Abstract

Ventricular assist devices (VAD) represent a revolution for the management of severe heart failure. Their insertion requires the use of cardiopulmonary bypass. They are used either permanently for long term treatment of refractory heart failure or temporally as a bridge until cardiac transplantation and until cardiac recovery from reversible cardiomyopathy. Insertion of VAD is a risky surgery with high incidence of complication such as bleeding, cardiac tamponade, renal failure and device failure. Anesthetic management of patients with heart failure undergoing VAD insertion requires full review of the patient critical condition, understanding VAD physiology, extensive hemodynamic monitoring and a harmony between cardiac anesthesia and surgical teams. Marinating and protection the right ventricular function is highly important for the continuation of VAD function. The aim of this review is to put new insights on anesthetic management of VAD insertion and to show their different types and their physiology.

Keywords: Heart Failure; Ventricular assist devices; Anesthesia; Insertion

Introduction

Heart failure (HF) is a chronic disease with progressive deterioration occurring over years or decades. The increase in the incidence of HF can be attributed to high survival after cardiac insults like myocardial infarction, improvement of drug treatment of circulatory disorders, and population aging [1]. Although the great advances in medical therapy and management, HF still is a leading cause of high incidence of hospitalization, readmission and mortality [2].

Stages of Heart Failure

The American College of Cardiology and American Heart Association have designed a conceptual classification to help understanding the disease progression in four stages. Stage A represents a patient who are at high risk for HF but has no organic heart disease or symptoms of HF, for example, patients with hypertension, atherosclerosis disease, or diabetes mellitus. Stage B includes patients with organic heart disease but without overt clinical presentation, such as myocardial infarction and left ventricular remodeling. Stage C are those patients who have organic heart disease with previous or current symptoms of HF, such as shortness of breath and fatigue. Stage D represents patients with end-stage HF and sever symptoms at rest despite maximal medical therapy [3,4].

In stage D, different management approach should be employed including end-of-life care, heart transplant, permanent circulatory mechanical support, or drugs [5]. Medical treatment for chronic heart failure includes angiotensin-converting enzyme inhibitors (ACEIs), beta blockers, proper use of diuretics to relieve volume overload, and digoxin which may help to improve resistant symptoms and reduce hospital, anesthesia and surgical readmission rates. For all patients with wide QRS more than 150 milliseconds, biventricular pacing should be considered and for some patients with QRS of 120 to 150 milliseconds according the condition.

More recently, sinus-node inhibitor ivabradine, and LCZ696, which combines angiotensin II inhibition with a neprilysin inhibitor, have been demonstrated to hold promise for HF patients [6-9]. However for patients with end-stage heart failure the only effective treatment is surgical intervention. Heart transplant is the gold...
standard for treatment of end-stage HF. The left ventricular assist device is an increasingly used option for patients in Stage D HF to prolong life in lieu of heart transplant. Ventricular assist devices (VADs) are mechanical systems that reduce the workload of the heart, permitting the ventricle to rest, whilst maintaining cardiac output and perfusion of vital organs. They have subsequently gained popularity in both acute and chronic heart failure as potentially life-saving treatment modalities [10].

**Development of INTERMACS**

The Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) was designed to facilitate communication with other colleagues regarding any patient with failing response to optimal medical therapy and needs further discussion of possible implantation of mechanical circulatory support devices (MCDs). Also is used for adjustment of pre-operative risk and clarification of target populations for future devices. INTERMACS has been acquiring data on most patients with implanted MCDs under the sponsorship of the National Heart, Lung, and Blood Institute (NHLBI) [11]. These profiles should facilitate collection of outcome data, help to address the varied needs of advanced heart failure patients and hoped to increase clarity of clinical profiling which will illuminate the progress of other surgery, other devices and all treatments for the advanced stages of heart failure [12,13].

