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Cardiac failure and left ventricular assist devices

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Congestive heart failure (CHF) is a clinical syndrome defined as chronic, inadequate myocardial contraction and relaxation that results in decreased cardiac output. It has multiple etiologies, including coronary artery disease causing ischemic changes, valvular heart disease, viral cardiomyopathies, and congenital disease. A major cause of morbidity and mortality in the United States, CHF affects more than 4 million people each year and causes approximately 40,000 deaths. The incidence of CHF increases in patients aged 65 years and older, and the 5-year, after-diagnosis mortality rate is approximately 50%. In 2001, the estimated direct and indirect costs of CHF were $21 billion [1].

Primary medical therapy for CHF has always been pharmacologic, and the armamentarium available to clinicians has grown during the past 40 years—from digitalis and mercurial diuretics to the current, more potent diuretics, beta blockers, angiotensin converting enzyme (ACE) inhibitors, and aldosterone receptor blockers, among others [2]. For some patients, inotropic therapy is administered on a long-term basis. In addition to primary therapy for coronary or valvular disease, other therapeutic options are available, including cardiac resynchronization therapy by way of biventricular pacing [3].

Since 1967 when Barnard [4] performed the first successful human heart transplant, the ultimate surgical therapy for cardiac failure has been cardiac transplantation. Unfortunately, the number of patients in the United States who potentially could benefit from cardiac transplantation is estimated to be anywhere from 4,000–40,000 (or more) in 2001, whereas the number of donors has remained fixed at 2,000–2,500 per year [1]. As a result, a significant number of patients die while awaiting cardiac transplantation. This disparity between availability and demand—and the subsequent mortality risk—has made mechanical support of the failing circulation a viable option.

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Historical evolution and indications

The first left ventricular assist devices (LVADs) were proposed for use as temporary cardiac support after cardiac surgery. In 1966, DeBakey and colleagues implanted a blood pump in a patient who had undergone double valve replacement, making this the first successful use of an LVAD for postcardiotomy heart failure [5].

In 1964, the Artificial Heart Program of the National Heart Institute (now called the National Heart, Lung, and Blood Institute) initiated a program to support the development of devices for use as bridges to transplantation. The first contract for LVAD development was granted to the Texas Heart Institute in 1975 [6], and significant further progress has been made at several institutions. Currently there are at least nine devices suitable for short-, medium-, or long-term support (Table 1).

Current indications for LVAD insertion include reversible ventricular dysfunction occurring after cardiac surgery, temporary support for patients awaiting heart transplantation, and destination therapy for patients who are not candidates

Table 1
Cardiac support devices

Device	Type	Position	Drive mechanism
IABP (various manufacturers)	Counterpulsation	Intraaortic	Pneumatic (helium)
Centrifugal pump (various manufacturers)	Kinetic displacement; nonpulsatile	Extracorporeal	Electric
HeartMate IP (Thoratec)	Implantable; pulsatile	Abdominal (pre- or intraperitoneal)	Pneumatic (air)
HeartMate VE (Thoratec)	Implantable; pulsatile	Abdominal (pre- or intraperitoneal)	Electric (pneumatic in emergencies)
Thoratec VAD (Thoratec)	"Paracorporeal"; pulsatile	Exterior to abdominal wall	Pneumatic (air)
Novacor LVAS (World Heart Corp.)	Implantable; pulsatile	Abdominal (pre- or intraperitoneal)	Electric
ABIOMED BVS 5000 (ABIOMED, Inc.)	Extracorporeal; pulsatile	Extracorporeal	Pneumatic (air)
Jarvik 2000 (Jarvik Heart, Inc.)	Intracorporeal; axial flow; nonpulsatile	Implantable intracardiac pump; outlet graft to descending or ascending aorta	Electric
DeBakey VAD (MicroMed, Inc.)	Intracorporeal; axial flow; nonpulsatile	Implantable intracardiac pump; outlet graft to ascending aorta	Electric

IABP, intraaortic balloon pump; IP, implantable pneumatic; LVAS, left ventricular assist system; VAD, ventricular assist device; VE, vented electric.

