Surgical management of end-stage heart failure

This review explores the history, methods and evidence surrounding the various surgical therapeutic options which are available for patients with end-stage heart failure.

Heart failure can be defined as '… a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood' (Hunt et al, 2005). It is typically thought of as a benign condition managed by cardiologists. Yet mortality rates are greater than bowel or breast cancer, in men and women respectively, and it accounts for nearly 5% of hospital admissions (Stewart et al, 2001, 2002). Furthermore, it is managed by junior doctors, acute physicians, GPs, intensivists and specialist and district nurses.

The evidence for mortality and morbidity benefit of beta-blockers and angiotensin-converting enzyme inhibitors is well documented, but what if the heart continues to fail despite maximal tolerated doses of medications? In these refractory cases, the patient may be admitted for non-invasive ventilation, intravenous inotropes and diuretics and if suitable may be considered for complex device therapy such as cardiac resynchronization therapy. However, where medical management has failed, the physician will refer to the expertise of the surgeon. The cardiothoracic surgeon can offer a raft of different therapies for heart failure, from the common such as coronary artery bypass grafts, to the uncommon such as left ventricular restoration, and from the relatively minimal, such as mitral valve repair, to the definitive such as orthoptic heart transplant (*Figure 1*).

Transplant

Transplantation remains the gold standard treatment of end-stage heart failure, once the condition is refractory to both maximal medical and device therapy. Since the first orthoptic human heart transplant in 1967 techniques have advanced considerably and, while the operative technique varies, the overall principles between surgeons and centres around the world are the same.

Selection

Identification of suitable patients must be based on likely gains in survival and quality of life compared with organ-conserving treatment options. Patients should be referred for transplantation before complications such as cardiorenal syndrome have developed. Most patients referred have a diagnosis of chronic heart failure as a result of left ventricular dysfunction which is not attributable to structural or coronary artery disease which can potentially be corrected. Although there are exceptions, most fulfil the conventional criteria as follows:

- Impaired left ventricular systolic function
- New York Heart Association (NYHA) class III or IV symptoms
- Receiving optimal medical treatment
- Cardiac resynchronization therapy, implantable cardioverter defibrillator or cardiac resynchronization therapy and defibrillator device already implanted if appropriate

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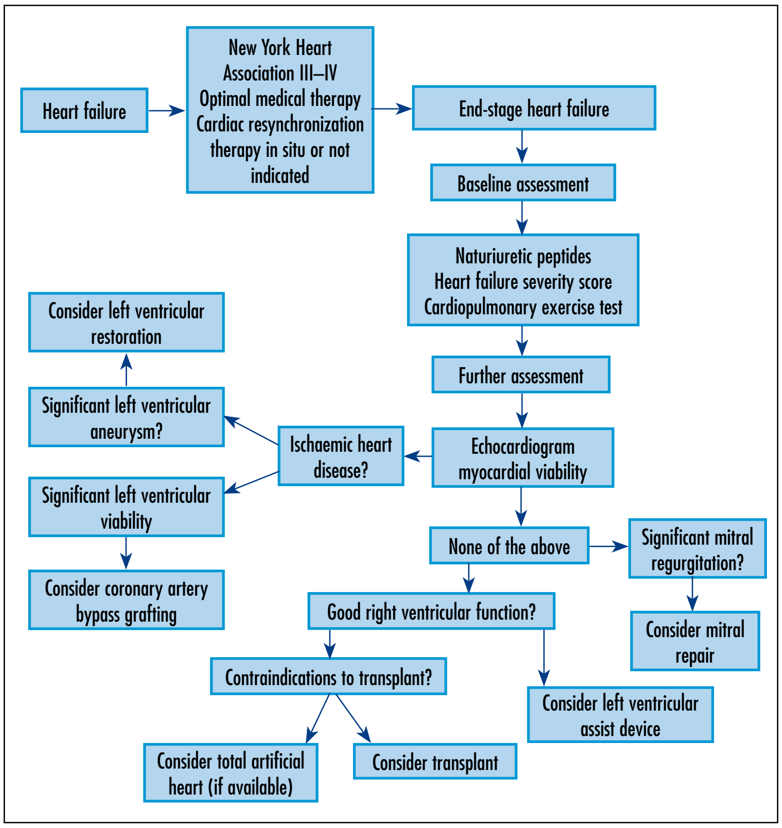
Figure 1. Management options for the patient with end stage heart failure.



