We are IntechOpen,
the world's leading publisher of
Open Access books
Built by scientists, for scientists

6,900
Open access books available

185,000
International authors and editors

200M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

CLARIVATE ANALYTICS

BOOK
CITATION
INDEX

INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Mechanical Circulatory Support Devices

Sagar Kadakia, Vishnu Ambur, Sharven Taghavi,
Akira Shiose and Yoshiya Toyoda

Additional information is available at the end of the chapter

Abstract

Heart failure (HF) is a global public health concern that has the potential to reach epidemic proportions. The gold standard for treating end-stage HF remains heart transplantation. Unfortunately, given the scarcity of available organs, alternative means for providing cardiac support are required. Mechanical circulatory support devices (MCSDs) have the potential to treat many patients with end-stage HF. They replace some of the mechanical functions of the failing heart to improve cardiac output and organ perfusion. These include the intra-aortic balloon pump, extracorporeal membrane oxygenation, ventricular assist devices, and the total artificial heart. In this chapter, we will discuss a brief history of MCSD, available devices, indications, patient selection, surgical procedures, postoperative management, complications, and outcomes.

Keywords: heart failure, intra-aortic balloon pump, extracorporeal membrane oxygenation, ventricular assist device, total artificial heart

1. Introduction

Heart failure (HF) is a global public health concern, affecting 26 million people worldwide at an estimated cost of $108 billion in 2012. It typically affects the elderly, and both the right heart and left heart can be involved. In right HF, the right ventricle cannot effectively pump blood to the lungs. In left HF, which is more common, the left ventricle cannot effectively pump blood to meet the body's demands. In our aging world, where 22% of the population is expected to be over the age of 60 by 2050 [1], HF has the potential to reach epidemic proportions. Although medications such as beta-blockers, angiotensin-converting enzyme inhibitors, and angiotensin II receptor blockers can alleviate the symptoms of HF and improve mortality [2], the gold

standard for treating end-stage HF remains heart transplantation. Unfortunately, given the scarcity of available organs, only about 4500 heart transplantations were performed world-wide in patients of all ages in 2013 [3]. The demand for heart transplantations is expected to far exceed the available supply for the foreseeable future.

Mechanical circulatory support devices (MCSDs) replace some of the mechanical functions of the failing heart to improve cardiac output and organ perfusion. They have the potential to treat many patients with end-stage HF who cannot be transplanted either due to lack of available organs or socioeconomic reasons. Between June 2006 and December 2014, 15,745 patients were implanted with ventricular assist devices (VADs) and the total artificial heart (TAH) in the United States. These numbers have been rising steadily, from approximately 1000 devices implanted in 2009 to approximately 2500 devices implanted in 2014 in the United States [4]. Besides the VAD and the TAH, other types of MCSD exist, including the intra-aortic balloon pump (IABP) and extracorporeal membrane oxygenation (ECMO). The IABP, ECMO, and VAD can be utilized as a bridge to recovery (BTR). All four types of MCSD can be utilized as a bridge to transplantation (BTT), whereas only VADs can be utilized as destination therapy (DT).

In this chapter, we will discuss a brief history of MCSD, available devices, indications, patient selection, surgical procedures, postoperative management, complications, and outcomes.

1.1. History

MCSDs trace their origin to the early days of cardiac surgery. In 1953, Dr. Gibbon successfully utilized the heart–lung machine, which he developed over the course of two decades, to repair an atrial septal defect (ASD) in an 18-year-old woman [5]. This patient did well and was discharged home. Although his subsequent efforts to utilize the heart–lung machine to close ASD were met with poor results, secondary to misdiagnosis and bleeding complications, he laid the foundation for modern-day open-heart surgery with cardiopulmonary bypass [6]. Soon afterwards, it became clear that failure to wean off cardiopulmonary bypass was a significant problem in the field of cardiac surgery. Dr. Spencer ushered in the use of mechanical support for postcardiotomy cardiac recovery in 1959. He utilized left atrial–femoral cardio-pulmonary bypass to provide temporary cardiac support in postoperative patients. This early work in mechanical support set the stage for mechanical support for cardiogenic shock [7]. In 1966, Dr. DeBakey performed the first successful implantation of a VAD. He implanted a paracorporeal VAD from the left atrium to the right subclavian artery in a patient with cardiogenic shock after a double valve replacement. The patient required mechanical support for 10 days and ultimately recovered [8]. Dr. Kantrowitz was the first to successfully utilize the IABP in 1967. His patient was a 45-year-old woman who was in cardiogenic shock secondary to an acute myocardial infarction. She remained on the IABP for 7 hours and, during this time, was weaned off all vasopressors ([9]. In 1984, Dr. DeVries reported on the first clinical use of the TAH. Although the patient ultimately died after 112 days from multiorgan system failure, the TAH remained functional and uninvolved in any thrombotic or infectious proc-esses [10].

Our modern era of MCSD began in 2001 with the Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure (REMATCH) trial. This landmark study compared patients with advanced HF who underwent left ventricular assist device (LVAD) implantation versus maximal medical therapy. It showed a survival benefit of 52% in the LVAD group versus only 25% in the medical therapy group at 1 year ($p = 0.002$). Likewise, using a variety of quality-of-life questionnaires including the SF-36, Minnesota Living with HF, and Beck Depression Inventory, the patients who underwent LVAD implantation had a statistically significant improved quality of life when compared to the medically managed patients [11]. This pivotal study played a key role in the approval of the LVAD for DT in the United States in November 2002 [12].

Each of these technological advancements in mechanical support has been monumental. Equally important was the creation of a national database, the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS), in the United States. In 2005, this was developed as a joint venture between the National Heart, Lung, and Blood Institute, the Food and Drug Administration, the Centers for Medicare and Medicaid Services, and the University of Alabama at Birmingham. This database serves as a registry for all patients who have been implanted with VAD and TAH. It collects data from the index hospitalization, follow-up appointments, and all major adverse events to improve clinical outcomes and promote research into new devices [4, 13].

2. Devices

2.1. Intra-aortic balloon pump

2.1.1. Background

The IABP is the most frequently utilized MCSD, having been in clinical practice for more than 40 years. When mechanical support is indicated, it is often the first one employed as it could be readily inserted and has a relatively low complication rate. Upwards of 70,000 patients are supported annually with the IABP in the United States [14]. It serves as a temporary MCSD that can be placed quickly at the bedside, in the interventional cardiology suite, or in the operating room to improve shock and promote organ perfusion.

2.1.2. Basic principles

The IABP results in decreased myocardial oxygen demand and an increase in myocardial oxygen supply. These physiologic effects are achieved through a reduction in the afterload and an increase in coronary perfusion [15]. The IABP functions by reducing left ventricular afterload, which leads to a decrease in left ventricular wall stress. Since wall stress is proportional to oxygen consumption, this reduction in afterload and wall stress results in decreased myocardial oxygen consumption. In addition, the IABP increases aortic diastolic pressures.

Since the coronary arteries are perfused during diastole, this increase in aortic diastolic pressures leads to increased coronary perfusion and thus myocardial oxygen supply [16, 17]. The combined physiologic effects of afterload reduction and augmented coronary perfusion lead to an improvement in cardiac output.

Figure 1. IABP Console (Image courtesy of Teleflex.com).

The sequential inflation and deflation of the IABP is synchronized to the electrocardiogram (EKG), pacemaker, or to the arterial pressure tracing. This results in counterpulsation and the various hemodynamic changes that are observed. IABP inflation is timed to coincide with the closure of the aortic valve. As seen on the aortic pressure tracing, this coincides with the dicrotic notch. Alternatively, it is timed to coincide with the T-wave of the EKG. With counterpulsation, the IABP rapidly inflates. This inflation increases the intra-aortic pressures and displaces a volume of blood equivalent to the volume of the balloon (usually 30–50 mL in adults, 2.5–25 mL in children) away from the balloon. This results in increased coronary perfusion.

Figure 2. IABP Screen with EKG and aortic pressure tracing (Image courtesy of Teleflex.com).