Table 2
Contraindications for left ventricular assist device use

Irreversible chronic renal failure
Severe pulmonary disease unrelated to cardiac function
Irreversible liver failure
Metastatic cancer
Sepsis
Irreversible neurologic deficits

for heart transplantation. The intraaortic balloon pump (IABP) is used commonly for patients with postcardiotomy heart failure, but longer-term devices are needed in a small number of cases. For patients temporarily supported by LVADs, myocardial function sometimes improves sufficiently to allow removal of the LVAD, making transplantation unnecessary. The Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure (REMATCH) trial showed that when compared with medical therapy, LVADs could double survival at 1 year and triple survival at 2 years, which supports the use of LVADs as destination therapy in patients who are not candidates for cardiac transplantation [7]. Unfortunately, not all patients with severe CHF are suitable candidates for LVAD insertion (Table 2).

Types of left ventricular assist devices

Counterpulsation

The IABP (various manufacturers) is a mechanical circulatory support device widely used throughout the world for supporting patients after cardiac surgery, angioplasty, and myocardial infarction or with various low-output syndromes. It increases perfusion to the coronary arteries with diastolic inflation and decreases afterload and, thus, left ventricular work and oxygen consumption with systolic deflation, but it cannot provide complete mechanical support. The IABP is implanted retrograde by way of the femoral artery or occasionally antegrade by way of the ascending aorta in patients who have undergone cardiac surgery and have occluded femoral arteries. A complete discussion of this device is beyond the scope of this article.

Extracorporeal kinetic displacement

The centrifugal pump (various manufacturers) is used most commonly as part of a standard extracorporeal circuit for cardiopulmonary bypass (CPB), but it also is used for temporary left, right, or biventricular support in patients with postcardiotomy heart failure. A series of rotating vanes within the device's body move blood forward, producing nonpulsatile flows of up to 6 L/min. The centrifugal pump may be used as an LVAD without the oxygenator of the extracorporeal circuit in postcardiotomy patients who cannot be weaned from CPB or with an oxygenator for extracorporeal membrane oxygenation. Heparin is required during use. The device is afterload-dependent, with increasing peripheral resistance lowering output. Its main advantages are availability, familiarity, and relative simplicity. The patient's sternum cannot be closed while the device is in place. The device usually must be removed after a few days. A complete review of this device is also beyond the scope of this article.

Pulsatile pumps

Pulsatile pumps represent, in general, the second generation of LVADs. Most were developed in the 1980s and have been used since. They all provide pulsatile flow and are capable of generating normal cardiac output (6–10 L/min), given adequate venous return. The ultimate goal of their use is to restore normal perfusion, especially to end organs compromised by left ventricular failure.

Pulsatile pumps may be divided into two categories: extracorporeal or implantable. Extracorporeal pulsatile pumps provide short- to medium-term right, left, or biventricular support for patients with postcardiotomy heart failure, for patients with an implantable LVAD and compromised right ventricular function after LVAD placement, or as a bridge to transplant or recovery. Extracorporeal pulsatile pumps require long-term anticoagulation. They are insensitive to electromagnetic interference, which is important during the later stages of implantation

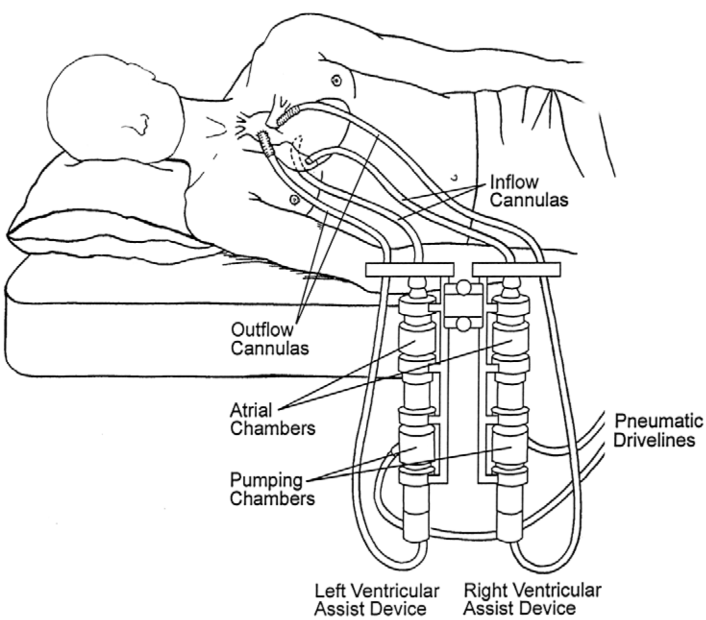

Fig. 1. The ABIOMED BVS 5000 (ABIOMED; Danvers, MA). A disposable, two-chamber, polycarbonate air-driven pump with polyurethane valves (the illustration shows biventricular support). With normovolemia, a stroke volume of 80 ml and a flow rate of 6 L/min are possible.

