Surgical Management of Heart Failure

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Abstract: Optimal management of heart failure is collaborative, with the involvement of specialist heart failure physicians, nurses, interventionalists, and surgeons. In addition to medical optimisation and cardiac resynchronisation therapy, surgery plays a valuable role in many patients. We herein study the evidence and the role of surgical intervention in functional mitral regurgitation, coronary revascularisation in ischaemic cardiomyopathy, and surgical ventricular reconstruction. Additionally, we describe techniques of temporary and durable mechanical circulatory support, with their relative advantages and disadvantages, and applications. Finally, we describe the history and nomenclature around heart transplants, their indications, techniques, present-day outcomes, complications, and new developments in the field.

Keywords: Surgical management, heart failure, mechanical circulatory support, mitral regurgitation, ischaemic cardiomyopathy, heart transplant.

1. INTRODUCTION

There is a wide spectrum of cardiac surgery for heart failure, from traditional Coronary Artery Bypass Grafting (CABG) to heart transplantation and Total Artificial Heart (TAH) implantation. The indication for each depends on the aetiology of the heart failure, individual patient risk profile, and severity of the disease. The shift towards less invasive options in cardiac surgery will allow for intervention at different timelines for patients. The decision to perform surgery and the operation that is to be performed should be evidence-based and discussed in by a heart team, including a cardiac surgeon, an interventional/structural cardiologist, and a cardiologist specialising in heart failure. We review the procedures most commonly performed to treat this challenging patient cohort.

2. SURGERY IN FUNCTIONAL MITRAL REGURGITATION

Understanding mitral valve anatomy is fundamental to functional (secondary) Mitral Regurgitation (MR) surgery. The mitral apparatus comprises the annulus, leaflets, chordae tendineae, and papillary muscles, as well as the Left Ventricle (LV). Unlike primary MR, secondary MR is predominantly due to ventricular pathology rather than leaflet pathology. A distorted LV alters the complex mitral apparatus geometry. LV dilatation and remodelling causes papillary muscle displacement, consequent tethering of the leaflets causing reduced coaptation, and enlargement and flattening of the usually saddle-shaped annulus [1]. Ventricular pathology may be focal, from ischaemia or infarction, or global, in non-ischaemic dilated cardiomyopathy [1]. Ischaemic cardiomyopathy may produce asymmetric leaflet tethering with eccentric MR from a regional wall motion abnormality, becoming more symmetric with increasing global systolic dysfunction, producing more central MR [1]. Dilated cardiomyopathy is typically symmetric [1, 2]. Increased LV sphericity causes annular dilatation and central MR [1]. Long-standing changes in flow dynamics can create leaflet pathology, often lengthened as an adaptive response to increase coaptation, 1,3 apart from any primary leaflet pathology in these patients [3].

Functional MR carries a poor prognosis [4]. It is unclear if this is independent of the associated LV dysfunction [5]. There is no clear evidence that surgical reduction of functional MR improves survival [5], whether isolated or with concomitant CABG. Recent European ‘Guidelines for the management of valvular heart disease’ (2017) caution a “lower level of evidence for treatment recommendations” and “highlight the importance of decision making by the Heart Team” [5]. The only Class I recommendation (Level C evidence) is an intervention for severe MR with concomitant CABG in patients with LV ejection fraction (LVEF) >30%. With LVEF <30%, surgery should only be considered for symptomatic patients with a revascularisation option with viability demonstrated (Class IIa, Level C) [5]. For isolated MR without revascularisation, surgery should only be considered in symptomatic patients with LVEF >30% despite optimal medical therapy (including cardiac resynchronisation therapy) where surgical risk is low (Class IIb, Level C) [5].

The selection of repair vs. replacement is also debatable. Unlike primary MR, where repair is the gold standard [5, 6], evidence varies for functional MR. A Cardiothoracic Surgical Trials Network randomised trial of 251 patients with severe ischaemic MR demonstrated insignificantly different 1-year mortality for repair versus replacement (14.3% cf
17.6% respectively) [7]. Both groups’ high mortality rates highlighted the poor prognosis in these patients. There was a higher rate of moderate or severe MR recurrence with repair (32.6% vs. 2.3%), though no differences in major adverse cardiac or cerebrovascular events, functional status, or quality of life were found [7]. The relatively good performance of repair may relate to the preservation of the chordal and entire subvalvular apparatus [7].

