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Perioperative Considerations in the Patient with a Left Ventricular Assist Device

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MECHANICAL support of the cardiovascular system is an important therapeutic modality for a growing number of patients with congestive heart failure. Certain patients with refractory end-stage failure who will likely succumb to their disease before a potential heart transplant may be effectively "bridged to transplant" by a left ventricular assist device (LVAD). Three such devices are currently approved by the US Food and Drug Administration for this indication. Several ongoing multicenter clinical trials are also evaluating LVAD therapy as an alternative to transplantation ("destination therapy"). Preliminary data from the Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure Trial indicate that an implantable LVAD prolongs survival and enhances quality of life in patients with end-stage heart failure.[1] The efficacy of destination therapy will undoubtedly lead to an expansion in the number of LVAD-supported patients in future years. Some of these patients will require operations for noncardiac, non-LVAD problems. Anesthesiologists should therefore be familiar with the unique considerations related to these patients and their devices.

Accordingly, this review presents important features of the commonly used devices, describes the effects of LVAD therapy on the pathophysiology of heart failure, and discusses the major perioperative considerations for patients who come to the operating room with an implanted LVAD. The discussion focuses on the patient with a chronically implanted device, not on the implantation procedure itself, which involves a different set of physiologic challenges and anesthetic considerations.

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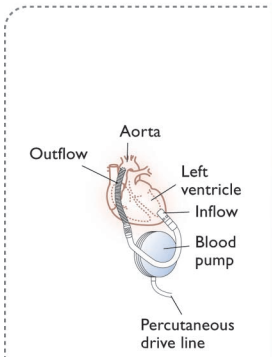
On November 6, 2002, the United States Food and Drug Administration approved the vented electric model of the HeartMate® LVAD (Thoratec Corporation., Pleasanton, CA) for 'destination therapy' in patients with end-stage heart failure who are not candidates for heart transplantation and who have a projected life expectancy of less than two years.

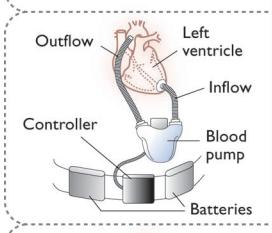
Device Features

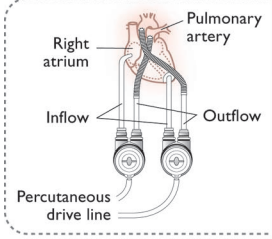
The three devices[2-5] currently approved by the US Food and Drug Administration for use as a "bridge to transplant" are the HeartMate® LVAD (Thoratec Corporation, Pleasanton, CA), the Novacor® LVAD (World Heart Corporation, Ottawa, Canada), and the Thoratec® assist device (Thoratec Corporation) (fig. 1). The therapeutic goal of all three devices is to assume the pump function of the failing left ventricle. To accomplish this objective, each device is designed to drain the left ventricular blood volume into a mechanical pump, which ejects the blood into the circulation *via* a conduit that connects to the aorta. The Thoratec® device is unique in that it may also be used to support the right ventricle (RV) with a second pump that is interposed between the RV and pulmonary artery.

Each device maintains unidirectional flow using valves in the inflow and outflow conduits that connect LV to pump and pump to aorta, respectively. Both the HeartMate® and Novacor® devices use porcine valves. The blood-contacting surfaces of the HeartMate® pump chamber and its conduits are textured to promote the formation of a pseudointimal lining that markedly reduces the thrombogenicity of the device. As a result of this surface and the use of tissue valves, most HeartMate® patients are treated with chronic aspirin therapy alone; the vast majority do not require chronic anticoagulation therapy unless another clinical indication exists. The pump chambers of the Novacor® and Thoratec® devices contain smooth blood sacs that do not form a pseudointima. In addition, the Thoratec® device uses mechanical valves to maintain unidirectional flow. As a result, patients with either Novacor® or Thoratec® devices must be chronically anticoagulated with warfarin.

