We propose a Novel Left Ventricular Assist System® (Novel LVAS®) as a bridge to cardiac transplantation and to functional heart recovery in advanced heart failure. This report regards the principles that led to its development. It is our hope that the design of a high-peak-output pump of smaller size will lead to improved functional capacity, when compared with currently available left ventricular assist bridges to heart recovery.

Several basic considerations went into the design of this system: 1) we did not want to cannulate the heart chambers; 2) in particular, we rejected the use of a left ventricular apical cannula for myocardial recovery, because it destroys the helical anatomy of the chamber; 3) we chose an atriotomy for blood inflow to the implanted pump; and 4) we synchronized the pump to the patient’s electrocardiogram, to ensure blood pump ejection in diastole. The key to success is the atriotomy, which creates an opening larger than the patient’s mitral valve. The atriotomy may be performed with the heart beating.

Bleeding from the left ventricular apical anastomosis is a fairly common occurrence in currently available left ventricular assist systems; subsequent transfusion can exacerbate right heart dysfunction and sensitize the immune system. These complications are avoided with our system.

The new system works either in partial mode or total mode, depending on whether partial or full left ventricular unloading is required. The Novel Left Ventricular Assist System is in its initial clinical trial stage, under the supervision of the author. (Tex Heart Inst J 2003;30:194-201)
The new system works either in partial mode or total mode, depending on whether partial or full left ventricular unloading is required.

Description of the System

The Novel LVAS is a pneumatically powered ventricular assist device consisting of a blood pump, a percutaneous drive line, an external power source, an intramyocardial electrode for electrocardiographic (ECG) synchronization, and a drive system. It is attached between the left atrium and the healthy descending thoracic aorta. If the descending thoracic aorta is diseased, the surgeon may use the abdominal aorta below the renal arteries. Alternatively, the ascending aorta may be used.

An atrial prosthesis (atrial cuff) has been developed (Fig. 1). It is sutured on the epicardial side of the left atrial wall with a continuous 4-0 polypropylene monofilament suture to encircle a large atriostomy (Figs. 2A–D).

The current system configuration incorporates a dual pusher-plate blood pump with either a 23-mm bileaflet mechanical prosthesis or a 25-mm porcine-valved aortic root (Fig. 3).

A shock absorber mechanism acts during the closing and opening phases of the biologic valve. Discounting its inflow and outflow valve chambers, the internal (blood) pump is 9 cm in diameter and 6 cm in height (Fig. 4). It weighs 140 g and is placed intraperitoneally (Fig. 5). When contrast material is injected into the inflow connector during pump filling, it displays a perfect vortex that washes the periphery of the blood chamber. The next systole ejects the contrast material completely into the aorta.

The housing is made either of pure titanium or of medical polyurethane (Figs. 4A and 4B) (Desmopan, Bayer Laboratory; Berlin, Germany). The blood chamber is made of medical segmented polyurethane (Bioprost; Berkeley, Calif).

The percutaneous drive line is 6 mm in its outer diameter. A distance of 3 meters separates the patient from the pneumatic driver, to enable the patient to move about.

The pneumatic driver incorporates security measures. The pneumatic assembly consists of 2 identical circuits, 1 electric and the other pneumatic. Thus, 2 small, independent, pneumatic units have been incorporated. The driver weighs 2,660 grams (without batteries) (Fig. 6).

The system provides a pump output in the range of 3.4 to 6 L/min under a mean arterial pressure (afterload) of 100 mmHg, with a minimum of 9 mmHg atrial filling pressure (preload) and 180 to 220 mmHg pneumatic pressure (systolic compression in the blood pump chamber) (Table I).
Fig. 2. Diagram of the atriostomy technique. A–D) Sequential steps of the method: A) Suture of the inner circle of the left atrial cuff. B) Suture of the outer circle. A pericardial washer may be incorporated into the suture line. C) Either a balloon-tipped catheter or an umbrella-shaped occluding device is inserted into the left atrial chamber. The atrial wall is excised into quadrants, which facilitates the removal of each triangular quadrant flush with the base of the inner circle of the implanted atrial prosthesis. Alternatively, a large excision of the lateral wall of the left atrium may be performed under extracorporeal circulation, with the heart fibrillated during the short atriostomy period. D) The atrial cuff has been connected to the inlet pump connector. E) Cross-section of the atriostomy and of the titanium frame (lined with the atrial cuff’s Dacron velour), which keeps the atriostomy open. F) Left atrium-to-descending aorta configuration of the Novel LVAS implanted through a left axillary thoracotomy. LA = left atrium.
The atrial prosthesis, lined internally with either Dacron double-velour or Biospan segmented polyurethane, is sutured to the epicardial side of the left atrial wall. Autologous or bovine pericardium is placed as a washer in the outer suture line. Only a small amount of BioGlue is applied on the suture line. (Late full-thickness aortic necrosis and false aneurysm formation have been reported after the use of BioGlue in the repair of acute dissection.) After the atrial cuff has been placed, a large core of left atrial wall is removed: either 2.5 cm in diameter with a surface area of 4.6 cm², or 3 cm in diameter with a surface area of 7 cm². During this procedure, the heart is beating and extracorporeal circulation is not employed. Alternatively, a large excision of the lateral wall of the left atrium may be performed under extracorporeal circulation with the heart fibrillated during the short atriostomy period. Bleeding from the left atrium is controlled from within the left atrial cavity with either a balloon-tipped catheter or an umbrella-shaped occluding device. Only light pressure need be applied to the intra-atrial occluding system.

