Anesthesia for Ventricular Assist Device Implantation
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What Are VADs

Ventricular assist devices (VADs) are medical devices that are used to support the patient’s acutely or chronically decompensated heart. Most often, these costly devices are implanted as temporary “bridges” to recovery or transplantation (BTT) [1]. Less commonly, they are implanted permanently as “destination therapy” (DT) [2]. Devices can be located outside the body (extracorporeal), inside the patient (intracorporeal), or immediately adjacent to the body (paracorporeal). They can be further sub-classified according to the type of blood flow provided (pulsatile or non-pulsatile/axial), driving power utilized (pneumatic or electric), or according to the level of anticoagulation required (low – anti-platelet agents only vs. full – anti-platelet agents, warfarin and/or heparin).
Three generations of VADs are currently in existence, either approved for therapy or in clinical trials. The first generation generally consists of pumps such as the Thoratec VAD® or HeartMate®, which frequently provide pulsatile flow. The second generation comprises smaller devices making use of electromechanical impellers to drive blood forward (e.g., HeartMate II, Jarvik 2000). The newest, third generation devices are an upgrade of the foregoing using bearingless, magnetically- and/or hydrodynamically suspended impellers that minimize heat generation and improve durability.

After implantation, patients who do well experience improved hemodynamics, reversal of end-organ dysfunction, increased exercise tolerance, and a better chance of survival. Indications for LVAD implantation include end-stage heart failure and cardiogenic shock of various etiologies. Preoperative risk factors predictive of worse outcome after LVAD implantation include poor renal function, right heart failure, liver dysfunction, coagulopathy and reoperation. Good anesthetic management of these patients is intensive, complicated, and integral to success.

For the anesthetic management of patients who already have an LVAD, we provide a selection of articles in the References section [3, 4].

Overview of Ventricular Assist Devices, Total Artificial Hearts, and VAD physiology

There are a number of different types of mechanical assist devices that are placed in patients with acute or chronic heart failure. A complete review of all of these devices, their advantages, disadvantages, indications and anesthetic implications is beyond the scope of this manual. The following tables will briefly review a few of these devices. Information in this section may rapidly become outdated as new devices are introduced/approved and older devices are retired. In addition, the type of device varies widely from center to center and from situation to situation. See the References section for articles [5, 6] that review information on ventricular assist devices.

<table>
<thead>
<tr>
<th>Table 1. Extracorporeal devices</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pump mechanism &amp; flow type</strong></td>
</tr>
<tr>
<td>TandemHeart® (CardiacAssist, Inc., Pittsburgh, PA)</td>
</tr>
<tr>
<td>Abiomed® BVS® 5000 (Abiomed Inc., Danvers, MA)</td>
</tr>
</tbody>
</table>
pneumatically-driven ventricle.
- An external console can support one or two VADs. It compensates for changes in both preload and afterload.
- After deployment, chest remains “open”, which increases infection risk and limits the use of the device.
- The device functions only in “volume” mode – emptying occurs only after the ventricular chamber is full.

Table 2. Paracorporeal devices

<table>
<thead>
<tr>
<th>Pump mechanism &amp; flow type</th>
<th>Configuration, special features</th>
<th>Anticoagulation</th>
<th>Approval status/clinical use</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Thoratec VAD®</strong> (Thoratec Corporation, Pleasanton, CA)</td>
<td>Pneumatic – pulsatile flow</td>
<td>LVAD, RVAD or BiVAD. Can be driven by a large stationary console or a smaller portable one. Filling is largely passive and depends on an adequate CVP (&gt;13). Two pumping modes: <strong>asynchronous or “fixed” mode</strong>, where the VAD rate and ejection time are set by the ser and the driver maintains those</td>
<td>Full</td>
</tr>
</tbody>
</table>

conditions indefinitely; and **volume or "auto" mode**, where ejection begins as soon as complete VAD filling occurs.

<table>
<thead>
<tr>
<th>Berlin Heart® EXCOR® and EXCOR® Pediatric <em>(Berlin Heart, Inc., Northborough, MA)</em></th>
<th>Pneumatic – pulsatile flow</th>
<th>LVAD, RVAD or BiVAD</th>
<th>Full</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Stationary and portable driving units available</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>pediatric sizes available with 10, 25, 30, 50 or 60 ml displacement</td>
<td></td>
</tr>
</tbody>
</table>


