Chapter 10
Tricuspid Annulus Measurements: Dynamic Changes in Health and Disease

Denisa Muraru and Luigi P. Badano

Abstract The most common cause of tricuspid regurgitation (TR) is the dilation of tricuspid annulus (TA). In patients with left-sided valve disease, an enlarged TA is currently regarded as a more reliable sign of associated tricuspid valve (TV) dysfunction, reflecting better the chronicity and the hemodynamic consequences of TR on TV apparatus over time than the actual severity of TR, which may vary significantly with loading conditions, respiration and technical factors. This paradigm shift has led to the more liberal indications for surgical annuloplasty at the time of left-sided valve surgical intervention and to the adoption of "prophylactic" TV annuloplasty if the TA diameter exceeds 40 mm (or 21 mm/m^2 body surface area), irrespective of TR severity. Due to limitations of conventional two-dimensional echocardiography in assessing maximal TA diameter, three-dimensional echocardiography is emerging as the future standard technique for accurately measuring the TA size. This chapter is focused on the characterization of TA geometry and function as a key component for maintaining the competency of the normal TV, and on its role in the pathophysiology and development of functional TR.

Keywords Tricuspid valve • Tricuspid annulus • Tricuspid regurgitation • Tricuspid insufficiency • Echocardiography • Three-dimensional echocardiography

Introduction

Since the '80s, it has been recognized that the most common cause of tricuspid regurgitation (TR) is not the intrinsic abnormality of the tricuspid valve (TV) itself, but the dilation of tricuspid annulus (TA) [1]. The awareness of the crucial role of TA dilation in the pathophysiology of functional TR has led to a renewed interest in the assessment of TA and to its identification as the main therapeutic target in symptomatic patients with significant TR. Unlike TR severity, TA size does not change as

D. Muraru, M.D., Ph.D. (✉) • L.P. Badano, M.D., Ph.D.
Department of Cardiac, Thoracic and Vascular Imaging, University of Padua, Padua, Italy
e-mail: denisa.muraru@gmail.com

O.I. Soliman, F.J. ten Cate (eds.), *Practical Manual of Tricuspid Valve Diseases*,
DOI 10.1007/978-3-319-58229-0_10

swiftly and, once dilated, will not return to normal [2]. In patients with left-sided valve diseases, an enlarged TA is currently regarded as a more reliable sign of associated TV dysfunction, reflecting better the chronicity and the hemodynamic consequences of TR on TV apparatus over time than the actual severity of TR. The latter may vary significantly with loading conditions, respiration and technical factors [3]. This paradigm shift coupled with solid evidences showing a high mortality risk in case of redo surgery to treat late TR, have led to the more liberal indications for surgical annuloplasty in the recent guidelines and to the adoption of "prophylactic" TV annuloplasty at the time of left-sided valve surgical intervention by many surgeons. Finally, most of the current devices for percutaneous treatment of TR in high surgical risk patients act by downsizing the TA through various methods [4].

This chapter focuses on the TA geometry and function as a key component of the TV apparatus for maintaining the competency of normal TV, and on its role in the pathophysiology and development of functional TR. A thorough understanding of these mechanisms has profound implications for the imaging assessment of TV and to develop effective treatment options for patients with functional TR.

Normal Tricuspid Annulus: Size, Shape and Dynamics

Similar to the mitral annulus, the TA has a complex elliptical and saddle-shaped morphology [3]. Non-planar TA has higher points oriented in antero-posterior direction (i.e. anterior high point adjacent to the aortic valve and posterior high point at 180°) and lower points in medio-lateral direction [5] (Fig. 10.1).