The second phase of the counterpulsation occurs with deflation of the IABP. Deflation is timed to occur with the onset of the R-wave, or alternatively before the start of systole. Deflation occurs as late as possible, in order to maintain the increased aortic diastolic pressures. It is also timed to occur rapidly, which results in a vacuum effect that leads to a reduction in the afterload by movement of blood toward the balloon [18].

There are several factors that influence the efficacy of the IABP, including heart rate, rhythm, balloon volume, proximity to the aortic valve, and aortic compliance. As the heart rate increases, the amount of time the heart spends in diastole decreases. Thus, the IABP is less likely to function efficiently with tachycardia, since there would be less diastolic coronary flow augmentation. The optimal rate reported in the literature is from 80 to 110 beats/minute [19]. In addition, having a normal sinus rhythm allows for readily identifiable waves on EKG or the aortic pressure tracing, which improves the performance of the IABP. Since the balloon size is proportional to the amount of blood displaced, a larger balloon allows for increased coronary perfusion and a greater reduction in afterload. In addition, the proximity of the IABP to the aortic valve affects diastolic augmentation of coronary perfusion. There is greater diastolic augmentation when the balloon is positioned closer to the aortic valve. Finally, aortic compliance affects the function of the IABP. Increased aortic compliance, which is seen in younger patients, results in a decrease in diastolic augmentation [18, 20].

2.1.3. Components and insertion

The IABP system is composed of a dual lumen catheter, with the inner lumen serving to monitor aortic pressures and the outer lumen connected to a polyethylene balloon that inflates and deflates. It is also composed of a pump console that controls the inflation and deflation of

the balloon. The catheters are available in a variety of sizes, depending on the patient's height. Helium gas is utilized to inflate the balloon, since it has a low density and rapidly transfers from the console to the balloon. In addition, helium is inert and could be rapidly absorbed into the blood stream in the event the balloon ruptures.

The IABP can be placed via both percutaneous and open surgical techniques. In the percutaneous technique, the common femoral artery is punctured with an introducer needle. Using the modified Seldinger technique, a guidewire is inserted and then the IABP catheter is inserted over the wire. The catheter is then positioned about 1–2 cm distal to the left subclavian artery and confirmed with fluoroscopy or chest roentgenogram [18]. Care has to be exercised to prevent obstruction of the left subclavian artery and left carotid artery by a highly placed IABP. Care also has to be exercised to avoid placing the IABP too low, as it could occlude mesenteric and the renal arteries.

Alternatively, the IABP can be placed via open surgical techniques. This is typically reserved for patients with severe peripheral vascular disease (PVD) affecting the distal aorta, iliac arteries, or femoral arteries. In such cases, placement of an IABP can lead to critical limb ischemia. For these patients, the IABP can be inserted into the ascending aorta, aortic arch, common iliac artery, subclavian artery, axillary artery, and brachial artery [21].

2.1.4. Indications and contraindications

The indications for the IABP include postcardiotomy syndrome, prophylactic support for high-risk percutaneous coronary interventions, myocardial infarction or its mechanical complications, unstable angina, cardiogenic shock, and as a bridge to heart transplantation. The Benchmark Registry, a multi-institutional study with nearly 17,000 patients, investigated the patient demographics, outcomes, and complications of the IABP. They found that the indications for the IABP were most frequently hemodynamic support during or after cardiac catheterization (21%), cardiogenic shock (19%), and weaning from cardiopulmonary bypass (16%) [22].

There are several important contraindications to using the IABP. These include aortic regurgitation, since the IABP would result in increased regurgitation during diastolic augmentation. In addition, severe aortic diseases, such as aneurysm or aortic dissection, are a contraindication. In this setting, the placement of an IABP could result in aortic rupture or extension of the dissection. Placing them in the femoral region is contraindicated in patients with known severe PVD [18, 23].

The authors believe the IABP should be the first MCSD that should be placed for typical cardiogenic shock, when no obvious contraindications exist. Despite the interpretation of the controversial IABP-Shock II trial, which showed that the IABP did not lead to a reduction in 12-month all-cause mortality in patients undergoing early revascularization for myocardial infarction complicated by cardiogenic shock [24], we believe the IABP should be the first to be placed for cardiogenic shock. We believe this for a number of reasons, including its availability at most hospitals, the ability to initiate its use quickly, and the ability to upgrade it to a more advanced MCSD if the cardiogenic shock remains poorly addressed.

2.1.5. Weaning and removal

Little data exists on how to wean patients off the IABP with minimal hemodynamic consequences. Generally, weaning is performed in one of two ways: rate reduction or volume deflation. In the first method, the ventricular assist rate is gradually reduced from full support of every beat (1:1) to cardiac support every other beat (1:2) to finally cardiac support every three beats (1:3). Once the patient demonstrates that he can tolerate this wean, the IABP is discontinued. The IABP can also be weaned by maintaining a ventricular rate of 1:1 and deflating the balloon over several hours. This leads to a decrease in the counterpulsation and a decrease in diastolic augmentation [25, 26]. There are limited studies in the literature that have assessed which weaning method is superior. Onorati et al. [26] showed that weaning using volume deflation led to improved hemodynamic and metabolic parameters in their study.

Once the patient has demonstrated that he can tolerate being weaned, the IABP is removed. The removal of percutaneously placed IABP can be performed at the bedside. After prepping the femoral artery entry site, the balloon catheter is disconnected from the console. It is then completely deflated and the catheter is removed. Retrograde bleeding is first allowed, which enables blood clots to flush into the wound. This potentially prevents distal embolization of blood clots. After this, antegrade flushing is allowed, to once again prevent distal embolization of blood clots. Finally, the puncture site in the femoral artery is compressed and pressure is maintained for about 30 minutes. Inadvertent placement of the IABP above the inguinal ligament can result in a retroperitoneal bleed, while poor technique at the time of removal can result in a femoral artery pseudoaneurysm and distal embolization with leg ischemia.

2.1.6. Complications

The overall complication rate has been reported to be low. The Benchmark Registry found that the incidence of all complications to be 7%. This study found that severe complications occurred in 2.8% of the patients. Severe complications were defined as severe bleeding (with hemodynamic instability requiring transfusions or surgical interventions), major limb ischemia (with loss of pulse or sensation or the presence of pallor), balloon rupture, or in-hospital mortality related to the IABP. Multivariate logistic regression analysis identified several predictors of major complication, including female gender, PVD, body surface area <1.65 m^2, and age >75 years [22].

Additional complications include renal artery occlusion and renal failure if the IABP is placed too distally. By manipulating the aorta with the guidewire and catheter, there is always the possibility of distal embolism, resulting in bowel ischemia or lower extremity ischemia.

In the Benchmark Registry, the incidence of balloon rupture was found to be 1% [22]. Balloon rupture usually could be detected by the presence of blood either in the IABP driveline or in console alarms. Balloon rupture is thought to occur secondary to abrasive contact between the balloon and atherosclerotic plaque in the aorta. It necessitates the immediate removal of the IABP to prevent thrombus formation around the balloon and distal embolization.

2.2. Extracorporeal membrane oxygenation

2.2.1. Background

Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) is another modality for short-term mechanical support in patients with HF refractory to medical management. Initial reports of its success were described by Baffes et al. in the 1970s in the pediatric population [27]. Since that time, its applications have been broadened to include adult patients with reversible cardiogenic shock and as a bridge to VAD implantation or transplantation. It can also serve as rescue therapy in patients with cardiopulmonary arrest [28]. Chen et al. showed that VA-ECMO could be utilized in patients in cardiopulmonary arrest after 10 minutes of unsuccessful advanced cardiovascular life support. It was shown that VA-ECMO could extend the duration of cardiopulmonary resuscitation (CPR) with 50% survival with acceptable neurologic status at 30 minutes [29]. A recent meta-analysis showed ECMO in adult patients in cardiac arrest had statistically significant improved outcomes if they were younger (age 17–41) and had a shorter duration of ECMO support (0.9–2.3 days) [30].

Figure 3. ECMO circuit with pump and heater.