**Table 1: INTERMACS profile.**

| INTERMACS profile descriptions                                                                 | Time frame for intervention                                      |
|------------------------------------------------------------------------------------------------|------------------------------------------------------------------|
| Profile 1: Critical cardiogenic shock Patients with life-threatening hypotension despite rapidly escalating inotropic support. | Intervention needed within hours.                                |
| Profile 2: Progressive declining function despite intravenous inotropic support.                  | Intervention needed within few days.                             |
| Profile 3: Stable but inotrope dependent Patient with stable blood pressure, organ function, nutrition, and symptoms on continuous intravenous inotropic support. | Intervention elective over a period of weeks to few months.     |
| Profile 4: Patient can be stabilized close to normal volume status but experiences daily symptoms of congestion at rest or during ADL. | Intervention elective over period of weeks to few months.       |
| Profile 5: Comfortable at rest and with ADL but unable to engage in any other activity, living predominantly within the house. | Variable urgency, depends upon organ function, and activity.     |
| Profile 6: Patient without evidence of fluid overload is comfortable at rest, and with activities of daily living and minor activities outside the house but fatigues after the first few minutes of any meaningful activity. | Variable, depends upon organ function, and activity level.       |
| Profile 7: This level includes patients who are without current or recent episodes of unstable fluid balance, living comfortably with meaningful activity limited to mild physical exertion. | Transplantation or circulatory support may not currently be indicated. |

**Modifiers for Profiles**

- Possible Profiles to Modify TCS: Temporary Circulatory Support can modify only patients in hospital includes IABP, ECMO, Impella. Possible Profiles to Modify 1, 2, 3 in hospital.
- A-Arrhythmia: can modify any profile. Recurrent ventricular tachyarrhythmias that have recently contributed substantially to clinical compromise. Any profile.
- FF-Frequent Flyer: can modify only outpatients, designating a patient requiring frequent emergency visits or hospitalizations for diuretics, ultrafiltration, or temporary intravenous vasoactive therapy. 3 if at home, 4, 5, 6. A frequent flyer would rarely be profile 7.

**History and classification**

**First-generation devices**

These mimic normal cardiac function and circulation by providing pulsatile blood flow at physiological rates. The energy source used to eject blood from the pumping chamber may be pneumatic, hydraulic, or by mechanical pusher plate [14,15]. These mechanisms produce audible pump operation. Durability beyond 2 years is rare and operations for device exchange carry high risk (Figure 1).

![First-generation devices.](image1.png)

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Second-generation devices

Flow is continuous and non-pulsatile, which reduces the pump size and the need for external venting. The term second generation describes rotary pumps, typically with axial blood flow, which have an internal rotor within the blood path that is suspended by contact bearings. These bearings are generally mechanical (ball, ruby, or fluid design) [16,17]. Spinning of the internal rotor and flow generation is by magnetic coupling of the rotor magnet to an external rotor (Figure 2).

![Figure 2: Second-generation devices.](image)

Third-generation devices

These rotary pumps have non-contact bearings and utilize centrifugal blood flow, incorporating either magnetic or hydrodynamic levitation of an internal impeller. Impeller rotation achieves blood flow through magnetic coupling to the pump motor [18,19]. Greater blood flow can be achieved around the impeller, which reduces thrombus formation and the intensity of antithrombotic therapy required (Figure 3).

![Figure 3: Third-generation devices.](image)

The following selected definitions are important to the understanding of how mechanical support devices operate:

- RPM: the revolutions per minute (RPMs), which determine pump flow.

- Flow: the continuous flow from the LVAD is created by a spinning impeller, which generates forward flow. The device flow = Rotor speed/(Pump inflow – Pump outflow).

- Pump power: LVAD pump power is a measure of the current and voltage applied to the motor and varies directly with pump speed and flow.

- Pulsatility index: (PI) corresponds to the magnitude of flow pulse through the pump [20,21].

**Indications for a Left Ventricular Assist Device**

**Strong indications:** Bridge to transplant, destination or bridge to recovery [22]. All must apply

- a. NYHA IV for 60–90 days.

- b. Maximal tolerated medical therapy and CRT/ICD if indicated.

- c. Chronic inotrope dependence.
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Support. Neurological assessment is documented, via transient mean pulmonary artery pressures <25 mm Hg, and low inotropic support. Neurological assessment is documented, via transient mean pulmonary artery pressures <25 mm Hg, and low inotropic support.

**Contraindications for a Left Ventricular Assist Device**

Some may be relative, especially as technology improves [24].