Conversely, although annuloplasty rings were used in all repairs to stabilise and “cinch in” the annulus, there was no detail regarding important adjuncts such as addressing leaflet tethering and papillary elongation [8]. A meta-analysis of repair versus replacement for ischemic MR identified reduced peri-operative and late mortality following repair versus replacement, more pronounced with longer follow-up beyond 3 years [9]. However, patients within the replacement cohorts had more comorbidities than those with the repair. Although propensity matching was performed, this is inferior to randomisation [9].

The landscape is, however, changing. Two recent randomised trials - the French MITRA-FR and the United States’ COAPT - examined edge-to-edge percutaneous mitral repair using the MitraClip (Abbott) device in patients with symptomatic heart failure and severe (MITRA-FR) or moderate-to-severe and severe (COAPT) functional MR [4]. MITRA-FR randomised 304 patients to either optimal medical therapy (OMT) plus MitraClip or OMT alone; COAPT randomised 614 patients similarly. In MITRA-FR, the MitraClip reduced MR to Grade 2+ or less in 92% of patients by hospital discharge but with no impact on all-cause mortality or heart failure rehospitalisation [4]. In COAPT, the MitraClip similarly reduced MR periprocedurally to Grade 2+ or less in 95% of patients. In contrast, however, MitraClip implantation showed a substantial reduction in the 2-year all-cause mortality (29.1% vs. 46.1%) and the composite of death and heart failure rehospitalization (45.7% vs. 67.9) [4]. Further information awaits the eventual publication of the RESHAPE-HF-2 and MATTERHORN trials [4]. Not all mitral valves are anatomically amenable to edge-to-edge percutaneous repair. Ascertainment who will derive the most benefit is challenging [10]. Percutaneous Transcatheter Mitral Valve Replacement (TMVR) techniques are at the preclinical and clinical trial stage, showing “promising results.” However, “further development, testing, and trials need to be conducted before TMVR can become a sensible MR treatment” [11].

3. SURGICAL REVASCULARISATION IN ISCHAEMIC CARDIOMYOPATHY

Revascularisation in unstable coronary artery disease and management of the stable symptomatic coronary disease is well-established. However, prior to the publication of the ‘Surgical Treatment for Ischemic Heart Failure’ (‘STICH’) trial in 2011, there was limited evidence regarding surgical revascularisation in ischaemic cardiomyopathy. The STICH trial’s first hypothesis was that CABG plus OMT would improve long-term survival compared to OMT alone [12].

STICH evaluated adults with LVEF ≤ 35% with coronary disease suitable for surgical revascularisation, excluding those requiring surgical aortic valve replacement, recent myocardial infarction, significant left main disease, or mechanical support [12]. 1,212 patients were randomised to either CABG plus OMT or OMT alone [13]. 52% in each group only had Canadian Cardiovascular Society (CCS) angina class O or I angina [13]. The median LVEF was 28% [14]. 30-day operative mortality was 5.1% [13]. At first publication in 2011 (median follow-up 56 months), there was no difference in death from any cause or from cardiovascular causes. CABG did, however, confer an advantage for death from any cause plus either hospitalisation for heart failure, hospitalisation for cardiovascular causes, hospitalisation for any cause, or need for revascularisation [13]. The “STICH Extended Study” (STICHES)-published in 2016 extended median follow-up to 9.8 years [14]. At this time, CABG conferred advantage across all endpoints evaluated - death from any cause (mortality rate 58.9% for CABG patients vs. 66.1% for OMT alone), death from cardiovascular causes, hospitalisation for any cause, cardiovascular causes or heart failure, need for subsequent revascularisation, nonfatal myocardial infarction and nonfatal stroke [14]. STICH and STICHES provided high-quality evidence for revascularisation in ischaemic cardiomyopathy, even without angina.

STICH also evaluated myocardial viability. Of the 1,212 patients, 601 underwent viability testing using Single-Photon Emission Computed Tomography (SPECT), dobutamine Transthoracic Echocardiogram (TTE), or both [15]. 487 (81.0%) demonstrated viability; 114 (19.0%) demonstrated non-viability; these were almost evenly distributed between the CABG and the OMT alone groups. The presence or absence of myocardial viability did not affect the benefit of CABG over OMT alone [15]. This subset study was probably underpowered to detect the impact of viability on outcomes. However, the subset of patients amenable to surgical ventricular reconstruction (SVR), i.e., those with anterior wall akinesia/dyskinesia, implying full-thickness infarction in the left anterior descending artery (LAD) territory, particularly benefited from CABG [14]. This counters the traditional impression that the prognostic advantage of CABG mainly relates to grafting LAD territory with the left internal thoracic artery, suggesting that LAD territory viability may be inconsequential.

