Long-term biventricular support with rotary blood pumps: prospects and pitfalls

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INTRODUCTION

Congestive heart failure affects 23 million people worldwide, including 7.5 million in North America (670,000 new cases per year) and 7 million in Europe [1]. Since 10% of those over 65 suffer from systolic left ventricular dysfunction, the number of patients with heart failure will double over the next 25 years. At any time, 10% of the heart failure cohort are categorized Stage D with advanced structural heart disease and symptoms at rest, despite detailed medical and cardiac resynchronization therapy. Twenty percent of these are younger than 65 years of age (around 140,000 in both the USA and Europe).

The prognosis of Stage D heart failure is grim. In the REMATCH study, only 8% of the medically treated patients were alive at 2 years and continued to suffer in the interim [2]. A recurrent sentiment expressed by the Stage D patients is that they would sacrifice some duration of survival for a period of symptomatic relief [3]. Cardiac transplantation provides this for a highly selected tiny minority [4]. In contrast, cardiac resynchronization therapy is disappointing. A meta-analysis of 14 trials that randomized resynchronization against medical treatment showed only 59% of the NYHA IV device patients to have borderline symptomatic improvement to NYHA III with no survival benefit [5]. Boyle et al. [6] compared functional outcomes for NYHA IV patients after resynchronization or implantation of a left ventricular assist device (LVAD). At 6 months, resynchronized patients achieved only an additional 6 m in the 6 min walk test (insufficient to affect their daily living) and remained NYHA III or IV. In contrast, LVAD patients improved by 341 m, achieving NYHA I or II status. The study could not be randomized because 90% of the LVAD candidates were bed-bound on intravenous inotropes and could not walk beforehand.

Long-term mechanical circulatory support is an increasingly realistic ‘off the shelf’ alternative to cardiac transplantation and an effective solution for those rendered ineligible through common heart failure comorbidities [7–9]. The treatment aims are uncontroversial. The first is to provide symptomatic relief and the second to achieve at least 5-year survival with a good quality of life. The third aim is cost containment by reducing the number of hospital admissions to palliate intolerable levels of breathlessness and fatigue. Hospital events account for $20.9 billion of an overall $35 billion heart failure budget in the USA.

The rationale for a blood pump deployment is clear. The failing heart beats more than 120,000 times per day pumping around 7000 l of blood against an increasing afterload. As the heart dilates, the ventricular wall tension, myocardial energy and oxygen consumption increase, while sub-endocardial blood flow decreases. The LVAD unloads the failing ventricle resulting in beneficial structural and functional changes in the diseased myocardium [10–12]. Systemic blood flow returns to physiological levels to preserve the coronary and vital organ perfusion [13]. When native heart contractility and segmental wall motion improve, there is less propensity for intraventricular thrombus formation and thromboembolism.

In September 2011, the Society of Thoracic Surgeons and the Food and Drugs Administration (USA) assembled a ‘Think Tank’ to focus on the need for long-term mechanical circulatory support in the USA. The group acknowledged the increasing safety profile and reliability of contemporary implantable rotary blood pumps together with the potential for stem cells to broaden the horizons of heart failure management. A need for 40,000 long-term LVADs per year was predicted, which would account for 50% of the heart failure annual budget. Between 15 and 30% of patients require biventricular support and there is no satisfactory long-term mechanical solution for these patients. The pulsatile total artificial hearts have numerous restrictions which confine their use as a bridge to transplantation [14]. INTERMACS data indicate that all total artificial heart patients are transplanted or dead within 6 months [15].

WHICH PATIENTS NEED BIVENTRICULAR SUPPORT

Biventricular support is necessary when the right ventricle is severely impaired by the underlying disease process. While this seems obvious, the decision to deploy an RVAD concomitant with an LVAD implantation is very difficult. Frequently, the right
heart is supported secondarily, but the staged approach conveys increased morbidity and mortality [16].

The right ventricle serves to maintain low systemic venous pressure and provide flow through the lungs. It has two functionally and anatomically different cavities adapted to generate sustained low pressure perfusion through the low-resistance, high-compliance pulmonary vascular bed [17]. The sinus (body) contains the tricuspid valve papillary muscles and generates systolic pressure. The pulse pressure is attenuated by the cone (outflow tract). The normal right ventricular ejection fraction is between 40 and 60%. A pulmonary perfusion pressure of 20 mm Hg permits optimum gas exchange. Elevated pressure causes histological damage to the microvasculature and eventual pulmonary hypertension.

