Left Ventricular Assist Device Implantation and Management: How I Teach It

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Over the last 2 decades use of left ventricular assist device (LVAD) therapy for support of the failing left ventricle has become an accepted treatment. Device indications generally fall into 2 broad categories defined by the Centers for Medicare & Medicaid Services as bridge to transplantation, for patients who are considered appropriate transplantation candidates, and destination therapy. Destination therapy is used in patients who are not candidates for heart transplantation at the time of device implantation (because of age, comorbidities, or psychosocial concerns) and for whom this therapy is the definitive treatment. The reality is that this binary categorization remains fluid, and patients can change from 1 category to the other, depending on their overall clinical course during the period of LVAD support.

To gain a better understanding of the acuity of patients at the time of device implantation and the associated outcomes, patients have been categorized according to the Interagency Registry for Mechanically Assisted Circulatory Support (Intermacs) criteria.[1] To date, the largest numbers of patients undergoing LVAD therapy fall into the categories of levels 1 to 3, with level 3 patients those who are deemed stable on inotropes. Level 1 patients are in acute cardiogenic shock with manifestations of end-organ dysfunction. Although carefully selected level 1 patients continue to undergo implantation with durable LVADs, most of them transition through a period of support with temporary assist devices before definitive treatment with an LVAD. The traditional rationale is that in many cases, these are patients with acute presentations and rapid deterioration in whom prompt hemodynamic stabilization becomes imperative as a lifesaving measure and who have no time for a full LVAD evaluation and a more complex operation. In addition, the presentation precludes any sort of optimization before definitive VAD surgery.

Current LVAD therapy is based on continuous-flow, centrifugal rotary technology. The 2 dominant devices currently in use are the HVAD (Medtronic, Minneapolis, MN) and the Heartmate 3 (Abbott, Abbott Park, IL). Although there are subtleties in the mechanism for the rotor's suspension, which largely dictates the size of the permissible gap between the rotor and the housing, the clinical advantages of 1 pump technology over the other are still not defined. Both devices allow for intrapericardial implantation, thereby eliminating the need for creating a separate pump pocket. Clinically relevant points with both pumps are afterload sensitivity (with high afterload on the pump, overall output is diminished) and the problem that excessive speed can lead to left ventricular (LV) suction, commonly occurring in the setting of diminished return of blood to the left side (hypovolemia, tamponade, or more commonly right ventricular failure [RVF]). These are extremely important factors to consider intraoperatively as the patient is separated from cardiopulmonary bypass and is transitioning to LVAD support.

Preparation

Preparation for LVAD implantation begins well before the day of surgery. In the best circumstances, patients are undergoing an outpatient evaluation, and once their workup is complete they are presented in a multidisciplinary meeting for final acceptance. In many cases, this meeting will be in the context of consideration for multiple advanced therapies, including heart transplantation, LVAD implantation, or other mechanical circulatory support.

Our meetings consist of heart failure cardiologists and advanced practice providers, cardiac surgeons and residents, intensivists, pharmacists, social workers, finance professionals, administrators, and consulting services whose input may be valuable in decision making. This input can include consultants from neurology, gastroenterology, hepatology, nephrology, infectious disease, and psychiatry, depending on an individual patient's disorder. Once we agree that advanced therapies are medically appropriate, we review the patient's support structure to ensure that a caregiver has been identified to help the patient transition to life at home once the patient is ready for discharge. Our heart failure advanced practice providers and social workers are instrumental in reviewing these details, and their input is carefully considered.

For the surgeons, review of the available imaging at this time is particularly valuable. We are most interested in identifying any ancillary procedures that will be necessary and assessing the suitability of the right ventricle for LVAD implantation. In particular, we evaluate the aortic valve and make a plan to correct any aortic insufficiency that is greater than mild in degree. This is especially

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important in patients undergoing destination therapy, in whom heart transplantation will not be available as a bailout should aortic insufficiency worsen after LVAD implantation.[2] For the tricuspid valve, we have adopted the strategy of repair if the regurgitation is severe or if there is moderate regurgitation and a tricuspid valve annulus greater than 40 mm in diameter.[3] For the mitral valve, we tend to be conservative and repair even severe mitral regurgitation only if there is a component of prolapse that would be expected to result in severe residual mitral regurgitation despite adequate unloading of the left ventricle by the VAD.