- Evidence of a poor prognosis based on poor cardiopulmonary exercise test result, elevated B-type natriuretic peptide despite maximal medical treatment, or a low heart failure survival score or other prognostic scoring system measurement (Mehra et al, 2006).

There are also cases where urgent referral is appropriate, e.g. those requiring continuous inotrope infusion or intra-aortic balloon pump to prevent multi-organ failure or persistent cardiogenic shock. Organ allocation schemes give priority to patients who need the most urgent treatment – those requiring continuous inotrope infusion, intra-aortic balloon pump support or who have had acute placement of a ventricular assist device.

Outcomes

Transplantation is better than medical therapy alone, leading to fewer hospitalizations, functional improvement and enhanced quality of life, with 1- and 10-year survival of 83% and 50% respectively (Trulock et al, 2005). The median survival for those who survive the first year following heart transplantation is currently 13 years (Taylor et al, 2009). Patient comorbidities must be evaluated before consideration for transplantation as these independently affect survival and quality of life (Kirklin et al, 2001).

Complications

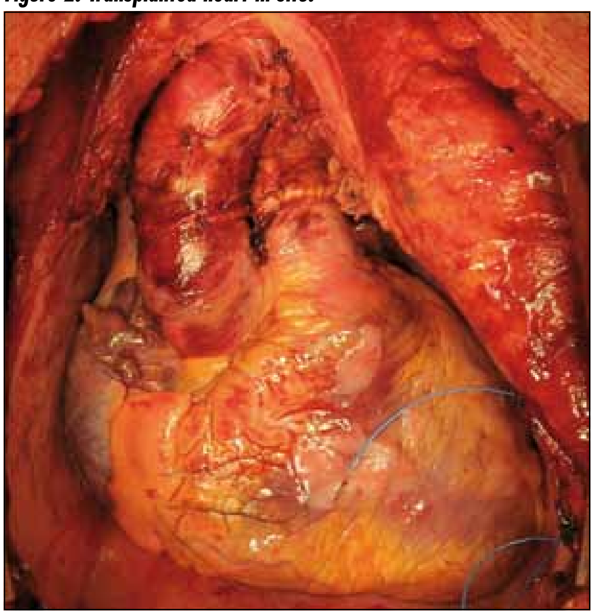
The most threatening long-term complication to heart and patient, following heart transplantation, is coronary graft atherosclerosis transplant graft vasculopathy, also termed chronic rejection. This atherosclerosis is microvascular, diffuse and not amenable to re-vascularization, unlike typical coronary artery disease.

The future

An average of 105 heart transplants (*Figure 2*) have been performed in the UK per year for the last 5 years, spread over the six UK adult heart transplant centres (Johnson, 2010). One problem with transplantation, that is unlikely to be resolved in the near future, is a lack of donor organs. This is the main limitation to its use as a treatment for all patients with heart failure. The allocation of donor hearts is governed by equity (equal access for all patients with priority given to those closest to death) and utility, which maximizes patient and graft survival.

Heart transplantation should be the optimal and definitive treatment for most patients with refractory end-stage heart failure, but until the practical issue of a lack of donors has been addressed, alternative treatments must be sought.

Figure 2. Transplanted heart in situ.