The failing right ventricle dilates to maintain the stroke volume. This remodelling disturbs the peristaltic contraction pattern, causing an accelerated rise in the pulmonary artery pressure and flow. The interventricular septum contractile function is an important determinant of the right ventricular function [18]. Between 20 and 70% of the right ventricular function is derived from the left ventricle. After an LVAD implantation, global right ventricular contractility is impaired by the leftward septal shift, but the myocardial power output is maintained through a decrease in the right ventricular afterload and the elevated preload [19, 20]. The pulmonary vascular resistance falls after an LVAD implantation and lower right ventricular pressure reduces the risk of dysrhythmias by decreasing the duration of monophasic right ventricular action potentials [21].

Any fall in the right ventricular contractility in LVAD patients is balanced by a decreased pulmonary artery pressure (after load) secondary to a low left atrial pressure [22]. An increased LVAD systemic flow boosts the venous return to the right atrium and requires increased right ventricular work [23]. An acute right ventricular failure following a rotary LVAD implantation is associated with high mortality [24]. Consequently, every effort should be made to determine the need for biventricular support before the LVAD insertion.

A long-standing left ventricular failure with mitral regurgitation causes pulmonary hypertension, right ventricular hypertrophy and an increased right ventricular stroke work index. The trained right ventricle compensates for the increased venous return partly because the LVAD reduces the pulmonary vascular resistance [25]. In contrast, a high central venous pressure with a low pulmonary artery pressure in chronic heart failure suggests impaired right ventricular function [26]. In rotary LVAD patients with a mean systemic pressure of 70–90 mmHg, a high CVP reduces tissue perfusion gradient and limits the rate of hepatic renal, gastrointestinal and cerebral recovery. Numerous studies have attempted to define the preoperative pathophysiological characteristics that indicate the need for an RVAD [25–29]. Although tricuspid regurgitation may reflect an elevated right ventricular afterload and remodelling, it does not necessarily indicate poor right ventricular function [30]. Pulmonary hypertension and right ventricular dilatation cause an annular enlargement and secondary tricuspid regurgitation [31]. When the tricuspid annulus dilates, it is the mural part that increases in length [30]. The annulus becomes dilated, flattened and circular. The papillary muscle attachments arise directly from the upper septum, and are little affected by remodelling. Right atrial hypertension and a systolic reversal of flow in the vena cava are responsible for end-organ venous congestion. The regurgitant fraction also impairs the forward flow through the lungs, thereby reducing LVAD filling and the cardiac output.

Uncorrected moderate or severe tricuspid regurgitation perpetuates a right ventricular dysfunction, causing progressive heart failure and a premature death. When the right ventricle fails, an increased diastolic pressure can cause a septal shift, which may impair LVAD filling [24]. When the LVAD reduces the left ventricular volume, tricuspid regurgitation may be exacerbated acutely because of the leftward shift of the intraventricular septum and an increased venous return in response to the LVAD flow [32]. This is an indication for a concomitant tricuspid valve repair during the LVAD insertion, not for the implantation of an RVAD [31]. There is understandable reticence to complicate or prolong LVAD surgery in the high-risk heart failure patient, but tricuspid repair improves pulmonary blood flow and LVAD filling. Navia et al. [33] recently reviewed the efficacy of different types of tricuspid repair and demonstrated that a rigid prosthetic ring annuloplasty and the Kaye technique of bicuspidization by the obliteration of the mural leaflet provided a similar satisfactory long-term outcome. The Kaye technique is simple, rapid and reliable. In the Navia series, 75% of the patients with a bicuspidization commissurotomy remained free of significant tricuspid regurgitation as did 69% of the patients with a ring annuloplasty.