At this time, we also review computed tomographic imaging if the patient requires reoperative sternotomy. This approach facilitates our operative planning in terms of cannulation strategy, and it allows us to assess the risk of injury to the right ventricle on reentry. If the patient has a previous coronary artery bypass graft, we request and review the preoperative coronary angiogram. In particular, we want to identify the course of the left internal mammary artery graft and to know whether there are other patent grafts that may be injured during dissection.

A new area of interest for us and others is in the treatment of atrial fibrillation in patients undergoing LVAD implantation. Our current strategy is to ligate the left atrial appendage with a clip in our patients with atrial fibrillation who undergoing LVAD implantation. There is some evidence that this method many decrease the stroke risk in LVAD recipients.[4] Whether extrapolating current recommendations for aggressive surgical management of atrial fibrillation in general adult cardiac surgery to LVAD-treated patients would be beneficial remains to be seen. However, given that stroke remains one of the most common complications after LVAD implantation, exploring all options to reduce this risk seems appropriate.

In assessing a patient as a surgical candidate for LVAD, nothing is clinically more important than the status of the right ventricle and the ability to predict whether significant RVF will dominate the complex postoperative course of the patient. The reported incidence of early RVF after LVAD implantation ranges from 6% to 44%, varying mostly as a result of differences in the definition of RVF. The most comprehensive definition of RVF has been the revised definition provided by Intermacs, which defines RVF as a persistence of signs and symptoms of right ventricular (RV) dysfunction as evidenced by central venous pressure (CVP) greater than 18 mm Hg with a cardiac index of less than 2.0 L/min/m^2 in the absence of increased left atrial filling pressure. Placement of an RV assist device, or the use of intravenous inotropes or nitric oxide for more than 14 days after implantation of an LVAD, also fulfills the definition.

The pathophysiologic mechanisms that lead to manifestations of RVF are multifactorial, with some related to preexisting conditions and others to the hemodynamic changes brought on by the device. First, in patients with the generic "dilated" cardiomyopathies (whether related to genetic predisposition, chemotherapy-induced, or resulting from other mechanisms), it is expected that the same cardiomyopathic processes that affect the left ventricle will also involve the right ventricle. To a large extent, the decrement in RV systolic and diastolic function and RV dilation is determined by this process and can be further exacerbated by the afterload imposed by chronic pulmonary hypertension. Similarly, in patients with ischemic cardiomyopathy, coronary artery perfusion may be compromised by atherosclerotic disease that can also be further exacerbated by decreased perfusion pressure related to high CVP and low systemic blood pressure, which are commonly found in patients considered for LVAD therapy.

Cardiopulmonary bypass can exacerbate RVF by causing an acute increase in pulmonary vascular resistance, likely secondary to inflammatory mediators. Blood transfusion and protamine administration also have been shown to cause pulmonary vasoconstriction and exacerbate RV dysfunction. Implantation of an LVAD itself may exacerbate RVF by several mechanisms. The effect of ventricular interdependence is most prominent in the setting of loading changes after LVAD implantation. The interventricular septum contributes substantially to RV systolic pressure and stroke volume and also serves as its support structure against which the RV free wall contracts. Excessive LV unloading results in leftward shift of the interventricular septum, thus jeopardizing efficient RV contraction and output. In addition, augmentation of cardiac output and systemic blood flow by the LVAD increases the amount of venous return to the failing right ventricle. In the absence of a significant and immediate reduction in RV afterload, RVF may be exacerbated.