when the patient has to return to the operating room for other reasons [8]. The ABIOMED BVS 5000 (ABIOMED, Inc.; Danvers, Massachusetts) (Fig. 1) and the Thoratec ventricular assist device (Thoratec Corporation; Pleasanton, CA) (Fig. 2) are examples of extracorporeal pulsatile pumps.

The implantable pulsatile LVADs, including the Novacor (World Heart Corporation; Ottawa, Ontario, Canada) (Fig. 3) and the pneumatic (IP) and vented-electric (VE) HeartMates (Thoratec Corp.; Pleasanton, California) (Fig. 4), are placed in the abdomen, either preperitoneally or occasionally within the peritoneum. They are intended for left ventricular support only and are used as a bridge to transplantation or to recovery. On the basis of data from the REMATCH trial, the HeartMate VE also has been approved for use as destination therapy. Because these pumps require percutaneous drive cables that must exit the skin, there is risk for infection. Implantable pulsatile pumps require minimal anticoagulation but are susceptible to electromagnetic interference [8].

Nonpulsatile pumps

The newest types of LVADs are electrically powered devices that produce axial, nonpulsatile blood flow. With variable pump speeds, the Jarvik 2000 (Jarvik Heart

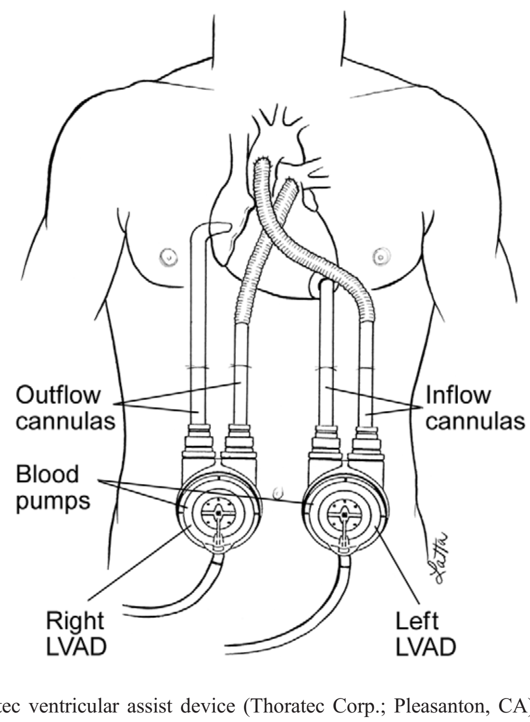

Fig. 2. The Thoratec ventricular assist device (Thoratec Corp.; Pleasanton, CA). A single-chamber pump with mechanical inflow and outflow valves. Alternating positive and negative air pressure within a flexible sac moves blood through the pump. A flow rate of 7 L/min is possible.

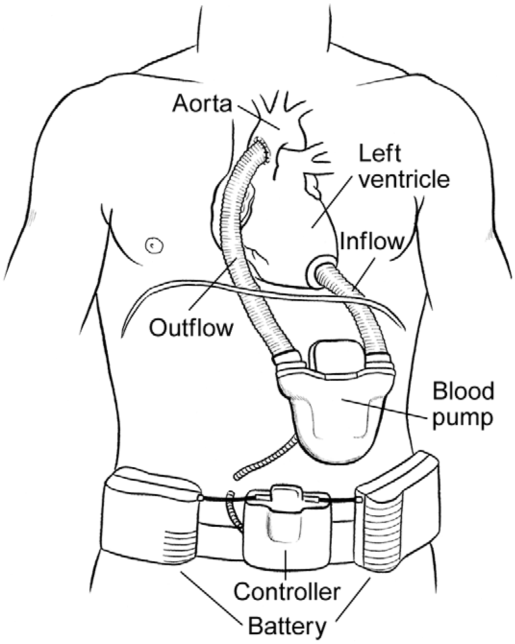

Fig. 3. The Novacor LVAS (World Heart Corporation; Ottawa, ON, Canada). An electrically driven, pusher-plate actuated pump with porcine-valved conduits for inflow (from the left ventricle) and outflow (to the ascending aorta).