It is worth noting, however, that the knowledge we have accumulated on the mitral annulus shape and dynamics does not apply to the TA, due to significant

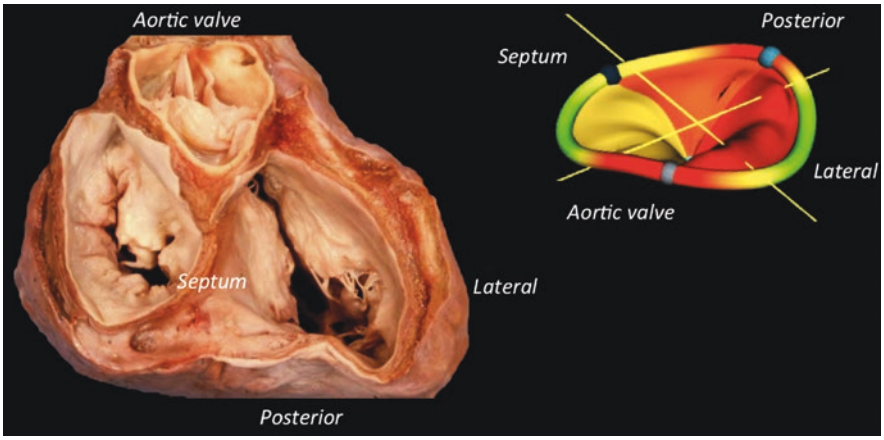

Fig. 10.1 Heart specimen (*courtesy of Prof Cristina Basso, Cardiac Pathology, University of Padua*) seen from atrial perspective, showing the tricuspid valve morphology and its spatial relationships with surrounding structures. At the *right*, a three-dimensional model of a normal tricuspid valve obtained by 3D echocardiography, illustrating the non-planar shape of tricuspid annulus (high annular points are color-coded in *red*, while low annular points are represented in *green* color; *yellow leaflet*—septal; *red leaflet*—anterior; *orange leaflet*—posterior)

differences in the anatomy and function, and in the spatial anatomic relationships of these two valves. For instance, mitral valve has two fibrous trigons (a right and a left one), while the TV has only one right fibrous trigon (Fig. 10.1). Between the two trigons, the mitral annulus has a fibrous part in continuity with the aortic valve, which is not surrounded by myocardium, possibly explaining the smaller systolic shortening of the mitral annulus area (28% on average) [6] in comparison with TA area (35%), which is surrounded by myocardium to a larger circumferential extent. In normal hearts, the mitral annulus is smaller and has a systolic longitudinal excursion towards the apex, while the TA is larger and has a more active and complex motion, combining longitudinal displacement and tilting (larger displacement at the free wall side than at the septal side). All these differences suggest that the normal anatomy and pathophysiology of TV should be understood and evaluated on its own, without translating prior knowledge derived from the mitral valve studies.

Using 2D echocardiography, the normal TA diameter in adults is 28 ± 5 mm and significant dilation is defined by a diastolic diameter >40 mm or >21 mm/mm^2 in the apical four-chamber view. Of note, it has been demonstrated that 2D echocardiography underestimates the maximal dimension of TA in comparison with 3D echocardiography (Fig. 10.2), cardiac magnetic resonance (CMR) and multidetector computed tomography (MDCT) measurements [7–9].