Table 3. Intracorporeal devices

<table>
<thead>
<tr>
<th>Pump mechanism &amp; flow type</th>
<th>Configuration, special features</th>
<th>Anticoagulation</th>
<th>Approval status/clinical use</th>
</tr>
</thead>
<tbody>
<tr>
<td>IMPELLA® LP2.5 and LP5.0 <em>(Abiomed Inc., Danvers, MA)</em></td>
<td>Minimally-invasive, percutaneous catheter LVADs (2.5 and 5 l/min, respectively). Insertion is similar to an IABP but device rests across the aortic valve, with the tip in the LV cavity.</td>
<td>Full</td>
<td>FDA approved for investigational use; FDA safety trial completed; clinical scenarios include support for patients during high-risk PCI, post PCI, and with AMI with low cardiac output (Impella® LP2.5), and postcardiotomy, myocarditis, cardiogenic shock, and bridge-to-next decision (Impella® LP5.0)</td>
</tr>
<tr>
<td>Thoratec IVAD® <em>(Thoratec Corporation, Pleasanton, CA)</em></td>
<td>Implantable (or paracorporeal) successor to the Thoratec VAD®. Identical operation principle. Can be implanted in</td>
<td>Full</td>
<td>FDA approved for BTT and post-cardiotomy recovery.</td>
</tr>
</tbody>
</table>
LVAD, RVAD or BiVAD configuration.

HeartMate® IP (Thoratec Corporation, Pleasanton, CA) ¹

Implanted pneumatic – pulsatile flow

LVAD only.
- antithrombogenic inner surface composed of sintered titanium microspheres
- Only device that uses low-level anticoagulation.
- Like the Thoratec VAD®, these devices have two pumping modes. In the fixed mode, the device ejects at a preset rate (between 50 and 120 bpm) regardless of the degree of filling of its blood chamber, i.e., stroke volume varies. SV will depend on the rate of filling ("preload") of the device.
- In auto mode, it only ejects when the chamber is 96% full, i.e. SV remains fixed at 76ml, but the rate varies. Auto rate mode maximizes the amount

FDA approved for BTT and post-cardiotomy recovery.
<table>
<thead>
<tr>
<th>LVAS Model</th>
<th>Description</th>
<th>Anticoagulation</th>
<th>FDA Approval</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HeartMate® XVE LVAS</strong> (Thoratec Corporation, Pleasanton, CA)</td>
<td>Extended lead vented electric – pulsatile flow</td>
<td>LVAD only.</td>
<td>Low-level</td>
</tr>
<tr>
<td></td>
<td>▪ LVAD only.</td>
<td></td>
<td>FDA approved for BTT and DT</td>
</tr>
<tr>
<td></td>
<td>▪ Antithrombogenic inner surface composed of sintered titanium microspheres</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>▪ Only device that uses low-level anticoagulation.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>▪ Only device approved for DT in the U.S.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>▪ Driveline connects to an external, wearable controller and battery, allowing the patient to ambulate.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Novacor® LVAS</strong> (World Heart Inc., Oakland, CA)</td>
<td>Electric dual pusher plate – pulsatile flow</td>
<td>LVAD only.</td>
<td>Full</td>
</tr>
<tr>
<td></td>
<td>▪ LVAD only.</td>
<td></td>
<td>FDA approved for BTT; destination trial ongoing</td>
</tr>
<tr>
<td><strong>Arrow LionHeart™ (Arrow International Inc., Reading, PA)</strong></td>
<td>Pusher plate – pulsatile flow</td>
<td>totally implantable LVAD</td>
<td></td>
</tr>
<tr>
<td></td>
<td>▪ TETS (Transcutaneous Energy Transmission System)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>▪ operates in full-to-empty mode (fixed stroke volume)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>▪ asynchronous with native heart</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Device</td>
<td>Impeller Type</td>
<td>FDA Approval Status</td>
<td>Clinical Use</td>
</tr>
<tr>
<td>---------------------------------------------</td>
<td>----------------</td>
<td>-------------------------------------------------------------------------------------</td>
<td>------------------------------------------</td>
</tr>
<tr>
<td>HeartMate® II (Thoratec Corporation, CA) 7</td>
<td>Impeller pump – axial flow</td>
<td>FDA approved for investigational use; clinical trials ongoing</td>
<td>Preclinical trials, destination/bridge</td>
</tr>
<tr>
<td>DeBakey VAD® (MicroMed Cardiovascular Inc., Houston, TX) 8</td>
<td>Impeller pump – axial flow</td>
<td>The inducer-impeller uses a patented ceramic bearing system; has a flow probe for real-time direct flow measurement reduced surgical time compared to 1st generation LVADs due to smaller size; virtually silent</td>
<td>FDA approval for investigational use; clinical trials for BTT/DT use ongoing</td>
</tr>
<tr>
<td>HeartMate® III (Thoratec Corporation, Pleasanton, CA) 9</td>
<td>Centrifugal</td>
<td>Yes</td>
<td>Preclinical trials, destination/bridge</td>
</tr>
<tr>
<td>HeartWare HVAD™ (HeartWare Ltd., Sydney, Australia; formerly</td>
<td>Centrifugal pump with a</td>
<td>Implantable in the pericardium</td>
<td>Clinical trial in Europe/Australia</td>
</tr>
<tr>
<td>Pump mechanism &amp; flow type</td>
<td>Configuration, special features</td>
<td>Anticoagulation</td>
<td>Approval status/clinical use</td>
</tr>
<tr>
<td>----------------------------</td>
<td>---------------------------------</td>
<td>----------------</td>
<td>---------------------------</td>
</tr>
<tr>
<td>Biventricular, pneumatic, pulsatile pump</td>
<td></td>
<td>Full</td>
<td>Approved as BTT in patients who are in need of biventricular support</td>
</tr>
</tbody>
</table>

