patients without a VAD.

If the hand pump is used and a palpable pulse cannot be generated, chest compressions should be started immediately. Chest compressions should also be started immediately for cardiac arrest in patients with an RVAD. The pediatric advanced life support algorithm should be used for the management of resuscitation, including the administration of medications and defibrillation as needed. The VAD team should be alerted at the time of resuscitation, and the patient should be transferred emergently to an intensive care unit.

If loss of power is the reason for driver malfunction, initial support with the hand pump should initiated while a new IKUS driver unit is being reconfigured. If unconsciousness is not related to device function and the patient has a good pulse, other causes of loss of consciousness (eg, cerebrovascular event, hypoglycemia, sedative use, hypoxemia) should be investigated and treated.

SUMMARY

This document represents consensus opinion among MCS specialists and resuscitation and EMS experts concerning responding to the unique needs of MCS-supported patients with cardiac arrest/circulatory collapse. Every effort was made to include key organizations and providers for patients with MCS. Because this is an evolving field, devices vary from center to center and population to population. For that reason, an attempt was made to discuss cardiac arrest in broad terms without focusing on the nuances of each individual device. The Table provides a summary that highlights the basic differences in performing resuscitation in patients with MCS.

KNOWLEDGE GAPS

- What are the risks/benefits of chest compressions to patients with a VAD?
- The group’s current consensus was that withholding chest compression in a patient with a VAD who is truly in circulatory failure that is not attributable to a device failure would cause more harm to the patient than the potential to dislodge the device.
- Does an MAP of ≥50 mm Hg or a PetCO₂ of ≥20 mm Hg act as a good identifier of patients with marginal/adequate cardiac output and those for whom the risk of chest compressions may be outweighed by the benefits?
- The group’s current consensus was that an MAP of 50 mm Hg or a PetCO₂ of >20 mm Hg was a reasonable measure of perfusion and that the addition of chest compressions, as opposed to other interventions, would not be expected to further benefit the patient.
**Table. Basic Differences in Performing Resuscitation in Patients With Mechanical Circulatory Support**

<table>
<thead>
<tr>
<th>Mechanical Support Type</th>
<th>Perfusion (Pulse Check)</th>
<th>ECG</th>
<th>Defibrillation/Cardioversion</th>
<th>Chest Compressions</th>
<th>ACLS Drugs</th>
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<td>p-RVAD</td>
<td>Pulsatile</td>
<td>Present</td>
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<td>p-LVAD</td>
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<td>p-BIVAD</td>
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<td>Present</td>
<td>Acceptable</td>
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<tr>
<td>cf-RVAD</td>
<td>Pulsatile</td>
<td>Present</td>
<td>Acceptable</td>
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<tr>
<td>cf-LVAD</td>
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<tr>
<td>cf-BIVAD</td>
<td>Absent pulsatile</td>
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<tr>
<td>TAH</td>
<td>Pulsatile</td>
<td>Absent</td>
<td>Unacceptable</td>
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</table>

ACLS indicates advanced cardiovascular life support; BIVAD, biventricular assist device; cf, continuous flow; LVAD, left ventricular assist device; p, pulsatile; RVAD, right ventricular assist device; and TAH, total artificial heart.

• What are the emergency experiences of pediatric patients supported by MCS?
  
  This was truly a consensus opinion because few data exist.

• What is the best way to identify a VAD-supported patient, and should it be standardized? If so, how should that happen? Who would monitor/oversee it?
  
  The group’s current consensus was that there should be a standard across the VAD and TAH centers to supply identification necklaces or bracelets to discharged MCS patients. The identification should include the device type, the center contact information, and the patient’s advance directives.

  Each clinical scenario requires an individualized treatment plan. We hope that this consensus document provides guidance and standardization in an area where there is a recognized lack of published evidence yet a clinical need for direction. MCS centers are encouraged to publish their experiences and to educate others about potential best practices.

**ACKNOWLEDGMENT**

This scientific statement is dedicated to the memory of Dr. Richard Kerber, who died suddenly while this manuscript was in press. Dr. Kerber was a lifelong volunteer to the AHA, and under his leadership, the Emergency Cardiovascular Care Committee made groundbreaking progress in bringing CPR to the forefront of the AHA and emergency care. His contributions to the science of resuscitation, in particular defibrillation, led the field in mapping a better understanding of the mechanisms of this lifesaving treatment. He was also a nationally recognized expert echocardiographer whose work over decades had a substantial impact on the field of cardiology. Dr. Kerber’s greatest legacy, however, will be as an unwavering friend and colleague and a tireless teacher and mentor to generations of physician-scientists. He taught by example as well as word, and his passion for resuscitation science and mentorship will be carried on by the countless lives he touched. The field has truly lost a giant. May our dear friend rest in peace.

**FOOTNOTES**

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest. This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on August 1, 2016, and the American Heart Association Executive Committee on September 23, 2016. A copy of the document is available at http://professional.heart.org/statements by using either “Search for Guidelines & Statements” or the “Browse by Topic” area. To purchase additional reprints, call 843-216-2533 or e-mail kelle.ramsay@wolterskluwer.com.


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<td>University of Iowa</td>
<td>None</td>
<td>None</td>
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<td>None</td>
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</tr>
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</table>

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be “significant” if (a) the person receives $10,000 or more during any 12-month period, or 5% or more of the person’s gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10,000 or more of the fair market value of the entity. A relationship is considered to be “modest” if it is less than “significant” under the preceding definition.

*Modest.
†Significant.
CPR With Mechanical Circulatory Support

CLINICAL STATEMENTS

REFERENCES


20. Peberdy et al


Cardiopulmonary Resuscitation in Adults and Children With Mechanical Circulatory Support: A Scientific Statement From the American Heart Association

Mary Ann Peberdy, Jason A. Gluck, Joseph P. Ornato, Christian A. Bermudez, Russell E. Griffin, Vigneshwar Kasirajan, Richard E. Kerber, Eldrin F. Lewis, Mark S. Link, Corinne Miller, Jeffrey J. Teuteberg, Ravi Thiagarajan, Robert M. Weiss, Brian O’Neil and On behalf of the American Heart Association Emergency Cardiovascular Care Committee; Council on Cardiopulmonary, Critical Care, Perioperative, and Resuscitation; Council on Cardiovascular Diseases in the Young; Council on Cardiovascular Surgery and Anesthesia; Council on Cardiovascular and Stroke Nursing; and Council on Clinical Cardiology

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