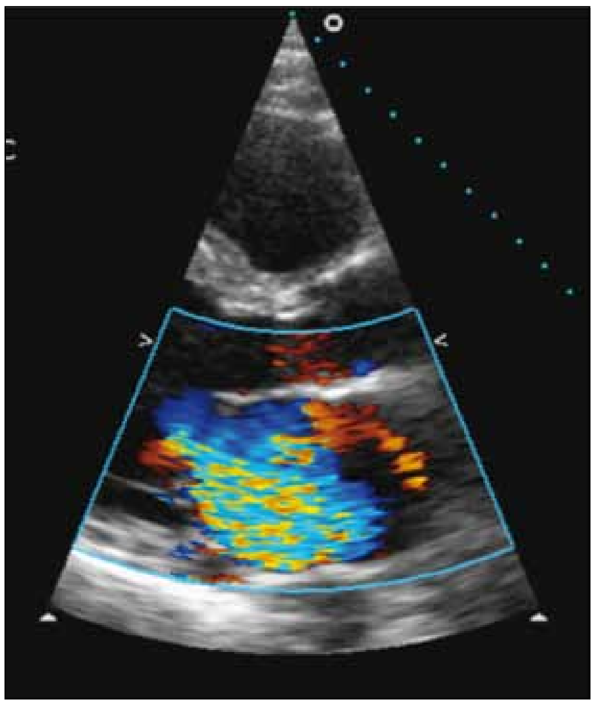
Mitral valve surgery

Valvular dysfunction can be a cause or consequence of heart failure. Mitral regurgitation can occur acutely or chronically, but the onset and progression of symptoms depends on the rate of increase in left atrial pressure and the compliance of the left atrium.

Aetiology

In chronic (secondary or geometric) mitral regurgitation, typically found in heart failure, there is a gradual increase in regurgitant flow into the left atrium leading to atrial enlargement and an increase in the left atrial and pulmonary venous compliance. As the left ventricle dilates in heart failure, the annulus also dilates and the papillary muscles are displaced from the annulus, with tethering of the subvalvular apparatus, leading to failure of coaptation and mitral regurgitation. Mitral regurgitation (*Figure 3*) in these patients is primarily a function of

Figure 3. Parasternal long axis echocardiogram demonstrating severe mitral regurgitation.



distorted left ventricular geometry rather than a primary valve problem. Volume overload causes progressive mitral regurgitation which leads to increased regurgitation of blood into the left atrium, causing progressive annular dilatation and worsening mitral regurgitation. Secondary mitral regurgitation sets up a vicious cycle and has been associated with worsened survival even with mild mitral regurgitation and few overt symptoms of heart failure (Trichon et al, 2003). Signs and symptoms of heart failure may not become apparent until much later in the disease process despite a significant degree of mitral regurgitation and volume overload.

Pathophysiology

Mitral regurgitation contributes to both increased back pressure and decreased forward flow. Compensatory increased heart rate and total stroke volume leads to increased left ventricular workload from volume overload. Once the volume overload exceeds the compensatory adaptation of the left ventricle, it is forced to dilate and as this dilatation progresses, the wall tension increases and coronary flow reserve decreases, on top of an increased left ventricular workload. This chronic energy imbalance accelerates the damage to the left ventricular muscle and the loss of left ventricular contractile functional reserve.

Outcomes

Mitral valve repair in patients with end-stage heart failure improves ventricular function and overall survival. It is associated with progressive reduction in left ventricle end-diastolic volume and increases left ventricular ejection fraction and sphericity index, all factors consistent with reverse remodelling (Romano and Bolling, 2004). There is also a significant improvement in NYHA functional class (Bolling et al, 1998). Patients are able to tolerate higher doses of medication following repair and have an improved quality of life (Bishay et al, 2000; Acker et al, 2006). The appropriate timing of intervention for secondary mitral regurgitation is controversial because of the high operative risk and poorly defined late outcome measures.

Aims

The rationale for repair or replacement is to reverse the cycle of volume overload, to allow ventricular unloading and to promote myocardial remodelling. Treatment of valvular mitral regurgitation aims to establish a zone of coaptation of the two mitral leaflets according to the functional anatomy. The aim in secondary mitral regurgitation is to re-establish the zone of coaptation and decrease the annular diameter in order to reshape the dilated left ventricle.