3 Original image on: [http://news.bbc.co.uk/1/hi/health/3980263.stm](http://news.bbc.co.uk/1/hi/health/3980263.stm)
8 Original image on: [http://www.micromedtech.com/products2.html](http://www.micromedtech.com/products2.html)
10 Image used with the permission of HeartWare Ltd., Sydney, Australia. Original image on: [http://www.heartware.com.au/IRM/content/usa/media_images.html](http://www.heartware.com.au/IRM/content/usa/media_images.html)
<table>
<thead>
<tr>
<th>AbioCor® (Abiomed Inc., Danvers, MA)</th>
<th>Highest bridge-to-transplant rate (79%) of all approved BTT devices [1]</th>
<th>Biventricular support (TAH)</th>
<th>Fully implantable, with a TET (transcutaneous energy transmission) system</th>
<th>Next generation AbioCor® II is under development.</th>
<th>Full</th>
<th>FDA Humanitarian Device Exemption approval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydraulically coupled, asynchronous</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

13 Original image on: [http://www.syncardia.com/](http://www.syncardia.com/)

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### Criteria for LVAD placement

Following is a list of inclusion and exclusion criteria for the placement of LVADs as a BTT. (From Oz et al., 1995 [8], Mancini et al., 2005 [6])

Table 5. Current Guidelines for the Placement of LVAD as a Bridge to Transplantation

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Patient is a transplantation candidate</td>
</tr>
<tr>
<td>2. Systolic blood pressure &lt;80 mmHg with either:</td>
</tr>
<tr>
<td>▪ Cardiac index &lt;2.0 L/min/m2 or</td>
</tr>
<tr>
<td>▪ Pulmonary capillary wedge _20 mmHg</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Exclusion criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Technical considerations</td>
</tr>
<tr>
<td>▪ Body surface area &lt;1.5 m2</td>
</tr>
<tr>
<td>▪ Aortic insufficiency</td>
</tr>
</tbody>
</table>
2. Severe right-side heart failure

3. Factors increasing the risk of perioperative complications
   - Right atrial pressure >16 mm Hg
   - Prothrombin time >16 s
   - Reoperation
   - White blood count >15
   - Urine output <30 ml/h
   - Mechanically ventilated patient
   - Temperature >101.5°F

Columbia Presbyterian Criteria for LVAD Destination Therapy Patients. Used WITHOUT permission from Mancini et al., 2005 [6].

<table>
<thead>
<tr>
<th>Inclusion criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class IIIB or IV CHF</td>
</tr>
<tr>
<td>Inotrope dependent (failed ≥1 attempt at weaning, i.e., documented hypotension, end-organ failure, refractory CHF with downtitration)</td>
</tr>
<tr>
<td>Maximal medical therapy with VO2 &lt;10 mL/kg/min (if not able to tolerate β-blockers, then VO2 &lt;12 mL/kg/min)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Exclusion criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transplantation candidates</td>
</tr>
<tr>
<td>Acute cardiogenic shock</td>
</tr>
<tr>
<td>Renal dysfunction: dialysis, CVVH, or Cr &gt;3 mg/dL</td>
</tr>
<tr>
<td>Hepatic failure: ALT, AST &gt;3 times normal, INR &gt;2.5</td>
</tr>
<tr>
<td>BMI &lt;18 or &gt;35 kg/m2</td>
</tr>
<tr>
<td>Prolonged ventilatory support</td>
</tr>
<tr>
<td>FEV1 &lt;1</td>
</tr>
<tr>
<td>PVR &gt;8 and/or severe RV dysfunction with anticipated RV support</td>
</tr>
<tr>
<td>Comorbidity with life expectancy &lt;2y</td>
</tr>
<tr>
<td>Acute condition: GI bleed, infection</td>
</tr>
<tr>
<td>Neurological: Mini Mental Exam score &lt;20, prior CVA with significant residual</td>
</tr>
<tr>
<td>High surgical risk (ascending aortic carotid artery, &gt;2 prior cardiac surgeries)</td>
</tr>
<tr>
<td>Severe PVD (limb ulcers, amputation)</td>
</tr>
<tr>
<td>Heparin-induced thrombocytopenia</td>
</tr>
<tr>
<td>Psychosocial factors</td>
</tr>
</tbody>
</table>
CVVH indicates continuous venovenous hemofiltration; Cr, creatinine; ALT, alanine transaminase; AST, aspartate transaminase; INR, international normalized ratio; BMI, body mass index; PVR, pressure-volume relationship; RV, right ventricular; GI, gastrointestinal; CVA, cerebrovascular accident; and PVD, peripheral vascular disease.