The 22-mm-diameter inflow pump connector is made of woven Dacron double-velour, which promotes the growth of autologous neendocardium to deter thromboembolism; alternatively, it is rigid and lined with Biospan.

A spiral loop (MP 35 N) of alloy wire 0.4 mm in diameter (Fort Wayne Metals; Fort Wayne, Ind) is sutured to the external surface of the woven Dacron double-velour graft. This prevents the inflow connector from collapsing during pump filling. It should be noted that the woven graft requires preclotting.
The inflow of the blood pump is improved when slight tension is applied on the atrial prosthesis, sufficient to produce a funnel shape at the level of the atriotomy (Figs. 2E and 2F).

- The 22-mm-diameter outflow connector is sutured to the descending aorta.
- The LVAS has been developed to work with high efficiency at a low frequency rate, 50 to 65 beats/min. The aim of the low-frequency pump is to facilitate the synchronization of the LVAS with the patient’s ECG. Both a peak in blood flow and a peak in aortic pressure during the diastolic period of the patient’s cardiac cycle (chronic counterpulsation) are obtained. The low frequency of the pump also preserves the integrity of the blood cells.
- A remarkable feature of the drive system is that each pneumatic unit works for a period of 10 to 15 min. Then a timer switches automatically to the other unit. This prevents overheating and component fatigue, and it enables the use of smaller units. Should one of the systems fail, the other will continue indefinitely and an alarm will warn of the problem.

The implantable pump is inserted through a left vertical axillary thoracotomy. The incision is extended approximately 7 cm downward in the abdominal wall, following the direction of the external oblique muscle. This is an anatomical approach, which preserves thoracic muscles and nerves. The chest cavity is entered first through the 4th intercostal space to expose the left atrium and descending aorta, and then through the 7th space to provide safe passage of the pump connectors through the diaphragm. The internal pump is placed inside the abdominal cavity.

**Discussion**

Heart failure produces an attenuation of the normal excitation/contraction coupling mechanism and a severe alteration in myocyte biology. It leads progressively to myocyte hypertrophy, myocytolysis, and interstitial fibrosis, setting the stage for heart chamber enlargement and myocardial hypertrophy.

Chamber enlargement in heart failure produces myocyte stretching and induces the release of intracellular angiotensin II, which causes severe arterial hypertension. Interstitial fibrosis and stiffening of the heart leads to systolic dysfunction, followed by diastolic dysfunction and remodeling of the heart to a more spherical state. This is accompanied by mitral regurgitation and diminished stroke volume.

The implantation of left ventricular assist systems has been associated with reversal of left ventricular remodeling, reduction of ventricular size, improved contractility, and regression of myocyte hypertrophy. Experimental and clinical LVAS implantation

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**Fig. 6** The photographs show A) the hospital drive unit and a volunteer carrying the knapsack (ambulatory) drive unit, and B) the home drive unit.
has reduced pulmonary artery wedge and mean pulmonary arterial pressures, resulting in afterload reduction of the right ventricle. Moreover, there have been dramatic improvements in cardiac index and central hemodynamics.

Although there is no doubt concerning the clinical changes that take place when intracardiac pressures and circulatory flows are restored to more normal levels, there remains an important question regarding which LVAS mode—total or partial—provides the more physiologic bridge to myocardial recovery.

