Use of continuous flow (CF) left ventricular assist devices (LVAD) has resulted in increased survival, lower rates of complications, and greater stability than with previous pulsatile devices. As a result, CF devices predominate, and the total number of device implants has increased noticeably because patients can be supported for longer periods of time.1 Given the extended survival of patients supported with CF LVAD therapy, previously uncommon disorders are now being seen with increasing frequency. Development of LVAD-acquired aortic insufficiency (AI) has been reported in patients with long-term LVAD support as either a bridge to transplant or destination therapy.2,3 Based on extrapolation from cohort studies, the AI does not improve over time.2 Regurgitant blood flow from the LVAD outflow graft is recycled back into the LV, resulting in reduced forward cardiac output and thus end-organ malperfusion, increased LV preload, and increased LVAD pump work.4 Development of LVAD acquired AI is therefore a potential obstacle to successful long-term LVAD support.5

In this issue of the Journal, Imamura et al6 report that native aortic valve (AV) opening during LVAD support is profoundly associated with reversal of LV systolic function, especially in patients with shorter preoperative duration of heart failure. In patients whose native AV remains closed, the low pulsatility of axial flow pumps may facilitate aortic root remodeling and post-LVAD AI development, resulting in poor quality of life.6 Other cohort studies to date have sought to identify the baseline characteristics and serial correlates of LVAD-acquired AI. Its development has been associated with multiple baseline characteristics: device type (more likely with continuous than with pulsatile), larger baseline aortic root diameter, smaller body surface area, lower body mass index, female sex, and lower preoperative ejection fraction. Notably, none of the studies reported a significant association between the degree of baseline AI and future identification or progression of disease.2–4 The result that Imamura et al6 present could be an important predictor of LVAD-acquired AI after device implantation.

With CF LVAD, the AV is exposed to increased aortic diastolic pressures and increased pressure differential across the AV, known as the transvalvular pressure. In the presence of continuous transvalvular pressure overload, the radial length of the AV tends to expand over time, leading to remodeling and malcoaptation, annular deformation, and ultimately regurgitation. With prolonged valve closure, the leaflets are exposed to more stagnant blood flow, leading to hemolysis, thrombosis, fusion of the valve leaflets,8 and ultimately leading to poor

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**THERAPY OF LVAD-ACQUIRED AI**

| A. Non Invasive therapy |   |
|-------------------------|--|
| 1 Device management     | 2 Medical therapy |

When A-① ② are insufficient

| B. Invasive therapy (Redo open surgery) |   |
|---------------------------------------|--|
| 1 Valve replacement                   | 2 Valve repair |
|                                       | 3 Valve closure |

When B-① ② ③ are high risk

| C. Minimally Invasive therapy (Percutaneous transcatheter) |   |
|------------------------------------------------------------|--|
| 1 Valve closure                                           | 2 Valve implantation |

**Figure.** Therapies for LVAD-acquired AI (ISHLT 2013 guideline).11 AI, aortic insufficiency; LVAD, left ventricular assist device.
coaptation and greater AI. The retrograde flow from the outflow cannula, which is often positioned several centimeters away from the valve, disrupts the normal flow vortices and “washes out” the bulbous geometry of the aortic root, leading to thrombus formation. Under high-flow LVAD conditions, cardiac output is fully supported by the device propelling antegrade flow from the ventricle to the aorta, leaving the valve in a constant state of closure in the setting of an elevated transvalvular gradient, all of which contribute to LVAD-acquired AI.

Patients with LVAD-acquired AI have decreased exercise capacity compared with those without AI. The development of LVAD-acquired AI during prolonged LVAD support is also associated with a poor prognosis and increased adverse cardiac events (cerebral thrombosis, congestive heart failure, volume overload, and arrhythmia requiring readmission).2,3

Therapy of LVAD-Acquired AI (Figure)

A. Noninvasive Therapy

1. Device Management A ramped-speed study using echocardiography and hemodynamic data can directly determine the optimum pump speed for cardiac support in each patient. During the study, several variables are evaluated, including LV size, septal position, AV opening frequency, and blood pressure. Taken together, these variables help determine the optimal pump speed, which should be lowered to reduce transvalvular pressure gradient without compromising systemic perfusion and not result in further elevation of LV filling pressures and poor pump rotor washing. Jorde et al report that AV opening is only a major driver of the development of AI in hearts that have poorly optimized loading conditions.4 Loading conditions should be optimized during CF-LVAD support, and intermittent opening of the AV should be performed.5

2. Medical Therapy Flow of CF LVADs mainly causes diastolic pressure and flow increase. When the pump speed is increased, the systolic pressure remains relatively constant, while the diastolic pressure increases and the pulse pressure narrows. ISHLT guidelines recommend a mean arterial pressure goal ≥80mmHg in patients with CF LVADs.6 Maintaining optimal blood pressure will optimize cardiac support, decrease the incidence of stroke, and theoretically help to control the degree of AI.7

B. Invasive Therapy

In general, surgical correction should be considered for AI that is severe, symptomatic, and refractory to medical and/or device therapy. Options for surgical correction of LVAD-acquired AI are: bioprosthetic valve replacement, valve repair, or valve closure. Surgical valve closure is often complicated by recurrence of AI; operative mortality can be as high as 7%.8,9

C. Minimally Invasive Therapy

In patients with LVAD-acquired AI for whom redo sternotomy is contraindicated, minimally invasive percutaneous approaches provide an alternative with potential for functional recovery. Several centers have reported the use of percutaneous left ventricular outflow tract occluders to successfully treat severe AI in the immediate post-procedural period.10 Long-term implications of AV closure have yet to be determined, and device malfunction could result in immediate hemodynamic collapse and death. Transcatheter AV implantation has been reported as successful in providing relief of symptoms in LVAD-acquired AI.11 There is only minimal data on the use of percutaneous devices to repair LVAD-acquired AI, but this technique is expected to become the treatment of choice for significant LVAD-acquired AI once additional experience has been gained.

Conclusions

There are various therapies for the treatment of LVAD-acquired AI, ranging from noninvasive treatment, minimally invasive treatment, and invasive treatment. A major goal is to reduce further the risks associated with invasive and minimally invasive treatments of LVAD-acquired AI. For that purpose, it is necessary to identify pre-implantation those patients who will inevitably develop LVAD-acquired AI, and then perform AV surgery concurrently with LVAD implantation only on those patients. Therefore, more studies are needed to determine the predictors of LVAD-acquired AI before LVAD implantation.

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