Inc.; New York, NY) (Fig. 5) and the DeBakey VAD (MicroMed Inc.; Woodlands, Texas) can produce high flows with essentially all cardiac output moving through the pump or can operate at reduced speeds to serve as a ''booster'' to cardiac output, with some blood flow through the pump and some through the patient's own aortic valve [9,10]. The Jarvik 2000 is an intraventricular device, whereas the DeBakey VAD is an extracardiac device. The Jarvik 2000 is implanted through a left thoracotomy, with the outflow graft from the pump anastomosed to the descending aorta; alternatively, the outflow graft may be anastomosed to the ascending aorta through a sternotomy. The DeBakey VAD requires a sternotomy for its left-ventricle-to-ascending-aorta course. Presently these pumps are approved in the United States as bridges to transplant. In Europe they are used for permanent support.

LVAD physiology and preoperative considerations

Chronic heart failure may result from congenital heart disease, ischemic heart disease, idiopathic disease, infection, or other causes—all of which usually result in cardiomyopathy. Until recently, the focus has been mainly on systolic pathology, but systolic and diastolic functions usually are affected in patients

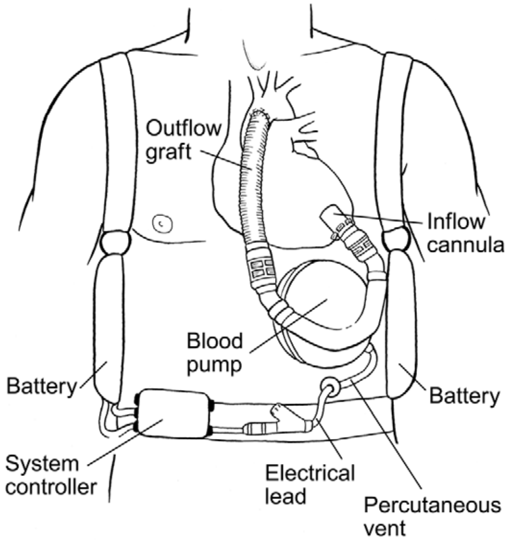

Fig. 4. The HeartMate vented electric (VE) or implantable pneumatic (IP) left ventricular assist systems (Thoratec Corp.; Pleasanton, CA). An electrically (illustrated) or pneumatically driven pusher-plate pump with porcine-valved inflow and outflow conduits. Blood flows from the left ventricle to the ascending aorta. Blood contacting surfaces are nonthrombogenic and minimal anticoagulation is necessary.

with CHF, although diastolic dysfunction may exist alone. The resulting lowered cardiac output commonly produces ejection fractions less than 20%, with little cardiac reserve or ability to increase output through usual physiologic means. CHF patients have low stroke volumes that make them heart rate-dependent to maintain cardiac output. Although they depend on adequate preload, increasing preload does not significantly increase cardiac output, because their myocardium cannot respond by the standard Starling mechanism. Conversely, decreasing heart rate and increasing diastolic filling does not increase stroke volume either, because of lack of preload response. Increasing heart rate and decreasing diastolic time causes a decrease in output, as does increases in afterload [11]. Autonomic tone is increased, with resulting vasoconstriction and eventual down-regulation of catecholamine receptors and myocardial norepinephrine [12].

Previous cardiovascular surgeries may result in problems at the time of LVAD insertion. Patients who have had a prior sternotomy have significant pericardial adhesions that can complicate opening the sternum and cardiac dissection. The possibility of catastrophic hemorrhage is always present and should be anticipated.