2.2.2. Basic principles

VA-ECMO is a form of advanced cardiopulmonary life support which functions essentially like cardiopulmonary bypass. Blood is drained from a central vein, circulated in the ECMO circuit, and then returned to the arterial system. In neonates and children, the carotid artery is

typically accessed for arterial cannulation. In adults, arterial cannulation is obtained via the femoral, axillary, or carotid artery. Venous access is obtained via the femoral or internal jugular vein. It provides cardiac support by augmenting cardiac output and respiratory support by assisting in gas exchange. VA-ECMO can be utilized for a period of days to weeks [31].

2.2.3. Components and insertion

The ECMO circuit consists of a blood pump and membrane oxygenator with a heat exchanger connected to the VA-ECMO cannulas. The membrane oxygenator has a membrane that readily allows diffusion of oxygen and carbon dioxide across it. Oxygen can be added to the system by increasing the amount of oxygen supplied to the oxygenator. Carbon dioxide can be adjusted by changing the gas flow rate or the "sweep." The heat exchange helps maintain normothermia, as heat is readily lost through the circuit.
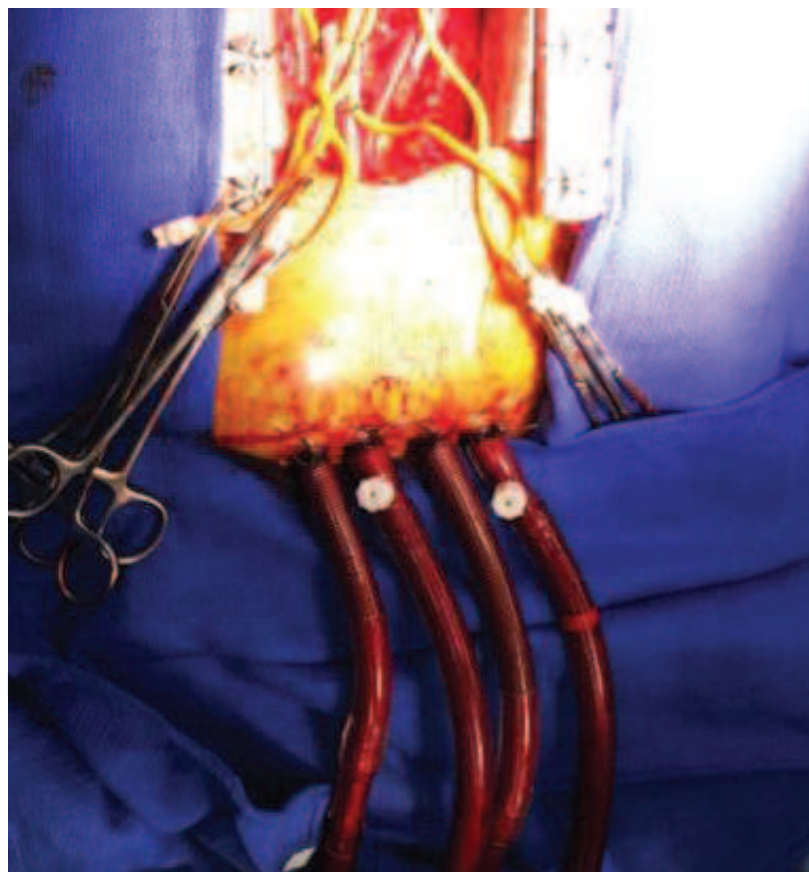

Figure 4. Biventricular assist devices (Centrimag).

Cannulation insertion techniques include both percutaneous and open surgical approaches. Percutaneous cannulas are inserted using the Seldinger technique and can be placed readily at the bedside. After gaining access, the vessels are serially dilated and the cannula is then placed over the guidewire. The patient requires full anticoagulation with heparin. The femoral vessels are most commonly used in the adult. For adults, the arterial cannula size ranges

between 12 French (Fr) and 22 Fr and the venous cannula size ranges between 18 Fr and 28 Fr. An arterial perfusion cannula (typically 5–8 Fr) can also be added to perfuse the lower extremity, as lower extremity ischemia is a source of significant morbidity and mortality. A similar venous drainage line can be placed to drain blood from the lower extremity, as venous outflow obstruction too can lead to lower extremity ischemia [32].

Figure 5. Biventricular assist devices (Centrimag).

Our institutional practice has been to use a 15 Fr arterial cannula placed via the Seldinger technique for patients who need modest support. This allows us to decannulate the patient at bedside without needing to perform a cutdown. When patients require more robust cardiac support, we use larger cannulas, typically 17–21 Fr. We prefer to perform open insertion when these larger cannulas are required. It also requires decannulation in the operating room. We prefer to initiate VA-ECMO in the operating room whenever possible, provided the patient is stable enough to tolerate transport and waiting for operating room availability. Distal perfusion cannulas should be routinely placed. If they are not utilized, the lower extremity has to be monitored for ischemia. There has to be a low threshold for insertion of a distal perfusion cannula, as the morbidity and mortality of an ischemic limb in a patient on VA-ECMO can be catastrophic. We perform central VA-ECMO on our patients who have postcardiotomy cardiogenic shock or in patients who need robust support. We usually use 18–22 Fr arterial cannulas placed in the ascending aorta and 31–40 Fr venous cannulas placed in the right atrium.

2.2.4. Indications and contraindications

The indications for VA-ECMO in adult patients are cardiogenic shock from a variety of causes including acute myocardial infarction, myocarditis, drug toxicity, and pulmonary embolism. It also includes peripartum cardiomyopathy, decompensated chronic HF, postcardiotomy shock, and as a bridge to VAD implantation or heart transplantation. Typically, patients have low cardiac index (<2 L/min/m^2) and hypotension (systolic blood pressure <90 mmHg) despite inotropic agents, IABP, and adequate volume resuscitation [33]. Advantages of VA-ECMO over temporary VAD include ease of insertion bedside and not needing to transport a hemodynamically unstable patient to the operating room.

Absolute contraindications to ECMO include patients who are not candidates for transplantation or VAD implantation, disseminated malignancy, unwitnessed cardiac arrest, end-stage organ dysfunction, non-compliance, and patients with prolonged CPR without adequate tissue perfusion. Relative contraindications include advanced age, obesity, and contraindications to anticoagulation [33, 34].

2.2.5. Management

There are no randomized trials to date that have validated management guidelines. However, there are general management strategies that are implemented at most institutions. Such strategy is geared toward minimizing multiorgan system dysfunction. The complex management of a patient on VA-ECMO requires coordination and communication between the cardiothoracic surgeon, critical care intensivist, perfusionist, nurses, and ancillary staff.

For the respiratory system, successful management requires aggressive pulmonary toileting. This necessitates frequent endotracheal suctioning and possible bronchoscopy, positional changes, nebulizers, and chest roentgenogram. The fraction of inspired oxygen (FIO$_2$) is minimized in order to lessen oxygen toxicity. In order to lessen atelectasis, increased positive end-expiratory pressures (PEEPs) is often implemented [33]. Schmidt et al. showed that higher PEEP during the first 3 days on ECMO led to improved survival [35].

Successful cardiovascular support requires maintaining perfusion and aggressive volume resuscitation during the first few days on ECMO. Since ECMO promotes release of cytokines and a generalized systemic inflammatory response syndrome, adequate volume resuscitation with crystalloid or colloid is paramount. In addition, inotropic support is often required as the heart recovers.

The management of the renal system is often complex in the ECMO patient. Nearly 70–85% of patients on ECMO develop acute kidney injury (AKI) [36]. The first 2 days on ECMO usually require aggressive fluid resuscitation and is associated with oliguria. The diuretic phase usually begins after 2 days. Frequently, diuretics are utilized to improve mobilization of extravascular fluid. If AKI does not improve, renal replacement therapy often becomes indicated. However, the requirement for dialysis carries a significant mortality risk. Kielstein et al. showed that patients on ECMO who required dialysis had a 3-month survival rate of 17% while those on ECMO who did not required dialysis had a 3-month survival rate of 53% ($p = 0.001$). They also showed that duration of dialysis was associated with increased mortality [37].