Of the preoperative characteristics that indicate the need for an RVAD, decreased right ventricular stroke work index is probably the best. Such patients usually have hepatic and renal dysfunction and require preoperative ventilation or temporary circulatory support. The University of Pennsylvania established a risk-score model to predict which patients would require an RVAD [27]. They included six variables: cardiac index ≥2.2 l/min/m², right ventricular stroke work index ≤0.25 mm Hg/l/m², severe pre-VAD right ventricular dysfunction, creatinine >1.9 mg/dl, previous cardiac surgery and systolic blood pressure <96 mmHg. If a patient met the high-risk criteria for a variable, they were given a risk score of 1 for that component. If they were considered low risk, they were assigned a score of 0. These scores were then entered into a final risk score equation with a maximum score of 98 [27]. Applying a cut-off score of 50, where a score <50 predicted successful LVAD support and a score >50 predicted the need for an RVAD, the authors found a sensitivity and specificity of 83 and 80%, respectively. Investigators at the University of Michigan also established a right ventricular failure risk score derived by adding points awarded for the presence of each of the following variables: vasopressor requirement (4 points), aspartate aminotransferase (AST) >80 IU/l (2 points), bilirubin >2.0 mg/dl (2.5 points) and creatinine >2.3 mg/dl (3 points) [28]. In this study, patients with a score greater than 5.5 were at a 15-fold greater risk than those with a score less than 3.0 and about 3-fold greater risk than subjects with a score of 4.0–5.0 to develop right ventricular failure.

Although patients deemed high risk should be considered for an RVAD, many LVAD recipients (irrespective of their risk assessment) will only declare the need for RVAD support after the LVAD has been implanted. Survival data from the HeartMate II clinical trials clearly demonstrate higher survival in patients who do not manifest right ventricular failure in response to an LVAD implantation (1-year survival 78 versus 59%) [34]. Although it is clear that patients presenting with preoperative multi-organ failure require RVAD support, this should first be undertaken with a less expensive temporary system and not a long-term rotary VAD.
MECHANICAL AND PHYSIOLOGICAL ASPECTS OF BIVENTRICULAR ASSISTANCE

The potential for continuous flow pumps to autoregulate themselves similar to the native heart was explored experimentally by Saxton and Andrews in 1960 [35]. At that time, mammalian physiology was thought to require a pulsatile blood flow and pulsatile biventricular mechanical hearts were in development. The introduction of miniaturized continuous flow pumps into the clinical arena was met with great scepticism [36].

Intuitively, two small rotary blood pumps with portable power systems could be used for biventricular support. However, there are important differences between the rotary VAD performance in the pulmonary and systemic circulations.

The rotary blood pump output is dependent on the speed of the rotor and the pressure difference between the inlet and outlet of the pump [37]. At a fixed pump speed, an increasing gradient (higher blood pressure) decreases the flow, whereas a decreasing afterload boosts the flow. Thus, for a fixed impeller speed, the pump output will be much greater faced with pulmonary (right sided) versus systemic (left sided) vascular resistance. The power consumed by the motor rises with an increasing impeller speed and blood flow through the VAD. The power use will therefore be different for right- and left-sided VADs. The imbalance between the pulmonary and systemic flows is further exacerbated during exercise and other altered haemodynamic states.

A rotary VAD will pump what is delivered to it [37]. An increased preload boosts flow, and so there is inherent automated control when the intra-ventricular pressure rises during physiological stress [38]. Equally insufficient preload (low blood volume) allows the pump to suck in the heart wall and obstruct. The dynamic interaction between the systemic and pulmonary VADs is also affected by anatomical issues and the fact that the volume of the venous return to the left and right atria is different [36]. Between 2 and 3 ml per stroke volume (250 l per day) is distributed from the aorta to the bronchial tree but returns directly to the pulmonary veins and the left atrium. Thus, the left ventricular output is between 2 and 5% greater than the right ventricular output. Although the human heart is well adapted to accommodate the imbalance, the differences must be considered when implanting a rotary VAD in both the right and the left ventricles.

Rotary VADs were initially optimized for a blood flow of 5 l per minute against a pressure of 100 mmHg. In practice, the majority of patients utilize a flow of ~4.5 l per min against a pharmacologically adjusted mean systemic pressure of 80 mmHg [34, 39, 40]. The Berlin Heart ‘Incor’ set at 7500 rpm, will pump 3.5 l per minute against a mean arterial pressure of 110 mmHg, versus 6.0 l per minute against a mean pressure of 75 mmHg [41]. Similarly, a Jarvik ‘FlowMaker’ set at 12 000 rpm provides a pump flow of between 3 and 6 l per minute, depending upon the high or low systemic vascular resistance during stress or sepsis [40] (Fig. 1).