In its classic configuration (left ventricular apex to ascending aorta), the LVAS implant is directed toward total left heart unloading as a bridge to heart transplantation. The customary left ventricular apical cannula, with an internal diameter of 18 to 20 mm, destroys the helical shape of the chamber and its buttress wrapping. This affects the power of contraction and relaxation that the left ventricle exerts over its major axis of rotation (from apex to base).

External inspection of the left ventricle, from the apex toward the base, shows a clockwise and counterclockwise spiral formation of the myocardium, which is responsible for the heart's rotation during the cardiac cycle and for its ejection and suction activity. Any further loss of the power of contraction of the left ventricle can of course be catastrophic in a patient who is attempting myocardial recovery.

Furthermore, during total ventricular unloading, the main concerns are pulmonary hypertension and residual right ventricular dysfunction, which result chiefly from the loss of the septal component in right heart contractility. There is also a possibility that myocyte atrophy and myocardial fibrosis ensue from chronic full unloading of the heart, which could lead to detrimental hemodynamic effects as the patient is being weaned from the LVAS. This phenomenon might be analogous to the skeletal muscle atrophy that is associated with lengthy immobilization of fractured limbs.

A partial mode of left ventricular unloading is most conducive to functional heart recovery. The energy to propel the blood through the body should be supplied by both the LVAS and the biological ventricle. Our proposed system pumps the blood received from the native heart through a large atriostomy and adjusts its level of support during the long process of myocardial recovery. Our own studies, which have spanned more than 4 decades, support the principle of partially unloading the heart as a bridge to myocardial recovery.

Cannulation can cause the left atrium to collapse. We feel that the alternative left atriostomy method of installing the inflow pump connector shows great promise. The surface area of the atriostomy must exceed that of the patient's mitral valve, so that the blood will flow smoothly from the left atrium to the pump.

The avoidance of left ventricular and left atrial cannulation has the further advantage of greatly decreasing the danger of encephalic embolization. We suture the outflow pump connector to the descending thoracic aorta.

The left atriostomy is performed in a beating heart without extracorporeal circulation, but we theorize that partial extracorporeal circulatory support could be used to keep the heart beating when required in an unstable patient.

The drive unit is extremely safe, because of its identical electric and pneumatic circuits, which are on a timer that switches every 15 minutes from 1 circuit to another, to prevent overheating and component fatigue. It must be emphasized that all currently available LVASs, without exception, require percutaneous connection to either an external power source or an external electronic command. All percutaneous connections are similar in their potential complications,

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<th>Beats/min</th>
<th>Pump Filling Pressure (Preload) (mmHg)</th>
<th>Pump Mean Arterial Pressure (Afterload) (mmHg)</th>
<th>Pneumatic Pressure (Systolic) (mmHg)</th>
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mainly infection. The intracorporeal implantation of a cumbersome electric motor is unnecessary, and its dysfunction can be disastrous. An external drive unit is more amenable to quick change-out, should mechanical dysfunction occur.

An ECG-synchronized LVAS offers the best prospect of reversing profound heart failure—especially if the blood pump’s frequency rate is associated with the native heart rate of a patient under β-adrenergic blocker therapy, which helps adjust heart rate to the rate of the pump.

In future, LVAS concomitant transplantation into the injured myocardium of myogenic autologous skeletal myoblast or bone marrow might open a new era in the treatment of end-stage heart failure. At present, however, the heart failure mortality curve in essentially all series of medically and surgically treated patients shows an almost linear loss, culminating in a 5-year survival rate of 50% or less. Patients with advanced heart failure tend to die quickly. Therefore, we need to reexamine the current systems of mechanical cardiocirculatory assistance, with the goal of myocardial recovery even for patients with advanced, chronic heart failure.

The premise behind LVAS operation should be that patients—ambulatory and out of the hospital—will have at least 3 years of survival under mechanical cardiocirculatory assistance. This can be accomplished by using simpler, smaller, safer, more reliable, and easier-to-insert systems that will preserve the anatomy and the physiology of the heart.

We must act pre-emptively, before cardiac remodeling initiates a progressive myopathic process that will alter myocyte function irreversibly on cellular, biochemical, and genetic levels.

The basic physiology of the proposed system, which combines counterpulsation with the unloading of the heart, thereby augmenting coronary perfusion in diastole, provides a sound approach to functional heart recovery. The Novel LVAS would also have application as a bridge to heart transplantation, whether in cases of chronic heart failure or acute myocardial infarction in cardiogenic shock.

References