Perioperative Transesophageal Echocardiography in Heart Transplantation

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ABSTRACT

In recent years, perioperative transesophageal echocardiography has become well estab-lished and widely used in several heart surgical operations. Heart transplantation, which is the treatment of choice for patients with end-stage heart failure, may benefit from transe-sophageal echocardiography. It can be adopted for assessing donor organs in addition to the perioperative monitoring of heart transplant patients. In this review, the perioperative use of transesophageal echocardiography is discussed in heart transplantation.

Keywords: Heart transplantation, perioperative, transesophageal echocardiography

Introduction

Heart transplantation is an important form of surgical treatment for end-stage heart diseases that do not respond to conventional treatments. Since the first heart transplant in 1967, the recipient's life span has increased considerably due to developments in many areas such as organ dona-tion, surgical techniques, organ preservation, perioperative care, immunological risk assessment, immunosuppressive agents, and monitoring of organ functions.[1] In addition, with the expansion of donor criteria and an increase in data transfer in some countries, the annual number of heart transplantation cases has exceeded 5500 worldwide.[2] Be-cause these patients usually have additional diseases such as chronic lung disease and pulmonary hypertension in ad-dition to myocardial dysfunction, transesophageal echocar-diography (TEE) can be used to evaluate the cardiac func-tions of these patients in the perioperative period.[3]

Surgical Technique

Two basic types of heart transplantation techniques are used today. The standard technique (biatrial technique) was described by Lower and Shumway[4,5] in 1966, and the bicaval technique was described by Dreyfus et al.[6] in 1991.

In the standard technique, after median sternotomy and systemic heparinization, the ascending aorta and vena cava are cannulated. Once the donor heart is taken to the operating room, cardiopulmonary bypass (CPB) is initiated. After the aorta is cross-clamped, the heart of the recipient, anterior parts of the right and left atrium, ascending aorta, and pulmonary artery are removed, leaving as much tissue as possible. In the meantime, the donor heart is kept in cold saline, and the foramen ovale and heart valves are checked. The right atrial wall is opened from the inferior vena cava (IVC) to the right atrial appendage, and the left atrial wall is prepared by cutting. After the donor heart is placed in the thoracic cavity, left atrial anastomosis is performed. The right atrial, pulmonary artery, and aortic anastomoses are then completed. Ligaments that were previously placed on both vena cavae are opened, and after the air in the heart is evacuated, decannulation is achieved by exiting the CPB. The sternum is closed using the standard technique (Fig. 1).

In the bicaval technique, unlike the standard technique, the incision is made under the superior vena cava (SVC) in the upper part and under the IVC in the lower part. While the right atrium is removed, a portion of the SVC and IVC is left to facilitate donor and recipient anastomoses. The left atrium of the donor and recipient are anastomosed, as in
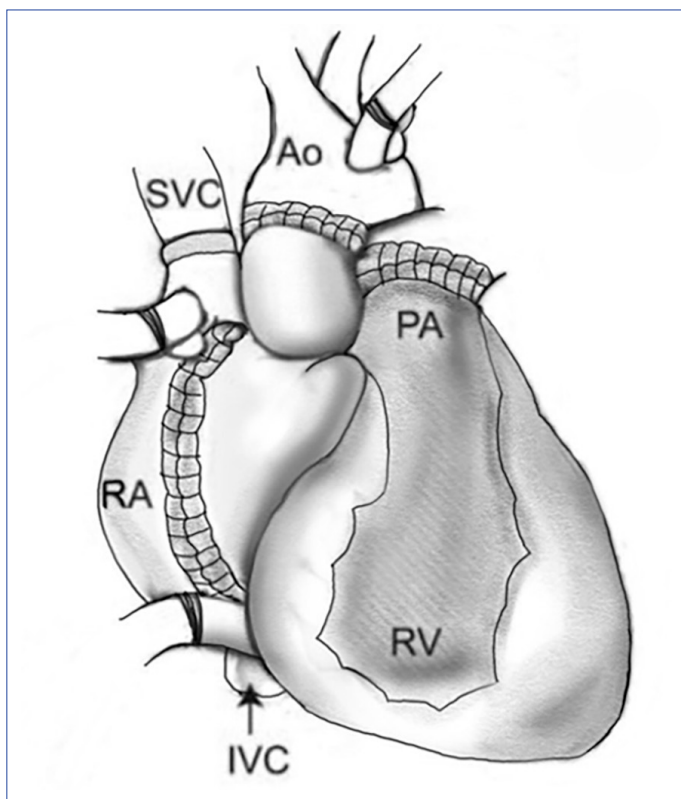
Figure 1. Classic biatrial technique in heart transplantation.

Ao: Aorta; IVC: Inferior vena cava; PA: Pulmonary artery; RA: Right atrium; RV: Right ventricle; SVC: Superior vena cava.

Figure 2. Bicaval technique in heart transplantation.

Ao: Aorta; IVC: Inferior vena cava; PA: Pulmonary artery; RA: Right atrium; RV: Right ventricle; SVC: Superior vena cava.

the standard technique. SVC and IVC anastomoses are then performed (Fig. 2). The sternum is closed using the standard method. In this technique, the atrium geometry is not distorted. In this way, complications such as arrhythmia, tricuspid regurgitation (TR), stasis, and embolism are less common. For these reasons, the bicaval technique is the preferred method in use today.

Perioperative Tee

The American Society of Anesthesiologists and the Society of Cardiovascular Anesthesiologists recommend the use of TEE in heart transplantation operations (Table 1).[7]

TEE Before CPB

TEE can be used to evaluate the donor heart.[8] In addition to systolic and diastolic functions, large-vessel anomalies, patent foramen ovale, valve function, and anatomy can also be examined.[9,10] Normal ejection fraction, the absence of left ventricular hypertrophy, and congenital and valve lesions in the donor's heart are indicators of better outcomes after transplantation.[11,12]