There are subtle structural differences between the rotary VADs in clinical use. The Berlin Incor and HeartMate II have an inflow cannula situated between the native left ventricle and the pump itself [34, 41]. This functions as a resistor which holds more blood within the left ventricular cavity and helps achieve a higher left ventricular end-diastolic pressure (LVEDP). The physiological response to exercise is determined by the pump input pressure which is pulsatile when the contractility of the native ventricle improves. The HeartWare and Jarvik FlowMaker VADs are inserted directly into the native left ventricle without an inflow cannula and more readily unload the ventricle [40, 42]. Pulsatility from the native left ventricle is conveyed directly through the pump to the circulation without the damping effect of an inflow conduit.

The first reported biventricular implant occurred as a staged salvage procedure at the Texas Heart Institute in 2003 [43]. A 21-year old man with idiopathic dilated cardiomyopathy received a Jarvik Flowmaker LVAD but suffered worsening right ventricular failure with a hepatorenal dysfunction. A second Jarvik Flowmaker was implanted into the right atrium without cardiopulmonary bypass on postoperative day 15. In combination, the VADs provided a cardiac index >3.0 l/min/m² with a halving of the central venous pressure. Unfortunately, he died from sepsis and hepatic failure after 12 days of biventricular support. This clinical case promoted further laboratory work to determine how best to balance the pulmonary and systemic circulations.

EXPERIMENTAL EVIDENCE

In 2005, Frazier et al. employed a 75 kg calf model where both native ventricles were excised and replaced with a Jarvik Flowmaker [44]. The pumps worked in series without any modulation from the native heart. To accommodate the differences between the pulmonary and systemic vascular resistance, the left-sided pump was set at a constant speed of 14 000 rpm, while the right impeller speed was varied between 8000 and 12 000 rpm. The systemic blood flow was thus determined by the pulmonary blood flow and the venous return to the left atrium. The left-sided VAD was seen to imitate the normal Frank-Starling mechanism and both VADs were capable of
autoregulating the flow. The haemodynamic balance was optimized with right atrial and left atrial pressures between 5 and 15 mmHg. Pharmacological control was used to provide aortic and left atrial pressure of 100 ± 15 and 15 ± 5 mmHg, respectively. The pulmonary artery and right atrial pressure were 20 ± 5 and 15 ± 5 mmHg, respectively, with VAD flows varying between 8 and 10 l per minute. The left LVAD flow was constantly higher than the right (0.5 ± 0.2 l per min), demonstrating automatic adjustment of the drainage of bronchial arterial blood to the left atrium. Both VADs responded directly to alterations in the preload and the afterload by automatically increasing or decreasing the flow simultaneously without altering the impeller speed. There was no systemic or pulmonary venous congestion. This proof of concept study over 20 days demonstrated that biventricular continuous flow VADs could function as a total artificial heart without an atrial shunt to accommodate an imbalance through the bronchial flow.

In 2009, the same team repeated the ventricular excision experiment in a 92 kg calf using two HeartMate II VADs [45]. On this occasion, the left impeller speed was varied between 10 000 and 15 000 with a lower right impeller speed of 6000 and 13 000 rpm to compensate for the low pulmonary vascular resistance. The left impeller speed was adjusted to provide a constant mean blood pressure of 80–95 mmHg. An increase in the speed of one impeller caused an instantaneous increase in the preload for the second. The increased VAD preload boosted the flow though the impeller speed remained the same. The calf survived for 48 days and was subjected to a treadmill exercise test. Exercise boosted the flow through both VADs without changing the pump speed. There was an increase in the total body oxygen consumption, while the blood lactate levels remained at baseline, confirming an appropriate cardiac output response. Again this experiment demonstrated an intrinsic VAD flow pressure sensitivity which provides an automated balance between the systemic and pulmonary circulations irrespective of the bronchial blood flow. The VAD preload and afterload are the major determinants of the flow and require careful pharmacological adjustment in the clinical setting [36].

While the impeller speed and the outflow pressure can be reduced in the VADs that have magnetic or mechanical bearings (Berlin Heart Incor, Jarvik Flowmaker, HeartMate II), the hydrodynamically suspended impeller in the HeartWare VAD requires a minimal rotational speed to maintain non-contact suspension [46]. The speed cannot be reduced sufficiently and safely to accommodate a low pulmonary vascular resistance. An alternative approach is to band the right-sided outflow graft to increase the VAD afterload. Timms et al. tested this method in mock circulation loops using two identical Medos Deltastream pumps [47]. With the right and left pumps set at the same speed to provide a 6 l/min flow, a band was applied to the right outflow graft until the mock pulmonary artery pressure fell to 15 mmHg. Haemodynamic balance was obtained when the 10 mm outflow graft diameter (area 78.5 mm²) decreased to 5.4 mm (22.9 mm²). Although the banding approach has direct clinical relevance, it could prove disadvantageous by promoting thrombosis of the outflow graft. Equally, the pressure differential across the band will change when the pulmonary vascular resistance falls during prolonged unloading.