Many predictive risk models for RVF have been developed over the years, all based on retrospective analysis of patients in whom RVF has developed compared with patients in whom it has not. Part of the major limitation of all of these models is that they largely disregard many of the physiologic changes that are brought about by the impact of anesthesia, changes in intrathoracic pressure with positive-pressure ventilation, and volume shifts. In addition, these models rarely account for the impact of the pump on ventricular volume, pressure, and interventricular septal function. In evaluating the right ventricle, we use a combination of hemodynamic values (pulmonary artery pulsatility index, ratio of CVP to pulmonary capillary wedge pressure) and echocardiographic data (RV/LV diameter, severity of tricuspid regurgitation, objective assessment of RV free wall contractile function, tricuspid annular plane systolic excursion) to assess the severity of RVF. In the absence of any standardized approach, this has largely been subjective and experienced based. Hence, the most important factors in selecting patients are focused on optimization to prevent the need for RV assist devices. These approaches include aggressive preoperative diuresis to lower the CVP, ideally to less than 10 mm Hg without a concomitant rise in creatinine, and liberal use of inotropic support (to optimize peripheral perfusion while decongesting the liver). Additional considerations include intraoperative minimization of transfusion, use of inhaled

pulmonary vasodilators throughout the procedure, and maintenance of mean systemic blood pressure greater than 70 mm Hg to optimize RV performance.

How I Teach It

Once the patient is anesthetized and intubated, appropriate monitoring lines, including an arterial line and pulmonary artery catheter, are placed. A transesophageal echocardiography probe is inserted, and a final assessment of the heart is performed. In particular, RV function, aortic valve, mitral valve, tricuspid valve, atrial septum, left atrial appendage, and LV apex are all evaluated for pathologic findings that will change the operative plan. Once the final surgical plan is confirmed, the patient is prepared and draped in sterile fashion. A standard median sternotomy is performed. The pericardium is entered, and a pericardial well is created. The patient is heparinized (activated clotting time $>$ 480 seconds), and the ascending aorta and right atrium are cannulated for bypass.

Before the initiation of bypass, the pump is inspected to ensure proper connections. The outflow graft is wrapped with Gore-Tex (W. L. Gore & Associates, Newark, DE) pericardial membrane (0.1 mm), and a separate piece of Gore-Tex is wrapped around the driveline about halfway up the velour. The purpose of this maneuver is to avoid significant intrapericardial adhesions to these structures for future reoperation. Once the pump is prepared, full cardiopulmonary bypass is initiated. We continue low–tidal volume ventilation throughout the cardiopulmonary bypass run that allows us to continue inhaled pulmonary vasodilators (i.e., epoprostenol or nitric oxide) in situations where they are necessary. The field is flooded with carbon dioxide running at 4 to 6 L/min.

To expose the LV apex, a deep pericardial silk suture is placed in the posterior pericardium below the level of the left inferior pulmonary vein and is retracted upward and attached to the drapes. Warm, moist laparotomy pads are used as needed to bring the apex to the midline. We then use transesophageal echocardiographic guidance to identify a position for apical cannulation that will allow us to direct the pump inlet toward the mitral valve. This point is then marked, and the apical connector is brought onto the field. Four anchoring mattress sutures (2-0 Tevdek [Teleflex Medical OEM, Gurnee, IL] with Teflon [Chemours, Wilmington, DE] felt pledgets) are placed on the 4 opposite corners and tied down. A silk suture is then placed in the apex, and a small portion of the myocardium is resected with a No. 11 blade to create a circular ventriculotomy. We find that this technique makes insertion of the coring knife less traumatic. The apex is then cored in standard fashion with the coring knife, and we inspect the apex for any thrombus or trabeculations that need to be resected. It is important to ensure that the inflow pathway is clear because the degree of decrease in the volume of the left ventricle is unpredictable, and unresected trabeculae may interfere with the LVAD inflow. Once this is completed, soft-tip cardiotomy suction is placed just at the apex, to minimize the accumulation of LV air and reduce the time spent removing air from the left ventricle later. We then place the remaining 8 apical pledgeted sutures in the usual fashion. We prefer to place these sutures with the heart decompressed to minimize the chance of tearing the LV wall. Once these sutures are placed and tied down, we bring the pump onto the field and tunnel the driveline. We prefer the left side, midway between the costal margin and the umbilicus, just medial to the midclavicular line, but this position may be adjusted for body habitus or patient preference. After the driveline is tunneled, we place the pump into the left ventricle and secure the pump to the apical connector. At this stage we check the apex for hemostasis because this is the best time to fix any bleeding from this site. Once we are satisfied with the apex, we remove the lap pads and deep pericardial suture and drop the left ventricle back into anatomic position.