Both the HeartMate® and Novacor® pumps are fully implanted in the body. The pumps are placed in the left upper quadrant of the abdomen using an intraperitoneal or preperitoneal technique. With each of these devices, only the driveline, which connects the pump to the control unit, traverses the skin. The Thoratec®, on the other hand, is a "paracorporeal" device, in that the inflow and outflow conduits traverse the skin to connect with the pump, which lies externally on the skin of the upper abdomen. Any product containing acetone (*e.g.*, tape remover or nail polish remover) must be strictly







	Pump Location	Actuation Mode	Need for Anti-coagulation	Potential for Electromagnetic Interference	Preload / Afterload Sensitive
Heartmate IP®	Implanted (intra- or pre-peritoneal space in LUQ of abdomen	Pneumatic	Usually No	Yes	Yes / Yes
Heartmate VE®	Implanted (intra- or pre-peritoneal space in LUQ of abdomen	Electric, but can be converted to Pneumatic	Usually No	Yes	Yes / Yes
Novacor®	Implanted (intra- or pre peritoneal space in LUQ of abdomen	Electric Only (cannot be operated pneumatically)	Yes	Minimal / No	Yes / Yes
Thoratec®	Paracorporeal (lies externally on the upper abdomen)	Pneumatic	Yes	Minimal / No	Yes / Yes

Fig. 1. Effects of chronic circulatory support with a left ventricular assist device (LVAD) on the pathophysiology of end-stage heart failure. Chronic LVAD therapy leads to reversal of both myocardial and systemic changes that are associated with heart failure.

avoided in patients with a Thoratec® LVAD because the exposed polycarbonate housing may crack in the presence of liquid or vapor acetone.

All three devices provide pulsatile flow. Intermittently, the blood chamber in the LVAD pump is rapidly compressed, leading to ejection of a "stroke volume" that is determined both by the preejection chamber volume and the outflow resistance. The HeartMate® delivers a maximum stroke volume of 85 ml, the Novacor® delivers a maximum stroke volume of 70 ml, and the Thoratec® delivers a maximum stroke volume of 65 ml. The heart continues to beat in its own rhythm. While the left ventricle may at times contract simultaneous with pump ejection, it generally remains volume unloaded throughout the cardiac cycle and thus usually has negligible contribution to aortic flow in the absence of LVAD problems. The RV continues to function as the pump for the pulmonary circulation, except in cases where the RV is supported by a second Thoratec® pump.

All three pumps normally function in a "fill-to-empty" (or "automatic") mode, in which the blood volume within the pump chamber is automatically emptied into the circulation each time the control unit senses that the chamber is nearly full. Total LVAD flow in this mode depends on the rate of the filling–emptying cycle, assuming that pump emptying is complete in each cycle. The cycling time in this mode fluctuates with changes in pulmonary venous return, which in turn is determined both by the patient's intravascular volume state and the capabilities of the RV. When pumping in the "automatic" mode, both the HeartMate® and Novacor® devices are capable of generating flows in excess of 10 l/min., while the Thoratec® can generate flows of approximately 7 l/min. Each pump may also be programmed to operate in a fixed-rate mode, such that the filling–emptying cycle remains constant. Depending on the set rate, flow in this mode may be limited by underfilling of the pump in each cycle. In either pumping mode, automatic or fixed-rate, LVAD pumping is not synchronized with the rhythm of the heart.

With all three devices, the pump is connected to a control unit by a driveline. The Novacor® control unit is