Management of the gastrointestinal system and nutrition focuses on maintaining the integrity of the gastrointestinal mucosa, in order to lessen translocation of bacteria. This is accomplished with proton pump inhibitors or histamine blockers and enteral nutrition when possible [38]. However, parenteral nutrition is often required to supplement nutrition, as enteral nutrition is often interrupted. A recent study by Ridley et al. showed that enteral nutrition was interrupted for a median of 8 hours on 53% of the days. This was secondary to high gastric residual volume or fasting for a procedure or diagnostic test [39].

Neurologic complications are very common with ECMO and the clinician needs to have a low threshold to pursue imaging to rule out intracranial hemorrhage and acute stroke. Recently, all patients who received ECMO between 2001 and 2011 were selected from the Nationwide Inpatient Sample. Neurologic complications included acute ischemic stroke, intracranial hemorrhage, and seizures were evaluated. Of the 23,951 patients included in the study, 10.9% of patients suffered seizures, 4.1% suffered strokes, and 3.6% suffered intracranial hemorrhage. Patients who suffered intracranial hemorrhage were found to have a higher mortality rate, length of stay, and discharge to a long-term facility than those who did not have intracranial hemorrhage. Similarly, patients who suffered acute ischemic stroke had higher rates of discharge to long-term facilities and length of stay than patients who did not have an acute ischemic stroke. No difference in outcomes was found between those who had seizures and those who did not [40].

2.2.6. Weaning

Currently, there is a lack of established guidelines on weaning patients from VA-ECMO. Generally, patients have to demonstrate hemodynamic stability, recovery of organ dysfunction, resolution of pulmonary edema, and be in a euvolemic state. Echocardiogram is an invaluable tool for assessing cardiac recovery. Different institutions utilize different weaning parameters. Generally, weaning is accomplished once echocardiogram shows improvement of cardiac function. ECMO support is then gradually weaned, with flows reduced to 50% and then 25%. If the patient tolerates this, the circuit is then clamped between 30 minutes to 4 hours. The cannulas must be flushed with heparinized saline frequently to prevent thrombosis. Once the patient tolerates this, decannulation can be performed at bedside or in the operating room for large cannulas [41].

2.2.7. Complications

The most common complications in patients on VA-ECMO are bleeding and thrombosis. Bleeding requiring surgical treatment has been reported to occur in nearly 34% of patients on VA-ECMO [42]. It is a major concern for patients on VA-ECMO since the patients are anticoagulated with heparin and have platelet consumption and dysfunction [33]. Anticoagulation is a critical component to prevent circuit thrombosis, although there are no clear guidelines for standardized goals. However, most institutions use heparin with an activated partial thromboplastin time (aPTT) of 60–80 seconds. There is also an increased risk of disseminated intravascular coagulation (DIC) and heparin-induced thrombocytopenia (HIT). HIT mandates anticoagulation with a non-heparin-based agent. Available medications include bivalirudin

and argatroban. Additional complications include infection, neurological complications, and limb ischemia. Limb ischemia is a function of cannula size and positioning in relation to the patient's vasculature. Reperfusion cannulas to perfuse distal to the entry site have decreased the risk of this complication [32, 43].

2.3. Ventricular assist devices

2.3.1. Background

VAD technology has advanced rapidly since the landmark REMATCH trial, which demonstrated survival and quality of life improvement in patients who underwent implantation of an LVAD compared to patients receiving maximal medical therapy. It also highlighted several limitations of pulsatile devices, namely device failure and thromboembolism [11]. Advancement in technology has led to the development of continuous-flow devices. These devices have been associated with significantly improved survival free from stroke and device failure at 2 years, when compared to pulsatile devices [44]. Overall survival with the current continuous-flow VADS in use today is reported to be 80% at 1 year and 70% at 2 years post implantation [4].
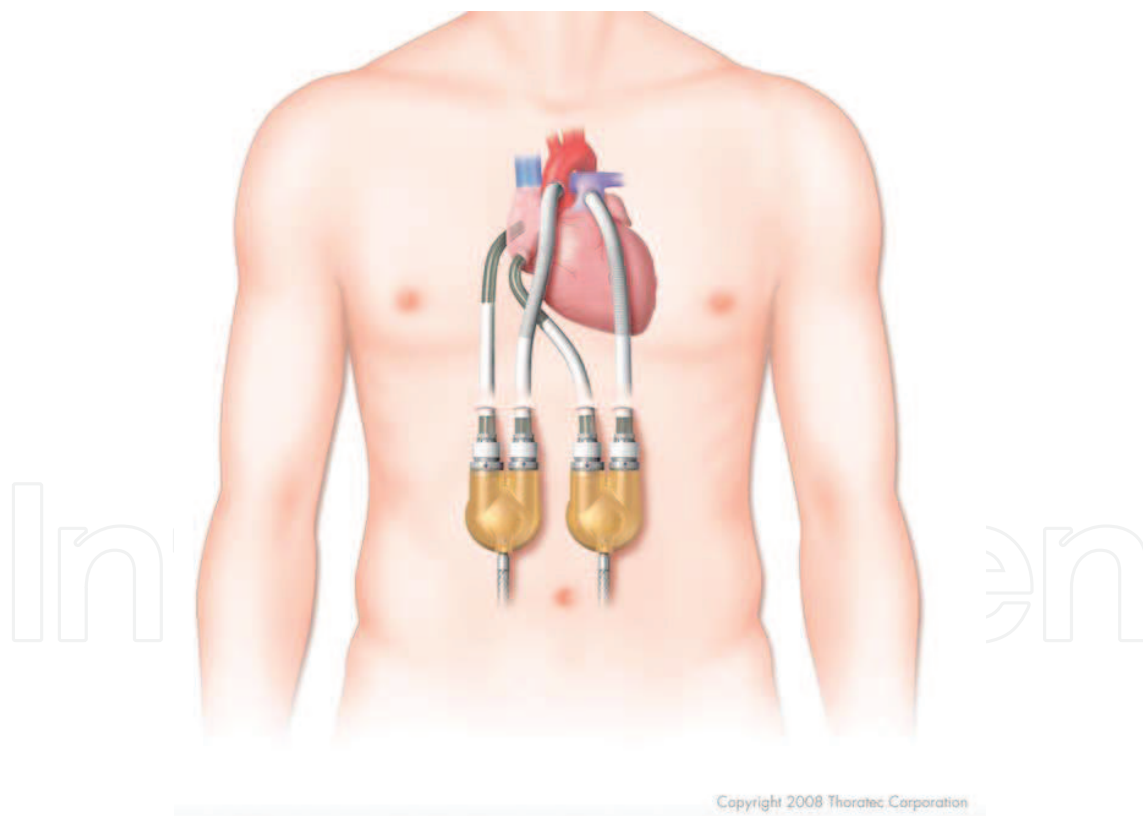

Copyright 2008 Thoratec Corporation

Figure 6. Paracorporeal ventricular assist devices (Reprinted with the permission of Thoratec Corporation).

Most frequently, VADs are implanted to assist the left ventricle. The inflow cannula is inserted into the left ventricle and the outflow cannula is inserted into the ascending or descending aorta. They can also provide right ventricular and biventricular support. In a right ventricular

assist device (RVAD), the inflow cannula is inserted into the right atrium or right ventricle and the outflow cannula is inserted into the pulmonary artery.