- Acute cardiogenic shock or arrest with uncertain neurologic status.
- Irreversible contraindication to heart transplant if destination or recovery is not the aim.
- Non-systolic HF.
- Co-existing illness with life expectancy < 2 years.
- Terminal severe comorbidity, e.g. metastatic or advanced cancer, severe liver disease or severe lung disease.
- Active uncontrolled systemic infection or significant risk of infection.
- Active severe bleeding.
- Right HF not secondary to left HF.
- Moderate or severe aortic insufficiency that will not be corrected.
- Anatomical considerations such as hypertrophic cardiomyopathy, large ventricular septal defect.
- Psychosocial limitations, e.g. inability to comply with medical regimen or device and driveline maintenance or inability of patient or companion to maintain LVAD operation and interpret alarms [25].

**Indications to enable heart transplant. Either must apply**

- Moderate or severe aortic insufficiency that will not be corrected.
- Anatomical considerations such as hypertrophic cardiomyopathy, large ventricular septal defect.

**Anesthesia for VAD Insertion**

**Preoperative considerations**

Patient optimization targets platelet count >150 x 10^3 mm^-1, serum albumin >33 gm litre^-1, normal liver enzyme levels, estimated glomerular filtration rate > 50 ml kg^-1 min^-1, hematocrit >34%, mean pulmonary artery pressures <25 mm Hg, and low inotropic support. Neurological assessment is documented, via transient

cessation of sedation if the patient is intubated. Premedication is avoided to prevent cardiac or respiratory depression [26].

**Monitoring and vascular access**

Standard cardiac monitoring, including five-channel electrocardiography, is used. Existing I.V. access should be changed if there is an infection risk and lines sent for culture. Central venous access is mandatory, both for pulmonary artery catheterization and fluid resuscitation, with meticulous attention to sterility. Mixed venous oxygen saturation assesses the adequacy of cardiac output, aiming for >65%. Transesophageal echocardiography (TEE) is used throughout the perioperative and immediate postoperative period. Assessment of coagulation via thromboelastography (TEG) provides a baseline to compare with after operation [6].

**Induction and maintenance**

Induction must prevent haemodynamic decompensation from reduced preload and contractility and is best performed inside the operating theatre with titrated increments of induction agents and inotropic support. Inhalation or I.V. anesthesia is suitable, but nitrous oxide best avoided. External defibrillation pads are attached. Antibiotic prophylaxis is customized to the institution’s pathogenic flora and includes broad-spectrum gram positive bacterial and anti-fungal cover. Body warming devices are used to maintain normothermia.

Surgical access is commonly via median sternotomy, but left thoracotomy with lung isolation via a double-lumen endotracheal tube is an option. Bleeding is common so aprotinin anti-fibrinolysis and cell salvage are routinely used. Full heparinization is required, irrespective of preoperative coagulation [27].

**Perioperative period**

TEE is used to exclude valve lesions, shunts and intracardiac thrombi as discussed above. CPB is generally but not universally used for placement of inflow cannulae within the ventricle, but duration can be minimized by tunnelling the percutaneous lead and completing the outflow cannula anastomosis before instituting bypass.

Inflow cannulae are directed posteriorly towards the mitral valve to prevent obstruction. Before VAD activation, all air should be removed, and the heart filled with blood. Deairing is via backflow of blood from the aorta to a Luer lock connection on the outflow cap and by elevating the LV apex. The VAD is initiated at low speed with a cross-clamp on the outflow cannula and a needle vent. CPB flow is then reduced followed by removal of the graft cross-clamp. The LV should be full and off CPB before the pump speeds are increased to avoid entraining air into the system, which is further minimized by flooding the surgical field with saline or carbon dioxide [6].

**Weaning from CPB**

Atelectatic lung is expanded before weaning bypass to prevent Valsalva-induced hypotension. CPB weaning concerns are from...
RV failure, pulmonary hypertension, systemic vasodilatation, and bleeding. TEE is vital for deairing, RV assessment, and to check cannulae positions and flow [28]. RV dysfunction can be difficult to predict, thus monitoring and prophylaxis against right heart failure must be instituted in all cases before separation from bypass, using inhaled nitric oxide, I.V. nitrates and phosphodiesterase inhibitors. RV dysfunction results in chamber dilatation and leftward ventricular septal deviation on TEE, predicting imminent failure. Atrial or AV sequential pacing is useful to maintain adequate cardiac output. Protamine must be used to reverse heparinization but can worsen RV function and cause pulmonary hypertension [29].