Maintaining the balance between the systemic and pulmonary circulations is something that the native heart does well, but pulsatile total artificial hearts do poorly [14, 48]. In contrast, the pressure responsiveness indicates that rotary VADs can be used for long-term biventricular support without having to manually alter the impeller speed. If the right-sided VAD delivers blood to the left atrium too rapidly, the left atrial pressure gradually rises; if too slowly, the left atrial pressure falls. Thus, each VAD plays an important role in determining the flow of the other. During exercise, the systemic venous compliance decreases, providing increased return to the right atrium. In conjunction with the chronotropic response to exercise this causes the VAD output to increase as illustrated by the calf treadmill experiment [45]. If the left VAD flow rate decreases, less blood is pumped from the left atrium causing the preload to rise sufficiently to maintain a physiologically acceptable steady flow rate. Operating the left and right VADs at the pressure-sensitive regions of their respective pressure flow curves enhances autoregulation [37]. A natural flow balance is achieved making a complex control system unnecessary, although the biventricular system remains vulnerable at the extremes of hypertension or hypovolaemia.

**CLINIC EXPERIENCE WITH BIVENTRICULAR ROTARY VENTRICULAR ASSIST DEVICES**

Following Frazier’s salvage case, elective biventricular implants commenced in Europe with the Jarvik FlowMaker and HeartWare VADs [43]. Some of these were published and we are aware of others that were not [49, 50]. In 2009, the Deutsches Herzzentrum Berlin began a bridge-to-transplant programme with biventricular assist using the HeartWare HVAD [42] (Fig. 2). From their own experimental work, the group recognized that even with the lowest recommended rotor speed of 2400 rpm, the pulmonary flow would be too high. Therefore, the 10 mm outflow graft from the right HVAD was banded down to 5 mm for patients with a normal pulmonary vascular resistance and to 6–7 mm in those with pulmonary hypertension. This was achieved by side-clamping and then narrowing the Dacron graft over 35 mm in length with a continuous suture. To prevent the pump inflow cannula abutting on the septum, two 5-mm silicon suture rings were added to the original HVAD implantation ring. The right HVAD inflow was then connected to the anterior free wall of the right ventricle just below the outflow tract. This site yielded the greatest distance between the tip of the inflow cannula and the intraventricular septum. The left HVAD was implanted into the apex of the ventricle as usual. Seventeen patients aged 29–73 years received biventricular support between September 2009 and November 2010. The majority had idiopathic-dilated cardiomyopathy or ischaemic cardiomyopathy and were INTERMACS Class I or II [51]. The thirty-day survival was 82% and patients were anticoagulated to a target international normalized ratio of 2.8 to 3.5. A daily dose of acetylsalicylic acid 100 mg was added to this. The LVAD pump speed was set to provide the maximum flow with the left atrial pressure of 8–13 mmHg. All RVADs could be operated in a speed ranging between 2400 (lowest) and 3800 rpm. At these settings, the RVADs provided a flow between 3.5 and 5.5 l/min. Typically, LVAD flows ranged between 3.8 and 6.9 l/min. Daily echocardiography during the first 7 days allowed the pump speed settings to be adjusted according to the filling status of both ventricles and the position of the interventricular septum. In one patient, the right flow inflow cannula abutted onto the septum and the pump was switched to the right atrium. This configuration supported him for 230 days until successful
transplantation. Patients with pulmonary hypertension experienced a steady decline in the pulmonary vascular resistance (800–160 dynes) and required a reduction in the RVAD speed (3200–2400) if the RVAD flow exceeded the LVAD flow. Pulmonary congestive symptoms did not occur. In two patients, the right ventricular function and size normalized (after 60 and 370 days) resulting in frequent suction occlusion of the VAD flow. The VADs were stopped and the flow thrombosed without adverse sequelae. Twelve patients were discharged home with the longest survival of 420 days (Fig. 3). Their 6-month survival was 50%, the same as for the INTERMACS recorded biVAD patients with an extracorporeal RVAD.