4. SURGICAL VENTRICULAR RECONSTRUCTION

With modern techniques for acute revascularisation in ST-elevation myocardial infarction, including PCI and thrombolysis, ventricular aneurysms have become uncommon [16]. SVR is, therefore, rarely performed in most centres. 90% of aneurysms are anterior (LAD territory); 10% arise posteriorly or laterally [17]. Unlike scar or infarct, an aneurysm has a more delineated area of a thinned fibrous scar-with few muscle fibres [16]. Often, aneurysm diagnosis can only be definitively made during surgery [16]. The aki netic/dyskinetic area of the LV directly wastes systolic work; with increasing size, there is also remote LV remodel-
SVR aims to reduce LV size and excise or exclude this area of the fibrous scar; “plastic” as opposed to “linear” techniques also aim to reapproximate normal geometry [18]. Linear repair, described by Cooley and colleagues (1958), entails longitudinal incision of the aneurysm, parallel and lateral to the LAD, with excision of most of the aneurysm [16, 17]. The defect is then sutured closed, taking these through the septum medially, excluding as much scar as possible [16, 17]. Incorporating the LAD into the suture line eliminates the possibility of LAD grafting [17]. “Plastic” techniques described by Dor and McCarthy [17] entail opening the aneurysm adjacent to the LAD. Mural thrombus is excised. A purse-string endoventricular suture from the junction of the aneurysm and healthy myocardium is drawn tight and tied, reducing the “orifice” area of the aneurysm [16, 17]. A patch (e.g., polyester lined by autologous pericardium) can be sutured in place, closing the residual defect (Dor) [16, 17] or further purse-strings to tighten the orifice and eliminate the defect (McCarthy) [17]. The residual rim of scar tissue is then closed over this repair [17]. A pre-shaped balloon (e.g., 50-60ml) can be utilised to size the ventricular cavity [16]. The LAD is preserved for revascularisation [17]. Randomised trials are likely needed to compare the techniques [17].

Evidence varies regarding the utility of SVR. Hypothesis 2 of the STICH trial was that SVR plus CABG (n=499) compared to CABG alone (n=501), in patients with an anterior LV aneurysm, would improve survival free of cardiac hospitalisations [12]. The results published in 2009 with a median follow-up of 48 months demonstrated that although SVR improved LV geometry, this did not translate to any significant clinical difference in symptoms, death, or hospitalisation [19]. However, there have been criticisms of the handling of Hypothesis 2 in this paper. Dor and colleagues described SVR results in 117 operated patients who would have been excluded from the STICH trial for reasons including unsuitable vessels for CABG, recent myocardial infarction, and pre-operative inotropy [20]. The 1-year mortality rate was 5.1%. LVEF and LV geometry improved markedly in the matched group, no survival benefit could be demonstrated. It is purported that advocates and regular performers of SVR claimed LV aneurysm, would improve survival free of cardiac hospitalisations [16]. Mural thrombi may form, and ventricular arrhythmias may occur [16].

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With minimal evidence and infrequent occurrence, SVR features minimally in heart failure management guidelines. American guidelines have a Class IIb recommendation for SVR in “carefully selected patients” for specific indications, including intractable heart failure and ventricular arrhythmias” [21]. European guidelines state that SVR “seems of no benefit,” however, it may be considered in patients with large aneurysms and intractable heart failure or recurrent ventricular arrhythmias” [22].

5. MECHANICAL CIRCULATORY SUPPORT

Mechanical Circulatory Support (MCS) can be broadly divided into temporary and durable categories. These differ in both technology and application.

