

# Haemodynamic management of patients with left ventricular assist devices using echocardiography: the essentials

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Mechanical circulatory support with continuous-flow left ventricular assist devices (LVADs) has emerged as a viable treatment modality for patients with advanced heart failure. LVAD support results in unique haemodynamic and echocardiographic alterations that must be understood to provide optimal care for these patients. In this review, we propose essential echocardiographic and haemodynamic elements for the assessment of optimal LVAD function based on the literature and the use of simulation software. A key element of LVAD physiology remains the interaction between an unloaded left ventricle and a loaded right ventricle. The echocardiographic assessment and treatment of the pathophysiology of the right-sided part of the heart remains critical to maintaining optimal LVAD support.

## Keywords

ventricular assist device • mechanical circulatory support • echocardiography

## Introduction

Use of a left ventricular assist device (LVAD) has become an important option for the treatment of patients with advanced heart failure refractory to medical therapy. It is estimated that more than 5000 pumps are implanted annually worldwide.<sup>1</sup> This trend of increasing LVAD therapy was facilitated by the introduction of these devices as a bridge to transplantation and as destination therapy for transplant-ineligible patients. The role of cardiologists is expected to become even more important in the management of patients with LVADs, and optimal haemodynamic management in these patients requires good understanding of both LVAD physiology and the related echocardiographic measurements.

## Goal of LVAD therapy

Modern LVADs are small devices implanted through the apex in the left ventricle (LV) where they unload the LV and pump blood into the aorta, creating a continuous systemic blood flow. LVAD physiology can best be described as the optimal loading balance between the right and left Ventricle during Assist Device support (LVAD). This

balance leads to increased circulatory output and improved organ perfusion.

## Anatomy of the LVAD

The most commonly used continuous-flow LVADs in Europe are HeartWare (HeartWare, Inc., Framingham, MA, USA), HeartMate 2, and HeartMate 3 (St. Jude Medical Inc., St. Paul, MN, USA).

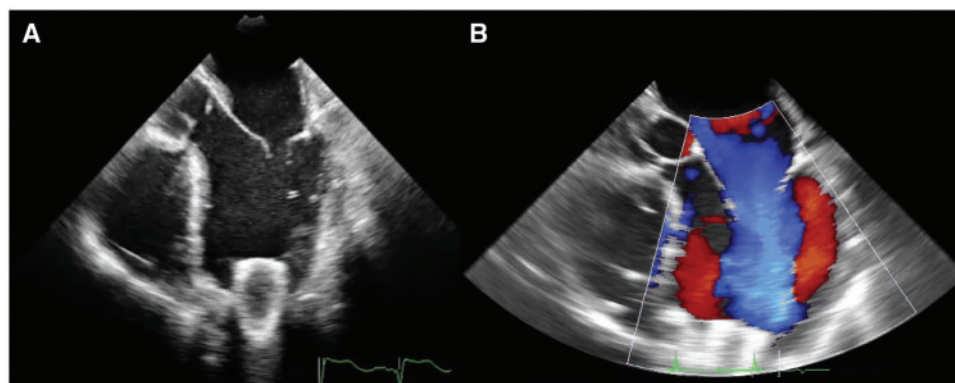
The anatomy of the LVAD apparatus consists of the device and the surroundings including the LV, right ventricle (RV), and the neighbouring valves, especially the aortic valve (AV). The complete apparatus should be considered a multilayer system with the LVAD in the centre surrounded by the LV, with the RV as the outer layer. The latter is very important because the RV is the preload source for the LV and the flow-limiting pump for the system.

## Left ventricular assist device

Current continuous-flow LVADs have three components: the LV inflow cannula inserted into the LV apex, the outflow cannula anastomosed to the ascending aorta, and the impeller implanted in the pericardial sac above the diaphragm.

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**Figure 1** (A) Central position of the apical or inflow cannula inserted into the apex of the LV and directed toward the MV. (B) Colour Doppler flow interrogation of the apical cannula showing laminar flow.

### Inflow cannula

The inflow or apical cannula is implanted through the apex of the LV and should be directed toward the mitral valve (MV). The position of the cannula can best be assessed using echocardiography in the mid-oesophageal four-chamber (ME-4C) view (Figure 1). When using colour Doppler flow, the flow should be laminar toward the inflow cannula. Pulsed-wave Doppler (PWD) interrogation of the inflow cannula should reveal two velocities: a low diastolic and a higher systolic velocity.<sup>2,3</sup> The dominant systolic velocity should be below 1.5 m/s (Figure 2A), and higher inflow velocities can indicate flow obstruction. The presence of a diastolic velocity component is necessary for good functioning in continuous-flow LVAD. Very low systolic velocities and an almost absent diastolic velocity during optimal rotational speed settings are considered abnormal, and immediate further evaluation is warranted.