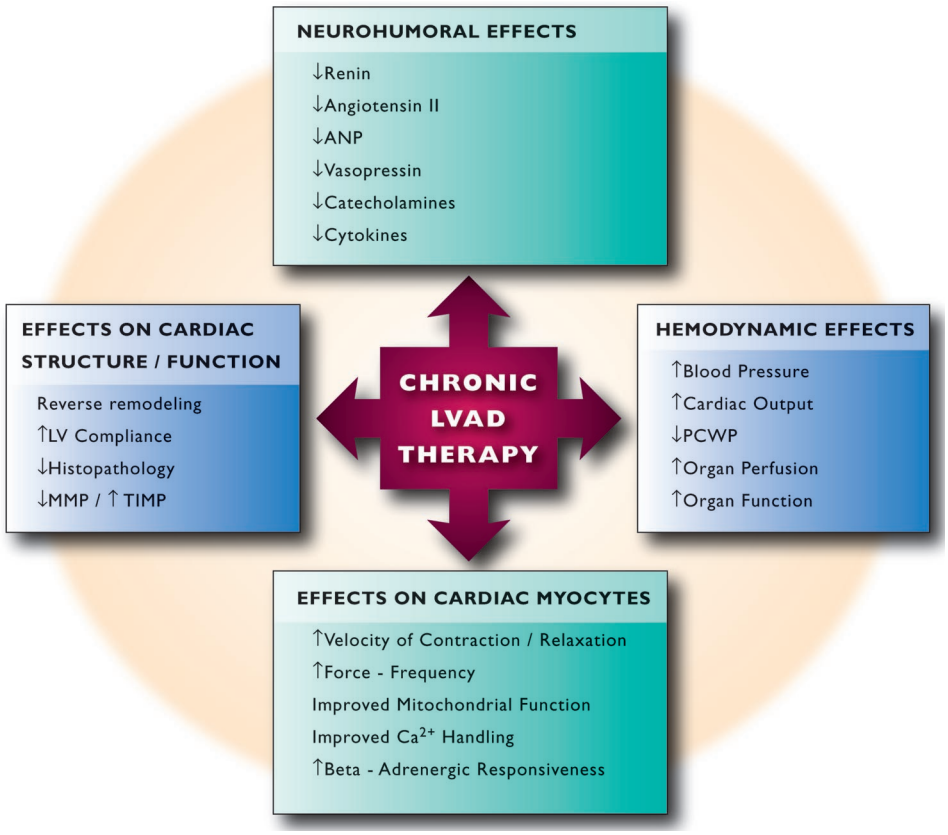


Fig. 2. Important features of the Heartmate®, Novacor®, and Thoratec® left ventricular assist devices. The anesthesiologist must consider the potential impact of these features when caring for a patient with one of these devices in place for the treatment of end-stage heart failure.

portable and may be worn on a belt or vest. The pump is powered by rechargeable, wearable batteries. The Thoratec® controller–driver is portable, although not wearable. The pump is powered pneumatically; blood is ejected when the blood sac inside the rigid pump housing is compressed by pressurized air supplied by the driver. The HeartMate® is available in two models, one powered pneumatically (IP model) and the other electrically (VE model). The IP model functions much the same as the Thoratec®, *via* a portable, although not wearable controller–driver. The VE model pump is controlled by a wearable processor and is powered by rechargeable batteries, similar to the Novacor®. The Thoratec® and both HeartMate® models may be operated manually, using a hand-held, pneumatic pump if the control unit or power source fails. The Novacor® has no mechanism for manual actuation, and a back-up controller must therefore be available in case the unit fails.

Effects of Chronic Left Ventricular Assist Device Therapy on the Pathophysiology of Heart Failure

Chronic implantation of a LVAD has a profound impact on the pathophysiology of end-stage heart failure (fig. 2). In assuming the function of the failing left ventricle, the LVAD restores blood pressure and cardiac output to near normal values, and it improves function of other organ systems, particularly the liver and kidneys.[2,3] Relief from LV pressure–volume overload also leads to reversal of many of the structural and functional abnormalities of the failing heart itself. Left ventricular dilatation in particular is reduced ("reverse remodeling"),[6,7] and the improvement in LV geometry is associated with both increased chamber compliance[6] and reduced myocardial damage.[7]

Chronic hemodynamic unloading is associated with recovery of cellular function as well. Cardiomyocytes obtained from heart failure patients who were treated with chronic LVAD therapy exhibit accelerated contraction and relaxation, improved force–frequency relations, more normal calcium (Ca^{2+}) handling and enhanced β-adrenergic responsiveness.[8] LVAD therapy also restores mitochondrial function in cardiac myocytes from patients with heart failure.[9] Improvements in the force–frequency relation are accompanied by increases in the expression of genes encoding for proteins involved in Ca^{2+} cycling, including the sarcoplasmic reticular Ca^{2+}-ATPase subtype 2a, the ryanodine receptor, and the Na^{+}–Ca^{2+} exchanger.[10] In addition to these changes in the contractile machinery of the heart, chronic LVAD therapy reverses heart failure–induced changes in the

supporting cardiac matrix, as evidenced by reduced concentrations of matrix metalloproteinases and increased tissue inhibitors of matrix metalloproteinases.[11] These data demonstrate that LVAD therapy improves a wide variety of intrinsic myocardial properties that are known to be adversely affected during the progression to end-stage failure.

An interesting feature of LVAD therapy is that only about 10% of patients require any form of mechanical RV support, and the majority of those patients only require it during the early postimplant period.[12–14] The ability of a LVAD to maintain cardiovascular performance in patients with biventricular failure is primarily caused by the beneficial effects of LV unloading on RV performance through both series ventricular interactions and favorable alterations in intraventricular septal function.[15–18] It is important to note, however, that despite improvements in RV mechanical function produced by chronic LV unloading, underlying abnormalities in myocardial architecture, contractility, metabolism, and gene expression are not reversed to the same extent in the RV as in the LV.[18]