5.1. Temporary

Temporary MCS devices provide temporary for right, left or biventricular failure. This includes prophylaxis in high-risk PCI, cardiogenic shock and arrest, and acute decompensated heart failure, including post-cardiotomy [23]. In decompensated heart failure and cardiogenic shock, temporary MCS can stabilise critically ill patients for organ recovery, allowing time for decision-making regarding durable MCS insertion - a “bridge to a bridge” [24, 25]. Generally, temporary devices are used for hours up to 30 days. There are five main device categories: Intraaortic Balloon Pump (IABP); TandemLife (LivaNova) percutaneous pumps (for left or right heart support); Extracorporeal Membrane Oxygenation (ECMO) in central or peripheral venoarterial configurations for heart-lung support; Impella (Abiomed) percutaneous devices (for right or left heart support); and temporary LVADs (utilising the Thoratec CentriMag [Abbot] pump) [23, 24].

The IABP comprises a balloon catheter positioned in the proximal descending thoracic aorta, inflated cyclically with helium, and a console, set to inflate/deflate the balloon according to either arterial pressure or ECG signal. Inflating in diastole and deflating in systole, the IABP increases diastolic pressure, reducing systolic afterload. This increases diastolic coronary perfusion and Mean Arterial Pressure (MAP) and reduces ventricular load and myocardial oxygen consumption [23]. IABP is a percutaneous device, most commonly transfemoral. IABP has recently been downgraded in international guidelines after the publication of the IABP-SHOCK II trial and recent 6-year outcome update [26]. This multicentre trial randomised 600 patients with cardiogenic shock complicating acute myocardial infarction, undergoing early revascularisation, to IABP vs. control. IABP had no impact on all-cause mortality at 30 days, 1 year, or 6 years [26]. Consequently, IABP is “not routinely recommended in cardiogenic shock” (Class III recommendation, Level of Evidence B) [25].

TandemLife (LivaNova) provides percutaneous left atrial-to-femoral artery (TandemHeart) and right atrium-to-pulmonary artery (Protek Duo) circulatory support pumps for left and right heart bypass, respectively [27]. The former entails a transseptal puncture, with drainage cannula in the left atrium and return cannula in the femoral artery. The latter utilises a single cannula either via the internal jugular or femoral vein, with drainage holes in the right atrium and return into the pulmonary artery [27]. Although indicated for temporary left or right heart bypass for only 6 hours [27], the TandemHeart has been used for several days [28], pro-
neurological outcomes have been observed in some groups, genic shock or arrest [35]. However, improved survival and controlled studies have been conducted, likely due to logistical factors (IABP and Impella) nor create single-ventricle by- or thoracotomy) is required [32]. Surgical placement (sternotomy for drainage, passage extracorporeally through the pump and axial pumps [40]. Of all primary LVAD implants particularly in in-hospital cardiac arrest [35]. Most common indications include cardiogenic shock due to fulminant myocarditis, acute myocardial infarction, post-heart transplantation, or post-cardiotomy [34]. Utilising pumps such as the CentriMag, ECMO can provide support for up to 30 days [24]. Cannulae can be centrally placed surgically, with cannulation of the right atrium for venous drainage and aorta for arterial antegrade return, or percutaneously or via cut-down of peripheral vessels, commonly the femoral artery and vein, providing retrograde arterial flow. For the latter, an additional “backflow” cannula is required for antegrade femoral arterial flow, preventing ischaemia of the cannulated leg. Aside from bleeding and peripheral vascular complications, the ‘Achilles heel’ of VA ECMO is the lack of direct ventricular drainage and offloading, particularly in a compromised left ventricle. This can increase afterload, causing the closure of the aortic valve [24]. Strategies to unload the left ventricle include inotropy, systemic vasodilation, IABP, balloon atrial septostomy, surgical left ventricular vents, and percutaneous ventricular support such as the Impella devices (‘ECPella’) [24, 35]. Decision-making for VA-ECMO is complex and multidisciplinary, involving intensive care specialists, cardiac surgeons, and cardiologists. Tools such as the SAVE score may predict patients with the highest likelihood of survival with VA-ECMO [34].

5.2. Durable

Durable MCS may provide long-term myocardial support for patients with advanced heart failure despite optimal medical management, from patients with advanced New York Heart Association (NYHA) III symptoms to those deteriorating despite temporary MCS (INTERMACS’ patient profiles 1 to 7) [36]. Although internationally durable MCS has been approved and utilised as ‘Destination therapy,’ i.e., permanent support where heart transplant is not an option in Australia, use is confined to ‘Bridging therapy’ - an interim measure to either heart transplant, recovery, or transplant candidacy in those not yet eligible [37]. Driving its development is the increasing number of patients in the developed world with advanced heart failure, where a limited organ donor pool stimulates the need for alternative or bridging therapies to heart transplantation [38]. The current options are Ventricular Assist Devices (VADs) to augment native ventricular activity, implantable in left (LVAD), right (R-VAD), or biventricular (biVAD) conformations, and TAH, replacing the ventricles in an orthotopic location.

