I. INTRODUCTION

Orthotopic heart transplantation (OHT) is a proven surgical option for selected patients with advanced heart failure refractory to surgical or medical management and poor short-term prognosis but good indications for long-term survival (1). Transesophageal echocardiography (TEE) plays an important role during the process of heart transplantation. Therefore, a clear understanding of the surgical procedure helps to provide key diagnostic information and
appropriate monitoring parameters before, during and after the procedure. This chapter will review the surgical technique and the role of TEE in the perioperative management of heart transplant patients.

II. DONORS AND RECIPIENTS

The availability of donor organs has led to a plateau of the annual number of heart transplants at 5,000 cases worldwide (2). Broader donor eligibility criteria, to include older donors (> 50 years old), marginal donors and resuscitated hearts, have yet to have a substantial impact on the cardiac donor pool (2,3). Currently older donors (50-60 years old) have increased to account for 12% of all donors (4). A marginal donor heart is defined as an organ that fails to meet one or more of the traditional criteria for an optimal donor (3). Marginal donor hearts may have coronary artery disease, reduced left ventricular ejection fraction (LVEF), left ventricular (LV) hypertrophy, and successful resuscitation after asystole or exposure to high dose inotropes. A structurally normal young heart with LV dysfunction can recover normal function after hemodynamic and metabolic management. This form of donor heart resuscitation involves invasive monitoring to optimize fluid status, arginine vasopressin to maintain a normal systemic vascular resistance (SVR) and intravenous treatment of steroids, insulin and thyroid hormone (3).

Donor ischemic time is defined as the time from aortic cross-clamp at harvest to cross-clamp release following implantation. Donor ischemic time has increased beyond the gold standard of four hours without a significant increase in mortality (5).

Immediately prior to transplantation recipients should have a pulmonary artery catheter (PAC) inserted to assess pulmonary artery (PA) pressures. Elevated PA pressures, whether fixed or reactive, are a risk factor for post-transplant right heart failure and may preclude transplantation (6). General guidelines for unacceptable PA pressures and a relative contraindication to heart transplantation include: a) absolute systolic PA pressure > 60mmHg, b) calculated pulmonary vascular resistance (PVR) > 6 woods or PVR index > 6, c) transpulmonary gradient (TPG) = mean PA pressure - mean pulmonary capillary wedge pressure (PCWP) > 16-20 mmHg (6). A vasodilator challenge using nitroprusside, nitroglycerin or nesiritide and diuretics may lower PA pressures though these patients are still
at higher risk of early mortality (7).

III. SURGICAL CONSIDERATIONS

There are two surgical techniques of OHT currently used in clinical practice: the standard technique originally described by Lower and Shumway in 1966 (8,9) and the bicaval technique described by Dreyfus et al. (10) in 1991.

A. Standard Technique

A standard anesthetic preparation for routine cardiac surgery is performed including the use of a TEE probe for monitoring. The right internal jugular vein is left undisturbed for later use for endomyocardial biopsies. The Swan–Ganz catheter is initially inserted to measure real time RV and PA pressure but is pulled back during surgery. Both groins are prepared for emergency cannulation and initiation of cardiopulmonary bypass (CPB) if needed.

Through a median sternotomy following full systemic heparinization, standard cannulation of the ascending aorta (Ao) and both vena cava is performed. Cardiopulmonary bypass is initiated when the donor heart arrives in the Operating Room (OR). Cross-clamping of the Ao is followed by removal of the recipient’s failing heart leaving in place the posterior aspect of the left and right atria (LA, RA), and keeping as much as possible of the recipient’s ascending Ao and PA tissue. Mild systemic hypothermia is easily reached and maintained throughout surgery (34°C).

While the cardiectomy is performed, the donor heart is inspected and prepared in a cold bath saline solution. The integrity of the atrial septum is ensured and corrected if, for instance, a foramen ovale is detected. The tricuspid and mitral valve (TV, MV) apparatus are also carefully inspected. Proper ligation of the superior vena cava (SVC) is secured, the right atrial wall is opened from the inferior vena cava (IVC) to the right atrial appendage (RAA) and the LA wall is trimmed and prepared. The ascending Ao and the PA are completely dissected and separated to facilitate proper exposure and suturing approach (Fig. 22.1).