In addition to the LVAD type, brand-specific characteristics influence the Doppler signal. Doppler evaluation of both the inflow pattern of the apical cannula and the mitral inflow pattern is limited in patients supported by the HeartWare device (Figure 2B). The artefact specific to the HeartWare device is called the 'waterfall' artefact when using colour Doppler flow or PWD.<sup>4</sup> With the HeartMate 3, the PWD pattern shows not only the systolic–diastolic inflow variation but also intermittent changes in flow (Figure 2C). The HeartMate 3 device is programmed to change rotor speeds every 2 s to generate a pulsatile effect and to avoid possible thrombus formation in the system.

### Outflow cannula

The outflow cannula is grafted on the ascending aorta, and the distal part of the outflow graft can be assessed using echocardiography in the mid-oesophageal long-axis (ME-LAX) view in almost every patient. Colour Doppler flow can be used to find the exact location of the outflow graft and to align for the use of continuous-wave Doppler (CWD).<sup>2,3</sup> CWD interrogation should also reveal diastolic and systolic velocities during optimal unloading of the LV, but with a prominent diastolic velocity (Figure 3). CWD-derived velocities may vary not only with pump speed but also with changes in preload. If only systolic velocities are detected, the rotational speed of the

device is probably too low, and the unloading of the LV is insufficient. If only diastolic velocities are observed, the rotational speed is likely set too high for the preload present at the time. An outflow graft peak systolic velocity >2 m/s may be abnormal and might warrant further investigation.<sup>2,3</sup>

The systolic–diastolic variation in the outflow Doppler pattern is somewhat confusing because of the continuous-flow system. Currently, automated blood pressure monitors and/or Doppler can be effective for blood pressure measurements for the majority of LVAD patients. The mean arterial pressure is usually measured when these techniques are used.<sup>5</sup>

## The LVAD surroundings

### The left ventricle

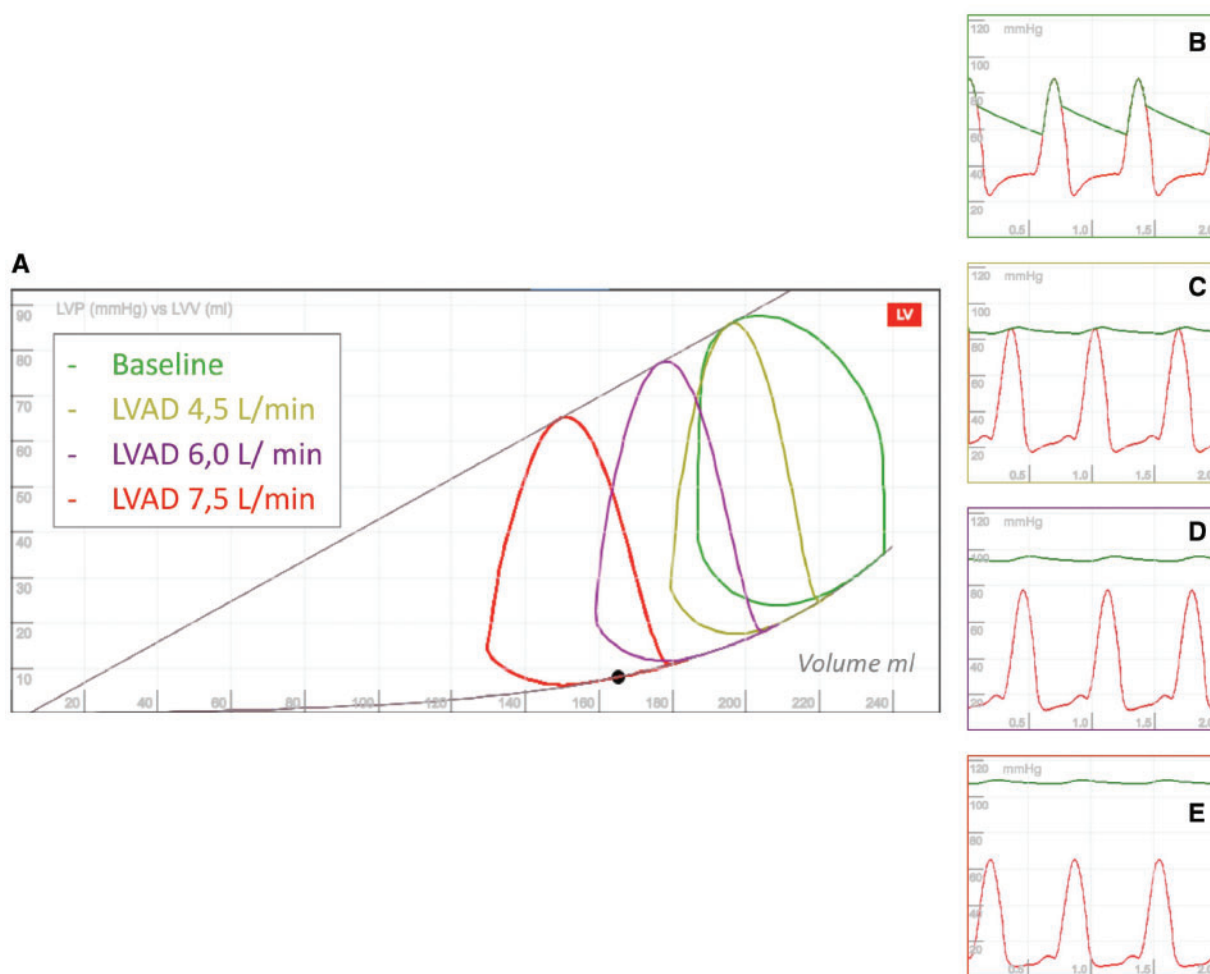
The primary goal of LVAD therapy is to unload the LV and support circulatory blood flow. Optimal circulatory output requires both adequate LV preload and unloading. During LVAD support, the peak LV pressures and left atrial pressures decrease together with preload reduction of the LV. Concurrently, the pressure in the aorta will rise, leading to an LV–aortic uncoupling of pressures<sup>6</sup> (Figure 4).