The landmark trial supporting LVAD in advanced heart failure was the ‘REMATCH’ study (2001), utilising a first-generation pulsatile-flow device, which randomised 129 patients ineligible for heart transplant with NYHA IV symptoms to either optimal medical therapy or LVAD. LVAD conferred a “clinically meaningful survival benefit and an improved quality of life,” including 48% reduction in risk of all-cause death at 2 years [39]. Subsequently, VAD technology has rapidly advanced. VADs are described as ‘pulsatile flow’ volume-displacement devices or ‘continuous flow’ rotary devices. The latter can be subdivided into centrifugal and axial pumps [40]. Of all primary LVAD implants...
(n=20,469) in the Intermacs registry between June 2006 and December 2017, most (n=19,206) were continuous flow [41]. Pulsatile flow devices incorporate inflow and outflow unidirectional valves with a chamber between which volume changes between ejections. Continuous flow devices utilise rotating impellers driving forward flow continuously [40]. Continuous flow device output is determined by inflow pressure (preload), outflow pressure (systemic vascular resistance/afterload), the pressure gradient across these, and the rotation speed of the impeller (set by the clinician) [40].

Three devices approved by the Therapeutic Goods Administration (TGA) are the third-generation HeartWare HVAD (Medtronic) and the HeartMate 2 and 3 (second- and third-generation) (Abbott). The HeartWare HVAD and the HeartMate 2 were compared as destination therapy in the randomised ‘ENDURANCE’ trial (2017) of 446 patients. At two years, the HVAD group demonstrated more strokes (29.7% vs. 12.1%), the Heartmate II exhibited greater device malfunction or failure requiring replacement (16.2% vs. 8.8%); however, there was no difference in overall survival (55.4% for HVAD vs. 59.1% for HeartMate 2) [42]. The Heartmate 3 Fig. (1) and 2 were recently compared in the ‘MOMENTUM 3’ study (2019), in which 1,028 patients were randomly assigned to receive one or the other device as bridging or destination therapy. The primary end-point was survival at 2 years free of disabling stroke or reoperation to replace or remove a malfunctioning device. The magnetically-levitated centrifugal Heartmate 3 pump was superior to the axial flow HeartMate 2 pump, with 76.9% in the former as compared to 64.8% in the latter for the primary end-point at two years. Additionally, the incidence of any stroke, major bleeding, and gastrointestinal bleeding was lower-with HeartMate 3 [43]. Extrapolating the ENDURANCE and MOMENTUM 3 trial results suggests the overall superiority of the HeartMate 3 over the HVAD device.

Although the proportion of biVAD implants to LVAD implants is low (1,013 vs. 19,116 over approximately 11 years in the USA as described in the Intermacs annual report [41]), and isolated RVAD implantation is rare, the decision to implant an RVAD when implanting an LVAD is complex. Right ventricular failure may coexist or be consequent to left ventricular failure. A continuous LVAD requires sufficient inflow generated by the right ventricle to sustain forward flow. After LVAD implantation, the right ventricle may benefit from unloading of left-sided pressures. However, the leftward shift of the interventricular septum with tricuspid annular distortion and the higher systemic venous return may cause progressive right ventricular failure. This can cause difficulty weaning from cardiopulmonary bypass, increased perioperative morbidity, and mortality [44]. Early utilisation of right ventricular MCS (temporary or durable) may improve outcomes compared to delayed biVAD support in right ventricular failure after LVAD insertion [45]. However, patients requiring biVAD demonstrate lower survival than those requiring only LVAD [41]. Temporary right ventricular MCS is favoured if there is uncertainty, bridging to recovery or durable RVAD [37]. Prediction scores e.g., the CRITT score (CVP >15mmHg, severe right ventricular failure, pre-operative intubation, severe tricuspid regurgitation, and tachycardia >100) are of modest use in determining the need for biVAD support [46]. The HeartMate 3 and HVAD have both been used for biVAD support as off-label usage [37].