The donor heart is brought into the thoracic cavity and the LA anastomosis is performed first with 3.0 polypropylene continuous sutures. To keep the heart as cold as possible during surgical implantation standard cold blood cardioplegia, at a rate of 100–200
mL/min, is administered retrograde through a coronary sinus catheter in addition to application of ice slush onto the right ventricle (RV) (11). The RA anastomosis (Fig. 22.2) is completed with 3.0 polypropylene sutures, leaving a small gap for the coronary sinus catheter prior to completion of both the PA and aortic anastomoses. Donor and recipient pulmonary arteries are prepared, trimmed and anastomosed with a continuous 4.0 polypropylene suture.

Next, snares on both vena cava are released, the coronary sinus catheter is removed, the operating table is put in the Trendelenburg position and the ascending Ao clamp is released. With gentle massage of the heart, air is evacuated from the LV and the ascending Ao through the suture line via the puncture used to inject the antegrade cardioplegic solution at the donor site. The hole created in the left atrial appendage (LAA) to decompress the LV during the harvest of the donor heart is closed with a 3.0 polypropylene suture.

The patient is weaned from the CPB with a heart rate averaging 100 beats/min achieved by an isoproterenol infusion or atrial pacing and a good cardiac performance with systolic blood pressure (SBP) >90–100 mmHg. Atrial and aortic cannulae are removed while 500 mg of solumedrol is administered and protamine is injected intravenously. The Swan–Ganz catheter is refloated through the PA to monitor cardiac output (CO) and PA pressure. Two chest tubes are inserted in the mediastinum. Temporary pacing wires are sutured onto the RV. The sternum is then closed according to a standard technique.

B. Bicaval Technique

This alternative technique of cardiecotomy and anastomoses is now more widely accepted as it minimizes the problems arising from oversized post-transplant atria, such as stasis, spontaneous echo contrast and embolic complications (10). The right aspect of the LA is entered as for a routine MV surgical approach. The incision is extended superiorly under the SVC and inferiorly under the IVC (Fig. 22.3). During excision of the RA, generous portions of the SVC and IVC are left to facilitate donor and recipient anastomoses. The donor LA is anastomosed to the recipient LA as described in the standard method. The IVC and SVC are then anastomosed with 4-0 polypropylene suture. The rest of the procedure continues as described above for the standard approach to OHT.
IV. TRANSESOPHAGEAL ECHOCARDIOGRAPHY IN THE PERIOPERATIVE PERIOD

The intraoperative use of TEE is a Society of Cardiovascular Anesthesiologists (SCA) category 2 indication during OHT (12). The role of TEE is less valuable during the pre-CPB period as the recipient’s failing heart is completely removed. Rarely, thrombotic material in the native heart can be mobilized during manipulation and lead to pulmonary embolism (Fig. 22.4). During the CPB weaning process, TEE helps to define the etiology of hemodynamic instability, assess the efficacy of de-airing procedures and confirm the integrity of surgical anastomosis in the post-transplantation period.

Hemodynamic instability after heart transplantation may arise from a single or multiple causes such as primary graft failure, hyperacute rejection, RV dysfunction (Fig. 22.5), hypovolemia, vasodilatation, tamponade, right ventricular outflow tract (RVOT) obstruction (Fig. 22.6) or left ventricular outflow tract (LVOT) obstruction (Fig. 22.7).

Nonspecific graft failure is still the most common cause of death in the early postoperative period (13). Severe systolic dysfunction in the recipient may result from inadequate myocardial protection during the harvesting of the donor heart (role of cardioplegic solution, temperature, and duration of ischemia) and/or during transplantation. In rare instances, hyperacute rejection, rather than insufficient myocardial protection, may be responsible for the heart failure.