Because the heart will be removed, the indication for the use of TEE before CPB in the recipient is limited. TEE can be used to control the diagnosis leading to transplantation
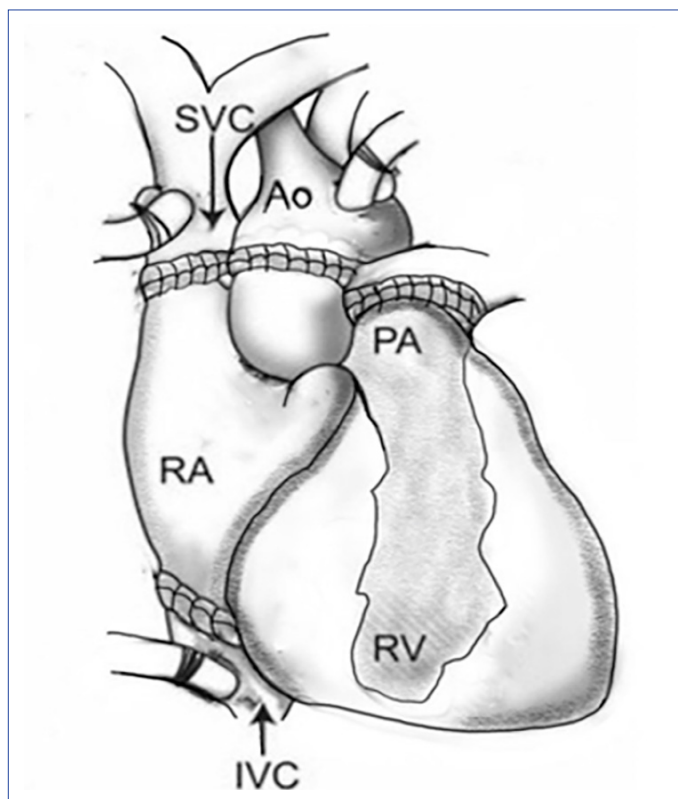
and to identify complications associated with this diagnosis. Most indications for heart transplantation in adults are in patients with a diagnosis of nonischemic dilated cardiomyopathy and ischemic cardiomyopathy. Complications such as pleural effusions are common in these patients and should be drained during surgery. In addition, in patients with low ejection fraction, thrombus may form in the ventricles, left atrial appendage, or on the pacemaker and defibrillator leads due to slow blood flow. With surgical manipulations, these thrombi may cause embolism.[13] TEE can identify these thrombi in the recipient before CPB, and precautions can be taken (Fig. 3).

Anastomotic sites such as the aorta, pulmonary artery, and vena cava can be checked for the presence of atherosclerosis and thrombus. The presence of mechanical circulatory support devices and implantable rhythm devices may complicate TEE examination.[14] Approximately 50% of patients awaiting heart transplantation benefit from the support of mechanical devices such as intra-aortic balloons, left/right ventricular assist devices (LVAD/RVAD), and extracorporeal membrane oxygenation in the preoperative period (Table 2).[15]

Problems such as hypovolemia, cannula malposition, and right ventricular dysfunction seen in LVAD patients can

Table 1. Role of transesophageal echocardiography in cardiac transplantation

Before transplantation	Donor:
	• Determination of left and right systolic and diastolic functions
	• Evaluation of mitral and tricuspid valves
	• Patent foramen ovale
	Recipient:
	• Thrombus
	• Pleural effusion
Perioperative period	• Monitoring following cardiac surgery
	• Removal of air from the heart cavities
	• Identification of stenotic anastomoses
	• Identification of patent foramen ovale
Early and late postoperative period	• Determination of left and right systolic and diastolic functions
	• Evaluation of mitral and tricuspid valves
	• Evaluation of atrial anastomosis, throm-bus, and spontaneous echocardiographic contrast
	• Aneurysm and pseudoaneurysm in great vessel anastomoses
If hemodynamic instability	• Determination of left and right ventricu-lar acute systolic and diastolic dysfunc-tion
	• Outflow obstruction in the left and right ventricles
	• Severe mitral or tricuspid regurgitation
	• Hypovolemia and vasodilation
	• Cardiac tamponade
	• Inferior vena cava stenosis

be quickly diagnosed and treated with TEE imaging before CPB. LVAD-related mitral regurgitation (MR) or aortic regurgitation, which can be seen especially in LVAD patients, can be detected.[16]

TEE during and After CPB (Intraoperative and Postoperative Period)

During and after withdrawal from CPB, hemodynamic instability may occur due to primary graft failure, hyperacute rejection, ventricular dysfunction, hypovolemia, vasodilation, tamponade, and right ventricular or left ventricular outflow tract obstructions. TEE is useful for detecting these causes.[14] A detailed TEE examination should be conducted to check for left and right ventricular systolic and diastolic functions and all valve functions (Table 3).

Ventricular dysfunction and hyperacute/acute rejection

During the removal of the donor heart and its transplantation to the recipient, ventricular dysfunction may develop as a result of prolongation of the ischemia period and insufficient myocardial protection. Primary graft failure caused by left ventricular, right ventricular, or biventricular dysfunction occurs in 30% of patients. Although isolated left ventricular dysfunction is rare, right ventricular dysfunction is more common in isolated or biventricular dysfunction. [17,18] Right ventricular function may also occur due to an acute increase in pulmonary vascular resistance, external compression during closure of the thorax, air embolism to the right coronary artery, reperfusion injury, and mechani-

cal ventilation with high positive end expiratory pressure. [13] It is responsible for 50% of cardiac complications and 19% of mortality in the early postoperative period (Fig. 4).[19] González Saldivar et al.[20] also reported right ventricular failure due to stenosis of the pulmonary artery anastomosis.

Pulmonary hypertension, defined as pulmonary vascular resistance (PVR) greater than 480 dynes s/cm^5, is considered a contraindication for heart transplantation. Patients with normal preoperative PVR have a significantly lower risk of postoperative right ventricular failure. Right ventricular failure may develop due to pulmonary hypertension of the recipient whose severity has not been previously recognized. It has been reported that pulmonary artery pressure may approach normal values after heart transplantation in patients with moderate pulmonary hypertension (mean pulmonary artery pressure < 50 mm Hg). In parallel, in 80% of patients, PVR returns to normal within 1 year. Right and left ventricular filling pressures were found to decrease to the upper normal limit within 2 weeks after surgery and remain unchanged for more than 1 year. Although abnormal diastolic flattening of the interventricular septum is found in all patients, this rate decreases to 75% in the first month and to 42% in the first year.[21]