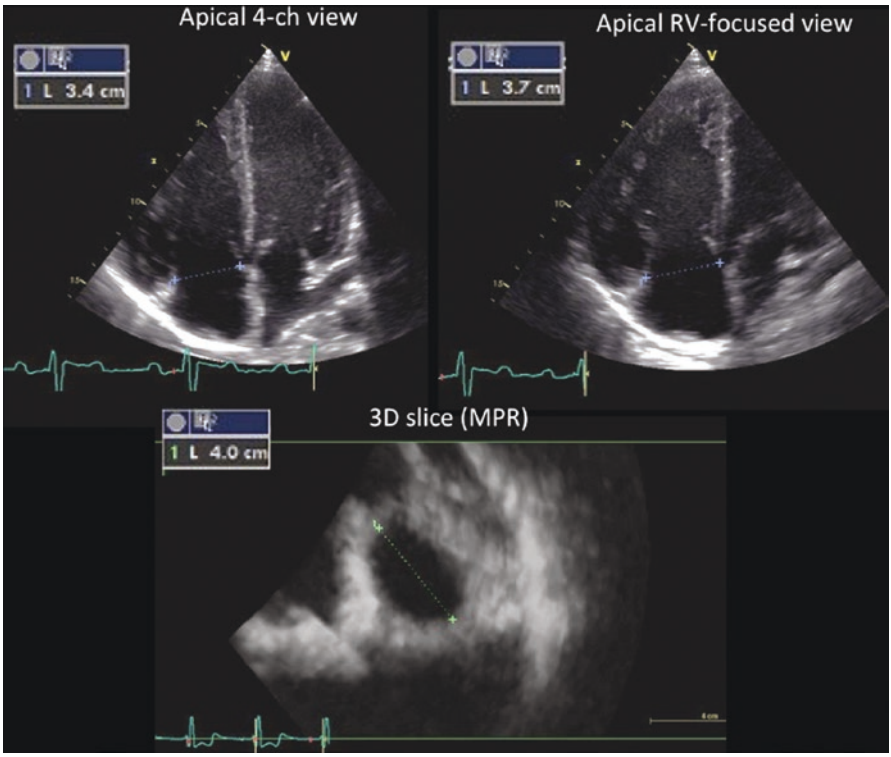

Fig. 10.2 Measurements of tricuspid annulus diameter by 2D echocardiography (**a**, **b**) and 3D echocardiography (**c**) in a patient with pulmonary arterial hypertension and functional tricuspid regurgitation. 2D echocardiography underestimated the maximal size of tricuspid annulus (particularly in the standard four-chamber view), with respect to the measurement obtained by 3D echocardiography

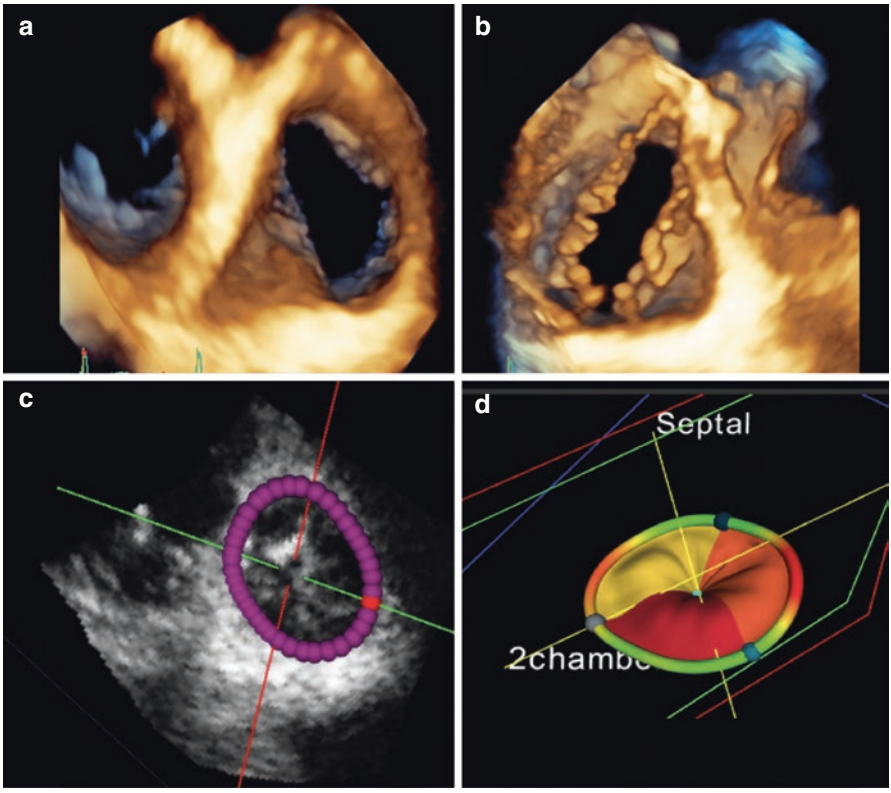

Fig. 10.3 Tricuspid valve anatomy by transthoracic 3D echocardiography in a patient with functional tricuspid regurgitation, as seen from atrial (**a**) and ventricular (**b**) perspective. Using custom prototype software (*courtesy of Federico Veronesi, University of Bologna, Italy*), the tricuspid annulus (**c**, **d**) and leaflets (**d**) geometry can be semi-automatically quantified