Right ventricular failure may occur in the recipient with pre-existing and underestimated pulmonary hypertension (PH) (Fig. 22.5). Registry data from the International Society of Heart and Lung Transplantation show that, despite advances in perioperative management, RV dysfunction accounts for 50% of all cardiac complications and 19% of early death after heart transplantation (2,13,14) (Fig. 22.8). There is approximately a fourfold higher mortality among transplanted patients with fixed PH compared with patients without PH (13). Pulmonary hypertension, defined as a PVR >480 dyne s/cm⁵ or 6 Wood units, is currently considered a contraindication to OHT. Patients with marked PH (PVR >640 dyne s/cm⁵ or 8 Wood units) should be considered for heart and lung transplantation and, more recently, long term LV assistance followed with transplantation. To date, there have been no reports in the literature of a threshold beyond which donor RV heart failure could occur which would constitute a clear contraindication to heart transplantation. A normal preoperative PVR does
provide some potential protection from acute increase in PVR causing RV failure after heart transplantation.

Organ preservation and CPB may have deleterious effects on ventricular function. Indeed CPB has been shown to cause an increase in PVR (13). Reviews have been published on the management of RV failure after heart transplantation and the role for inhaled nitric oxide (NO) treatment (13,15,16).

Transesophageal echocardiography is also useful to evaluate surgical anastomosis in the post-transplantation period (Fig. 22.9). The main PA anastomosis appears as a suture ridge within the vessels. Stenosis should be ruled out by two-dimensional (2D) color Doppler imaging to detect turbulent flow and by continuous wave (CW) Doppler to measure the systolic gradient across the anastomosis. The PAC can also be used to document any gradient across the RV and the PA (Fig. 22.10) (17). The mid-esophageal long-axis (LAX) or two-chamber views show an elongated LA now composed of both donor and recipient atrial tissue (Fig. 22.11). The suture line within the LA and RA also appears predominantly as an echodense ridge (Fig. 22.9). Acquired cor triatriatum may develop secondary to infolding of the redundant tissue from excessive donor atrial tissue (Fig. 22.7). Anastamoses of both cavae are examined to rule out stenosis.

V. ECHOCARDIOGRAPHY IN THE POSTOPERATIVE PERIOD
The early postoperative changes following heart transplantation include RV remodeling, abnormal septal motion, increase in LV mass, presence of pericardial fluid and abnormal LV and RV filling patterns (Fig. 22.12) (18).

A. Right Ventricular Remodeling, Tricuspid Regurgitation, and Abnormal Movement of the Ventricular Septum
The time course of the resolution of PH and RV remodeling after OHT has been described by Bhatia et al. (19). In 24 patients with moderate PH (mean PA pressure <50 mmHg) after heart transplantation, the PH resolved rapidly, with the mean PA pressure approaching normal values at two weeks after surgery. In parallel, the PVR had returned to normal in 80% of patients by one year. Patients with the highest preoperative PVR had the greatest decrease
after the transplantation (ranging from 10% to 84%). Right and left heart filling pressure also decreased to a nadir in the upper normal range at two weeks after surgery and remained unchanged over one year of follow-up.

The donor RV remodels in response to recipient perioperative PH. Initially enlarged, it dilates further during the first month and subsequently returns to its immediate perioperative dimensions at one year after surgery (Fig. 22.13) (19). The RV wall thickness does not increase significantly probably because the PVR declines to normal level. Thus, at one year, while resting hemodynamics are normal, the donor RV remains enlarged, probably as an adaptation to chronic volume overload.

Tricuspid regurgitation (TR) is common after heart transplantation due to the afterload mismatch and RV dilatation. The incidence of TR (grades 1–3) changes from 67% on day one after heart transplantation to 36% at one year. Nevertheless, TR is generally well tolerated and rarely of clinical significance (19). Finally, abnormal diastolic flattening of the ventricular septum was present in all patients immediately postoperatively (Fig. 22.10). This proportion decreased to 75% at one month and 42% at one year. This decline parallels the decrease in TR and RV dimensions. On the other hand, systolic paradoxical septal motion was uncommon, probably because the mean PA pressure decreases significantly immediately after OHT (19).

B. Left Ventricular Mass and Mitral Valve Function

In the immediate postoperative period, both LV wall thickness and mass increase, most likely from perioperative myocardial ischemia and edema. Systemic hypertension and side effects from cyclosporine and corticosteroid administration can further contribute to LV mass augmentation seen at one year. Acute increase in myocardial thickness may also occur later with allograft rejection (Fig. 22.8) (20). Left ventricular contractility and contractile reserve both appear to be normal after heart transplantation (21).