Although rare in the intraoperative stage, hyperacute rejection is also possible. It may present with acute left ventricular systolic or diastolic dysfunction.[22] Therefore, ventricular dysfunctions should be checked both visually and parametrically, especially in cases of unexplained hemodynamic instability.
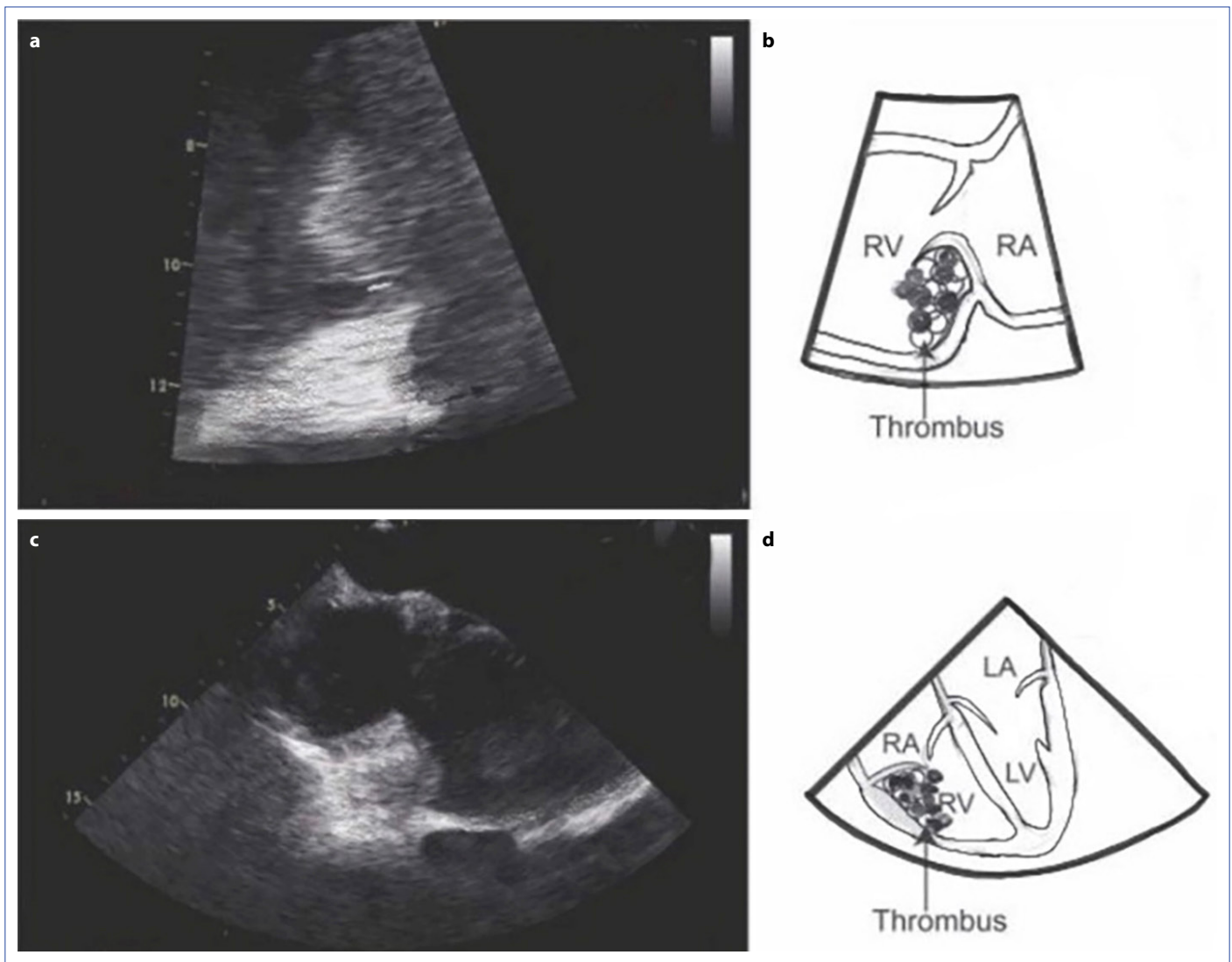
Figure 3. Thrombus in the right ventricle before heart transplantation. Thrombus under the anterior leaflet of the tricuspid valve in a 52-year-old female patient. **(a, b)** Midesophageal right ventricular view (114°). **(c, d)** Modified four-chamber view.

RA: Right atrium; RV: Right ventricle; LA: Left atrium; LV: Left ventricle.

Myocardial mass usually increases in the early postoperative period due to perioperative ischemia and edema. The continued increase in ventricular mass may indicate acute rejection. Acute rejection is usually seen in the first year after transplantation and is the most common cause of death during this period. The use of cyclosporine is associated with less myocardial edema in the case of rejection. Therefore, the evaluation of ventricular systolic function and myocardial mass has become less sensitive for detecting early rejection. In addition, the use of the myocardial performance index in recognizing acute rejection is controversial because impairment in systolic and diastolic functions may also result from other causes, such as ischemia and sepsis. Doppler measurements to evaluate diastolic function can be used to detect acute rejection. However, because the transplanted heart may be moving in a large pericardial

space, tissue Doppler imaging measurements should be interpreted with caution. Although the European Society of Cardiovascular Imaging does not find it appropriate to use a single parameter for graft rejection, if impairment in systolic and diastolic functions can be detected in several measurements, the probability of rejection is considered high.[17] Dilation, tricuspid annular planes systolic range of motion < 15 mm, and right ventricular ejection fraction <45% may indicate acute graft rejection.[23]

In moderate to severe rejection, there is at least a 15% decrease in mitral deceleration or isovolemic relaxation time. Because wide variations in Doppler measurements can be observed in patients, isolated measurements may not be predictively sensitive. Comparing each patient's baseline measurement with subsequent measurements is more sensitive in tracking graft rejection. Although the Doppler

Table 2. Points to consider in transesophageal echocardiography imaging in the recipient before heart transplantation

Aim of Imaging	Type of Imaging	Modality
Ascending aorta and main pulmonary artery imaging	ME: ascending aorta short axis/long axis UE: aortic arch short axis	• 2D, bi/multiplane imaging • 3D
Mass/thrombus in anastomosis areas	TG: basal RV, RV inflow/outflow Epicardial	
IVC and SVC imaging	I: Bicaval (for SVC, the probe is pulled slowly;	• 2D, bi/multiplane imaging
Mass/thrombus/stenosis in anastomotic areas	for IVC, the probe is advanced slowly), short axis to the ascending aorta	• Color flow Doppler
Intracardiac thrombus evaluation	ME: 4/5 chambers, mitral commissural,	• 2D, bi/multiplane imaging
LAA	2 chambers, short axis	• 3D
LV apex		• Spectral doppler (LAA velocities)
Extracardiac structures	ME: 4/5 chambers (turn the probe left for	• 2D
Pleural effusion	left pleural space, right for right pleural space)	
Ascites	TG: Ascites	

2D: Two-dimensional; 3D: Three-dimensional; ME: Midesophageal; UE: Upper esophageal; TG: Transgastric; RV: Right ventricle; IVC: Inferior vena cava; SVC: Superior vena cava; LAA: Left atrial appendage; LV: Left ventricle.