VAD Physiology

For left ventricular support, blood is drained from the left atrium or left ventricular apex to the pump and returned to the ascending aorta. For right ventricular (RV) support, blood is drained from the right atrium or RV to the pump and returned to the main pulmonary artery. For example, in the HeartMate® XVE, blood enters the pump through an inflow cannula that is sutured into the apex of the left ventricle. One-way valves ensure that the blood exits the device via the outflow cannula during device systole. The outflow cannula is anastomosed end-to-side to the ascending aorta. In most cases, the device assumes most of the work of the LV and delivers the majority of the total cardiac output into the ascending aorta.

Although different devices from different manufacturers use different strategies to accumulate and eject blood, the principles of VAD function are similar. In most cases, VADs are operated in an automatic (variable) rate, fixed volume mode (a.k.a. “full-to-empty” mode). In the variable-rate/fixed-volume mode, the pump will automatically eject as soon as the pump chamber is full (also see Table 3 for a description of these operating modes). Decreased pump output can be due to patient and mechanical factors that result in excessively slow or incomplete pump filling or in prolonged or incomplete pump emptying. The two most important factors leading to decreased pump output are hypovolemia and increased afterload.

Left ventricular assist devices will support the left ventricle, but the right ventricle has to do all its own work. Therefore, it is critical to avoid pulmonary hypertension (excess RV afterload) and avoid volume overload (excess RV preload), both of which can precipitate right ventricular failure. CVP and TEE monitoring is very important to assess RV function during weaning from CPB. With chronic LVAD therapy, the reduction in LVEDP will translate into reduced RV afterload. Therefore, over time even poor RVs improve in the presence of a well-functioning LVAD.

VAD Operation – notes on commonly used devices

Thoratec VAD®

Monitoring:

- Flows on the console are not an accurate measure of flow except in the case where the VAD is emptying completely; The device assumes a 65cc stroke volume.
- Can assess VAD emptying qualitatively by shining light at an oblique angle along the anterior surface of VAD and assessing size of “light flash”, which should be greater than the size of a quarter dollar.
- For the implantable Thoratec IVAD, there are pairs of LEDs that sit on top (separate) from the console that indicate whether emptying is adequate
- Switching from asynchronous (fixed-rate) mode to volume mode will not change the proportion of time spent in systole (this is fixed at 300 msec). Therefore, decrease in heart rate with volume mode will not improve emptying (it will only improve filling) Time in systole can be changed manually to optimize filling or emptying
- Volume mode can be used in a hypovolemic patient to ensure better filling of VAD. VAD rate will decrease in the hypovolemic patient if VAD is in volume mode until it loses it “fill” signal and switches to its fixed rate
• Conversely, if there is inadequate emptying, VAD rate will increase in volume mode (since it takes very little time for the VAD to fill if it is not emptying).
• Mixed venous O2 is a good monitor for overall adequacy of flow and systemic perfusion.

Improving Emptying:
• Can unload RVAD with nitric oxide
• Can unload LVAD with nitroprusside if systemic pressure is adequate
• If pharmacologic measures are not tolerated or don’t work, then one can increase the driving pressure. Typically, driving pressure is set at 100 mm Hg plus systemic systolic pressure (for LVAD) or 100 mm Hg plus PA systolic pressure (for RVAD).

Improving Filling:
• Volume administration; ensure that patient is not hypovolemic
• The Thoratec console adds a small amount of negative pressure during the filling part of the VAD cycle, which is meant to only overcome the resistance of the filling chamber. To improve filling, one can adjust negative pressure: normally set at –20 with chest open and –40 when chest is closed. The risk of increasing negative suction is that air could be entrained around the inflow cannula insertion site, causing air embolism.

Abiomed® BVS® 5000

Monitoring:
• Flows on the console are accurate for the Abiomed® BVS® 5000. The console measures the volume of driving gas that is being used to displace an equal volume of blood during each systole.

Improving Emptying:
• Pharmacologic interventions are the same as with the Thoratec® VAD. Driving pressure is fixed: 320 mmHg for LVAD and 220 mmHg for RVAD.

Improving Filling:
• Lower the position of the bladder with respect to patient’s body. VAD fills by gravity. Note that when bladder position is lowered, the afterload against which the VAD pumps is increased.
• DO NOT place the patient in Trendelenburg while the bladders are fixed to the table.