Effective unloading will reduce the LV size and can be assessed using the ME-4C view. It is critical to evaluate the position of the interventricular septum, which should be positioned in the middle during optimal LV unloading. Inadequate unloading will cause the septum to remain shifted to the right. Excessive unloading will shift the septum to the left and impede right ventricular function (Figure 5). Unloading of the LV is not only determined by the rotational speed of the system but also by the systemic vascular resistance (SVR), RV function, pulmonary arterial pressure, and the presence of tricuspid regurgitation (TR). Reducing SVR while increasing rotations can lead to suction events since both act synergistically to unload the LV

$$\text{LV unloading} = \frac{\text{Rotor speed} \times \text{PVR} \times \text{TR}}{\text{Preload} \times \text{SVR} \times \text{RVC}}$$

Preload, LV volemia; PVR, pulmonary vascular resistance; TR, Tricuspid regurgitation; SVR, systemic vascular resistance; RVC, right ventricular contractility (Figure 6).





**Figure 4** (A) LV-aortic uncoupling during LVAD support. Corresponding LV and aortic pressure waveforms at baseline (B), 4.5 L/min (C), 6.0 L/min (D), and 7.5 L/min (E). With increased flow, the degree of LV unloading and uncoupling between aortic and peak LV pressure generation is greater (Harvi Software by Burkhoff).<sup>6</sup>

### Mitral valve

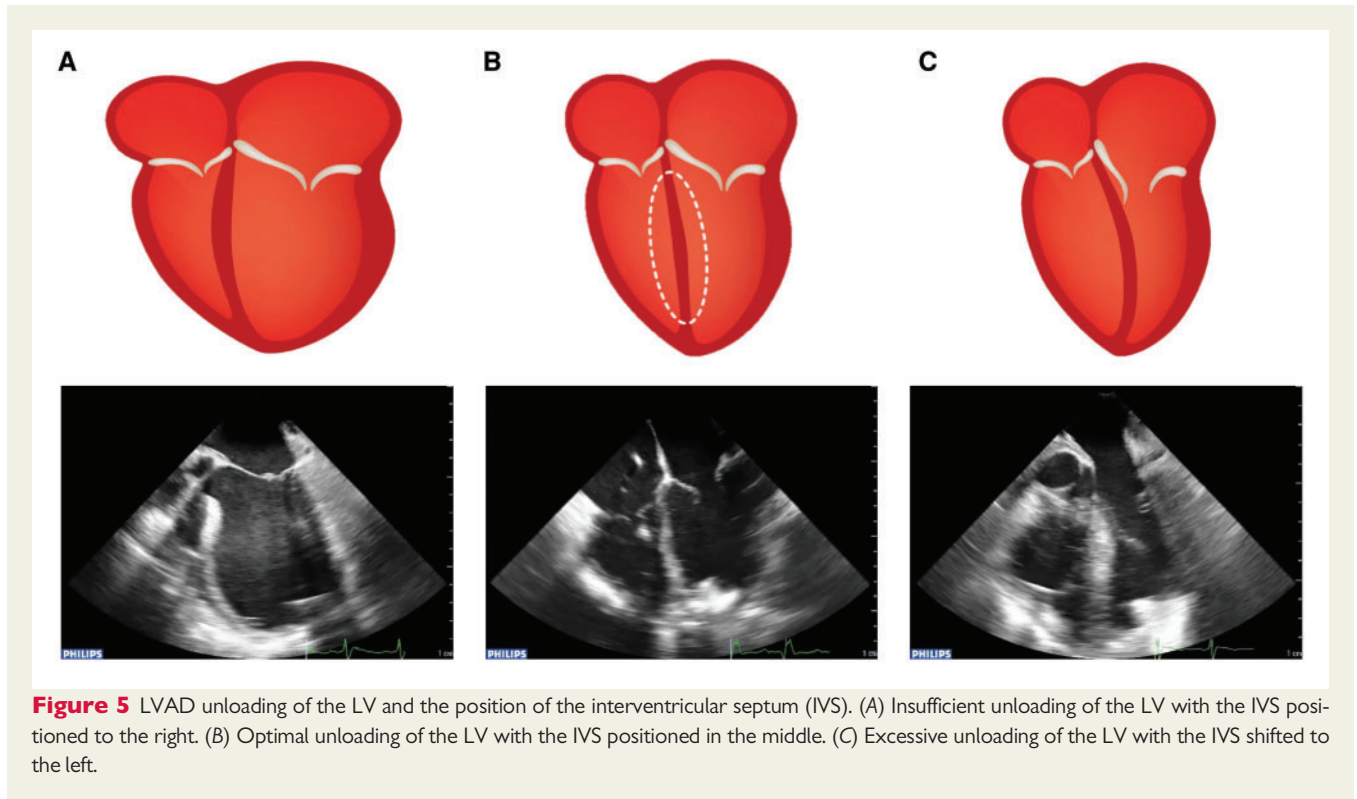
Mitral regurgitation (MR) is often present in dilated and dysfunctional LV planned for LVAD implantation. LV unloading during LVAD support generally leads to LV size reduction and improves MV coaptation by also reducing the mitral annular size. Therefore, MR usually disappears completely during adequate unloading. The presence of persistent rather than mild MR might indicate insufficient unloading of the LV (Figure 7).