Table 3. Points to consider in TEE imaging after heart transplantation

Aim of Imaging	Differential Diagnosis	Type of Imaging	Modality
Left ventricle Dimension Systolic function: Regional (wall motion anomalies) and global (FAC, SV, EF) Diastolic function (TMF, mitral annulus velocities)	• Primary graft dysfunction • Hyperacute rejection • Insufficient myocardial protection 2 chambers, deep 5 chambers	ME: 4/5 chambers, 2 chambers, short axis TG: basal short axis, mid short axis,	• 2D, bi/multiplane imaging • 3D • Spectral Doppler • Tissue Doppler
Right ventricle Size (IVC and movement) Systolic function (free wall, FAC, TAPSE) RVOT SV	• Air in the coronary arteries • Severe pulmonary hypertension • Insufficient myocardial protection	ME: 4-chamber RV inflow/outflow TG: basal RV, RV inflow, RV inflow/outflow	• 2D, bi/multiplane imaging • Color flow Doppler • Spectral Doppler
Detailed evaluation of the valves	• Primary valve anomalies • Secondary causes such as annular dilatation due to heart failure	ME and TG	• 2D • Color flow Doppler • 3D • Spectral Doppler
Ascending aorta and main pulmonary artery anastomoses	• Stenosis/thrombus (significant narrowing, increase in the flow rate) • Dissection (intimal flap)	ME: ascending aorta short axis/long axis UE: aortic arch short axis TG: basal RV, RV inflow/outflow Epicardial	• 2D, bi/multiplane imaging • Color flow Doppler • Spectral Doppler
IVC and SVC anastomoses	Stenosis/thrombus (significant narrowing, increase in the flow rate)	ME: bicaval (for SVC, the probe is pulled slowly; for IVC, the probe is advanced slowly), short axis to the ascending aorta	• 2D, bi/multiplane imaging • Color flow Doppler
Left and right atria	• Stenosis (increase in flow rate) in left atrial anastomosis • Thrombus • PFO, ASD	M=4 chambers, 2 chambers, short axis	• 2D • Color flow Doppler

TEE: Transesophageal echocardiography; 2D: Two-dimensional; 3D: Three-dimensional; ME: Midesophageal; TG: Transgastric; FAC: Fractional area change; SV: Stroke volume; EF: Ejection fraction; TMF: Transmitral flow; RV: Right ventricle; IVC: Inferior vena cava; FAC: Fractional area of change; TAPSE: Tricuspid annular planes systolic range of motion; RVOT: Right ventricular outflow tract; UE: Upper esophageal; SVC: Superior vena cava; PFO: Patent foramen ovale; ASD: Atrial septal defect.
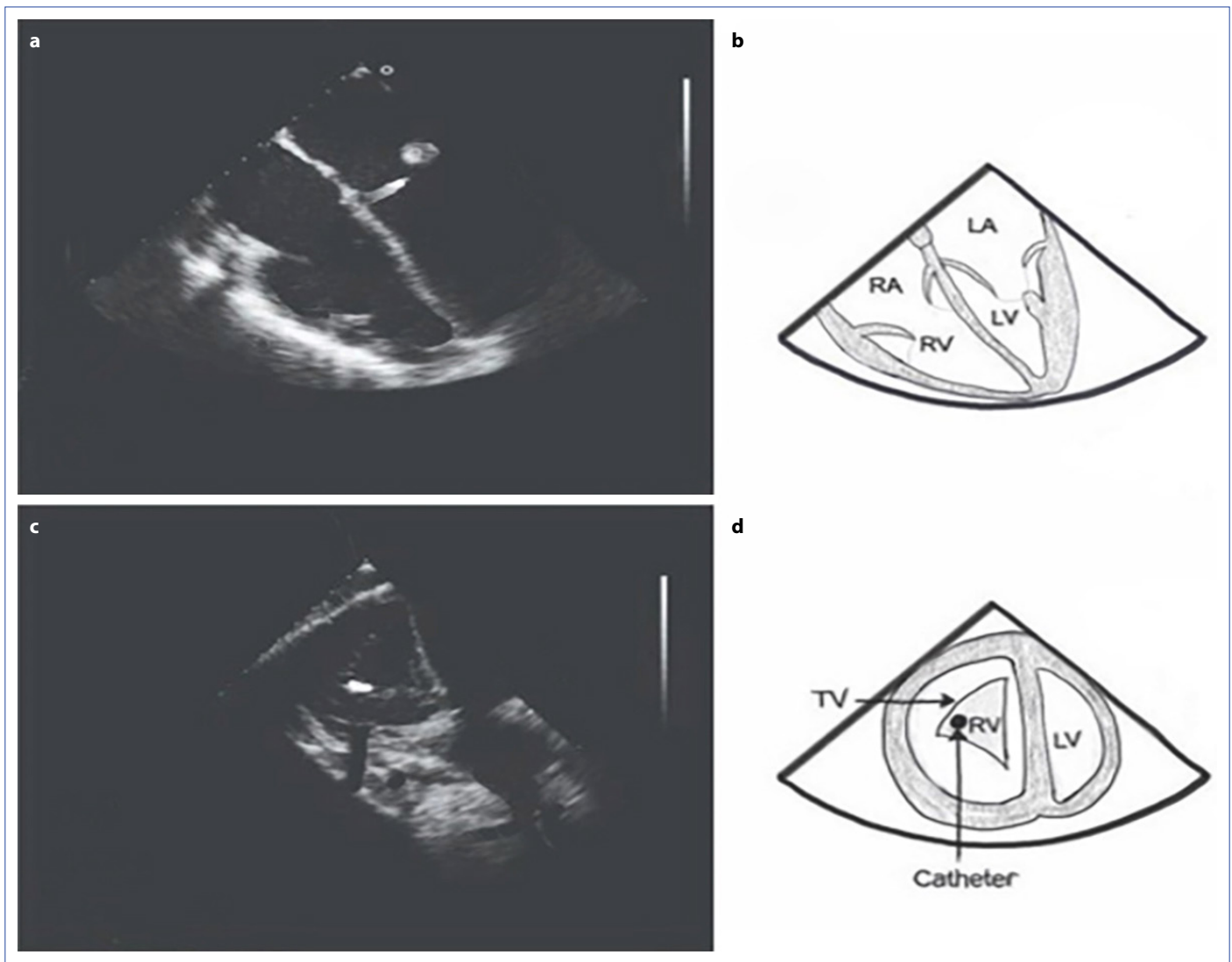
Figure 4. Right ventricular failure after heart transplantation. **(a, b)** Midesophageal four-chamber view with a dilated hypokinetic right ventricle. **(c, d)** Transgastric view: D-shaped right ventricle due to flattened ventricular septum.