In addition to direct cardiac and hemodynamic effects, chronic LVAD therapy alters the systemic humoral responses to heart failure. Marked reductions in the plasma concentrations of renin, angiotensin II, epinephrine, norepinephrine, atrial natriuretic peptide, and arginine vasopressin concentrations have been observed as hemodynamics improve.[19] Thus, chronic mechanical unloading of the LV may substantially attenuate deleterious systemic and local neuroendocrine effects associated with end-stage heart failure. Implantation of a LVAD also attenuates the systemic proinflammatory response, as demonstrated by the reductions in the cytokines interleukin-6 and -8 concomitant with improved hemodynamics.[20] These data support the hypothesis that the inflammatory cascade may play an important role in the regulation of cardiac dysfunction in heart failure, and that LVAD therapy is capable of reversing these detrimental effects.

With improved hemodynamics and reversal of both neuroendocrine and inflammatory responses to chronic heart failure, exercise tolerance dramatically improves in the vast majority of LVAD patients, and many resume active exercise programs. Thus, many LVAD patients undergoing non-LVAD surgery may present in better physiologic condition than other heart failure patients who are not receiving chronic mechanical support.

Perioperative Management of the Patient with a Left Ventricular Assist Device

The first priority for the anesthesiologist caring for a patient with a chronically implanted LVAD is to identify the "LVAD team" in his or her hospital. A specialized team of healthcare professionals that may include cardiothoracic surgeons, nurses, engineers, and cardiopulmonary perfusionists is usually responsible for the management of LVAD patients in the vast majority of medical centers that offer chronic mechanical circulatory support for the treatment of end-stage heart failure. This LVAD team is an invaluable resource for information about the intricacies of LVAD management, as is the LVAD manufacturer.

Securing a reliable power supply to assure continuous operation of a mechanical assist device is the next consideration for the LVAD patient presenting for surgery. The VE (electric) HeartMate® and the Novacor® pumps may be powered using portable rechargeable batteries for at least 6 h, and the manufacturer of the Novacor® LVAD contends that conversion to an alternating current power supply is unnecessary during surgical procedures (David Thomas, World Heart Corp., Ottawa, Canada, written communication, February 2002). Nevertheless, it may be more prudent to convert the battery-powered LVAD to an alternating current power source once the patient has arrived in the operating room. The IP (pneumatic) HeartMate® and Thoratec® LVAD drivers are normally powered by 120-volt alternating current but also have auxiliary battery power supplies that provide between 30 and 40 min of operation for patient transport to and from the operating room. Both pneumatic consoles provide visual messages and auditory alarms when battery power is nearly exhausted. Restoration of alternating current power should be established on arrival of the patient to the operating room.

The potential for electromagnetic interference with LVAD function by external defibrillation or electrocautery should be recognized by the anesthesiologist.[21] The electronics for the timing and driving of the Novacor® and Thoratec® devices are housed externally and are thus shielded from electrical interference. In contrast, some of the timing circuitry for both IP (pneumatic) and VE (electric) HeartMate® pumps are located within the implanted unit, and both defibrillation and electrocautery can affect their function. As a result, device settings and connections may require adjustment, but it must be emphasized that such maneuvers should only be done with consultation or supervision of the institution's LVAD team. For defibrillation, the electrical component of the IP-HeartMate® driveline (which consists of a hose for delivery of compressed air and an electrical wire for timing circuitry) should be disconnected from the driver console. The console may be programmed to function in a fixed-rate mode or the pump may be hand-operated during defibrillation. The VE-HeartMate® driveline does not have a separate timing connection, and the device should thus be shut down and the controller disconnected before defibrillation. The device may be hand-operated with the manual pneumatic driver during defibrillation. Electrocautery may also interfere with the electronics of both HeartMate® devices and may produce erratic pump output, particularly with the VE (elec-

tric) model. Thus, the manufacturer recommends that the electric model be set to function in the fixed-rate mode as opposed to the "fill-to-empty" mode during surgical procedures in which the use of electrocautery is anticipated. In addition, the grounding pad for the cautery should be placed such that the current will flow away from the implanted pump. Bipolar cautery should be used if possible, since current flows only between the tips of the bipolar instrument, but bipolar cautery is impractical for many surgical procedures because it is much less powerful than monopolar cautery.