An alternative to biVAD is TAH, a form of ‘cardiac replacement therapy’ [38]. Internationally, fewer than 20 centres report the use of the most common device, the Syncardia TAH (Syncardia Systems) [38]. In the Intermacs report where 20,469 patients had primary durable MCS implanted for left ventricular support between 2006 and 2017, 19,116 underwent LVAD implantation, 1,013 had biVAD, and only 339 had TAH [41]. The first Australian device was implanted at St. Vincent’s Hospital in Sydney in 2010 [47]. The devices are sutured to the native atria, the native ventricles excised, thereby replacing the ventricles and outflow valves. These can only be utilised as a bridge to transplant (or destination therapy) with no capacity for recovery. Other than the indications for biVAD implantation, a rare indication for TAH is isolated cardiac neoplasia with no reconstructive options [38].

6. HEART TRANSPLANT

Heart transplant was first described in 1905 by Carrel and Guthrie in dogs [48]. The first human heart transplant - utilising a chimpanzee “xenograft” heart was unsuccessfully performed in 1964 by Hardy [48]. The first successful human heart transplant using a human heart was performed by Barnaard in South Africa in late 1967. 102 patients underwent heart transplants internationally by the end of 1968 [48]. Understanding the immune response through parallel research in other organ transplants (including skin and kidney) facilitated this. A critical milestone was the introduction of cyclosporine in 1981 [48], which “accelerated the evolution of cardiac transplantation from the experimental

![Fig. (1). Artistic representation of the Abbott HeartMate 3 LVADTM [55] (A higher resolution / colour version of this figure is available in the electronic copy of the article).](image-url)
phase to a clinically useful treatment modality for patients with advanced heart failure" [48].

The four broad indications for cardiac transplantation are: 1) advanced heart failure despite optimal medical therapy, cardiac resynchronisation therapy and surgery as indicated; 2) refractory angina despite optimal medical therapy with no further PCI or surgical revascularisation options; 3) recurrent life-threatening ventricular arrhythmias refractory to medical and implantable defibrillator therapy; and rarely, 4) non-resectable cardiac tumour with very low metastatic probability [48]. Advanced heart failure may be from ischaemic or non-ischaemic dilated cardiomyopathy, hypertrophic or restrictive cardiomyopathy, or end-stage congenital heart disease (although severe pulmonary hypertension may warrant heart-lung on bloc transplant). Advanced heart failure may be described using INTERMACS Profiles [36] or the American or European Guidelines [21, 25], utilising symptomatic criteria (such as advanced NYHA III onwards) [36], functional physiologica criteria (e.g., VO2 max of less than 12-14 ml/kg/min) [21], as well as the potential need for inotropy or circulatory support in critical stages [36]. To warrant the risks of transplant, this must then be coupled with sufficiently low expected survival, such as predicted by the Heart Failure Survival Score (HFSS), to warrant transplant risks [49], maximal predicted benefit [48, 49] as well as sufficient psychosocial support and robustness [48]. Contraindications broadly include age (above approximately 65 years), irreversible non-cardiac organ dysfunction, recent malignancy, and inadequate psychosocial support or robustness [48].

Multiple terminologies are utilised in heart transplant. "Orthotopic": replacement of the recipient heart with the donor heart in the "same place"; "heterotopic": placement of a donor heart in different location, i.e. as a parallel circuit. Of 2,974 heart transplants performed in Australia and New Zealand from 1984-2018, only 31 were heterotopic; the remainder orthotopic [50]. "Allograft": a heart of the same species (i.e., human); "xenograft": a transplanted organ of another species (still experimental) [48]. Another distinction regards ‘Donation after Brain Death’ (DBD) versus “Donation after Circulatory Death” (DCD). Most heart transplants are from DBD donors, but to increase the donor pool, Australia pioneered the use of DCD hearts, publishing their first series in 2015 [51]. They were followed by four centres in the UK [51], and in the past few months by the first DCD heart transplant in the USA [52]. Whereas in DBD donors, procurement proceeds during intact (supported) ventilatory and circulatory function, DCD donation entails certifying futility, withdrawal of ventilatory and circulatory support, and natural progression of circulatory death with the loss of cardiac output. If this occurs within a pre-specified time frame (usually 30 minutes for cardiac donation, minimising ‘warm ischaemic time’ whilst the heart is warm though malperfused) [52], the donor is rapidly wheeled to the operating theatre for procurement. Remote DCD procurement generally necessitates perfusion, minimising ischaemic time for transport [51, 52]. Excellent results demonstrate survival and rejection episodes matching the DBD cohort [51].