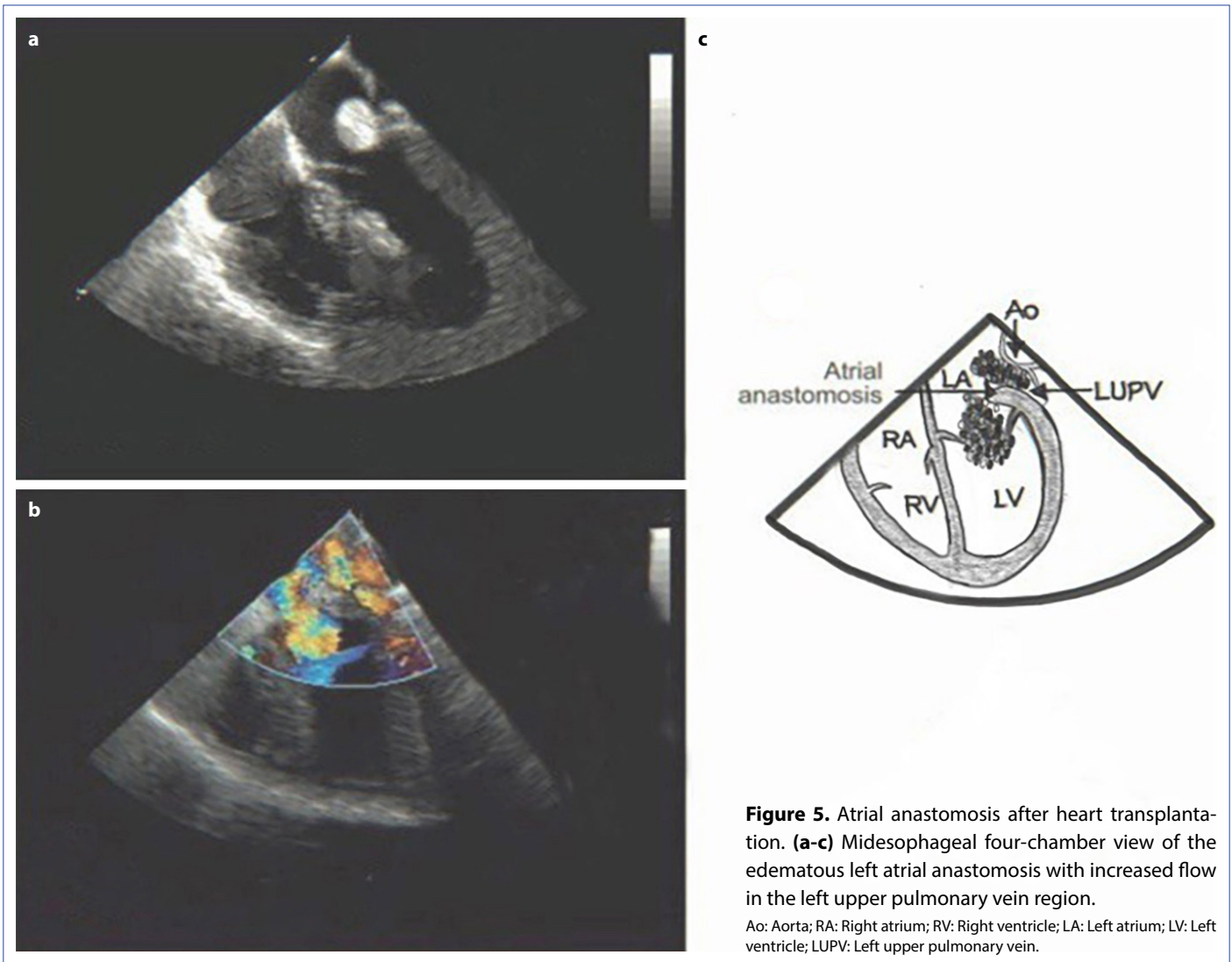RV: Right ventricle; LV: Left ventricle; RA: Right atrium; LA: Left atrium; TV: Tricuspid valve.

measurement of diastolic function has not been able to replace endomyocardial biopsy, it can be performed in addition to this examination.[24]

Although there is no rejection in the early postoperative period after heart transplantation, diastolic function may change. In addition, although the initial interrupted flow Doppler evaluation of mitral flow shows restrictive features, it turns into a nonrestrictive pattern after 6 weeks, and postoperative diastolic function parameters improve and left heart filling pressures decrease.[25] Persistent severe diastolic dysfunction with restrictive filling 6 months after transplantation has been associated with shorter survival times, which is independent of graft rejection.[26]

The bicaval technique ensures the preservation of normal left atrial shape and dimensions, allowing ventricular filling dynamics to continue closer to normal physiology.[24] Ventricular filling is more affected by the mechanical activity of the residual atrial tissue in the recipient than by the donor heart atrium. In addition, parasystolic contraction of this tissue also alters pulmonary venous flow. Contraction of the receptive atrial tissue in late systole results in an increase in the diastolic component (D wave), and if it occurs in early systole, the systolic component (S wave) decreases.[27]

The right ventricle begins to remodel in the postoperative period, adapting to the patient's pulmonary pressure. The initially enlarged and then dilated right ventricle returns to its perioperative dimensions 1 year after surgery. As the PVR decreases to normal levels, the right ventricular wall thickness does not increase significantly. In the first year, the right ventricle remains slightly enlarged, possibly due

Figure 5. Atrial anastomosis after heart transplantation. **(a-c)** Midesophageal four-chamber view of the edematous left atrial anastomosis with increased flow in the left upper pulmonary vein region.