The hemodynamic management of the LVAD patient during non-LVAD surgery is important because the pump function of each device depends on both filling volume and outflow resistance. HeartMate®, Novacor®, and Thoratec® pumps all exhibit sensitivity to changes in preload, especially when functioning in the "fill-to-empty" mode. While these devices exhibit no Starling effect with respect to stroke volume or stroke work, they can only pump the volume delivered to them, and inadequate filling will result in decreased flow through a decrease in stroke rate. Maintenance of adequate preload is thus critically important. Direct decreases in pump flow occur when preload declines as a consequence of decreased venous return secondary to increased venous capacitance, alterations in body position that reduce venous return (*e.g.*, lateral decubitus[22] or reverse Trendelenburg position), inadequate administration of intravenous fluids, or uncontrolled surgical bleeding. The preload sensitivity of these devices suggests that invasive monitoring of RV and pump filling pressures using a central venous or pulmonary artery catheter or two-dimensional transesophageal echocardiography may be indicated for procedures in which substantial shifts in intravascular volume are anticipated. Anesthetic induction using sedative–hypnotic drugs that increase venous capacitance (*e.g.*, sodium thiopental, propofol) or rapid administration of other vasoactive drugs that produce selective dilation of the venous circulation (*e.g.*, nitroglycerin) may also produce acute hemodynamic decompensation in the LVAD patient because pump blood flow decreases during these conditions.

In addition to this preload dependence, all commercially available assist devices exhibit sensitivity to changes in afterload. As a result, hypertension should be specifically avoided because emptying of the LVAD is reduced by increases in arterial pressure. Incomplete LVAD ejection not only decreases forward flow but also promotes blood stasis within the device and increases the risk of thrombus formation, even in the presence of systemic anticoagulation. Thus, attenuation of acute increases in sympathetic nervous system activity and its consequent effects on arteriolar tone produced by laryngoscopy, intubation, or surgical stimulation represents an important goal in the perioperative management of these patients. These objectives may be achieved by assurance of adequate anesthetic depth using volatile anesthetics in combination with an opioid or by the judicious administration of arterial vasodilators (*e.g.*, sodium nitroprusside, fenoldapam) to treat increases in arterial pressure. The drugs should be added cautiously, paying careful attention to resultant increased venous capacitance and decreased venous return.

In general, preload and afterload should be managed such that pump flow provides an acceptable cardiac output (which can be determined from thermodilution if flows are not displayed on the LVAD console) while blood pressure is maintained in the normal range. In the absence of hypertension, most cases of low LVAD flow can be corrected by volume expansion, although RV dysfunction must also be considered, as discussed below.

Right ventricular dysfunction is another potential cause of reduced LVAD output. As described above, RV function usually improves with chronic LVAD therapy, but the cellular and mechanical defects may not improve in the right ventricle as much as they do in the LV. Thus, the RV remains a potential hazard for the LVAD patient undergoing surgical procedures. Negative inotropic drugs (*e.g.*, volatile anesthetics, β_1-adrenoceptor antagonists, Ca^{2+} channel antagonists) and factors that can reduce RV output by increasing pulmonary vascular resistance (*e.g.*, hypoxemia, hypercarbia, acidosis) should be avoided. Progressive increases in central venous pressure and RV dilatation combined with simultaneous reductions in LVAD output (or thermodilution-derived cardiac output) are highly suggestive of RV dysfunction and may require intervention with positive inotropic drugs (*e.g.*, milrinone, dobutamine) or selective pulmonary vasodilators (*e.g.*, inhaled nitric oxide).

Management of anticoagulant therapy is another major issue that requires attention in the perioperative care of the LVAD patient. The patient with a Novacor® or Thoratec® device who is chronically treated with warfarin should be converted to intravenous heparin therapy before elective surgery, similar to the patient with a mechanical prosthetic valve.[23] The heparin should be discontinued during the immediate preoperative period and resumed when the risk of postoperative bleeding has diminished. Oral anticoagulation with warfarin may then be reinitiated, and the patient is weaned from heparin as the International Normalized Ratio approaches the therapeutic range. During emergency circumstances, withdrawal of oral anticoagulants cannot be accomplished before surgery, and transfusion of fresh frozen plasma is required to acutely restore deficient coagulation factors. In contrast to Novacor® or Thoratec® LVAD patients, the HeartMate® patient is generally maintained with chronic aspirin therapy alone, and excess perioperative bleeding may require platelet transfusion to obtain adequate hemostasis.

Summary

The perioperative management of the LVAD patient requiring elective or emergent surgery presents several unique challenges for the anesthesiologist. A thorough understanding of the normal operation of the LVAD and the factors that influence its performance is essential for the successful care of these complex patients. The anesthesiologist should consult the manufacturer and/or the institution's LVAD team to assist in perioperative management.

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