HeartMate® LVAD

• The HeartMate® console computes LVAD flow within +/- 5% (range, 1.8 to 10 L/min).
• This device is relatively afterload-independent. The “driving pressure” is fixed.
• Inadequate preload will result in slower heart rate and drop in CO. Aim for a HR in the 70-80 range.
• High flows can lead to LV collapse with shift of intra-ventricular septum towards LV resulting in RV dilation, increase RV wall stress, and RV failure. Therefore, will frequently use fixed mode to prevent RV failure.
Table 7. Hemodynamic requirements of common circulatory assist devices

<table>
<thead>
<tr>
<th></th>
<th>IABP</th>
<th>Abiomed® BVS® 5000 BiVAD</th>
<th>Thoratec® BiVAD</th>
<th>HeartMate® LVAD</th>
<th>Abiomed® BVS® 5000 RVAD HeartMate® LVAD</th>
<th>Abiomed® BVS® 5000 RVAD</th>
<th>ECMO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preload</td>
<td>No effect</td>
<td>CVP 12-15</td>
<td>CVP &gt; 13 recommended</td>
<td>Dependant</td>
<td>CVP 12-15</td>
<td>CVP 12-15</td>
<td>Normal</td>
</tr>
<tr>
<td>Afterload</td>
<td>IABP will reduce</td>
<td>Keep SBP&lt;150.</td>
<td>Keep SBP&lt;130.</td>
<td>Keep SBP&lt;120</td>
<td>Keep MAP&lt;100</td>
<td>Native LV</td>
<td>Normal</td>
</tr>
<tr>
<td>Position-dependent relative to patient</td>
<td>No</td>
<td>Yes: Caution with Trendelenburg</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
<td>Requires good cannula positioning</td>
</tr>
<tr>
<td>Effect of inotropes</td>
<td>Positive</td>
<td>No effect</td>
<td>No effect</td>
<td>Use for RV support</td>
<td>No effect</td>
<td>Use for LV support and VAD weaning</td>
<td>No effect</td>
</tr>
</tbody>
</table>

Anesthetic management for VAD implantation

For a more detailed discussion, see a selection of review articles [3, 9-11] in the References section.

Preoperative considerations

- Patients who are having an LVAD implanted have, by definition, severely compromised LV function. The fact that the patient is presenting for VAD implantation suggests that a further decompensation has occurred. Elicit possible causes of such decompensation: pneumonia, sepsis, or cardiac events such as arrhythmia and ischemia.
- Serum electrolytes can be abnormal due to alterations in the renin-angiotensin-aldosterone axis or chronic diuretic therapy.
- There is frequently elevated pulmonary vascular resistance, impaired RV function, impaired hepatic function and/or coagulopathy, renal insufficiency and impaired responsiveness to catecholamines.
- Patients may be on multiple inotropes, IABP, and/or a ventilator.
- Make sure that blood is available (packed red blood cells, FFP, and frequently also platelets may be required).
- Check and record the PA pressure, pulmonary vascular resistance and cardiac output. If available, obtain cardiac catheterization data, paying special attention to the transpulmonary gradient, PVR and response to pulmonary vasodilators.
- Document the latest echo report and note any evidence of RV failure, tricuspid regurgitation and/or pulmonary hypertension.
- Check to make sure that antibiotics have been given (e.g. vancomycin 1 gm and cefuroxime 1.5 gm). Check with local protocol whether antifungal prophylaxis should also be administered. Fungal infections are known to occur with VADs [12, 13].
Transport to OR

- Transport the patient to the operating room with full monitoring (EKG, arterial line, pulse oximeter) and with oxygen.
- Continue all inotropes; do not change the existing inotropic regimen.
- Do not administer any sedative or myocardial depressant before transporting the patient. If the patient is already on sedation, continue the same regimen.
- If transporting an intubated, ventilated patient, strive to maintain minute ventilation, FiO2 and PEEP requirements established in the ICU. Due to the extremely tenuous balance of these patients, seemingly small alterations may lead to significant instability.

Lines and monitors

- Large-bore IV access.
- Arterial line.
- TEE.
- Central venous catheter(s): #9 French introducer with a pulmonary artery catheter; PAC introducer and/or second CVC.
- Continuous cardiac output monitoring has been studied prospectively in 15 HeartMate® recipients [14]. The CCO system overestimated cardiac output by approximately 500 mL/min when compared with the LVAD-reported flow values. The use of CCO monitoring with a PAC is therefore not recommended where a reliable VAD flow reading is available (HeartMate®, Abiomed®).
- An oximetric PAC can be used if institutional CTICU protocol requires monitoring of mixed venous oxygen saturation.