Ao: Aorta; RA: Right atrium; RV: Right ventricle; LA: Left atrium; LV: Left ventricle; LUPV: Left upper pulmonary vein.
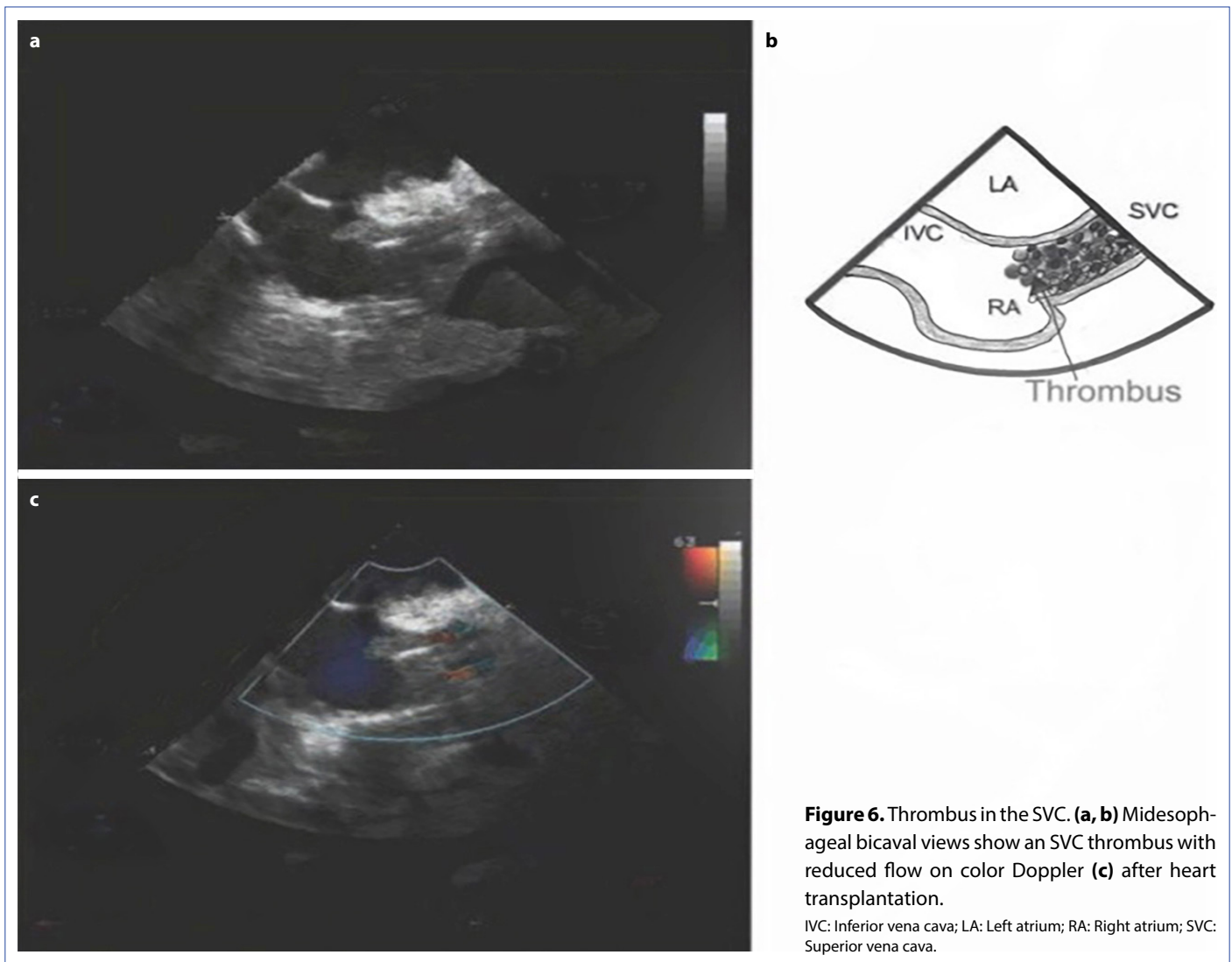
to chronic volume overload. The thickness of the wall and mass of the left ventricle typically increase with volume and ejection fractions within normal limits. The transplanted heart contracts by turning anteromedially during systole.[21]

Although fluoroscopy is used more frequently when performing endomyocardial biopsy for the follow-up of graft rejection, echocardiography is also a good alternative, especially in children and pregnant women, who avoid radiation exposure. In addition, because echocardiography provides better visualization of the soft tissue, injury to the tricuspid valve and papillary muscles is less common, and complications such as tamponade are noticed earlier.[19]

Evaluation of atrial functions, great vessels, and anastomoses

TEE can be used to evaluate anastomoses and great vessels immediately after transplantation. Left atrial anastomosis appears as a prominent protrusion on the posterior wall of the left atrium (Fig. 5). A thrombus may occur in the biatrial technique, especially in the left atrial appendage, due to the enlargement of the atrium.[28–31] A stenotic left atrial anastomosis can be diagnosed by imaging the left atrium in the midesophageal view. This may lead to flow obstruction (acquired core triatriatum); as a result, pulmonary venous hypertension and right ventricular failure may develop.[32] The presence of the atrial suture line, the portion of the recipient atrium that has limited contribution to contractility; increased atrial size; and asynchrony between the donor and recipient atria can also cause stasis and thrombosis. In this case, spontaneous atrial echo contrast (55%) is seen on TEE. Left atrial thrombus, which is often overlooked in transthoracic echocardiography, is detected in 38% of TEEs. Episodes of arterial thromboembolism are detected in 22% of patients with spontaneous echocardiographic contrast and left atrial thrombus.[33] The use of a modified bicaval surgical technique may reduce this incidence.[34]

Figure 6. Thrombus in the SVC. **(a, b)** Midesophageal bicaval views show an SVC thrombus with reduced flow on color Doppler **(c)** after heart transplantation.

IVC: Inferior vena cava; LA: Left atrium; RA: Right atrium; SVC: Superior vena cava.

TEE is superior to transthoracic echocardiography in demonstrating atrial septal thickening, swelling in the recipient and donor atria, and shunt at the atrial level.[33] TEE can also be used to determine patent foramen ovale after heart transplantation.[35]

Stenosis and/or thrombus may develop at anastomotic sites in great vessels (Fig. 6). In these regions, narrowing should not be observed in two-dimensional echocardiography, laminar flow should be observed in color Doppler, and no systolic gradient should be detected in continuous waves. Doppler flow rates should be measured within normal limits, unless there is incompatibility between donor and recipient aorta and pulmonary arteries.[19] Normal pulmonary artery Doppler velocity is <1 m/s, and normal ascending aortic Doppler velocity is <1.4 m/s.[36] Right ventricular dysfunction may occur as a result of diameter mismatch, narrowing of the sutures, torsion, or bending of the anastomosed pulmonary arteries.[20]

Studies have reported superior and inferior cava stenosis, especially in the bicaval technique. SVC stenosis to cerebral edema or SVC syndrome; IVC stenosis may cause postoperative liver and kidney failure.[37,38] If atrial enlargement is present, the pulmonary veins can be difficult to visualize.

Aortic dissection is a rare but catastrophic complication in the posttransplant period. Although it is usually seen in the recipient aorta, Lopez et al.[39] reported that dissection might also occur in the donor aorta, and early diagnosis with TEE allows for early intervention.

Valve regurgitation

TR is common after heart transplantation due to preload mismatch and right ventricular dilatation. In addition, it is the most common valve pathology after surgery. Depending on the diagnostic criteria and timing, the incidence of TR may be as high as 84%.[40–43] TR can occur due to geometric annular deformation (which is more common in the

biatrial technique) and annular dilatation in the presence of right ventricular dysfunction in the period immediately after transplantation.[40,44] It is generally well tolerated.[19]

Although TR is generally associated with the biatrial technique and graft vasculopathy, it may also be caused by pulmonary hypertension, right ventricular dilatation, and endomyocardial biopsies, causing endocarditis or chordal damage.[45] Significant development of TR may cause right heart failure, and valve repair or tricuspid replacement surgery may be required.[41] Bishawi et al.[46] showed that after weaning from CPB, 21% of patients had moderate or severe TR, and in 91% of these patients, this regurgitation regressed to a complete disappearance or to a mild level. Only 1% of patients required surgical tricuspid valve repair. Postoperative severe TR is a cause of mortality, with a rate of 62.5%.[45]

The incidence of mild or moderate MR ranges from 55% to 87%, without serious clinical findings.[21,28] It usually occurs without a structural abnormality in the mitral valve or left ventricular dysfunction because of increased posterior mitral valve tension resulting from biatrial enlargement, incomplete closure of the valve, and annuloventricular incompatibility. In addition, the presence of donor and recipient atria and sinus nodes contributes to MR formation by causing asynchrony and irregular atrial contraction.[21] Finally, MR may develop as a result of left ventricular outflow obstruction.