Mild to moderate mitral regurgitation (MR) has been reported to occur with a frequency ranging from 55% to 87% without major clinical implications (19,22). It usually occurs in a donor heart without structural abnormality of the mitral apparatus or LV dysfunction. The MR may arise from biatrial enlargement which exerts tension on the
posterior MV leaflet and causes incomplete closure of the valve and annuloventricular disproportion (19). Moreover, the presence of both donor and recipient atria and sinus nodes results in asynchrony and inhomogenous atrial contraction, which may also play a role in the genesis of MR (19). Finally, MR can occur in association with LVOT obstruction.

C. Pericardial Effusion
Pericardial effusions are commonly observed in 85% of patients after heart transplantation (23). The loss of lymphatic drainage and the discrepancy in size between the new donor heart and the large remaining pericardial cavity are plausible explanations (20). Even though pericardial effusions are common, rarely do they precipitate hemodynamic instability, such as cardiac tamponade. While large but slowly accumulating pericardial effusions usually cause little hemodynamic impairment, a loculated hematoma developing at a critical location may conversely cause acute cardiac tamponade (see Chapter 12). Thus, the relationship between the size of the effusion and the clinical outcome is likely to show a poor correlation (20). Most effusions are nearly, or completely, resolved by 30 days after surgery (23).

D. Left Ventricular Diastolic Function
Diastolic function is also altered in the heart transplant recipient (in the absence of rejection) during the early postoperative period. Initial pulsed wave (PW) Doppler examination of the mitral inflow shows a profile comparable to previously defined restrictive parameters (see Chapter 10); this evolves into a non-restrictive pattern over a six week period leading to progressive improvement in postoperative diastolic function parameters and decrease in left heart filling pressures (24). The presence of recurrent or persistent severe diastolic dysfunction with restrictive filling within six months after transplantation is associated with a reduced late-term actuarial survival, independent of graft rejection (25).

Evidence suggests that retaining normal LA size and shape by using the bicaval technique promotes ventricular filling dynamics which more closely approximates normal physiology (20). Ventricular filling is influenced by the mechanical activity of residual recipient atrial tissue over that of the donor heart. In addition, “parasystolic” contraction of residual recipient atrial tissue also modifies the pulmonary venous flow (PVF): recipient atrial
contraction occurring in late systole results in an increase in the diastolic component (D-wave); if it happens in early systole the systolic component (S-wave) is decreased. End-diastolic atrial contraction will increase the velocity observed during atrial reversal (AR) (18).

**E. Normal Echocardiographic Profile after Heart Transplantation**

One year after OHT, echocardiograms of recipients doing clinically well are characterized by increased LV wall thickness and mass. Left ventricular dimensions, volumes and ejection fraction are within normal limits. Right ventricular wall thickness and cavity size are increased with preserved RV systolic function. The transplanted heart also shows an anteromedial translational motion during systole. The atria of the transplanted heart have unique echocardiographic features. The anastomotic suture line is easily identified on 2D imaging as the waist of these hourglass-shaped atria (Fig. 22.11). This waist creates a natural point of subdivision within the native and donor atria. The markedly enlarged atrial volume results primarily from an increased LAX dimension (Fig. 22.11), although maximal width or short-axis dimension is also slightly larger than in controls (26). The increase in both donor and recipient atrial size is inversely correlated with survival (27).