### Aortic valve

During evaluation of the outflow cannula, the AV should be assessed for the presence of aortic regurgitation (AR). The valve should also be examined for opening during LVAD support.<sup>2,3</sup> The valve can remain consistently closed during LVAD support, might open intermittently, or might open with every beat. Ideally, LVAD support should be aimed at opening the AV every two or three beats. Opening of the valve with every beat suggests insufficient unloading in most circumstances, whereas a consistently closed valve often suggests

excessive unloading (LVAD unloading for the preload present at the time). AR, AV commissural fusion, and leaflet deterioration will develop more frequently during the course of LVAD support if the AV is consistently closed.<sup>7</sup> Using PWD, the LVAD outflow cannula diastolic acceleration is obtained by measuring the diastolic slope from the onset to the end of diastole. The systolic-to-diastolic peak velocity ratio (S/D ratio) can be obtained by dividing the peak systolic velocity by the end-diastolic peak velocity. An increase in diastolic acceleration and a decrease in the S/D ratio enable the detection of aortic regurgitant flow sooner than conventional echocardiographic parameters.<sup>8</sup>

The presence of AR will reduce the effective forward LVAD flow and, if severe, will compromise LV unloading. This phenomenon is referred to as valve ventricular assist device re-entry or simply 'valve-VAD re-entry' (Figure 8). If needed, the valve can either be replaced or repaired using a simple central coaptation stitch (Park stitch),<sup>9</sup> which not only eliminates the native AR but also allows partial opening of the valve. The valve can also be closed definitively, but this



**Figure 5** LVAD unloading of the LV and the position of the interventricular septum (IVS). (A) Insufficient unloading of the LV with the IVS positioned to the right. (B) Optimal unloading of the LV with the IVS positioned in the middle. (C) Excessive unloading of the LV with the IVS shifted to the left.

makes native LV output impossible in the event of LVAD failure. Interestingly, in approximately 25% of patients, mild to moderate AR will develop within 1 year of implantation.<sup>10–12</sup>

## The key to success is the right side of the heart

### Right ventricle

The most important determinant for successful LVAD support is a working RV because the RV is the flow-limiting pump for the system. The incidence of RV failure during LVAD support depends on the definition of RV failure and occurs in up to 40% of cases, of which 10–25% will even require mechanical RV support.<sup>13</sup>

LVAD support will unload the LV, but will also load the RV, and it is crucial to find an optimal balance between the loading conditions of both ventricles to generate an optimal circulatory output. The key element in LVAD therapy is the fact that the RV serves as the ‘pre-load donor’ for the LV. Most patients with RV dysfunction secondary to left heart disease will perform better after LVAD implantation.