Pulmonary regurgitation (42%) and aortic regurgitation (23%) can also be seen after heart transplantation.[40]

Pericardial effusion

Pericardial effusion occurs at a rate of 85% after heart transplantation.[47] Besides the absence of lymphatic drainage, this can be caused by the difference in size between the pericardial cavity and the donor heart. It usually does not progress to a serious level as tamponade. A slow accumulating effusion may cause minimal hemodynamic deterioration, whereas loculated fluid in a critical area may cause acute cardiac tamponade. Therefore, a weak correlation is observed between effusion size and clinical symptoms.[24] Most effusions disappear completely or almost completely 1 month after surgery.[47]

Coronary arteries

After heart transplantation, coronary fistulas, which can occur iatrogenically in 5% to 15% of patients, can be detected by TEE. Congenital coronary artery fistula is rarer. Its higher incidence in transplant patients has been attributed to right ventricular endomyocardial biopsies and is usually seen in the right coronary artery. Most of these fistulas open directly into the right ventricle and are detected during routine coronary angiography without significant hemodynamic findings.[48]

In summary, TEE may contribute to the evaluation of heart transplant patients. In addition to its use in perioperative monitoring of these patients, TEE can be adopted for assessing donor organs. The timely assessment of cardiac anatomy and physiology during the initial perioperative period and the detection of allograft rejection in the postoperative period are possible with TEE.

References

1. Goldraich LA, Leitão SAT, Scolari FL, Marcondes-Braga FG, Bonatto MG, Munyal D, et al. A comprehensive and contemporary review on ımmunosuppression therapy for heart transplantation. Curr Pharm Des 2020;26:3351–84.

2. Chambers DC, Cherikh WS, Harhay MO, Hayes D Jr, Hsich E, Khush KK, et al. The international thoracic organ transplant registry of the international society for heart and lung transplantation: Thirty-sixth adult lung and heart-lung transplantation Report-2019; Focus theme: Donor and recipient size match. J Heart Lung Transplant 2019;38:1042–55.

3. Thaden JJ, Malouf JF, Rehfeldt KH, Ashikhmina E, Bagameri G, Enriquez-Sarano M, et al. Adult intraoperative echocardiography: A comprehensive review of current practice. J Am Soc Echocardiogr 2020;33:735–55.e11.

4. Lower RR, Shumway NE. Studies on orthotopic homotransplantation of the canine heart. Surg Forum 1960;11:18–9

5. Shumway NE, Lower RR, Stofer RC. Transplantation of the heart. Adv Surg 1966;2:265–84.

6. Dreyfus G, Jebara V, Mihaileanu S, Carpentier AF. Total orthotopic heart transplantation: An alternative to the standard technique. Ann Thorac Surg 1991;52:1181–4.

7. American Society of Anesthesiologists and Society of Cardiovascular Anesthesiologists Task Force on Transesophageal Echocardiography. Practice guidelines for perioperative transesophageal echocardiography. An updated report by the American society of anesthesiologists and the society of cardiovascular anesthesiologists task force on transesophageal echocardiography. Anesthesiology 2010;112:1084–96.

8. Vandenbriele C, Brouckaert J, Hans G, Tchana-Sato V, Vandendriessche K, Gunst J, et al. The role of transesophageal echocardiography in guiding heart donation after circulatory death. Clin Transplant 2022;36:e14783.

9. Arain FD, Sohn J, Graber RG. Persistent left superior vena cava in a heart transplant recipient's native heart: A rare case diagnosed by transesophageal echocardiogram. Semin Cardiothorac Vasc Anesth 2020;24:374–7.

10. Salgado Filho MF, Siciliano A, Siciliano A, Oliveira AJ, Salgado J, Palitot I. The importance of transesophageal echocardiography in heart harvesting for cardiac transplantation. Rev Bras Anestesiol 2012;62:262–8.

11. Venkateswaran RV, Townend JN, Wilson IC, Mascaro JG, Bonser RS, Steeds RP. Echocardiography in the potential heart donor. Transplantation 2010;89:894–901.

12. Dronavalli VB, Banner NR, Bonser RS. Assessment of the potential heart donor: A role for biomarkers? J Am Coll Cardiol 2010;56:352–61.

13. Tan Z, Roscoe A, Rubino A. Transesophageal echocardiography in heart and lung transplantation. J Cardiothorac Vasc Anesth 2019;33:1548–58.

14. Nicoara A, Skubas N, Ad N, Finley A, Hahn RT, Mahmood F, et al. Guidelines for the use of transesophageal echocardiography to assist with surgical decision-making in the operating room: A surgery-based approach: From the American society of echocardiography in collaboration with the society of cardiovascular anesthesiologists and the society of thoracic surgeons. J Am Soc Echocardiogr 2020;33:692–734.

15. Kransdorf EP, Mehta HS, Shah KB, Vucicevic D, DePasquale EC, Goldraich L, et al. ISHLT transplant registry: Youthful investment-the path to progress. J Heart Lung Transplant 2017;36:1027–36.

16. Stainback RF, Estep JD, Agler DA, Birks EJ, Bremer M, Hung J, et al. Echocardiography in the management of patients with left ventricular assist devices: Recommendations from the American Society of Echocardiography. J Am Soc Echocardiogr 2015;28:853–909.

17. Nicoara A, Ruffin D, Cooter M, Patel CB, Thompson A, Schroder JN, et al. Primary graft dysfunction after heart transplantation: Incidence, trends, and associated risk factors. Am J Transplant 2018;18:1461–70.

18. Segovia J, Cosío MD, Barceló JM, Bueno MG, Pavía PG, Burgos R, et al. RADIAL: A novel primary graft failure risk score in heart transplantation. J Heart Lung Transplant 2011;30:644–51.

19. Badano LP, Miglioranza MH, Edvardsen T, Colafranceschi AS, Muraru D, Bacal F, et al. European Association of Cardiovascular Imaging/Cardiovascular Imaging Department of the Brazilian Society of Cardiology recommendations for the use of cardiac imaging to assess and follow patients after heart transplantation. Eur Heart J Cardiovasc Imaging 2015;16:919–48.