Others have reported either planned biventricular or staged implants with the HeartWare HVAD with or without outflow graft banding or a lower RVAD flow rate (5–6 L/min LVAD and 3–4 L/min RVAD) [49]. In one case following extensive acute myocardial infarction with ventricular septal defect, Strueber et al. excised the native ventricles and replaced them with two HVADs. [46] Again the survival or the support duration to transplantation was 4–6 months only. McGee et al. demonstrated a typical diurnal flow variation during rest and activity with the LVAD output ranging from 4.5 to 7.5 L/min at a pump speed of 2700 rpm and the RVAD flow of 4.2 to 5.4 L/min at 2500 rpm [50]. Comparison of the banding approach versus the fixed lowest RVAD flow rate indicates that banding provides a better flow consistency and the ability to balance the right and left circulations.

THE WAY FORWARD

There is an outstanding need for biventricular destination therapy. Preliminary findings support the feasibility of rotary blood pump support in this setting, but the time-limited bridge-to-transplant experience highlights important practical issues and reservations. A balance between pulmonary and systemic circulations is possible because of the autoregulatory properties of these VADs but they have to perform at the limits of their tolerance. This, together with the need to band the outflow graft of the RVAD, makes it less satisfactory for destination therapy. Contemporary RVADs do not fit well within the confines of the pericardium. Smaller pumps currently in development may improve this situation. Equally, there is room for new purpose-built devices to overcome the practical difficulties of using an LVAD as an RVAD.

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REFERENCES

Kucuker SA, Stetson SJ, Betker KA, Akgül A, Loebe M, Lafuente JA
Frazier OH, Myers TJ. Left ventricular assist system as a bridge to myo-

Kucuker SA, Stetson SJ, Betker KA, Akgül A, Loebe M, Lafuente JA et al.
Evidence of improved right ventricular structure after LVAD support in patients with end stage cardiomyopathy. J Heart Lung Transplant 2004;
23:28–35.

Kirklin JK, Naftel DC, Stevenson LW, Pagani FD, Miller MA et al. Early
planned institution of biventricular mechanical circulatory support
results in improved outcomes compared with delayed conversion

Laver MB, Strauss HW, Pohost GM. Right and left ventricular geometry:
adjustments during acute respiratory failure. Crit Care Med 1979;7:
509–19.

Klima UP, Lee MY, Guerrero JL, Laraia PJ, Levine RA, Vlahakes GJ. Italic

Kormos RL, Teukeberg JJ, Pagani FD, Russell SD, John R, Miller LW. Right
ventricular failure in patients with HeartMate II continuous flow left

Kotaka M, Stepanenko A, Potapov E, Krabatsch T, Redlin M, Mladenow A et al. Right to left ventricular end-diastolic diameter ratio and predic-

Potapov EV, Stepanenko A, Dandel M, Kuckuck M, Lehmkuhl HB, Weng Y et al. Tricuspid incompetence and geometry of the right ventricle as predictors of right ventricular function after implantation of a left


Santamore WP, Gray LA Jr. Left ventricular contributions to right ven-

Navi JA, Nowicki ER, Blackstone EH, Brozzi NA, Nento DE, Atik FA et al. Surgical management of secondary tricuspid regurgitation: annuloplasty, commissurotomy or leaflet procedure? J Thorac Cardiovasc Surg 2010;139:
1473–82.

Pagani FD, Miller LW, Russell SD, Naftel DC, John R, Boyle AJ et al. Extended mechanical circulatory support with a continuous-flow rotary

Santamore WP, Gray LA Jr. An ideal heart pump with hydrodynamic characteristics analogous to the mammalian heart. Trans Am Soc Artif


Hetzer R, Weng Y, Potapov EV, Pasic M, Drews T, Jurmann M et al. First experience with a novel magnetically suspended axial flow left ventric-


Frazier OH, Tuzon E, Cohn WE, Conger JL, Kadipasaoglu KA. Total heart


Hetzer R, Weng Y, Potapov EV, Pasic M, Drews T, Jurmann M et al. First experience with a novel magnetically suspended axial flow left ventric-


Frazier OH, Tuzon E, Cohn WE, Conger JL, Kadipasaoglu KA. Total heart


Hetzer R, Weng Y, Potapov EV, Pasic M, Drews T, Jurmann M et al. First experience with a novel magnetically suspended axial flow left ventric-