Many patients will show some degree of RV dysfunction, which may lead to insufficient LV preload. Under physiologic conditions, up to one-third of RV stroke work is performed by septal contraction, and it has been estimated that up to 20–40% of RV systolic pressure results from LV contraction through the septum.<sup>14</sup> Changes in RV and septal geometry increase tricuspid annular size, which may lead to the onset or worsening of pre-existing TR and limit effective forward RV output.<sup>15</sup>

Pre-procedural prediction of RV failure remains difficult. Physicians often rely on clinical signs, haemodynamic parameters, and

echocardiographic assessment. Although many echocardiographic measurements have been proposed to assess and predict RV function after LVAD implantation, there is no echocardiographic ‘holy grail’.

First, the RV dimensions should be considered relative to the size of the LV. A ratio of right-to-left ventricular end-diastolic diameter ratio (RV/LV ratio) over 0.75 is a strong predictor of RV failure after LVAD implantation.<sup>16,17</sup>

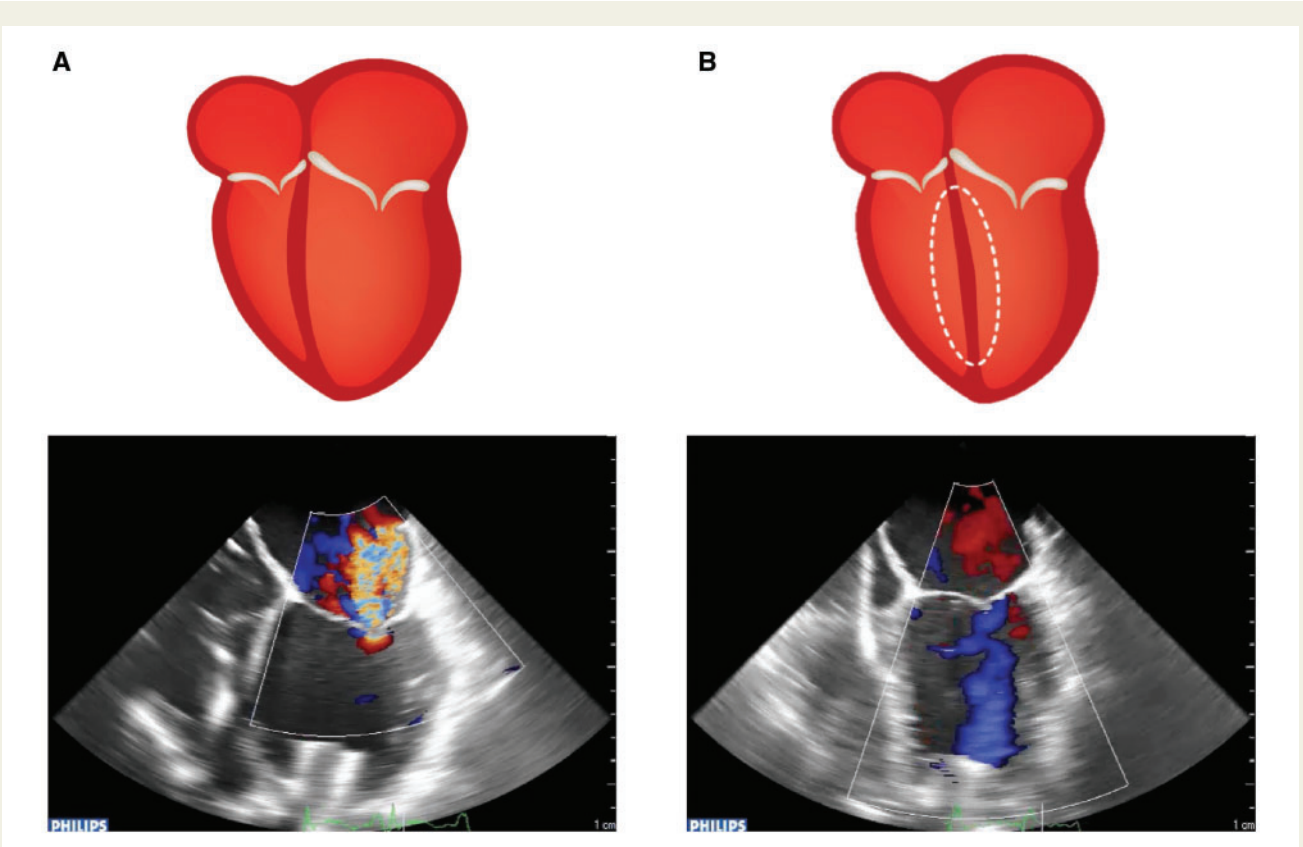
Next is the functional assessment of RV function for which the tricuspid annular plane systolic excursion (TAPSE) can be used. A normal TAPSE should be more than 15 mm, but a TAPSE of <7.5 definitely reflects poor RV function. The combination of both measurements with an RV/LV ratio of >0.75 and a TAPSE value below 7.5 mm highly predicts RV failure post-implantation (*Table 1*).