Figure 7. HeartMate XVE (left) and HeartMate II (right) (Reprinted with the permission of Thoratec Corporation).
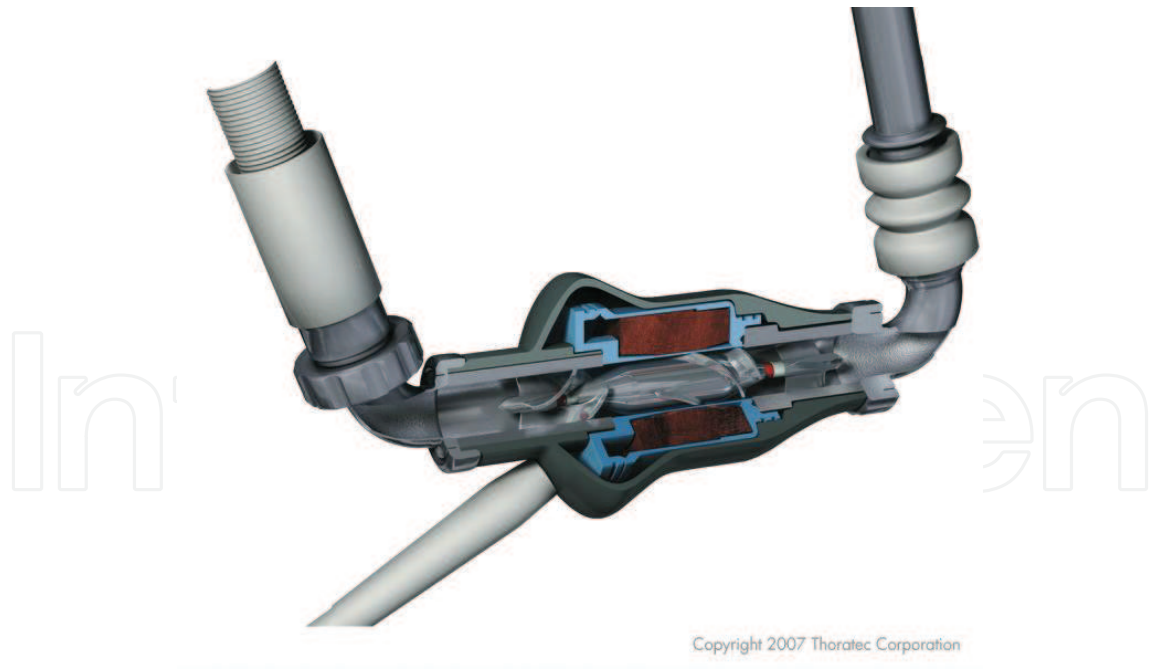
Figure 8. HeartMate II cross-section view with internal rotor (Reprinted with the permission of Thoratec Corporation).

VADs can be classified based on how they function mechanically. The first-generation VADs rely on pulsatile-flow technology. These VADs contain one-way valves and a flexible pumping chamber, which is compressed by an electric motor or pneumatic pressure. This forces blood

into the circulation. These patients will have a palpable pulse and a measurable blood pressure. The second-generation VADs are continuous axial flow devices while the third-generation VADs are continuous, centrifugal flow devices. These nonpulsatile, continuous-flow devices have internal rotors, which propel blood continuously. These patients have either weak, irregular pulses or non-palpable pulses.

In addition, VADs can provide both short-term and long-term circulatory support. Those designed for short-term circulatory support serve to restore organ perfusion quickly to relieve organ ischemia. They are used for BTR and BTT. These are non-implantable and provide support for days to weeks. Long-term VADs are implantable and are used for DT or BTT. The majority of VAD are implanted surgically. However, there are devices currently available that can be placed percutaneously.
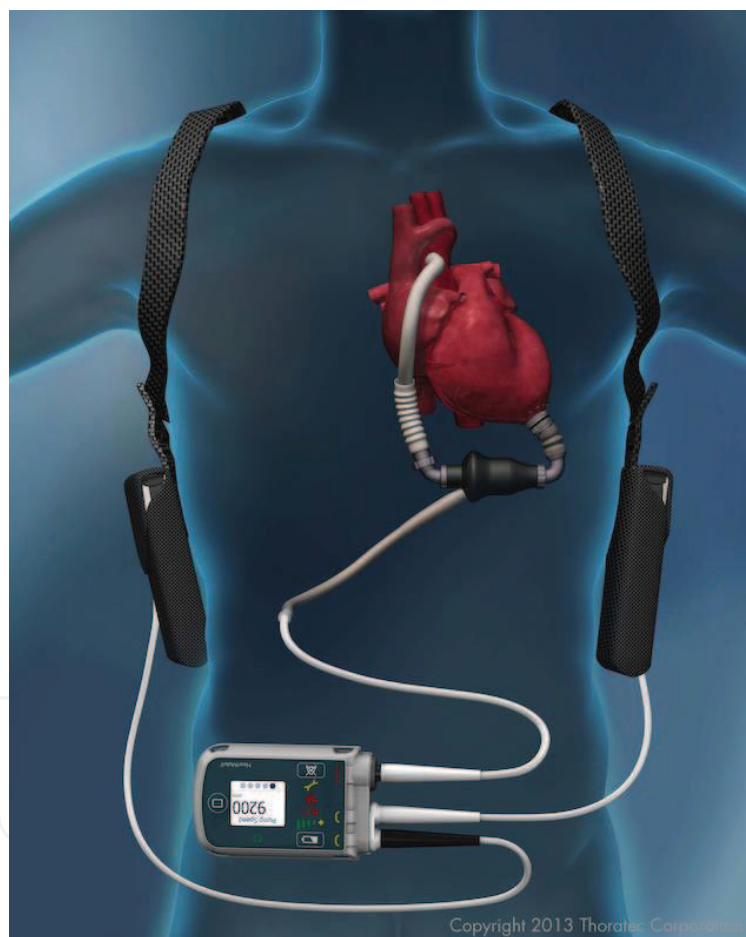

Figure 9. HeartMate II (Reprinted with the permission of Thoratec Corporation).

VADS are preload dependent and afterload sensitive. They can function independently of the EKG and most require anticoagulation. Generally, these devices are prone to infection, bleeding, hemolysis, thrombosis, cerebrovascular accidents, and mechanical malfunction. In addition, right ventricular dysfunction is a known complication of LVAD insertion, as the right ventricle has to increase its output to match the left heart and LVAD.

Figure 10. HeartMate 3 (Reprinted with the permission of Thoratec Corporation).

2.3.2. Indications for VAD

As mentioned earlier, indications for VAD can be divided into three main groups: BTR, BTT, and DT. Mechanical support with a VAD is used as a BTR in patients with acute decompensated HF. These patients typically have reversible causes of HF, such as postcardiotomy syndrome, medicine-induced cardiomyopathy, postpartum cardiomyopathy, and viral myocarditis. A retrospective study showed that patients who have acute fulminant myocarditis that progressed rapidly (median of 7 days from onset of symptoms to VAD implantation) had a greater likelihood of recovery of cardiac function and VAD explantation than patients who had a more indolent presentation (median of 22 days between onset of symptoms to VAD implantation). Those with a more indolent presentation were more likely to progress to needing a heart transplantation [45].

BTT remains the most common indication for MCSD. The Seventh INTERMACS report showed that implantation of VADs as BTT was about 51% in 2014 [4]. VADs allow patients with advanced HF to become healthier while awaiting heart transplantation. It has been shown that patients who receive a VAD as a BTT have improved functional capacity and quality of life [46].

DT refers to the use of VAD as definitive treatment for patients who do not qualify for heart transplantation. The Seventh INTERMACS report showed that implantation of VADs as DT has been steadily rising. In the year 2014, 45.7% of all VADs implanted in the United States have been for DT, up from 43.6% in 2013. This is in stark contrast to the 2008–2011 time period, when only 28.6% of all VADs implanted were for DT [4].

A fourth group, bridge to decision (BTD), is a new designation for those patients whose candidacy for heart transplantation or permanent VAD is still not determined, either due to

medical or socioeconomic reasons. A recent study investigating the CentriMag VAD, an external continuous-flow device, as BTD therapy in patients with refractory cardiogenic shock demonstrated survival of 69.2% at 30 days and survival of 48.6% at 1 year. This study showed that 30% of patients had sufficient myocardial recovery to allow explantation of the VAD. Another 15% of patients progressed to needing a permanent VAD and 18% of patients required heart transplantations [47].

2.3.3. Short-term VADs

Short-term VADs are available as pulsatile and nonpulsatile devices. The short-term pulsatile device includes the first-generation Abiomed BVS 5000 and the second-generation Abiomed AB 5000, which allows the patient to be more mobile. These are paracorporeal devices that can provide left ventricular, right ventricular, or biventricular support for several weeks. In a retrospective single institution review, the Abiomed BVS 5000 was inserted for precardiotomy cardiogenic shock in 18 patients and for postcardiotomy cardiogenic shock in 53 patients. Of these, 62% of the patients survived, with 41% of patients being successfully weaned after myocardial recovery, 11% receiving a long-term LVAD, and 10% receiving a heart transplantation [48]. Similar to other VADs, patients require anticoagulation while being supported by this device.