A reduction in cardiac output produces effects on the function of other major organ systems, especially hepatic and renal metabolism, which results in alteration in the pharmacokinetics of therapeutic drugs because of decreased volume

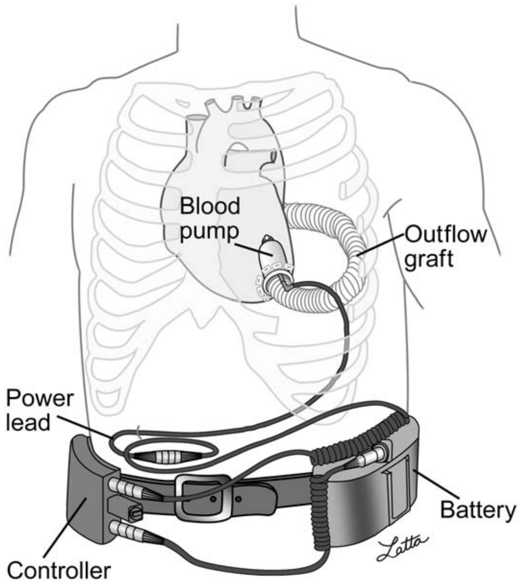

Fig. 5. The Jarvik 2000 (Jarvik Heart Inc.; New York, NY). An implantable, valveless, intraventricular pump that produces axial flow by means of a single vaned impeller (8,000–12,000 rpm). A flow rate of 7 L/min is possible.

distribution, altered protein binding, slowed metabolism, and decreased clearance and systemic elimination. Decreased clearance and elimination of various endogenous vasoactive substances, such as cytokines, also may affect adversely the patient's condition and render pharmacologic, therapeutic interventions less effective. Affected synthetic functions, with resultant alterations in the coagulation system, often produce significant preoperative coagulopathy.

At this stage, pharmacologic therapy for CHF includes several agents that in themselves may affect later management. Amiodarone, a common antidysrhythmic, may produce alpha and beta blockade and contribute to difficult to manage vasodilation during surgery (though this phenomenon has not been observed consistently) [13]. ACE inhibitors, which may produce partial blockade of the renin-angiotensin system and catecholamines, also may contribute to vasodilation. End-stage CHF patients often receive intravenous inotropic support in the form of milrinone, dobutamine, or dopamine, and infusion of these agents should be continued during the prebypass period. The perioperative risk for hemorrhage is increased if patients are on anticoagulant therapy (such as warfarin or clopidogrel) for chronic atrial fibrillation, generally sluggish blood flow, or other reasons [14].

By the time of device implantation, CHF can produce enough respiratory compromise that patients require ventilatory support. Further cardiac decompensation also may require preoperative insertion of an IABP.

Surgical approach

With the exception of the IABP, a median sternotomy is required for most LVAD insertions. As noted previously, the Jarvik 2000 usually is inserted through a left thoracotomy (an approach that requires lung isolation), but it can be inserted through a sternotomy if necessary. In general, all LVADs (except the IABP) require the aid of CPB for insertion, permitting placement of the inlet cannulas from the heart to the device and of the outlet cannulas to the aorta. In selected patients, however, it has been possible to insert the Jarvik 2000 without the aid of CPB.

Aside from the basic pathology affecting cardiac function, patients may have specific cardiac lesions that affect device performance. A patent foramen ovale may lead to right-to-left shunting after LVAD insertion when there are low left-sided and relatively high right-sided heart pressures, subsequently leading to systemic desaturation. Mitral stenosis, which retards flow into the LVAD, needs to be addressed intraoperatively. Aortic regurgitation causes retrograde flow from the aorta to the left ventricle across the incompetent valve, causing a net decrease in total device output, as blood is shunted continually through the device. Occasionally, coronary artery bypass may be necessary in cases of isolated coronary artery disease, especially of the right coronary system, to aid right ventricular function.

Preoperative evaluation

In addition to the standard laboratory studies and radiographic examinations, coagulation evaluation, cardiac catheterization, and echocardiographic examinations are useful (Table 3), especially for focusing on factors that affect right ventricular function (eg, response to pulmonary vasodilators).