With poor RV function, the most significant predictor of outcome is a decreased timing interval between the onset and cessation of TR flow corrected for heart rate—this is a surrogate for early systolic equalization of RV and right atrial pressure.<sup>13</sup> In patients with severe RV dysfunction, the rate of isovolumic contraction (IVC) and relaxation (IVR) is already poor, and the gradient between the right atrial pressure and RV pressure will decline as the stroke volume decreases, resulting in a shorter IVC and IVR period. Patients with a corrected TR duration of more than 461 ms will perform better<sup>18</sup> (*Figure 9*).

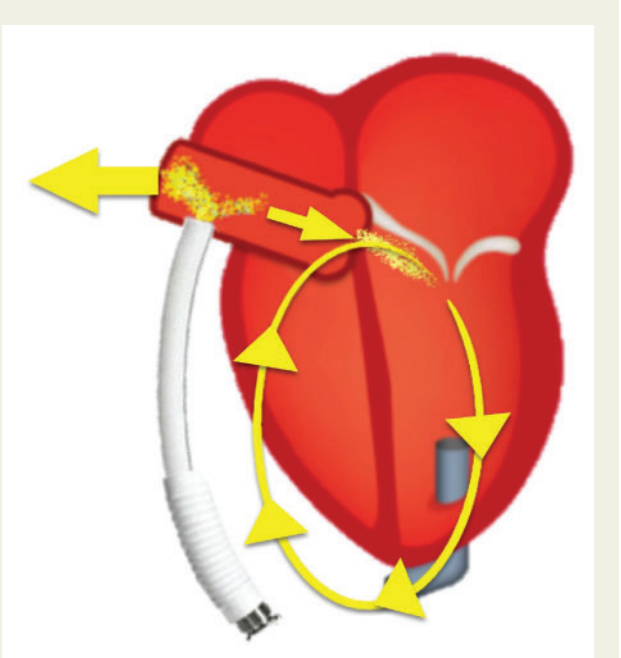
The three most important reasons for RV failure during LVAD support are:

- (1) Rotational speeds are too high for the LV preload present at the time.





**Figure 7** (A) ME-4C view showing insufficient unloading and greater than moderate mitral regurgitation (MR). (B) Optimal unloading of the LV without any MR.



**Figure 8** Mechanism of valve-VAD re-entry causing a decrease in systemic forward flow.

**Table 1** Evaluation of RV by combining the measurement of the ratio between RV and LV dimensions and TAPSE as a functional measure of RV function

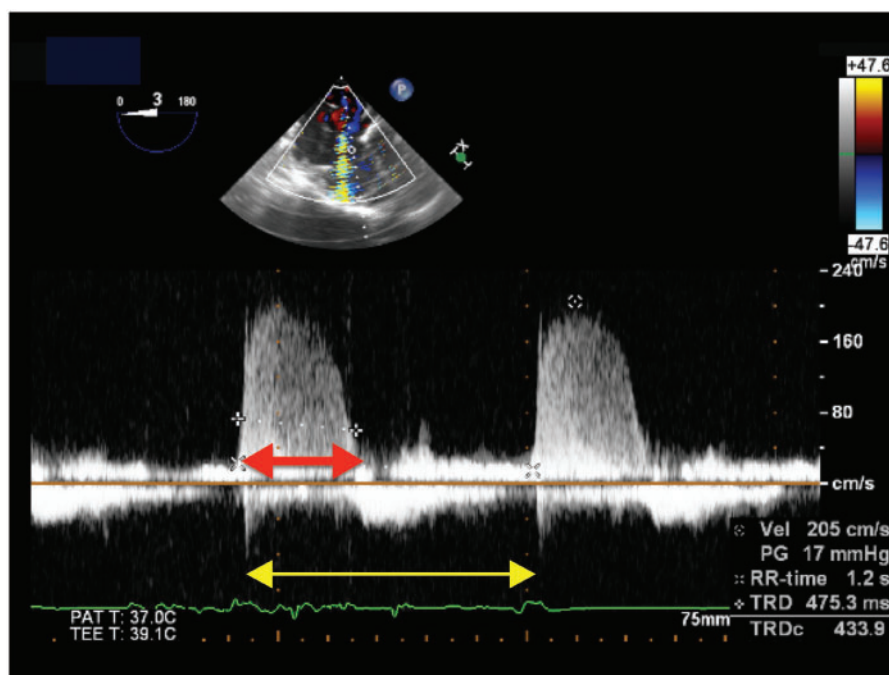
RV/LV ratio	<0.75	>0.75
TAPSE (mm)		
>7.5	Proceed	Caution
<7.5	Caution	RV support

- (2) Increased pulmonary vascular resistance (PVR), which not only impedes blood flow toward the LV but also increases RV afterload and wall tension.
- (3) Adequate RV performance requires adequate coronary perfusion.