Repair or replacement?

Previously, mitral replacement was the surgical treatment of choice before repair techniques were developed in the 1980s. In patients with left ventricular dilatation, an annuloplasty ring can now be used to reduce the annular diameter and, where appropriate, an edge-to-edge approximation of the anterior and posterior leaflets. This is known as an Alfieri repair. Prolapsing leaflets can be resected and repaired. If repair is not possible, replacement must be performed with retention of the subchordal attachments. Preservation of the anterior and posterior chordal attachments helps to maintain normal ventricular geometry and function following replacement.

Bach and Bolling (1996) challenged previous theories that surgical correction of ischaemic mitral regurgitation has a prohibitive mortality. They described reconstruction of the mitral valve annulus geometric abnormality with an undersized annuloplasty ring to restore valvular competency, alleviate excessive ventricular workload, improve ventricular geometry and improve overall ventricular function.

Mitral valve surgery either as a sole or joint procedure is a suitable treatment option for patients with both primary and secondary mitral regurgitation causing left ventricular dysfunction.

Coronary artery bypass grafts

Coronary artery disease is the underlying cause of heart failure in up to two-thirds of cases (Hunt et al, 2005). The mechanism of heart failure is either previous myocardial infarction with left ventricular dysfunction and remodelling, or the presence of hibernating myocardium as a result of chronic but potentially reversible ischaemic dysfunction.

Outcomes

There is a reduction in mortality and improved long-term survival following coronary revascularization compared with transplantation in heart failure (Hausmann et al, 1997). Revascularization leads to substantial improvement in left ventricular function, exercise tolerance and quality of life (Pagano et al, 1998). Another benefit is arresting the remodelling process in the non-ischaemic myocardium, reducing sub-endocardial ischaemia, subsequent hypertrophy and preventing further dilation. The benefits of revascularization as compared with transplantation may be time limited, with less than 50% of patients with advanced preoperative left ventricular dysfunction being free from heart failure symptoms at 5 years (Luciani et al, 1993). Also impaired left ventricular function confers a much higher risk of mortality and morbidity following coronary artery bypass graft, with more than a twofold increased mortality risk compared with patients who have preserved left ventricular function (Topkara et al, 2005; Ahmed et al, 2009). However, the Surgical Treatment for Ischemic Heart Failure (STICH) trial compared coronary artery bypass graft with best medical therapy in patients with heart failure, and showed a significantly increased mortality, over a median follow up of 56 months, in patients in the medical therapy group when adjusted for baseline characteristics (41% *vs* 36%, *P*=0.039). There was also a slight advantage for coronary artery bypass graft in cardiovascular-specific causes of death and hospitalization for cardiovascular causes (Velazquez et al, 2011).

Physiology

It was previously thought that viability of myocardium must be established preoperatively as scarred non-functional muscle will not benefit from revascularization. Pagley et al (1997) showed that myocardial viability index was the only independent predictor of event-free survival. However, the STICH trial found that myocardial viability studies do not identify patients who will benefit from surgical revascularization, thus all patients with heart failure should be assessed for angiographic coronary artery disease (Bonow et al, 2011).

Up to 50% of patients can be redirected towards coronary revascularization after assessment of myocardial viability and risk factors (Tjan et al, 2000). Hibernating myocardium is regions of heart muscle which are dysfunctional under ischaemic conditions, but regain normal function after blood flow has been restored. Most patients will have a mixture of scarred and viable tissue. There has been proven improvement in left ventricular regional and global function following revascularization of hibernating myocardium (Schinkel et al, 2010).

The risk of myocardial damage at the time of coronary artery bypass grafting must be weighed against the benefit of revascularizing hibernating myocardium when deciding to operate on patients with heart failure.

On or off pump?