Anesthetic management

Monitoring

The approach for monitoring patients during LVAD insertion is initially the same as that for any cardiac surgical procedure involving CPB. Additionally,

Table 3
Preoperative tests and examinations

Laboratory	Hb, Hct, electrolytes, BUN, creatinine, liver function
Coagulation	PT, PTT, fibrinogen, platelet count, platelet function
Echocardiography and cardiac catheterization	LV and RV filling pressures, PAP, PVR, transpulmonary gradient, response to pulmonary vasodilators

Abbreviations: BUN, blood urea nitrogen; Hb, hemoglobin; Hct, hematocrit; LV, left ventricular; PAP, pulmonary artery pressure; PT, prothrombin time; PTT, partial thromboplastin time; PVR, pulmonary vascular resistance; RV, right ventricular.

electroencephalographic monitoring is used for assessing adequate cerebral blood flow during CPB support. Pulmonary artery pressure (PAP) monitoring is not always mandatory but is often useful in pharmacologic manipulation of PAPs, especially early in the post-CPB period. Transesophageal echocardiography (TEE) is necessary in all patients to ascertain if a patent foramen ovale is present and to assess LVAD inflow, valve function before and after device insertion, left ventricular filling, right ventricular function, and completeness of air evacuation.

Induction and prebypass period

Many end-stage CHF patients are fragile, and any decrease in their high resting sympathetic tone produces hemodynamic deterioration. Conversely, some patients may be so intravascularly volume-overloaded that they can tolerate any induction agent and its resulting vasodilation. Midazolam, etomidate, or even thiopental may be used for induction alone, or in combination, with doses titrated to clinical effect. Fentanyl is added for additional analgesia, with total doses ranging from 15–20 mcg/kg. Muscle relaxation may be achieved with whatever agent indicated. Occasionally, rapid sequence induction may be required. The anesthesiologist should be mindful of slowed circulation time, which affects satisfactory drug effect during induction. Low-dose isoflurane (0.4%–1%) is usually well tolerated as a maintenance agent. If hemodynamic deterioration occurs, continued vasopressor support may be required until CPB can be initiated.

Prebypass considerations

After induction, additional monitors (eg, PA catheters) are inserted and TEE is performed. Large-volume infusion catheters also are placed. The authors cannot overemphasize the importance of adequate sites for volume administration, because significant hemorrhage may be encountered anytime after the start of surgery; the sites are particularly important in the postbypass period and in the intensive care unit.

Because coagulation abnormalities are an almost invariant part of the clinical course of these patients, the risk for postoperative hemorrhage is always high. Antifibrinolytics such as aprotinin or aminocaproic acid are a useful prophylactic adjunct. For the authors' practice, the lower dose regimen of aprotinin with a subsequent weight-based continuous infusion has proved beneficial. Adequate blood bank resources must be available for red blood cells and coagulation products, and there must be the capacity for the rapid return of coagulation tests.

Cardiopulmonary bypass

A standard crystalloid prime for the CPB circuit may be appropriate for some patients, but many, especially those with preexisting coagulation deficits or evidence of significant hepatic congestion, may benefit from use of a prime consisting mainly of fresh frozen plasma, which attenuates dilutional coagulopathy and is partially therapeutic if prothrombin time (PT) is elevated. The activated coagulation time (ACT) on bypass may be prolonged because of preexisting coagulopathy, but if aprotinin is used, the authors have elected to maintain the ACT at greater than 500 sec. The authors have found the HepCon Device (Medtronic; Minneapolis, MN) a useful adjunct to this end.

With a competent aortic valve, it is often unnecessary to cross clamp the aorta for device insertion, but this may be necessary if the patient is undergoing additional procedures. Bypass flows are maintained in the standard fashion (50 ± 10 ml/kg/min).

Weaning from bypass

LVADs must have adequate preload to function properly. The major determinant of adequate preload is the right ventricle's ability to pump volume across the pulmonary circuit, or at least to have a pulmonary circuit that does not present an impediment to blood flow (similar to Fontan physiology in congenital heart disease). To that end, successful weaning from bypass usually depends on adequate right ventricular function.

It may not be possible to determine preoperatively how well the right ventricle is functioning, because it may not be possible to gauge adequately the left ventricular contribution to the right ventricular output. Decompression of the left ventricle may cause the ventricular septum to shift to the left, decreasing right ventricular function. Because of the LVAD, improved left ventricular flow subsequently improves flow across the pulmonary circuit, but a high pulmonary vascular resistance combined with right ventricular dysfunction may still result in inadequate flows. Inotropic therapy aimed at supporting the right ventricle and decreasing pulmonary vascular resistance is appropriate. Milrinone, dobutamine, and isoproterenol may be administered alone or in combination in standard doses. In cases of frank right ventricular failure unresponsive to inotropes, a right ventricular assist device (RVAD) may be used temporarily; however, the need for temporary RVAD support is associated with significantly higher mortality [15].