If symptoms or signs of RV failure (central venous pressure > 18, wedge pressure < 18, and cardiac index < 2.0 L/min/m<sup>2</sup> in the absence of tamponade, ventricular arrhythmias, or pneumothorax) remain after optimal medical support, an RV assist device should be considered.<sup>19</sup>

**Tricuspid valve: TR**

Managing mild TR in patients planned for LVAD surgery is not straightforward. The presence of more than moderate TR will



**Figure 9** Measurement of the timing interval between the onset and the cessation of TR flow corrected for heart rate (TRDc) using CWD interrogation of the TR. Duration of TR (red arrow) vs. R–R interval (yellow arrow).<sup>15</sup>

impede forward RV flow together with a rise in right-sided filling pressures. The literature is confusing; whereas some reports suggest addressing the TR during the implantation procedure, others suggest leaving the TR with no benefit to tricuspid valve repair.<sup>20–24</sup>

When considering the effects of LVAD support on TR, it is clear that the unloading of the LV will reduce MR, left atrial pressure, pulmonary hypertension, and, to some extent, RV size.<sup>25,26</sup> All of these factors may worsen TR severity. A tricuspid annulus diameter >43 mm, measured in the ME-4C view, is associated with an increased risk for RV failure and 1-year mortality post-LVAD.<sup>27</sup>

Cardiovascular implantable electronic devices are frequently used in advanced heart failure and LVAD patients. The presence of wires through the TV can impede normal TV leaflet function, causing TR. About one-third of patients will develop more than moderate TR about 1.5 years after implantation.<sup>28–30</sup>

In the evaluation of TR, we must consider the fact that massive TR is often associated with a low Doppler jet velocity (<2 m/s) as there is near equalization of RV and right atrial pressures.<sup>25</sup> Moderate or severe TR by itself will reduce RV forward flow and lead to a moderately or severely reduced LV preload, respectively. Although the evidence is poor, greater than moderate TR should be considered for repair to optimize right ventricular function in LVAD patients, especially when associated with pulmonary hypertension and a dilated TV annulus.<sup>31,32</sup>

### Optimal unloading of the LV

During echocardiographic evaluation of optimal unloading of the LV, several parameters should be assessed (Figure 10):

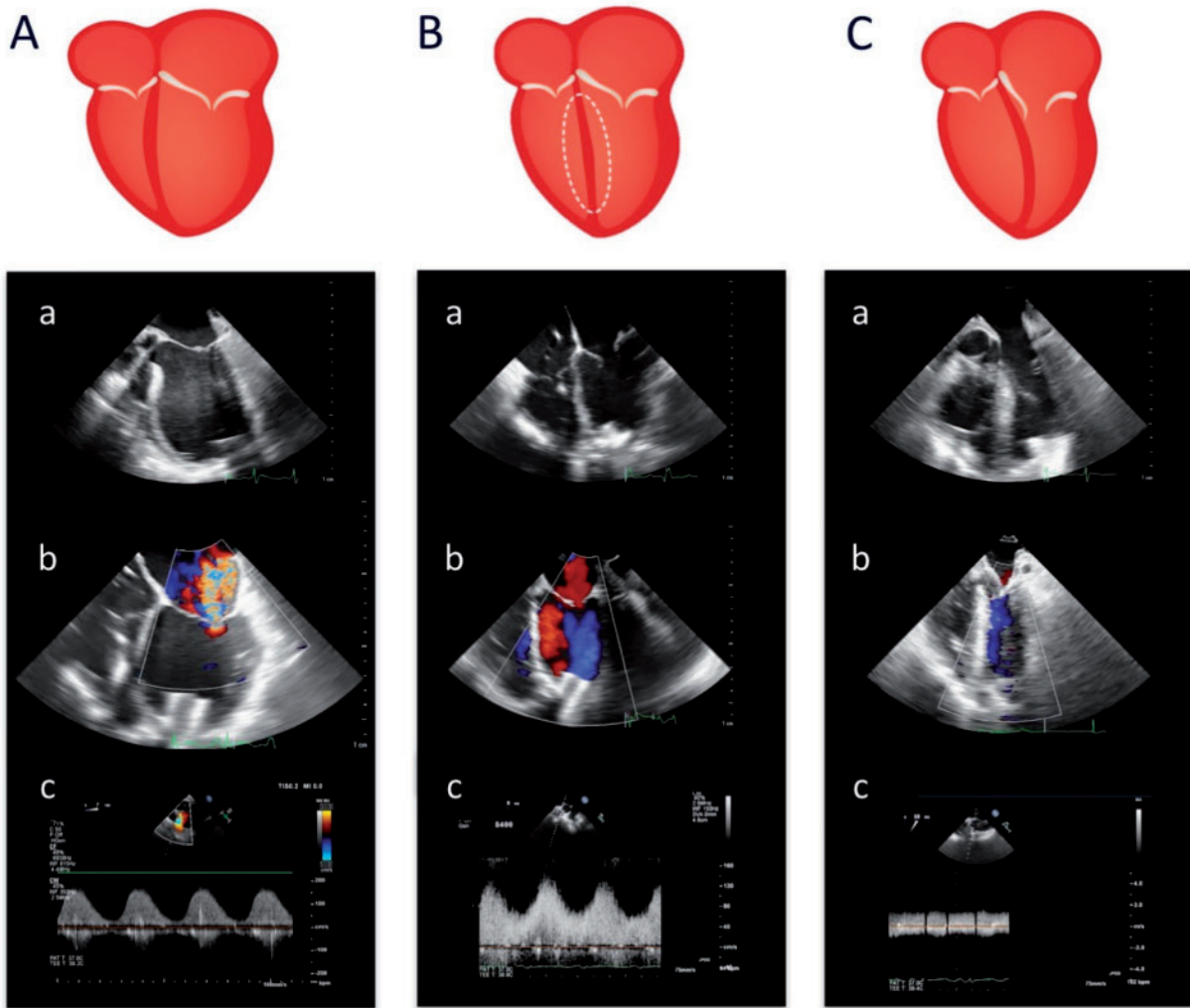
- (1) First, using the ME-4C view, the position of the interventricular septum should be positioned in the middle between the RV and the LV.
- (2) Second, the MR assessed by colour Doppler flow should almost disappear. The presence of more than mild MR reveals suboptimal unloading of the LV. The MV should open and close normally. If the MV does not close, excessive unloading is present.
- (3) Third, the AV needs to be assessed using the ME-LAX view. Optimally, the AV should open intermittently. More than mild AR leads to valve-VAD re-entry and compromises effective forward systemic flow.
- (4) Fourth, the outflow tract should be detected using colour Doppler flow, followed by CWD interrogation of the outflow jet. The Doppler pattern should ideally reveal both systolic and diastolic velocities. Only diastolic flow indicates excessive unloading for the preload present at the time.