Using transthoracic 3D echocardiography, maximal and minimal linear dimensions of normal TA are 42 ± 5 mm (22 ± 2 mm/m^2) and 36 ± 5 mm (19 ± 3 mm/m^2). TA circularity (minimum:maximum diameters) is around 0.85 in late diastole, reflecting its elliptical shape (Fig. 10.3). Furthermore, normal values of TA geometry are 12 ± 1 cm^2 (6 ± 1 cm^2/m^2) for maximal area at late diastole, 12 ± 1 cm (7 ± 1 cm/m^2) for maximal perimeter, while annular height between the highest and lowest point is around 7 mm (Fig. 10.1) [10, 11].

In healthy subjects, TA size and shape change significantly during the cardiac cycle. On average, TA linear dimensions and perimeter show >20% systolic shortening, while TA area shrinks by 35% during the cardiac cycle. TA area reaches a minimum in mid-to-late systole, then increases during isovolumic relaxation and diastole reaching a maximum value in late diastole after the onset of atrial contraction (end of P-wave) [5] (Fig. 10.4). Of note, the most significant reduction in TA size occurs in the pre-systolic phase of the cardiac cycle (after right atrial contraction and during isovolumic right ventricular contraction), with subsequent shortening during the first

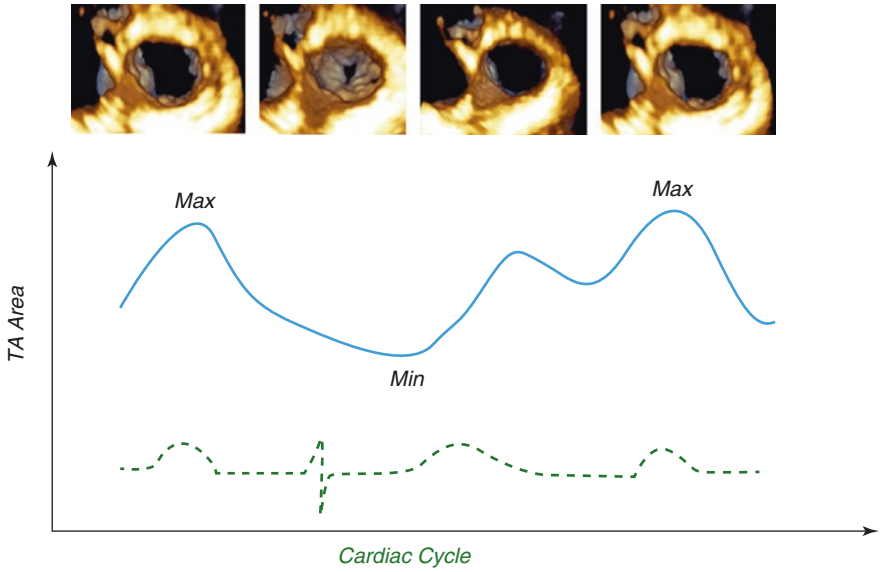

Fig. 10.4 Dynamic changes of tricuspid annulus (TA) area during the cardiac cycle. The maximal annular area is reached in late diastole, and the minimum area is reached during mid-late systole

part of systole. As seen in cross-section, TA shape becomes more circular during systole, and returns to more elliptical shape during diastole due to a relatively greater increase in antero-posterior dimension than in septo-lateral dimension.

TA size and function depend on gender, women having smaller and more dynamic TAs than men. However, indexing TA area by body size (i.e. body surface area) practically eliminates the differences between genders [11].

TA size depends also on the dimensions of right heart chambers, being more closely correlated with right atrial, than with right ventricular volumes [11, 12].

Changes in Tricuspid Annulus Geometry and Function in Functional Tricuspid Regurgitation

TA dilation, leaflet tethering, or both, can lead to secondary or functional TR.