The surgical revascularization technique used is still debated among surgeons. Many feel that beating heart, or off-pump coronary artery bypass graft, without cardiopulmonary bypass, is the procedure of choice in patients with heart failure to limit the myocardial injury associated with aortic cross-clamping and the ensuing global myocardial ischaemia, as well as cardioplegia-related dysfunction in an already poorly functioning heart. Several prospective non-randomized studies support the assumption that patients with the worst preoperative prognoses would benefit most from a less invasive procedure (Al-Ruzzeh et al, 2003; Puskas et al, 2007). Although heart failure patients could potentially benefit from the improvement in early cardiac function found following off pump coronary artery bypass graft, they may not be able to sustain an adequate intraoperative cardiac output during the cardiac manipulations necessary for off-pump surgery. Several studies have shown no benefit in performing off-pump coronary artery bypass graft compared with conventional techniques (Selvanayagam et al, 2004; Pegg et al, 2008).

That some heart failure patients benefit from coronary artery bypass graft is accepted, but how to select, image, operate and at what risk to quote patients, are still contentious issues.

Left ventricular restoration

The repair of left ventricular aneurysms was reported as early as the late 1950s and, since then, many different techniques have been introduced.

Pathophysiology

In ischaemic cardiomyopathy, myocardial damage is often associated with ventricular aneurysm formation resulting in ventricular enlargement, which can compromise myocardial contractility and lead to decreased cardiac output and clinical symptoms of heart failure. Remodelling leads to chamber dilatation with changes to the wall structure. The fibrotic heart then becomes spherical and loses its efficiency as a pump. Thus, according to Laplace's law, decreasing ventricular diameter will lead to decreased ventricular wall tension and a more uniform pattern of contraction, leading to improved systolic function.

Procedure

Fujimura et al (2001) introduced the concept of surgical ventricular remodelling with the partial left ventriculotomy for patients with idiopathic dilated cardiomyopathy. Dor et al (1985) had previously described surgical anterior ventricular endocardial restoration with exclusion of the non-contracting segments in a dilated ventricle, following an anterior myocardial infarction. In this procedure, a purse string suture is placed around the circumference of the non-viable scarred aneurysm to minimize the excluded area and the residual defect is covered with a patch. This results in normal geometry and improved systolic performance.

Help or hindrance?

The RESTORE group showed a significant decrease in left ventricular end diastolic volume index and an increase in left ventricular ejection fraction, as well as amelioration of mitral regurgitation in many cases. Overall it found left ventricular restoration to be highly effective in the treatment of ischaemic cardiomyopathy with excellent 5-year outcomes (but no different from other studies looking at coronary artery bypass graft alone in patients with poor left ventricular function) (Athanasuleas et al, 2004). In contrast, the STICH trial compared the efficacy of coronary artery bypass graft alone *vs* coronary artery bypass graft and surgical ventricular reconstruction in patients with coronary artery disease and left ventricular systolic dysfunction (Jones et al, 2009). The addition of ventricular restoration reduced left ventricular volume significantly, but this did not result in a measurable benefit for the patients. This is thought to be caused by reduced diastolic distensibility after volume reduction, such that when an increased cardiac output is required, the ventricle is not able to respond effectively by increasing end-diastolic volume. Overall they found no significant difference between the two groups in terms of symptom improvement, rate of death or hospitalization for cardiac causes. These findings have subsequently been contradicted by a continuation of the RESTORE trial (Isomura et al, 2011), which explains that surgical ventricular restoration must achieve a certain ventricular volume reduction to show symptomatic benefit, a factor not addressed specifically in the STICH trial.

While the procedure makes physiological sense, the evidence is currently unclear as to whether this translates into clinical benefit.