### Common complications after LVAD implantation

Complications can be divided into early and late. Tamponade and RV failure are two major problems that impede proper LVAD function in the early post-operative period.

### Tamponade

Post-operative bleeding and tamponade are considered major complications after LVAD implantation. Up to 20% of patients supported by LVAD may develop tamponade requiring re-exploration.<sup>33,34</sup> Contrary to classic tamponade in which fluid accumulates more often on the right side of the heart, the LVAD physiology leads to lower pressures on the left side of the heart. This unique haemodynamic environment created by the LVAD may lead to pericardial fluid or



**Figure 10** Echocardiographic assessment of left ventricular (LV) unloading. (A) Insufficient unloading of the LV. (a) Enlarged LV with the IVS positioned to the right. (b) Colour Doppler flow showing severe MR. (c) CWD interrogation of the outflow tract revealing only systolic velocities. (B) Optimal unloading of the LV. (a) Smaller LV with the IVS positioned in the middle. (b) Absence of MR on colour Doppler flow. (c) CWD interrogation of the outflow tract showing both systolic and diastolic velocities. (C) Excessive unloading of the LV. (a) Empty LV with the IVS shifted to the left. (b) Mitral valve remains in open position. (c) CWD interrogation of the outflow tract revealing only diastolic velocities.

blood accumulation at the level of the left atrium or near the LV while the RV remains unaffected.<sup>35</sup>

### LVAD thrombosis

LVAD thrombosis is defined as the presence of a thrombus in the conduit or pump, severe haemolysis, and symptoms of severe heart failure. When LV decompression cannot occur, the blood is pushed again through the AV, resulting in a palpable pulse. Colour Doppler flow of the inflow cannula can show turbulent flow or even backward flow as blood returns from the aorta to the LV. The inflow velocities will often be increased, typically by more than 2 m/s. Outflow graft velocities may be decreased and/or appear to have peak velocity variation.<sup>2,36</sup>

### Arrhythmia

Many patients scheduled for LVAD implantation already have a cardioverter defibrillator (ICD) implanted, which requires perioperative testing and reprogramming. It is recommended to reprogramme the ICD to minimize treatments for non-sustained arrhythmias and prioritize painless therapies such as anti-tachycardia pacing over painful shock therapy.<sup>37,38</sup>

Ventricular arrhythmias occur in about one-third of patients during LVAD support. An arrhythmogenic substrate associated with the underlying cardiomyopathy, ventricular scar tissue, and electrolyte abnormalities may contribute to ventricular arrhythmias in patients with an LVAD.<sup>30</sup> Furthermore, suction events during excessive ventricular unloading can lead to ventricular arrhythmias. After the onset of ventricular fibrillation, the LVAD will often support the LV output