Intraoperative Management

- If the patient has an internal cardioverter-defibrillator (ICD), then its defibrillator function needs to be turned off with a magnet to prevent inadvertent discharge.
- Induction should be gentle without significant myocardial depression. Hypotension is a serious risk during induction in this patient population. It should be helpful to keep in mind that any bolus of an agent intended to counteract hypotension will take significantly longer to take effect due to slow circulation time [15].
- Rapid sequence induction is NOT generally used, in order to minimize periods of apnea and the ensuing hypercarbia. The latter may increase PVR and further decrease left ventricular filling. Instead, a slow induction with cricoid pressure and ventilation is recommended [11].
- Administer adequate amnestic.
- Aprotinin is frequently used. Familiarize yourself with institutional and surgeon practice. As in other cardiac surgeries, for maximum safety it is suggested to administer the aprotinin test dose after heparinization and aortic cannulation, lest an anaphylactic reaction ensues that requires immediate institution of CPB.
- There is evidence to support vitamin K infusion IV throughout the case at 2 mg/hour for reduction of bleeding [16].
- An ICU ventilator may be required for NO administration. Check with the perfusion department or whoever manages the NO equipment at your institution and make adequate preparations.
- Full systemic heparinization is mandatory. Suggested initial dose is 300 units/kg. Maintain ACT about 4 x baseline.
- Prepare milrinone, norepinephrine, and vasopressin.
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Important goals for separation from CPB include:

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- RV afterload reduction, with NO if necessary. Due to its delivery only to ventilated areas of lung, NO has the added benefit of improving ventilation/perfusion matching and PAO2 [11]. A favorable response to NO therapy will be manifest as increased LVAD flow and a reduction in CVP. Nitric oxide causes suppression of nitric oxide synthase expression within hours, causing NO dependency to develop. It should therefore never be weaned quickly except in patients who were non-responders initially. In the latter group, it will add no benefit and should be discontinued promptly.
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Monitor for, and manage:

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- Hypovolemia, inadequate urine output
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- Evaluate inlet cannula position and blood flow velocity by CW Doppler (inlet velocity >2.5 m/s is abnormal). There are several reports in the literature of malpositioned [19] or even completely reversed [20] LVAD cannulae, leading to disastrous hemodynamic consequences. In both cases referenced here, the cannula malpositioning was accurately diagnosed with TEE, allowing corrective surgical measures to be taken.
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- Confirm proper LVAD function by evaluating LV decompression, aortic valve closure during LVAD systole, and outlet cannula presence in the aorta with appropriate flow.

Management of RV Failure

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To prevent and manage RV failure:

- Support systemic perfusion pressure with adequate vasoconstrictor drugs.
- Support the right heart with positive inotropic drugs. Milrinone is often the drug of choice due to its inodilator properties. Its effect on pulmonary vascular resistance can help offload the RV.
- Maintain a fine balance between avoidance of hypovolemia (in order to ensure LVAD filling) and avoidance of right heart volume overload, especially in the setting of a poorly diuresed patient with pre-operative CHF.
- Avoid excessive LVAD output. If the device empties the LV too vigorously or rapidly, the LV can collapse, displacing the interventricular septum towards the left ventricular cavity. This leads to RV dilation and significant tricuspid regurgitation. The inflow cannula may become obstructed leading to entrainment of air into the pump from around the sewing ring.
- With a well-functioning LVAD, ventricular arrhythmias may not lead to immediate hemodynamic collapse as in the unassisted patient. However, due to their detrimental effect on coronary perfusion and right ventricular function, they must be treated rapidly.
- Anticipate increased PVR due to blood products and protamine administration.
- Nitric oxide is frequently used to reduce pulmonary vascular resistance. For a more in-depth discussion of NO use and delivery systems, see chapter on Heart transplantation.
- If it appears that the above measures are insufficient to restore hemodynamic stability and proper LVAD function, a temporary RVAD may be lifesaving. Discuss early with the surgeon.

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Bleeding is often significant. A long CPB time, along with platelet activation and excess fibrinolysis worsen any pre-existing coagulopathy. It is necessary to anticipate this potential problem and order sufficient blood and blood products. The coagulopathy can extend well into the post-operative period and can lead to surgical re-exploration.

If possible, use leukocyte-filtered products to reduce the formation of reactive antibodies against blood and tissue antigens. Remember that the VAD is frequently implanted as a BTT, and future surgeries most likely will be needed. Alloimmunization will make future cross-matching of blood and a donor heart more difficult.

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Recognize that transfusion can increase preload and afterload on RV.

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Several reports indicate successful therapy with recombinant factor VIIa in VAD recipients with catastrophic bleeding, where all other measures to halt hemorrhage had failed [22, 23]. On the other hand, there is a report of a clotted-off Abiomed® BVS® 5000 device after rFVIIa administration, with a fatal outcome [24]. The use of rFVIIa is not FDA approved in this setting and cannot be recommended based on existing literature. Its humanitarian, “off-label” use in cases where all other care has failed to produce a result and death from hemorrhage seems imminent is left to the discretion of the attending clinician.