Another developing area to increase the donor pool is ‘Extended Criteria Cardiac Transplant’ (ECCT). This involves matching donors and recipients excluded from transplant due to age or comorbidity [53]. Duke University detailed their 10-year experience with ECCT. Recipients were included despite traditional exclusion criteria, e.g., age over 65 and comorbidities such as significant renal insufficiency and poorly controlled diabetes. Similarly, donor organs used would traditionally have been regarded as having unfavourable or high-risk characteristics, e.g., older donors, single- vessel coronary artery disease, left ventricular dysfunction, and high-dose inotropic support [53]. In their 454 patients, of whom 84 were on the ECCT pathway, standard criteria patient survival was higher than ECCT at 1 (89% vs. 86%) and 5 (77% vs. 66%) years. However, these outcomes still demonstrated an “acceptable alternative” for treatment of advanced heart failure in these patients [53].

With operating, there are two main techniques for recipients with normal anatomy - bicastral and biatrial implantation. Recipient cardiectomy entails aorto-bicastral cardiopulmonary bypass, aortic cross clamping, transection of the ascending aorta, pulmonary trunk, and then either transection of the cave (for the bicastral technique) with a left atrial cuff around the pulmonary veins remaining intact, or else cuffs kept of both the right atrium (with cavea intact) and left atrium (for the biatrial technique) [48]. For the bicastral technique, donor heart implantation usually proceeds with the left atrial anastomosis, followed by inferior vena cava, then the pulmonary trunk, then aortic (with de-airing manoeuvres followed by the release of aortic cross-clamp), then superior vena cava, and finally pulmonary trunk anastomosis. The biatrial technique usually starts with left atrial anastomosis, then right atrial, aortic, and finally the pulmonary trunk [48]. Post-operative care is similar to other cardiac surgical patients. The donor heart is entirely denervated, which may cause temporary bradycardia, requiring chronotropy [48]. The right ventricular function may be impaired by its greater susceptibility to graft ischaemia, or by pulmonary hypertension (pre-existing, related to cardiopulmonary bypass or protein reaction) [48]. Finally, graft ischaemia may reduce ventricular diastolic compliance, requiring higher filling pressure to maintain output [48]. The mainstay of post-operative care is infection prophylaxis and immunosuppressant use.

Of the centres captured by the International Society of Heart and Lung Transplant (ISHLT), the annual number of heart transplants exceeds 5000 [54, 55], with 100-150/year in Australia and New Zealand in recent years. 50 Global median survival is 12.5 years (for patients transplanted between 2002-2009); median conditional survival 14.8 years (i.e., for those surviving at 1 year post-transplant) [54]. As of the late 2000s, globally, the approximate survival rates are: 1-year 90%, 5-year 70%, and 20-year 20% [49]. More recent Australian/New Zealand data revealed 87.4% 1-year survival, 77.8% at 5 years, 64.7% at 10 years, 50% at 15 years and 36.6% survival at 20 years [50]. Whereas shorter-term survival is mostly eroded by early graft failure, rejection, and infection, mortality after 1 year is increasingly due to coronary...
disease in the transplanted heart - “cardiac allograft vasculopathy” (CAV) - and malignancy [48].

CONCLUSION

Alongside many medical developments, cardiac surgery has multiple options in its armamentarium for heart failure management. Surgical revascularisation of ischaemic myocardium increases survival, irrespective of myocardial viability. Coupling this with SVR, whilst predominantly a historical procedure, is of uncertain benefit. Surgical correction of functional MR, whether with repair or replacement, may improve symptoms. However, it displays no survival benefit. The management of secondary MR may be changing, with some evidence suggesting survival and morbidity benefit of percutaneous edge-to-edge repair. For patients with refractory heart failure, surgically-implanted durable VAD therapy offers improved survival over OMT and offers a bridge to potential transplantation. Temporary MCS can be offered to facilitate stabilisation or bridging to durable therapy, albeit limited data supports this, and observational data only supports the use of VA-ECMO. Heart transplantation is now accompanied by excellent outcomes, with approximately 50% of recipients still alive at 15 years. Patient complexity and the evolving therapeutic landscape necessitate multidisciplinary team management of heart failure, of which the surgeon must therefore play an integral part, with careful patient selection and tailoring of procedures accordingly.

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