20. González Saldivar H, Hortal J, Martín de Miguel I, Rodríguez-Abella H, Las CJ, Martínez-Sellés M. A reversible cause of acute right ventricular failure after heart transplant. Rev Esp Cardiol (Engl Ed) [Article in English, Spanish] 2017;70:677–9.

21. Bhatia SJ, Kirshenbaum JM, Shemin RJ, Cohn LH, Collins JJ, Di Sesa VJ, et al. Time course of resolution of pulmonary hypertension and right ventricular remodeling after orthotopic cardiac transplantation. Circulation 1987;76:819–26.

22. Kaczorowski DJ, Datta J, Kamoun M, Dries DL, Woo YJ. Profound hyperacute cardiac allograft rejection rescue with biventricular mechanical circulatory support and plasmapheresis, intravenous immunoglobulin, and rituximab therapy. J Cardiothorac Surg 2013;8:48.

23. Morita Y, Lencho T, Gunasekaran S, Modak R. Modified tricuspid annular plane systolic excursion using transesophageal echocardiography and its utility to predict postoperative course in heart transplantation and left ventricular assist device implantation. J Cardiothorac Vasc Anesth 2018;32:1316–24.

24. Burgess MI, Bhattacharyya A, Ray SG. Echocardiography after cardiac transplantation. J Am Soc Echocardiogr 2002;15:917–25.

25. StGoar FG, Gibbons R, Schnittger I, Valantine HA, Popp RL. Left ventricular diastolic function. Doppler echocardiographic changes soon after cardiac transplantation. Circulation 1990;82:872–8.

26. Ross HJ, Gullestad L, Hunt SA, Tovey DA, Puryear JB, McMillan A, et al. Early Doppler echocardiographic dysfunction is associated with an increased mortality after orthotopic cardiac transplantation. Circulation 1996;94(Suppl 9):II289–93.

27. Durennes ME. E´ chocardiographie transoesophagienne chez le transplante´ cardiaque. In: Cohen A, editor. E´chocardiographie transoesophagienne en cardiologie et en anesthe´sie. France: Edition Arnette; 1992. p.265–79.

28. Angermann CE, Spes CH, Tammen AR, Stempfle HU, Kemkes BM, Schütz A, et al. Transesophageal echocardiography after orthotopic heart transplantation. Int J Card Imaging 1990;5:271–4.

29. Ishizuka N, Nakamura K, Fujita Y, Tanimoto K, Niki K, Hosoda S, et al. Transesophageal echocardiographic findings in patients after heart transplantation. J Cardiol [Article in Japanese] 1997;29:163–70.

30. Polanco G, Jafri SM, Alam M, Levine TB. Transesophageal echocardiographic findings in patients with orthotopic heart transplantation. Chest 1992;101:599–602.

31. Wilhelmi M, Pethig K, Wilhelmi M, Nguyen H, Strüber M, Haverich A. Heart transplantation: Echocardiographic assessment of morphology and function after more than 10 years of follow-up. Ann Thorac Surg 2002;74:1075–9.

32. Oaks TE, Rayburn BK, Brown ME, Kon ND. Acquired cor triatriatum after orthotopic cardiac transplantation. Ann Thorac Surg 1995;59:751–3.

33. Derumeaux G, Mouton-Schleifer D, Soyer R, Saoudi N, Cribier A, Letac B. High incidence of left atrial thrombus detected by transoesophageal echocardiography in heart transplant recipients. Eur Heart J 1995;16:120–5.

34. Riberi A, Ambrosi P, Habib G, Kreitmann B, Yao JG, Gaudart J, et al. Systemic embolism: A serious complication after cardiac transplantation avoidable by bicaval technique. Eur J Cardiothorac Surg 2001;19:307–12.

35. Ouseph R, Stoddard MF, Lederer ED. Patent foramen ovale presenting as refractory hypoxemia after heart transplantation. J Am Soc Echocardiogr 1997;10:973–6.

36. Vegas A. Preoperative two-dimensional transesophageal echocardiography: A practical handbook. Germany: Springer; 2012.

37. Blanche C, Tsai TP, Czer LS, Valenza M, Aleksic I, Trento A. Superior vena cava stenosis after orthotopic heart transplantation: Complication of an alternative surgical technique. Cardiovasc Surg 1995;3:549–52.

38. Jacobsohn E, Avidan MS, Hantler CB, Rosemeier F, De Wet CJ. Case report: Inferior vena-cava right atrial anastomotic stenosis after bicaval orthotopic heart transplantation. Can J Anaesth 2006;53:1039–43.

39. Lopez CE, Lopez MJ, de Diego J, Cortina JM. Early acute aortic dissection of the donor aorta after orthotopic heart transplantation. Interact Cardiovasc Thorac Surg 2009;9:715–6.

40. Cladellas M, Oriol A, Caralps JM. Quantitative assessment of valvular function after cardiac transplantation by pulsed Doppler echocardiography. Am J Cardiol 1994;73:1197–201.

41. Kwon MH, Shemin RJ. Tricuspid valve regurgitation after heart transplantation. Ann Cardiothorac Surg 2017;6:270–4.

42. Wong RC, Abrahams Z, Hanna M, Pangrace J, Gonzalez-Stawinski G, Starling R, et al. Tricuspid regurgitation after cardiac transplantation: An old problem revisited. J Heart Lung Transplant 2008;27:247–52.

43. Chan MC, Giannetti N, Kato T, Kornbluth M, Oyer P, Valantine HA, et al. Severe tricuspid regurgitation after heart transplantation. J Heart Lung Transplant 2001;20:709–17.

44. Koch A, Remppis A, Dengler TJ, Schnabel PA, Hagl S, Sack FU. Influence of different implantation techniques on AV valve competence after orthotopic heart transplantation. Eur J Cardiothorac Surg 2005;28:717–23.

45. Berger Y, Har Zahav Y, Kassif Y, Kogan A, Kuperstein R, Freimark D, et al. Tricuspid valve regurgitation after orthotopic heart transplantation: Prevalence and etiology. J Transplant 2012;2012:120702.

46. Bishawi M, Zanotti G, Shaw L, MacKenzie M, Castleberry A, Bartels K, et al. Tricuspid valve regurgitation immediately after heart transplant and long-term outcomes. Ann Thorac Surg 2019;107:1348–55.

47. Weitzman LB, Tinker WP, Kronzon I, Cohen ML, Glassman E, Spencer FC. The incidence and natural history of pericardial effusion after cardiac surgery--an echocardiographic study. Circulation 1984;69:506–11.

48. Lowry RW, Young JB, Kleiman NS, Zoghbi WA. Transesophageal echocardiographic demonstration of right coronary artery-to-coronary sinus fistula in a heart transplant recipient. J Am Soc Echocardiogr 1993;6:449–52.