VAD Physiology

For left ventricular support, blood is drained from the left atrium or left ventricular apex to the pump and returned to the ascending aorta. For right ventricular (RV) support, blood is drained from the right atrium or RV to the pump and returned to the main pulmonary artery. For example, in the HeartMate® XVE, blood enters the pump through an inflow cannula that is sutured into the apex of the left ventricle. One-way valves ensure that the blood exits the device via the outflow cannula during device systole. The outflow cannula is anastomosed end-to-side to the ascending aorta. In most cases, the device assumes most of the work of the LV and delivers the majority of the total cardiac output into the ascending aorta.

Although different devices from different manufacturers use different strategies to accumulate and eject blood, the principles of VAD function are similar. In most cases, VADs are operated in an automatic (variable) rate, fixed volume mode (a.k.a. “full-to-empty” mode). In the variable-rate/fixed-volume mode, the pump will automatically eject as soon as the pump chamber is full (also see Table 3 for a description of these operating modes). Decreased pump output can be due to patient and mechanical factors that result in excessively slow or incomplete pump filling or in prolonged or incomplete pump emptying. The two most important factors leading to decreased pump output are hypovolemia and increased afterload.

Left ventricular assist devices will support the left ventricle, but the right ventricle has to do all its own work. Therefore, it is critical to avoid pulmonary hypertension (excess RV afterload) and avoid volume overload (excess RV preload), both of which can precipitate right ventricular failure. CVP and TEE monitoring is very important to assess RV function during weaning from CPB. With chronic LVAD therapy, the reduction in LVEDP will translate into reduced RV afterload. Therefore, over time even poor RVs improve in the presence of a well-functioning LVAD.

VAD Operation – notes on commonly used devices

Thoratec VAD®

Monitoring:
flows on the console are not an accurate measure of flow except in the case where the VAD is emptying completely; the device assumes a 65cc stroke volume.

- Can assess VAD emptying qualitatively by shining light at an oblique angle along the anterior surface of VAD and assessing size of "light flash", which should be greater than the size of a quarter dollar.

- For the implantable Thoratec IVAD, there are pairs of LEDs that sit on top (separate) from the console that indicate whether emptying is adequate.

- Switching from asynchronous (fixed-rate) mode to volume mode will not change the proportion of time spent in systole (this is fixed at 300 msec). Therefore, decrease in heart rate with volume mode will not improve emptying (it will only improve filling). Time in systole can be changed manually to optimize filling or emptying.

- Volume mode can be used in a hypovolemic patient to ensure better filling of VAD. VAD rate will decrease in the hypovolemic patient if VAD is in volume mode until it loses it "fill" signal and switches to its fixed rate.

- Conversely, if there is inadequate emptying, VAD rate will increase in volume mode (since it takes very little time for the VAD to fill if it is not emptying).

- Mixed venous O2 is a good monitor for overall adequacy of flow and systemic perfusion.

Improving Emptying:

- Can unload RVAD with nitric oxide.
- Can unload LVAD with nitroprusside if systemic pressure is adequate.
- If pharmacologic measures are not tolerated or don't work, then one can increase the driving pressure. Typically, driving pressure is set at 100 mm Hg plus systemic systolic pressure (for LVAD) or 100 mm Hg plus PA systolic pressure (for RVAD).

Improving Filling:

- Volume administration; ensure that patient is not hypovolemic.
- The Thoratec console adds a small amount of negative pressure during the filling part of the VAD cycle, which is meant to only overcome the resistance of the filling chamber. To improve filling, one can adjust negative pressure: normally set at –20 with chest open and –40 when chest is closed. The risk of increasing negative suction is that air could be entrained around the inflow cannula insertion site, causing air embolism.

**Abiomed® BVS® 5000**

Monitoring:

- Flows on the console are accurate for the Abiomed® BVS® 5000. The console measures the volume of driving gas that is being used to displace an equal volume of blood during each systole.

Improving Emptying:

- Pharmacologic interventions are the same as with the Thoratec® VAD. Driving pressure is fixed: 320 mmHg for LVAD and 220 mmHg for RVAD.

Improving Filling:

- Lower the position of the bladder with respect to patient’s body. VAD fills by gravity. Note that when bladder position is lowered, the afterload against which the VAD pumps is increased.
- DO NOT place the patient in Trendelenburg while the bladders are fixed to the table

**HeartMate® LVAD**

- The HeartMate® console computes LVAD flow within +/- 5% (range, 1.8 to 10 L/min).
- This device is relatively afterload-independent. The “driving pressure” is fixed.
- Inadequate preload will result in slower heart rate and drop in CO. Aim for a HR in the 70-80 range.
- High flows can lead to LV collapse with shift of intra-ventricular septum towards LV resulting in RV dilation, increase RV wall stress, and RV failure. Therefore, will frequently use fixed mode to prevent RV failure.