Nonpulsatile short-term devices that could provide univentricular and biventricular support include the Impella and TandemHeart. These devices require anticoagulation and could be readily placed in the interventional cardiology suite for acute HF. The Impella LP 2.5 is a catheter-based VAD that is inserted via the femoral artery. It is passed into the aorta, through the aortic valve, and into the left ventricle. The catheter has an inlet at its tip and an outlet more proximally. This device functions by pumping blood through the inlet located in the left ventricle and into the outlet located in the ascending aorta. This lessens the amount of work the left ventricle has to perform and augments cardiac output. The Impella LP 5.0 works similarly but is larger and requires a formal aortotomy or femoral artery cutdown.

However, the TandemHeart consists of a centrifugal pump and a cannula placed through the femoral vein and guided into the right atrium. From there, the cannula is placed transeptally into the left atrium. A femoral artery cannula is also placed and blood from the left atrium is passed into the femoral artery, thereby bypassing the left ventricle [49]. In the largest study of its kind, Patel et al. showed that percutaneous VADs such as the TandemHeart and Impella led to a statistically significant reduction in mortality when compared to the IABP in patients undergoing percutaneous coronary intervention [50].

2.3.4. Long-term VADs

Long-term VADS can be divided into three generations. The first-generation VADs rely on pulsatile flow technology. It includes the HeartMate XVE, a VAD that has been extensively studied. In fact, it was this device that was studied in the seminal REMATCH trial. It has a textured inner surface, which promotes pseudoneointimal lining formation throughout the pump. Consequently, anticoagulation is not necessary and these patients often only receive

antiplatelet therapy in the form of aspirin. The incidence of neurologic events remains low with this device. A small retrospective study with 21 patients showed no strokes or transient ischemic attacks during the average of 531 days of LVAD support. Only two of these patients developed metabolic encephalopathy, which resolved [51]. Major limitations of this device include increased incidence of infection, device malfunction, and its large size, which makes implantation into patients of a body surface area of less than 1.5 m^2 not feasible. Other first-generation devices include the Novacor, EXCOR, Thoratec IVAD, and Thoratec PVAD.

The second-generation VAD rely on a rotatory axial pump design. These are typically much smaller than the first-generation VADs because of the nonpulsatile flow design. By eliminating pulsatile flow, the need for having valves and chambers was eliminated. In addition, there are less moving parts leading to less hardware dysfunction. They also require less energy consumption. This group includes the HeartMate II, MicroMed DeBakey (now ReliantHeart), and Jarvik 2000. The HeartMate II has been extensively studied and has revolutionized the field of VAD. It was investigated against the HeartMate XVE by Slaughter et al. In their study, they showed that continuous flow LVAD had a statistically significant improved probability of survival free from stroke and device failure, when compared to pulsatile devices. In addition, the HeartMate II had actuarial survival rates of 68% at 1 year, compared to 55% for the HeartMate XVE. The survival benefit extended to 2 years, with 58% survival in the HearMate II cohort and 24% in the HeartMate XVE cohort [44].

The third-generation VAD also relies on continuous flow technology. However, instead of having a rotor in contact with blood, which results in hemolysis, there is a hydrodynamic or magnetic levitation component, which eliminates contact with blood. These include the DuraHeart, HeartMate 3, HeartWare, Evaheart, and INCOR. These remain investigational and in clinical trials in the United States. In Europe, several studies have already been conducted. A retrospective study from Italy reviewed the INCOR VAD in 42 patients. In their cohort, Iacovoni et al. showed survival of 74% at 1 year and 60% at 2 years. The most frequent adverse events included driveline infection, stroke, sepsis, and right HF. No episodes of pump thrombosis or gastrointestinal bleeding occurred [52].

2.3.5. Operative technique

Typically, VADs are placed via a median sternotomy and require cardiopulmonary bypass. A preperitoneal pocket is created to implant the device. Meticulous hemostasis is necessary since postoperative hematomas in the device pocket can predispose to infections. Alternatively, with the increasing miniaturization of the VADs, they can be placed within the pericardium.

With some of the newer models, implantation is possible via a thoracotomy incision and without cardiopulmonary bypass [53, 54].

2.3.6. Postoperative management

Postoperative care after VAD implantation requires a multidisciplinary approach to care. However, special attention has to be paid to blood pressure monitoring, anticoagulation, and right ventricular function. While arterial lines are in place, blood pressure can be titrated to

mean arterial pressure (MAP) of 70–80 mmHg. Once invasive lines are discontinued, a Doppler probe and sphygmomanometer can be utilized to measure blood pressure. Antiplatelet therapy and anticoagulation is started within a few days postoperatively, once risks of bleeding and coagulopathy have subsided. Antiplatelet therapy is started with aspirin. Anticoagulation is started with a heparin drip and the transitioned to oral warfarin. Alternatively, starting warfarin without a heparin bridge has been reported. It has been shown that such management reduces the need for blood transfusions without increasing risks for short-term thrombosis or thromboembolic events [55]. The right ventricle is supported with the use of inotropes and pulmonary vasodilators such as nitric oxide, prostaglandins, and milrinone. Despite maximal medical therapy, if central venous pressures remain consistently above 20 mmHg and the cardiac index remains below 2 L/min/m^2, implantation with a temporary RVAD may be indicated.

2.4. Total artificial heart

2.4.1. Background

The TAH is a mechanical support device, which has not yet gained widespread acceptance. Only 66 were implanted in the United States in 2013 [4]. It provides pulsatile biventricular support and is pneumatically powered. With this device, the right and left ventricle and all the heart valves are removed. The removal of the native ventricles and valves eliminates many of the problems seen with LVAD or biventricular support, namely right HF, valvular regurgitation, and arrhythmias. The TAH is connected directly to the atria. It is currently approved

Figure 11. Total Artificial Heart with battery pack (Image courtesy of Syncardia.com).

for support for biventricular HF as a BTT [56]. It is available as the SynCardia TAH and AbioCor.

2.4.2. Indications

The main indication for the TAH is as a BTT for biventricular failure. It can be used in patients who have contraindications to LVAD and biventricular assist devices implantation. Such patients include those infiltrative or restrictive cardiomyopathies, aortic regurgitation, severe cardiac arrhythmias, and left ventricular thrombus.

In a study by Copeland et al., 81 patients were implanted with the TAH as a BTT. Of these, the rate of survival to transplantation was 79%. The overall 1-year survival for patients implanted with the TAH was 70%. In those patients who received heart transplantation after a TAH, the 1-year survival was 86% and the 5-year survival was 64% [57].

2.4.3. Limitations and complications

Major limitations to its use include its large size, requiring the patient to have a body surface area of at least 1.7 m². It also requires extensive surgery to remove both of the ventricles and all the valves. It is also fraught with complications including infection, postoperative bleeding, and thromboembolic events [58].
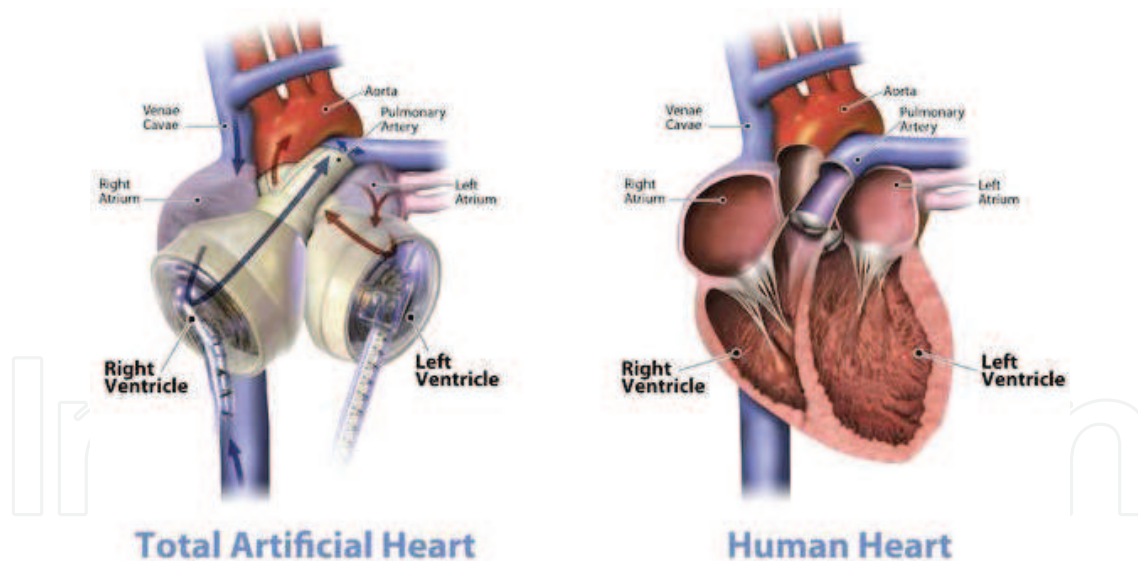

Figure 12. Total Artificial Heart (Image courtesy of Syncardia.com).