With functional TR, the TA becomes larger, flatter and more circular [5]. The annulus becomes more circular with TR worsening due to the dilation of the TA preferentially along its free wall distance. Specifically, there is greater enlargement of the TA antero-posterior diameter (antero-septal commissure to posterior leaflet distance) than the medio-lateral diameter (septal-to-anterior leaflet distance) [5] (Fig. 10.5). This is likely due to the anterior high point of the TA being adjacent to the fibrous skeleton of the heart, providing more resistance to dilation than along the free wall. The ratio between the antero-posterior dimension and the annular height

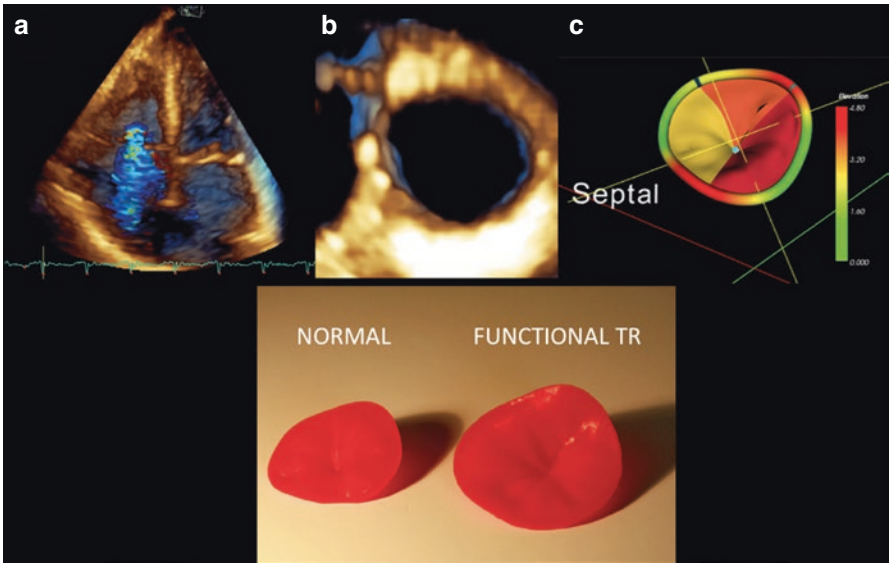

Fig. 10.5 Tricuspid annulus remodelling in severe functional tricuspid regurgitation (TR), as depicted by 3D echocardiography (**a**–**c**) and 3D printed models: the annulus becomes larger, rounder and flatter in comparison with normal annulus geometry

is a dimensionless index reflecting the TA remodelling ("stretch") in dysfunctional TV, which increases markedly in functional TR in comparison with controls (13 ± 5 vs. 4 ± 1, $p < 0.0001$) and together with TA area and circularity, are independent determinants of functional TR severity [5].

TA dilation is a constant feature in patients with functional TR and is poorly correlated with the severity of TR. Annular dilation is a reliable marker of TV dysfunction and a sign that the valve is prone to leak later on. In contrast with functional TR which may improve after the reduction of pulmonary pressures (i.e. after mitral valve surgery or after thromboendarterectomy in patients with chronic thromboembolic pulmonary hypertension [2]), TA dilation persists and will not return to normal values. The persistence of TA dilatation may explain why almost 40% of patients who underwent mitral valve surgery successfully may develop severe functional TR several years later despite absence of pulmonary hypertension [13]. However, almost 1 out of 2 patients *without* significant TA dilation at the time of mitral valve surgery (≥70 mm intraoperatively in Dreyfus series [14]) will still develop late TR, suggesting either that intraoperative TA diameter is an imperfect predictor of late TR, or that there are multiple contributing factors to TR late development in addition to TA size.