Potential Complications Associated with LVAD

Thrombosis

Patients with LVADs have an increased risk of thrombosis because the LVAD is foreign material in constant contact with the bloodstream. Patients are maintained on anticoagulation (usually warfarin) and antiplatelet therapy (usually aspirin). Patients who develop a thrombus may present with signs of HF. In the case of thrombosis, additional medical treatment such as heparin, glycoprotein 2b/3a antagonists, or tissue plasminogen activator may be necessary [30,31].

Infection

The current LVAD design is not a completely closed system as there is an open driveline exit. Trauma to this percutaneous exit site is a common cause for infection. The patient's condition can rapidly progress to sepsis and/or shock. Treatment involves establishing hemodynamic stability. Blood culture and imaging tests are necessary to evaluate for internal abscess or vegetation in or around the device. The patient should be treated with appropriate antibiotics and may require long term suppressive antibiotic therapy [32,33].

Gastrointestinal bleeding

The use of anticoagulation and antiplatelet therapy increases risk of bleeding, particularly in the gastrointestinal tract and brain. Also, chronic anticoagulation can lead to platelet dysfunction or acquired Von Willebrand disease. In severe cases, patients will demonstrate hypotension, hypovolemic shock, and rectal bleeding [34,35].

Stroke

Patients with LVADs are at increased risk for both ischemic and hemorrhagic stroke. Acute ischemic strokes result from thromboembolic events due to pump thrombosis, subtherapeutic anticoagulation, or a prothrombotic state associated with activation of the immune system. Alternatively, patients on long-term anticoagulation are at increased risk for hemorrhagic stroke. Hemorrhagic stroke can occur in these patients due to hemorrhagic transformation of an ischemic stroke, over anticoagulation, or infection [36].

Right HF

After LVAD implantation, there is increased output from the left ventricle that can lead to right ventricle failure (RVF). An echocardiogram and laboratory testing for liver enzymes are indicated if RVF is suspected. It can be treated with diuretics and modification of LVAD parameters. In severe cases, mechanical support of the right ventricle may be required with a biventricular assist device [37,38].

Ventricular dysrhythmias

LVAD-induced ventricular dysrhythmias can occur if the patient has a preload deficiency with decreased filling of the left ventricle due to hypovolemia. With diminished left ventricular filling, the inflow cannula can become drawn down into the cardiac ventricular tissue. The cannula's contact with the cardiac tissue can irritate the area and trigger a ventricular dysrhythmia. These dysrhythmias tend to be short and are repeated until the hypovolemia is corrected. Ventricular dysrhythmias can lead to cardiac arrest. Advanced cardiac life support measures that include chest compressions can be used in patients with LVADs [39].

The future

Design improvements in current VAD systems include totally implantable devices with transcutaneous energy transmission systems, which eliminate the need for a driveline insertion site and also designs allowing more acceptable side-effect profiles for thromboembolism, infection, and device failure.

In addition, there are two total artificial hearts in clinical use, which involve explantation of the native heart and replacement with the device. These are the CardioWest total artificial heart (Syncardia Inc.) and AbioCor (Abiomed Corporation).

The latter organization also produces the Impella, one of a group of minimally invasive, catheter-based assist devices, which is inserted via the femoral artery, the tip of which crosses the aortic valve to rest in the LV and drive forward flow (Figure 4).

Finally, with more devices in production, there is a need for regulation. At present, all devices are regulated in accordance with rules set out by the INTERMACS controlling use in the USA.
Conclusion

Perioperative anesthetic management of patients undergoing VAD implantation represent a challenging mission for the cardiac anesthesia team. This require full cooperation between surgical and anesthetic teams. Extensive hemodynamic monitoring and cardiopulmonary bypass support are essential.

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Conflict of Interest

No conflict of interest.

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