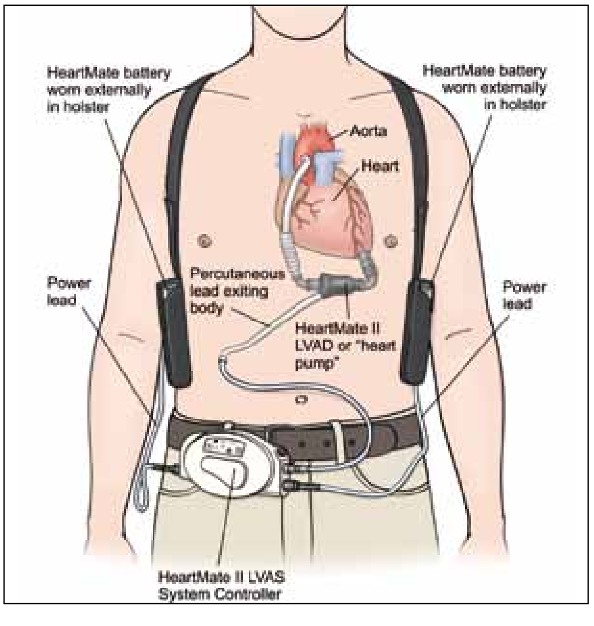
Ventricular assist devices

Ventricular assist devices were initially developed to give external mechanical support when unable to wean a patient from cardiopulmonary bypass (*Figure 4*). The first patient successfully supported to heart transplant was in 1978, and the improved systemic blood flow, and therefore coronary blood flow, gradually reverses multi-organ failure (Patel-Raman and Chen, 2010). They can be used as short-term or long-term support, and can support the right, left or both ventricles.

Procedure

In all devices, blood is removed from the failing ventricle to a pump which delivers it to the outflow vessel. In a left ventricular assist device, the inflow cannula is connected to the apex of the left ventricle and the outflow cannula is connected to the ascending aorta, devices are implanted through a median sternotomy and the pump is either placed outside the body, or in the peritoneum or the pericardial space. This unloading of the left ventricle improves contractile performance, reverses the down-regulation of beta-receptors seen in heart failure, and to some degree allows normalization of chamber geometry.

Figure 4. Diagrammatic representation of a left ventricular assist device in situ.



Bridge or destination?

Ventricular assist devices are now also used as destination therapy in those unsuitable for transplantation. In 2008, the International Society for Heart and Lung Transplantation reported that the proportion of patients supported with a left ventricular assist device before transplantation increased from 11% in 1999 to 22% in 2004 (Taylor et al, 2008).

Outcomes

Left ventricular assist devices function as a mechanical bridge to survival in patients with idiopathic dilated cardiomyopathy, with some ventricles reverting towards normal size and shape following implantation (Frazier et al, 1996). The therapeutic effect of remodelling after treatment with beta-blockers and angiotensin-converting enzyme inhibitors is amplified by mechanically offloading the left ventricle. There is evidence that prolonged, near-complete unloading of the left ventricle with the use of a left ventricular assist device is associated with structural reverse remodelling (Zafeiridis et al, 1998). Even in cases with high pulmonary vascular resistance, the right ventricle rarely needs support (Liden et al, 2009).

Continuous or pulsatile flow?

Pulsatile flow devices, such as the HeartMate XVE, consist of inflow and outflow cannulae and a pump to withdraw blood from the apex of the left ventricle and eject it out into the ascending aorta. The randomized control REMATCH trial showed that these devices relieved symptoms and improve survival compared with medical therapy (Long et al, 2005). However, device failure and pocket infection were common, limiting the long-term effectiveness of this device.

Continuous flow devices have now been developed to overcome some of the mechanical limitations of their predecessors. The HeartMate II is the only Food and Drug Administration approved continuous flow device for bridge to transplantation or destination therapy. Their use is associated with less frequent episodes of device failure, fewer infections and improved survival compared with the HeartMate XVE (Miller et al, 2007; Pagani et al, 2009).

Contraindications

Right ventricular function is one of the primary determinants of successful left ventricular assist device therapy, as the right ventricle must provide a reservoir of blood for the pump to eject. Patients with severe right ventricular compromise are unlikely to benefit from left ventricular assist device therapy alone and may benefit from a total artificial heart device.