**Table 7. Hemodynamic requirements of common circulatory assist devices**

<table>
<thead>
<tr>
<th>Device</th>
<th>Preload</th>
<th>Afterload</th>
<th>Native LV Position</th>
<th>Effect of inotropes</th>
<th>Use for LV support and VAD weaning</th>
</tr>
</thead>
<tbody>
<tr>
<td>IABP Abiomed® BVS® 5000 BiVAD Thoratec® BiVAD HeartMate® LVAD Abiomed® BVS® 5000 RVAD HeartMate® LVAD Abiomed® BVS® 5000 RVAD ECMO Preload No effect CVP 12-15 CVP &gt; 13 recommended Dependant CVP 12-15 CVP 12-15 Normal Afterload IABP will reduce Keep SBP&lt;150. Keep SBP&lt;130. Keep SBP&lt;120 Keep MAP&lt;100 Good cannula positioning Effect of inotropes Positive No effect No effect Use for RV support No effect Use for LV support and VAD weaning No effect</td>
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**Anesthetic management for VAD implantation**

For a more detailed discussion, see a selection of review articles [3, 9-11] in the References section.

**Preoperative considerations**

- Patients who are having an LVAD implanted have, by definition, severely compromised LV function. The fact that the patient is presenting for VAD implantation suggests that a further decompensation has occurred. Elicit possible causes of such decompensation: pneumonia, sepsis, or cardiac events such as arrhythmia and ischemia.
- Serum electrolytes can be abnormal due to alterations in the renin-angiotensin-aldosterone axis or chronic diuretic therapy.
- There is frequently elevated pulmonary vascular resistance, impaired RV function, impaired hepatic function and/or coagulopathy, renal insufficiency and impaired responsiveness to catecholamines.
- Patients may be on multiple inotropes, IABP, and/or a ventilator.
- Make sure that blood is available (packed red blood cells, FFP, and frequently also platelets may be required).
- Check and record the PA pressure, pulmonary vascular resistance and cardiac output. If available, obtain cardiac catheterization data, paying special attention to the transpulmonary gradient, PVR and response to pulmonary vasodilators.
- Document the latest echo report and note any evidence of RV failure, tricuspid regurgitation and/or pulmonary hypertension.
- Check to make sure that antibiotics have been given (e.g. vancomycin 1 gm and cefuroxime 1.5 gm). Check with local protocol whether antifungal prophylaxis should also be administered. Fungal infections are known to occur with VADs [12, 13].

Transport to OR
- Transport the patient to the operating room with full monitoring (EKG, arterial line, pulse oximeter) and with oxygen.
- Continue all inotropes; do not change the existing inotropic regimen.
- Do not administer any sedative or myocardial depressant before transporting the patient. If the patient is already on sedation, continue the same regimen.
- If transporting an intubated, ventilated patient, strive to maintain minute ventilation, FiO2 and PEEP requirements established in the ICU. Due to the extremely tenuous balance of these patients, seemingly small alterations may lead to significant instability.

**Lines and monitors**

- Large-bore IV access.
- Arterial line.
- TEE.
- Central venous catheter(s): #9 French introducer with a pulmonary artery catheter; PAC introducer and/or second CVC.
- Continuous cardiac output monitoring has been studied prospectively in 15 HeartMate® recipients [14]. The CCO system overestimated cardiac output by approximately 500 mL/min when compared with the LVAD-reported flow values. The use of CCO monitoring with a PAC is therefore not recommended where a reliable VAD flow reading is available (HeartMate®, Abiomed®).
- An oximetric PAC can be used if institutional CTICU protocol requires monitoring of mixed venous oxygen saturation.

**Intraoperative Management**

- If the patient has an internal cardioverter-defibrillator (ICD), then its defibrillator function needs to be turned off with a magnet to prevent inadvertent discharge.
- Induction should be gentle without significant myocardial depression. Hypotension is a serious risk during induction in this patient population. It should be helpful to keep in mind that any bolus of an agent intended to counteract hypotension will take significantly longer to take effect due to slow circulation time [15].
- Rapid sequence induction is NOT generally used, in order to minimize periods of apnea and the ensuing hypercarbia. The latter may increase PVR and further decrease left ventricular filling. Instead, a slow induction with cricoid pressure and ventilation is recommended [11].
- Administer adequate amnestics.
- Aprotinin is frequently used. Familiarize yourself with institutional and surgeon practice. As in other cardiac surgeries, for maximum safety it is suggested to administer the aprotinin test dose after heparinization and aortic cannulation, lest an anaphylactic reaction ensues that requires immediate institution of CPB.
- There is evidence to support vitamin K infusion IV throughout the case at 2 mg/hour for reduction of bleeding [16].
- An ICU ventilator may be required for NO administration. Check with the perfusion department or whoever manages the NO equipment at your institution and make adequate preparations.
- Full systemic heparinization is mandatory. Suggested initial dose is 300 units/kg. Maintain ACT about 4 x baseline.
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