Before weaning from CPB, air must be evacuated from the left ventricle and from the LVAD itself by filling the ventricle and the device with blood from the bypass circuit and venting the air from the outlet cannula. When most of the air is evacuated (it is almost impossible to evacuate all air), venous outflow to the CPB circuit is occluded gradually, allowing a further increase in preload. The LVAD then is started and the CPB flow gradually decreased until the LVAD has assumed adequate output (ideally 4–6 L/min for implantable pulsatile pumps such as the HeartMate).

LVAD flow is volume-dependent, so decreased flows usually represent a volume deficit, though right ventricular dysfunction also may be a cause. With pulsatile pumps, flows less than 3 L/min are a significant problem because of the risk for an occluded inflow cannula or for thrombosis. Preload must be maintained during the weaning process because low preload and resulting low flows can cause significant negative pressures to develop within the heart and pump system, which may result in air being pulled through the fresh suture lines or graft interstices, causing a systemic air embolus.

Initially during weaning, pulsatile LVADs are operated in a fixed-rate mode. After patients are weaned from CPB, the pumps are switched to an automatic fill-to-empty mode, in which the pump empties itself when its controller senses the pumping chamber is full. Cardiac synchrony is not used.

Despite adequate volume loading and LVAD output, systemic perfusion pressure may be inadequate in the early stages after pump insertion, which may be caused by vasodilatory shock from causes related to the patient's decompensated state, to altered drug metabolism of the ACE inhibitors or amiodarone, to side effects of milrinone or dobutamine, or to arginine vasopressin deficiency [16]. Vasopressin (1–6 U/h), norepinephrine (2–20 μg/min), or epinephrine (2–20 μg/min) may be titrated to improve perfusion pressure.

At this stage, TEE is especially important for assessing right ventricular function, possible right-to-left atrial shunting, the appearance of aortic or tricuspid valve incompetence, decompression of the left ventricle, inflow to the LVAD, and completeness of air removal.

If pulmonary artery pressures and vascular resistance remain high after the patient is weaned from CPB, pulmonary vasodilators, such as prostaglandin E_1, nitric oxide, or nesiritide may be useful. Adequate ventilation with good lung expansion and maintenance of P_ACO_2 in the low-normal range (to help reduce pulmonary vascular resistance) are always important. If dysrhythmia occurs, it generally does not interfere with LVAD function unless the pulmonary vascular resistance is high and right ventricular function thus is affected.

Postbypass period

In the postbypass period, reversal of anticoagulation and achievement of adequate hemostasis are a major focus. Surgical hemostasis may require extraordinary effort because of previous cardiovascular surgery, numerous suture lines, and preexisting and CPB-associated clotting defects. Volume requirements of blood and blood products can be high, and use of devices for rapid infusion of component therapy is needed. Because it may take 24–48 hours for patients to stabilize, primary chest closure may not be possible until cardiac function has stabilized and the risk for hemorrhage is controlled. Once sternal closure is possible, patients can be weaned from the ventilator. For successful extubation, it is often necessary to promote diuresis to remove residual pulmonary interstitial volume overload. The authors have found diuretics and nesiritide useful for this purpose, as is fenoldopam for increasing renal blood flow.

Postoperative period

LVAD support usually improves cardiac and multiorgan functions and may reverse abnormal processes of myocardial cellular structure. Neuroendocrine and inflammatory mediators also are normalized. Most patients who are stable after the immediate postoperative period show significant clinical improvement. Longer term, it is possible for patients with the VE HeartMate or the axial-flow pump systems to leave the hospital and continue therapy on an outpatient basis. Some patients have even been able to return to work.

Summary

LVADs represent advanced therapy for cardiac failure. The anesthesiologist's contribution to the pre-, intra-, and postoperative management of these challenging patients continues to grow as LVAD technology is refined and as more patients become eligible to receive these life-saving devices.

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