Complications

The main complications of these devices are cannula obstruction, right ventricular failure, arrhythmias, aortic valve disease, bleeding, thrombosis and infection.

Future

The REVIVE-IT trial is ongoing, exploring the potential benefits of left ventricular assist device therapy in patients with advanced heart failure who have not yet developed the serious consequences of this.

Provided patients have adequate right ventricular function, a left ventricular assist device is a real therapeutic option in end-stage heart failure and may even allow the heart to recover sufficiently to allow a left ventricular assist device to be withdrawn, e.g. following post-partum dilated cardiomyopathy.

Total artificial heart

Patients with severe biventricular heart failure may be poor candidates for isolated left ventricular mechanical support, and at present, no long-term implantable right ventricular assist devices are available. The total artificial heart is an alternative for patients who would require a biventricular assist device, as the use of left ventricular assist devices is associated with higher mortality rates in patients with right heart failure.

Procedure

The total artificial heart is made up of two pneumatically driven pumps with tilting disk valves and short outflow grafts. Both ventricles, the proximal segments of the aorta and pulmonary artery, and all four native valves are replaced. Following implantation, inotrope therapy and antiarrhythmics are no longer required. Currently the CardioWest total artificial heart is the only temporary device licensed for use as a bridge to transplantation and has been used at Papworth Hospital (Copeland et al, 2008). Owing to its size an adequate thoracic cavity space is required for implantation, precluding anyone with a body surface area of less than 1.7 m^2 or with an anterior-posterior thoracic diameter of less than 10 cm. However, many patients who would have not been considered for left ventricular assist device therapy are suitable for total artificial heart, for example those with right ventricular failure, myocardial wall rupture, cardiac rejection and refractory arrhythmias. The indications for total artificial heart implantation are severe heart failure requiring high dose inotrope therapy, vasopressors, with or without mechanical ventilation and the use of an intra-aortic balloon pump device or extracorporeal membrane oxygenation.

Outcomes

Use of the total artificial heart improves survival to transplant and also post-transplantation survival, with one study finding 79% survival to transplant with total artificial heart *vs* 46% in matched controls. Furthermore 69% *vs* 37% of matched controls reached a successful post-transplant survival, with 86% of patients with total artificial heart therapy surviving for the first year after transplantation (Copeland et al, 2004).

Complications

The rate of cerebrovascular accidents, one of the most feared complications, was no worse than with left ventricular assist device implantation, most occurring during implantation or explantation, with the cerebrovascular accident rate during device therapy less than 2% (Copeland et al, 2004).

Future

The currently available drivers require patients to remain in hospital, but there is an ongoing trial looking at the efficacy and safety of a portable 'take home' driver.

An artificial heart is no longer the preserve of science fiction; the total artificial heart is very much scientific fact. While future research is needed to reduce the size, and increase portability and availability, perhaps this will supplant the need for organ donation in the future.

Conclusions

The cardiothoracic surgeon has much to offer the end-stage heart failure patient who is refractory to conventional medical therapy and indeed the heart failure cardiologist who has exhausted all options. The heart transplant remains the gold standard, but as this is an increasingly scarce resource and with new developments such as the left ventricular assist device and the total artificial heart, the need for such highly emotive altruism may be replaced with technology. BJHM

KEY POINTS

- Left ventricular assist device is a destination therapy, not just a bridge to transplant.
- Heart transplant remains the gold standard for end-stage heart failure but the status quo over lack of donors remains a block.
- Total artificial heart, unlike left ventricular assist device, does not require intact right ventricular function.
- Myocardial revascularization may improve long-term survival and quality of life in certain patients with end-stage heart failure.
- The evidence on the benefit of left ventricular restoration surgery remains mixed.

Figure 2 *is reproduced from Wikipedia Commons (en.wikipedia.org/wiki/Heart_transplantation);* Figure 4 *is reproduced with permission of the Thoratec Corporation, California, USA.*

Conflict of interest: none.

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