**F. Abnormal Echocardiographic Findings Following Heart Transplantation**

The presence of the atrial suture line, increased atrial size with subcontractile portion of the recipient atrium and asynchrony between the donor and recipient atria contraction, promotes stasis. These factors may account for the high prevalence of atrial spontaneous echo contrast (55%) as assessed with TEE (28). Left atrial thrombi were observed in 38% by TEE and are often missed by transthoracic echocardiography (TTE). Thrombi were located in the donor LAA (10/18), on the posterior wall of the LA (6/18), on the donor component of the atrial septum (1/18) and on the left atrial suture (1/18) (28). Thrombi occurred only in patients displaying spontaneous echocardiographic contrast (Fig. 22.14). Episodes of arterial embolism were documented in 22 % of patients with both spontaneous echocardiographic contrast and LA thrombus (6% of heart transplantation recipients) (28). The use of the modified bicaval OHT technique seems to decrease the incidence of this problem considerably (29).
Transesophageal echocardiography has also been found to be superior to TTE in demonstrating thickening of the atrial septum, bulging of the recipient and donor atrial septum, and shunt at the atrial level (28). It may identify uncommon patent foramen ovale after heart transplantation (30). Coronary fistula is another finding detected by TEE after heart transplantation. The incidence of this iatrogenic complication has been estimated between 5% and 15%, a 20-fold increase over the incidence of congenital coronary artery fistula (31). The increased incidence in this group is attributed to injury from multiple routine surveillance RV endomyocardial biopsy procedures for detection of cardiac rejection, which frequently involves the right coronary artery (RCA). The vast majority of these fistulas communicate directly with the RV, are usually diagnosed by routine coronary angiography and are without hemodynamic significance (31). Finally pseudoaneurysm can occur after cardiac transplantation at any site of major vessel anastomosis (Fig. 22.11).

G. Detection of Acute Allograft Rejection and Coronary Artery Disease

Acute allograft rejection is common, particularly in the first year after transplantation. It constitutes the most frequent cause of death during this period. Morphologic features suggestive of allograft rejection include an increase in myocardial mass due to inflammatory cell infiltration and myocardial edema (Fig. 22.8) (Table 22.1). However, with the addition of cyclosporine to the immunosuppressive medical regimen, cellular rejection is associated with less myocardial edema: the evaluation of ventricular systolic function and myocardial mass has thus become obsolete as a means of detecting early rejection. Doppler parameters of diastolic function have recently been used to detect acute rejection (Table 22.2). It appears that moderate to severe rejection leads to a fall of at least 15% in the mitral deceleration or the isovolemic relaxation time. However, wide inter-patient variability in Doppler variables renders isolated measurement cut-offs less predictive. Each patient acting as her/his own control (from baseline) provides the basis for Doppler-based surveillance of allograft rejection. Currently, Doppler-derived parameters of diastolic function are used as an adjunct rather than a replacement for endomyocardial biopsy. Protocols now combine routine biopsies, with supplemental biopsy in the event of echocardiographic evidence of acutely restrictive physiology (20).
In patients who survive the first two years of transplantation, coronary artery disease (CAD) constitutes an important cause of mortality. There is increasing interest in the ability of dobutamine stress echocardiography to predict adverse cardiac events in OHT recipients. A normal dobutamine stress echocardiography result is a very powerful determinant of a benign clinical course with a negative predictive value in excess of 90–95% (20).

VI. CONCLUSION

In summary, echocardiography may significantly contribute to the evaluation of patients at various stages of heart transplantation (Table 22.3). During the initial perioperative period, it provides timely assessment of cardiac anatomy and physiology and bestows the opportunity to detect specific problems. Later, detection of allograft rejection and accelerated CAD benefits from the addition of this noninvasive diagnostic tool.
Figure 22.1 Classic biatrial technique of orthotopic heart transplantation.
In this technique, the sites of anastomoses between the donor heart and recipient heart are at the level of the atria (Ao, aorta; IVC, inferior vena cava; PA, pulmonary artery; RA, right atrium; RV, right ventricle; SVC, superior vena cava).

Figure 22.2 Atrial anastomosis.
(A,B) Mid-esophageal four-chamber view in a 37-year-old man after heart transplantation shows the thickened atrial anastomotic ridge. (C) Intraoperative view of the right atrial anastomotic line (LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle). (Courtesy of Dr. Denis Bouchard)

Figure 22.3 Bicaval technique of orthotopic heart transplantation.
In the bicaval technique, the donor vena cavae are anastomosed to the recipient superior vena cava (SVC) and inferior vena cava (IVC) (Ao, aorta; PA, pulmonary artery; RA, right atrium; RV, right ventricle).