3. Conclusion

With the increase in HF and the lack of available hearts for transplantation, MCSDs will continue to play a greater role as a BTR, BTT, or DT.

Author details

Sagar Kadakia[1], Vishnu Ambur[1], Sharven Taghavi[2], Akira Shiose[3] and Yoshiya Toyoda[3*]

*Address all correspondence to: yoshiya.toyoda@tuhs.temple.edu

1 Department of Surgery, Temple University School of Medicine, Philadelphia, PA, USA

2 Department of Surgery, Division of Cardiothoracic Surgery, Washington University School of Medicine, St. Louis, MO, USA

3 Department of Cardiac Surgery, Temple University School of Medicine, Philadelphia, PA, USA

References

[1] Ageing | UNFPA. United Nations Population Fund [Internet]. [Cited 2015 Dec 19]. Available from: http://www.unfpa.org/ageing

[2] Esper SA, Subramaniam K. Heart failure and mechanical circulatory support. Best Pract Res Clin Anaesthesiol. 2012 Jun;26(2):91–104.

[3] Lund LH, Edwards LB, Kucheryavaya AY, Benden C, Dipchand AI, Goldfarb S, et al. The Registry of the International Society for Heart and Lung Transplantation: thirty-second official adult heart transplantation report—2015; focus theme: early graft failure. J Heart Lung Transplant. 2015 Oct;34(10):1244–54.

[4] Kirklin JK, Naftel DC, Pagani FD, Kormos RL, Stevenson LW, Blume ED, et al. Seventh INTERMACS annual report: 15,000 patients and counting. J Heart Lung Transplant. 2015 Dec;34(12):1495–504.

[5] Gibbon JH. Application of a mechanical heart and lung apparatus to cardiac surgery. Minn Med. 1954 Mar;37(3):171–85; passim.

[6] Cohn LH. Fifty years of open-heart surgery. Circulation. 2003 May 6;107(17):2168–70.

[7] Spencer FC, Eiseman B, Trinkle JK, Rossi NP. Assisted circulation for cardiac failure following intracardiac surgery with cardiopulmonary bypass. J Thorac Cardiovasc Surg. 1965 Jan;49:56–73.

[8] DeBakey ME. Left ventricular bypass pump for cardiac assistance. Am J Cardiol. 1971 Jan 1;27(1):3–11.

[9] Kantrowitz A, Tjønneland S, Freed PS, Phillips SJ, Butner AN, Sherman JL, et al. Initial clinical experience with intraaortic balloon pumping in cardiogenic shock. JAMA. 1968 Jan 8;203(2):113–8.

[10] DeVries WC, Anderson JL, Joyce LD, Anderson FL, Hammond EH, Jarvik RK, et al. Clinical use of the total artificial heart. N Engl J Med. 1984 Feb 2;310(5):273–8.

[11] Rose EA, Gelijns AC, Moskowitz AJ, Heitjan DF, Stevenson LW, Dembitsky W, et al. Long-term use of a left ventricular assist device for end-stage heart failure. N Engl J Med. 2001 Nov 15;345(20):1435–43.

[12] Rector TS, Taylor BC, Greer N, Rutks I, Wilt TJ. Use of left ventricular assist devices as destination therapy in end-stage congestive heart failure: a systemic review. 2012 May [cited 2015 Dec 24]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK99056/

[13] Kirklin JK, Naftel DC. Mechanical circulatory support registering a therapy in evolution. Circ Heart Fail. 2008 Sep 1;1(3):200–5.

[14] Parissis H, Leotsinidis M, Akbar MT, Apostolakis E, Dougenis D. The need for intra aortic balloon pump support following open heart surgery: risk analysis and outcome. J Cardiothorac Surg. 2010 Apr 5;5:20.

[15] Powell WJ, Daggett WM, Magro AE, Bianco JA, Buckley MJ, Sanders CA, et al. Effects of intra-aortic balloon counterpulsation on cardiac performance, oxygen consumption, and coronary blood flow in dogs. Circ Res. 1970 Jun;26(6):753–64.

[16] Katz ES, Tunick PA, Kronzon I. Observations of coronary flow augmentation and balloon function during intraaortic balloon counterpulsation using transesophageal echocardiography. Am J Cardiol. 1992 Jun 15;69(19):1635–9.

[17] Dunkman WB, Leinbach RC, Buckley MJ, Mundth ED, Kantrowitz AR, Austen WG, et al. Clinical and hemodynamic results of intraaortic balloon pumping and surgery for cardiogenic shock. Circulation. 1972 Sep;46(3):465–77.

[18] Papaioannou TG, Stefanadis C. Basic principles of the intraaortic balloon pump and mechanisms affecting its performance. ASAIO J. 2005 May;51(3):296–300.

[19] Papaioannou TG, Terrovitis J, Kanakakis J, Stamatelopoulos KS, Protogerou AD, Lekakis JP, et al. Heart rate effect on hemodynamics during mechanical assistance by the intra-aortic balloon pump. Int J Artif Organs. 2002 Dec;25(12):1160–5.

[20] Weber KT, Janicki JS, Walker AA. Intra-aortic balloon pumping: an analysis of several variables affecting balloon performance. Trans Am Soc Artif Intern Organs. 1972;18(0):486–92.

[21] Sarıkaya S, Adademir T, Özen Y, Aydın E, Başaran EK, Şahin M, et al. Alternative non-femoral accesses for intra-aortic balloon pumping. Perfusion. 2015 Nov 1;30(8):629–35.

[22] Ferguson III JJ, Cohen M, Freedman Jr RJ, Stone GW, Miller MF, Joseph DL, et al. The current practice of intra-aortic balloon counterpulsation: results from the Benchmark Registry. J Am Coll Cardiol. 2001 Nov 1;38(5):1456–62.

[23] White JM, Ruygrok PN. Intra-aortic balloon counterpulsation in contemporary practice —where are we? Heart Lung Circ. 2015 Apr;24(4):335–41.

[24] Thiele H, Zeymer U, Neumann F-J, Ferenc M, Olbrich H-G, Hausleiter J, et al. Intra-aortic balloon counterpulsation in acute myocardial infarction complicated by cardiogenic shock (IABP-SHOCK II): final 12 month results of a randomised, open-label trial. Lancet Lond Engl. 2013 Nov 16;382(9905):1638–45.

[25] Manohar VA, Levin RN, Karadolian SS, Usmani A, Timmis RM, Dery ME, et al. The impact of intra-aortic balloon pump weaning protocols on in-hospital clinical outcomes. J Intervent Cardiol. 2012 Apr;25(2):140–6.

[26] Onorati F, Santini F, Amoncelli E, Campanella F, Chiominto B, Faggian G, et al. How should I wean my next intra-aortic balloon pump? Differences between progressive volume weaning and rate weaning. J Thorac Cardiovasc Surg. 2013 May;145(5):1214–21.

[27] Baffes TG, Fridman JL, Bicoff JP, Whitehill JL. Extracorporeal circulation for support of palliative cardiac surgery in infants. Ann Thorac Surg. 1970 Oct;10(4):354–63.

[28] Tramm R, Ilic D, Davies AR, Pellegrino VA, Romero L, Hodgson C. Extracorporeal membrane oxygenation for critically ill adults. Cochrane Database Syst Rev. 2015;1:CD010381.