Next, we identify our desired position on the ascending aorta for the outflow graft. A side-biting clamp is placed here, which is typically on the lateral wall of the mid-ascending aorta, approximately 2 cm above the sinotubular junction. The adventitia is cleared, and the aorta is entered with a scalpel. The aortotomy is extended with Potts scissors and rounded with an aortic punch to maximize the outflow area. We then fill the heart and outflow graft with volume and stabilize the outflow graft at the junction of the inferior vena cava and the right atrium. The graft is then maximally stretched to reach the aortotomy site, and a Kocher clamp is placed at a 45-degree angle on the graft. The graft is transected, and the volume is removed from the heart. The anastomosis is performed with a running 4-0 Prolene (Ethicon, Bridgewater, NJ) suture in standard fashion. Before tying the suture line, air is removed from the graft again. Once the suture line is tied, 2 needle holes are made in the graft, and the side-biting clamp on the ascending aorta is removed. Hemostasis of the outflow graft is confirmed, and an aortic root vent is then inserted. It is important that the outflow graft be the proper length because excessive length is prone to kinking, and a short, tight graft can compress the right ventricle.

The driveline is passed off the field, and the pump is started at low speed with the outflow graft clamped. We resume full ventilation and add volume to the heart. Inotropes and vasopressors are added to maintain mean arterial pressures in the 70s and a midline septum. The weaning phase requires constant communication and coordination among the surgeon, the anesthesiologist, and the perfusion team. This portion of the case is typically handled by the attending surgeon. The clamp on the outflow graft is slowly released, and the aortic root is inspected for the presence of air. If the aortic root and left ventricle are free of air, we increase the speed of the LVAD and reduce the flow on cardiopulmonary bypass. This requires a constant assessment of RV function, septal position, and hemodynamics. Once the patient is off cardiopulmonary bypass, the aortic root vent is removed, and the site is oversewn. The LVAD speed is then optimized as hemodynamics allow. Protamine is

administered, and the patient is decannulated in standard fashion.

We typically place a drain near the apex in addition to a substernal chest tube and a left pleural chest tube. Once hemostasis is satisfactory, we wrap the remaining Gore-Tex pericardial membrane around the remainder of the exposed portion of the outflow graft and secure it to the ascending aorta with a 4-0 Prolene suture. The ascending aorta is covered with mediastinal fat, and the chest is closed in standard fashion.

Comment

Since its inception, LVAD technology has improved with each new generation of devices. The pumps have become progressively smaller, and they are much more durable than their first iterations. Patient survival in the most recent trial is excellent.[5] Despite these advances, LVADs remained limited by their 3 most common complications: infections, strokes, and gastrointestinal bleeding. The future of LVADs will be determined by the ability to capitalize on technologic advances that will make the devices both easier to implant and more durable, and also by improvements in technology and patient management that will reduce these dreaded complications.

A primary factor in infectious complications remains the need for a driveline with the current technology. The ability to eliminate this driveline and the continual connection between implantable device and the outside world would be the most direct means to reduce infectious complications. Creating a reliable implantable power source without too many implantable components and of reasonable size remains the primary barriers to achieving this goal, but we believe the future holds great promise for making this a reality. Thrombotic complications, namely stroke, and bleeding complications, typically in the gastrointestinal tract, are closely related and will likely decrease with improved hemocompatibility of devices. Ongoing efforts to identify the optimum antiplatelet and anticoagulation regimens for each individual patient continue because striking the right balance between bleeding and thrombosis is important to reducing both these complications.

As the technology improves and the ability to augment cardiac output with smaller and more durable pumps becomes reality, we expect to see surgeons continue to find innovative and less invasive ways to implant devices. Currently, this involves smaller incisions and sternal-sparing approaches. Others are mastering the technical aspects of inserting LVADs without the need for cardiopulmonary bypass. The intersection between technologic and surgical innovation will lead to continued improvement in results with each new generation of devices.

Trainees who wish to push this field forward would do well to spend their time now learning the nuances of the technique for implanting the current-generation devices and immersing themselves in the pathophysiologic conditions that the devices are designed to treat. Regardless of the size and type of pump that is used to support the left ventricle, managing the right ventricle and associated structural heart disorders and identifying the right patients for device implantation will remain the surgeon's responsibility for the foreseeable future.

References

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