Functional TR is frequently associated with advanced stages of left-sided valve disease, myocardial or pulmonary diseases leading to increased pulmonary pressures, right ventricular dilation and/or dysfunction [3, 15]. Thus, it is an established belief that the dilation of right ventricle is the first mandatory step towards the TR development and univocally responsible for TA enlargement, even before significant TR is present [16]. However, this theory does not explain the occurrence of functional TR in chronic atrial fibrillation or its relatively low incidence in some conditions evolving with significant right ventricular dilatation (corrected tetralogy of Fallot, arrhythmogenic

cardiomyopathy etc) [17, 18] . Using 3D echocardiography-derived measurements of right heart chambers and TA in 59 patients with functional TR of various etiologies, we have found that right atrial volume was actually the most consistent determinant of TA area [19]. This may explain, at least in part, the onset of FTR in patients with dilated right atrium, but relatively small right ventricle (atrial fibrillation subgroup), and the absence of significant regurgitation in those with severely enlarged right ventricles, but preserved right atrial volumes (tetralogy of Fallot subgroup).

In patients with functional TR, there is also an impairment of TA "sphincteric" function and dynamics, with a significant decrease (up to 50%) in the systolic shortening of TA area and diameters [5].

Measurement of Tricuspid Annulus Dilation

Despite its importance for the diagnosis and therapy of functional TR, the sizing of TA remains an elusive goal. The best methodology for the noninvasive measurement of TA size is not clearly defined, and present thresholds for surgical annuloplasty based on TA diameter by conventional echocardiography are actually supported by limited evidence and have been questioned by several recent papers [20, 21].

So far, several approaches have been used to size the TA *in vivo* in TR studies. Dreyfus *et al.* have proposed the intraoperative sizing of TA from the antero-septal commissure to the antero-posterior commissure, and the 70 mm cut-off for defining significant TA dilation [14]. Performing an annuloplasty in all patients with TA above this threshold improved New York Heart Association functional class and prevented progression of functional TR at long-term follow-up. This approach was deemed reliable and reproducible by Dreyfus and coworkers [14], yet it has been criticized by others [20] due to its large variability with various degrees of TA stretching since the heart is unloaded and still during surgery.

Dreyfus et al. suggested that the intraoperative 70 mm diameter threshold corresponds to a 40 mm diameter measured in diastole by transthoracic 2D echocardiography between mid-anterior and mid-septal annulus in apical 4-chamber view [14, 22] (Fig. 10.2). This assumption actually has never been demonstrated experimentally and has been challenged recently by several recent 3D echocardiography studies showing that: (1) TA diameter in conventional 4-chamber view may represent the distance between septal leaflet and either anterior or posterior leaflet [23, 24] (Fig. 10.5); (2) the TA size by 2D echocardiography and surgical measurements correlate only modestly, and the difference between the two is much smaller (roughly 3 mm) [20] than the difference suggested in the seminal papers of Dreyfus GD *et al.* (30 mm) [14]; (3) 1 out of 5 healthy subjects have TA annulus >40 mm and would qualify for annuloplasty based on conventional 2D TA diameter [12]; (4) newer abnormality threshold for TA dilation could be larger—42 mm or 23 mm/m^2 [20].

Since the TA is a dynamic structure with an asymmetric saddle shape, even small variations in the angle of the ultrasound beam (Fig. 10.2) or in the timing of measurements can result in considerable discrepancies in TA size [12]. Although the TA measurement from apical four-chamber view seems to be preferred, being recommended by guidelines and different authors as more feasible and reproducible than