Figure 22.4 Right ventricular thrombus prior to orthotopic heart transplantation (OHT).
Zoomed mid-esophageal right ventricular view at 114 ° (A,B) and a modified four-chamber view (C,D) show a laminated thrombus under the anterior leaflet of the tricuspid valve in a 49-year-old woman prior to OHT (LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle).

Figure 22.5 Right ventricular failure after orthotopic heart transplantation (OHT).
A dilated hypokinetic right ventricle (RV) after OHT is seen in these mid-esophageal four-chamber (A,B) and transgastric (C,D) views. Note the D-shaped RV on the transgastric view (LA, left atrium; LV, left ventricle; RA, right atrium, TV, tricuspid valve).

Figure 22.6 Right ventricular outflow tract (RVOT) obstruction after orthotopic heart transplantation (OHT).
(A,B) Mid-esophageal right ventricular inflow/outflow view shows dynamic RVOT obstruction. (C) Systolic obliteration is shown on the corresponding M-mode (Ao, aorta; HR, heart rate; LA, left atrium; LV, left ventricle; MPA, main pulmonary artery; RV, right ventricle).

Figure 22.7 Atrial anastomosis after orthotopic heart transplantation (OHT).
Mid-esophageal two-chamber views in a patient with end-stage hypertrophic cardiomyopathy before (A,B) and after OHT (C,D) are shown. Note the left atrial suture anastomosis after the
procedure and the increase in the anteroposterior size of the left atrium (LA) (LAA, left atrial appendage; LV, left ventricle).

**Figure 22.12 Right ventricular diastolic function after orthotopic heart transplantation (OHT).**
(A) Hepatic venous flow (HVF) in a 37-year-old man after OHT shows an inverted systolic (S) waveform. The right ventricular pressure (Prv) waveform (B) obtained from the right ventricular pacing port of the pulmonary artery catheter (C) is also abnormal with rapid increase in diastolic pressure consistent with abnormal right ventricular filling (EKG, electrocardiogram; PA, pulmonary artery; PROX, proximal; RV, right ventricle).

**Figure 22.13 Right ventricular function after orthotopic heart transplantation (OHT).**
Serial right ventricular echocardiographic measurements after OHT (±1 SD) are compared with control values (n = 10). Shown are right ventricular end-diastolic dimensions (RVEDD in cm²), right ventricular end-diastolic area (RVEDA in cm²) and right ventricular wall thickness (RVWT in cm) (Adapted with permission from Bhatia et al. (19)).

**Figure 22.14 Superior vena cava (SVC) thrombus.**
(A,B) Mid-esophageal bicaval views show a SVC thrombus with reduced flow on color Doppler (C) after heart transplantation (IVC, inferior vena cava; LA, left atrium; RA, right atrium).

**Figure 22.15 Aortic pseudoaneurysm after orthotopic heart transplantation (OHT).**
(A,B) Mid-esophageal ascending aorta (Ao) long-axis view shows an aortic pseudoaneurysm in a 65-year-old man which developed nine months after OHT. The pseudoaneurysm was located at the anastomotic site of the native Ao. Blood flow in the pseudoaneurysm was present. (C) Tomographic scan of the pseudoaneurysm with both posterior and anterior extension close to the sternal border (LA, left atrium; RPA, right pulmonary artery; RV, right ventricle).
<table>
<thead>
<tr>
<th>Grade</th>
<th>1990 Description</th>
<th>2004 Description</th>
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<tr>
<td>Grade 0</td>
<td>No rejection</td>
<td>No rejection</td>
</tr>
<tr>
<td>Grade 1, mild</td>
<td>A - Focal</td>
<td>Focal perivascular and/or interstitial infiltrate without myocyte damage</td>
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<td></td>
<td>B - Diffuse</td>
<td>Diffuse infiltrate without myocyte damage</td>
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<tr>
<td>Grade 2 moderate (focal)</td>
<td>One focus of infiltrate with associated myocyte damage</td>
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</tr>
<tr>
<td>Grade 3, moderate</td>
<td>A - Focal</td>
<td>Multifocal infiltrate with myocyte damage</td>
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<tr>
<td></td>
<td>B - Diffuse</td>
<td>Diffuse infiltrate with myocyte damage</td>
</tr>
<tr>
<td>Grade 4, severe</td>
<td>Diffuse, polymorphous infiltrate with extensive myocyte damage ± edema, ± hemorrhage ± vasculitis</td>
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</table>