[29] Chen Y-S, Yu H-Y, Huang S-C, Lin J-W, Chi N-H, Wang C-H, et al. Extracorporeal membrane oxygenation support can extend the duration of cardiopulmonary resuscitation. Crit Care Med. 2008 Sep;36(9):2529–35.

[30] Cardarelli MG, Young AJ, Griffith B. Use of extracorporeal membrane oxygenation for adults in cardiac arrest (E-CPR): a meta-analysis of observational studies. ASAIO J Am Soc Artif Intern Organs 1992. 2009 Dec;55(6):581–6.

[31] Turner DA, Cheifetz IM. Extracorporeal membrane oxygenation for adult respiratory failure. Respir Care. 2013 Jun 1;58(6):1038–52.

[32] Russo CF, Cannata A, Vitali E, Lanfranconi M. Prevention of limb ischemia and edema during peripheral venoarterial extracorporeal membrane oxygenation in adults. J Card Surg. 2009 Apr;24(2):185–7.

[33] Makdisi G, Wang IW. Extra corporeal membrane oxygenation (ECMO) review of a lifesaving technology. J Thorac Dis. 2015 Jul 22;7(7):E166–76.

[34] Indications and Complications for VA-ECMO for Cardiac Failure [Internet]. American College of Cardiology. [cited 2016 Jan 18]. Available from: http://www.acc.org/latest-in-cardiology/articles/2015/07/14/09/27/indications-and-complications-for-va-ecmo-for-cardiac-failure

[35] Schmidt M, Stewart C, Bailey M, Nieszkowska A, Kelly J, Murphy L, et al. Mechanical ventilation management during extracorporeal membrane oxygenation for acute respiratory distress syndrome: a retrospective international multicenter study. Crit Care Med. 2015 Mar;43(3):654–64.

[36] Askenazi DJ, Selewski DT, Paden ML, Cooper DS, Bridges BC, Zappitelli M, et al. Renal replacement therapy in critically ill patients receiving extracorporeal membrane oxygenation. Clin J Am Soc Nephrol. 2012 Aug 1;7(8):1328–36.

[37] Kielstein JT, Heiden AM, Beutel G, Gottlieb J, Wiesner O, Hafer C, et al. Renal function and survival in 200 patients undergoing ECMO therapy. Nephrol Dial Transplant Off Publ Eur Dial Transpl Assoc—Eur Ren Assoc. 2013 Jan;28(1):86–90.

[38] Sen A, Callisen H, Alwardt C, Larson J, Lowell A, Libricz S, et al. Adult venovenous extracorporeal membrane oxygenation for severe respiratory failure: current status and future perspectives. Ann Card Anaesth. 2016;19(1):97.

[39] Ridley EJ, Davies AR, Robins EJ, Lukas G, Bailey MJ, Fraser JF. Nutrition therapy in adult patients receiving extracorporeal membrane oxygenation: a prospective, multi-centre, observational study. Crit Care Resusc J Australas Acad Crit Care Med. 2015 Sep; 17(3):183–9.

[40] Nasr DM, Rabinstein AA. Neurologic complications of extracorporeal membrane oxygenation. J Clin Neurol Seoul Korea. 2015 Oct;11(4):383–9.

[41] ELSO Guidelines [Internet]. [cited 2016 Jan 22]. Available from: http://www.elso.org/ Portals/0/IGD/Archive/FileManager/e76ef78eabcusersshyerdocumentselsoguideli-nesforadultcardiacfailure1.3.pdf

[42] Aubron C, Cheng AC, Pilcher D, Leong T, Magrin G, Cooper DJ, et al. Factors associated with outcomes of patients on extracorporeal membrane oxygenation support: a 5-year cohort study. Crit Care Lond Engl. 2013;17(2):R73.

[43] Jackson KW, Timpa J, McIlwain RB, O'Meara C, Kirklin JK, Borasino S, et al. Side-arm grafts for femoral extracorporeal membrane oxygenation cannulation. Ann Thorac Surg. 2012 Nov;94(5):e111–2.

[44] Slaughter MS, Rogers JG, Milano CA, Russell SD, Conte JV, Feldman D, et al. Advanced heart failure treated with continuous-flow left ventricular assist device. N Engl J Med. 2009 Dec 3;361(23):2241–51.

[45] Atluri P, Ullery BW, MacArthur JW, Goldstone AB, Fairman AS, Hiesinger W, et al. Rapid onset of fulminant myocarditis portends a favourable prognosis and the ability to bridge mechanical circulatory support to recovery. Eur J Cardio-Thorac Surg Off J Eur Assoc Cardio-Thorac Surg. 2013 Feb;43(2):379–82.

[46] Rogers JG, Aaronson KD, Boyle AJ, Russell SD, Milano CA, Pagani FD, et al. Continuous flow left ventricular assist device improves functional capacity and quality of life of advanced heart failure patients. J Am Coll Cardiol. 2010 Apr 27;55(17):1826–34.

[47] Takayama H, Soni L, Kalesan B, Truby LK, Ota T, Cedola S, et al. Bridge-to-decision therapy with a continuous-flow external ventricular assist device in refractory cardiogenic shock of various causes. Circ Heart Fail. 2014 Sep 1;7(5):799–806.

[48] Morgan JA, Stewart AS, Lee BJ, Oz MC, Naka Y. Role of the Abiomed BVS 5000 device for short-term support and bridge to transplantation. ASAIO J Am Soc Artif Intern Organs 1992. 2004 Aug;50(4):360–3.

[49] Gilotra NA, Stevens GR. Temporary mechanical circulatory support: a review of the options, indications, and outcomes. Clin Med Insights Cardiol. 2014;8(Suppl 1):75–85.

[50] Patel NJ, Singh V, Patel SV, Savani C, Patel N, Panaich S, et al. Percutaneous coronary interventions and hemodynamic support in the USA: a 5 year experience. J Intervent Cardiol. 2015 Dec;28(6):563–73.

[51] Slaughter MS, Sobieski MA, Gallagher C, Dia M, Silver MA. Low incidence of neuro‐ logic events during long-term support with the HeartMate® XVE left ventricular assist device. Tex Heart Inst J. 2008;35(3):245–9.

[52] Iacovoni A, Centofanti P, Attisani M, Verde A, Terzi A, Senni M, et al. Low incidence of gastrointestinal bleeding and pump thrombosis in patients receiving the INCOR LVAD system in the long-term follow-up. Int J Artif Organs. 2015 Nov 10;38(10):542– 7.

[53] Hetzer R, Potapov EV, Weng Y, Sinawski H, Knollmann F, Komoda T, et al. Implanta‐ tion of MicroMed DeBakey VAD through left thoracotomy after previous median sternotomy operations. Ann Thorac Surg. 2004 Jan;77(1):347–50.

[54] Haneya A, Philipp A, Puehler T, Ried M, Hilker M, Zink W, et al. Ventricular assist device implantation in patients on percutaneous extracorporeal life support without switching to conventional cardiopulmonary bypass system. Eur J Cardio-Thorac Surg Off J Eur Assoc Cardio-Thorac Surg. 2012 Jun;41(6):1366–70.

[55] Slaughter MS, Naka Y, John R, Boyle A, Conte JV, Russell SD, et al. Post-operative heparin may not be required for transitioning patients with a HeartMate II left ven‐ tricular assist system to long-term warfarin therapy. J Heart Lung Transplant Off Publ Int Soc Heart Transplant. 2010 Jun;29(6):616–24.

[56] Cook JA, Shah KB, Quader MA, Cooke RH, Kasirajan V, Rao KK, et al. The total artificial heart. J Thorac Dis. 2015 Dec;7(12):2172–80.

[57] Copeland JG, Smith RG, Arabia FA, Nolan PE, Sethi GK, Tsau PH, et al. Cardiac replacement with a total artificial heart as a bridge to transplantation. N Engl J Med. 2004 Aug 26;351(9):859–67.

[58] Gerosa G, Scuri S, Iop L, Torregrossa G. Present and future perspectives on total artificial hearts. Ann Cardiothorac Surg. 2014 Nov;3(6):595–602.