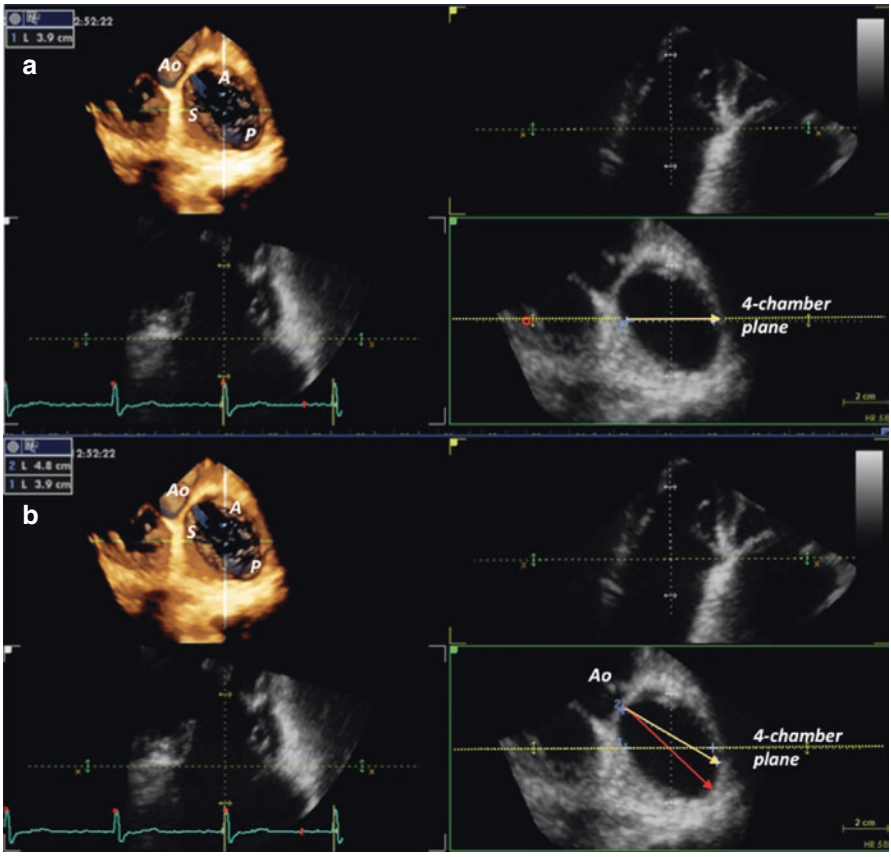

Fig. 10.6 Tricuspid annulus diameter in 4-chamber view (39 mm, panel **a**) is significantly smaller than the antero-posterior diameter (48 mm, *yellow arrow*—panel **b**) and both are smaller than the true maximal diameter of tricuspid annulus (50 mm, *red arrow*—panel **b**). All measurements pertain to the projected tricuspid annulus obtained from a 3D slice (MPR). *A* anterior, *Ao* aortic valve, *P* posterior, *S* septal

other views [8, 20], the best way and timing to measure the TA by conventional echocardiography still remain controversial [22] and require stronger evidence.

Two-dimensional echocardiography, either transthoracic or transesophageal, may underestimate the TA size. 3D echocardiography probably offers a more accurate evaluation of TA dilation by its ability to yield anatomically sound, quantitative measurements such as TA area, as well as true maximal and minimal diameters, irrespective of their spatial orientation [3]. In addition, semi-automated modeling of TV based on 3D echocardiography data offers unique quantitative measures of TA size and leaflet tenting volume, which account for the non-planarity of TA, as well as the asymmetry and inter-subject variability of leaflet size and tethering [25] (Fig. 10.3). Finally, current technology allows to obtain 3D prints of TV models and directly appreciate their different sizes and complex shapes (Figs. 10.6 and 10.7),

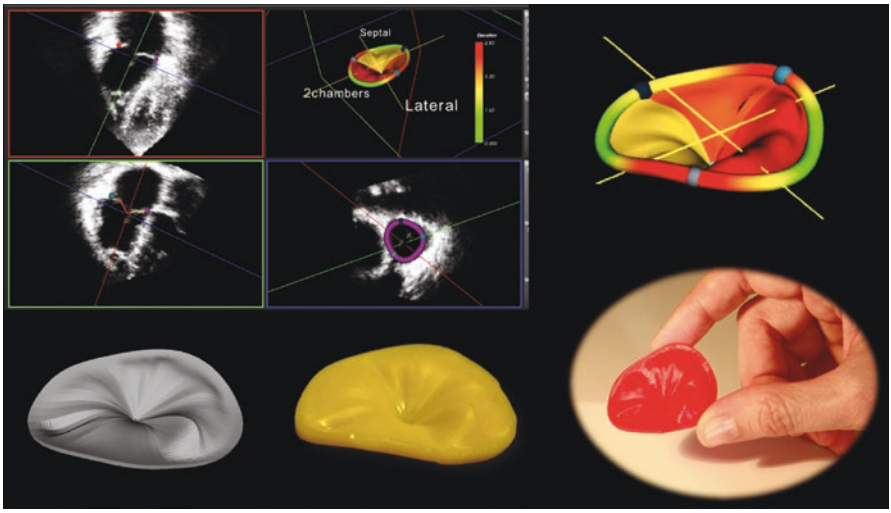

Fig. 10.7 Segmentation of normal tricuspid valve from transthoracic 3D dataset by custom software (*courtesy of Federico Veronesi, University of Bologna, Italy*), that can be used to obtain a 3D printed solid model of the valve