1The presence or absence of acute antibody-mediated rejection (AMR) may be recorded as AMR 0 or AMR 1, as required.
2Where "R" denotes revised grade to avoid confusion with 1990 scheme. Reproduced with permission from: Stewart et al. (32)
Table 22.2 Detection of Heart Transplant Rejection by Echocardiographic Indices

<table>
<thead>
<tr>
<th>Study</th>
<th>Design Number</th>
<th>Criteria Used for Rejection*</th>
<th>Events</th>
<th>Index</th>
<th>Cut-off Value</th>
<th>Se (%)</th>
<th>Sp (%)</th>
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<tbody>
<tr>
<td>Valantine et al. 1991 (33)</td>
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<td>2</td>
<td>PHT IVRT</td>
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<td>79</td>
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<td>11</td>
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<td>98</td>
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<td>PHT or IVRT</td>
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<td>Em IVRT PHT</td>
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<td>88</td>
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<td>Am</td>
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<td>Dandel et al. 2001 (39)</td>
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<td>35</td>
<td>Sm Em</td>
<td>&gt; 10% ↓</td>
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<td>MPI</td>
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<td>34</td>
<td>MPI PHT IVCT</td>
<td>ns +</td>
<td>- na</td>
<td>- na</td>
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<tr>
<td>Palka et al. 2005 (42)</td>
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<td>Em</td>
<td>&lt; 0.12 m/s</td>
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<td>Sun et al. 2005 (43)</td>
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<td>138</td>
<td>IVRT E/A PE</td>
<td>&lt; 90 msec &gt; 1.7 presence</td>
<td>3 independent predictors of rejection</td>
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* Criteria used to define significant rejection are based on the previous International Society of Heart and Lung Transplant (ISHLT) classification; Grade 3a or 2R rejection in the revised classification usually lead to an increase in immunosuppression (32). (Am, maximal tissue Doppler late diastolic velocity (mitral annulus); b, total number of biopsies in the study; E/A, ratio of early to late maximal diastolic velocity; Em, maximal tissue Doppler early diastolic velocity (mitral annulus); IVRT, isovolumic contraction time; LVMPI, left ventricular myocardial performance index; n, number; na, not available; ns or -, non significant; PE, pericardial effusion; PHT, pressure half time; Se, sensitivity; Sm, maximal tissue Doppler systolic velocity (mitral annulus); Sp, specificity; +, significant)
Table 22.3 Role of TEE in Cardiac Transplantation

Before the procedure in patient with ventricular assist device
(see Table 21.3)

Perioperative period:

- Monitoring after cardiac surgery (see Table 13.1)
- De-airing of all four cardiac cavities
- Detection of stenotic anastomosis (pulmonary artery, pulmonary veins, inferior and superior vena cava)
- Detection of patent foramen ovale

Early and late post-operative period: evaluation of

- Left and right systolic and diastolic function
- Mitral and tricuspid valves
- Atrial anastomosis, thrombus, spontaneous echocardiographic contrast
- Presence of coronary fistula
- Aneurysm and pseudoaneurysm of the large vessels anastomoses

If hemodynamic instability: detection of

- Left and right ventricular acute systolic and diastolic dysfunction
- Left and right ventricular outflow tract dynamic obstruction
- Severe mitral or tricuspid regurgitation
- Hypovolemia and vasodilatation
- Cardiac tamponade from circumferential or loculated effusion
- Inferior vena cava stenosis
References


40. Vivekananathan K, Kalapura T, Mehra M et al. Usefulness of the combined index of systolic and
diastolic myocardial performance to identify cardiac allograft rejection. Am J Cardiol 2002;90:517-520.


TIME AFTER TRANSPLANTATION

RVEDD (cm)

RVEDA (cm²)

RVWT (cm)

NORMAL 1 DAY 1 WEEK 1 MONTH 1 YEAR