elevating our impressions from textured flat-panel coloured perspectives to actual exploration of the complex geometry of the TV, with the potential to guide personalized care of patients with functional TR [26.]

It can be foreseen that 3D echocardiography will become the standard technique for assessing the TV geometry and accurately measuring TA dimensions [22]. If so, there are a few caveats that have to be considered.

First of all, since 3D echocardiography provides larger values for TA annulus size than 2D echocardiography (Fig. 10.2), specific abnormality thresholds of TA should be used if measurements are obtained by 3D echocardiography. Secondly, due to the non-planarity of TA, software-derived semi-automated measurements (Fig. 10.3) may provide different results than manual measurements of TA diameters and area planimetry performed on a 3D slice of TA (Fig. 10.5). Thirdly, preliminary data using dedicated algorithms specifically developed for TA quantification showed that TA reference values should be gender-specific and indexed by body surface area, suggesting that the "one-size-fits-all" approach suggested for 2D TA diameter no longer holds true for 3DTA measurements. Finally, despite the use of a dedicated software algorithm applied on 3D echocardiographic data is the only way to account for TA non-planarity and peculiar geometry, at present there is no such software commercially available.

Other studies used either multi-detector computed tomography (MDCT) or magnetic resonance imaging (CMR) for measuring the TA size. Interestingly, the anteroposterior TA dimension by MDCT, and not the septal-lateral dimension, was an independent determinant of TR severity [9]. However, their clinical use of CMR and MDCT is limited at present for patients with unsatisfactory quality images.

Tricuspid Annulus Deformation: A "New" Pathophysiologic Entity for TR Development?

By virtue of its ability to image large volumes of the heart including multiple cardiac structures in vivo, 3D echocardiography has recently provided new mechanistic insights regarding the existence of a reciprocal functional interaction ("coupling") between adjacent cardiac structures, such as aortic and mitral valves. Accordingly, it has been demonstrated that mitral valve repair with a rigid annuloplasty ring impacts on the function of untreated aortic valve [27] or that aortic stenosis impacts on mitral valve function through the calcification of aortic-mitral curtain [28].

As opposed to the mitral valve, the TV does not have a clear anatomic continuity with the aortic valve. However, looking at the base of the heart from the atrial perspective, one can appreciate that the posterior margins of the aortic root are wedged between the orifices of both atrio-ventricular valves (mitral and tricuspid, Fig. 10.1). Anatomically, the triangle between the non-coronary and the right coronary aortic sinuses incorporates the membranous septum, which is crossed on its right side by the hinge of the septal TV leaflet (which divides the septum into atrioventricular and interventricular components) [29]. Therefore, a possible geometric and functional interplay between TV and a dilated aortic root cannot be excluded.

Few isolated case reports advance the idea that TV dysfunction (regurgitation and/or stenosis) can rarely occur in acute type A aortic dissection [30], marked aortic tortuosity [31] or unruptured Valsalva sinus aneurysm [32, 33]. The suggested mechanism was the interference with the TV septal leaflet closure by the adjacent dilated aorta.

We recently studied and reported a clinical case of a patient with dilated ascending aorta and moderately-severe functional TR, that was likely due to the extrinsic compression and distortion of TV annulus by a dilated and tortuous aortic root [34] (Fig. 10.8). Using TA quantification by 3D echocardiography, we identified a small and severely elliptical TA which was clearly deformed by the adjacent aortic root, that most probably led to TR development by increasing the intercommissural distance and/or septal leaflet displacement.

Further studies are needed to clarify whether TA deformation by extrinsic compression may be an additional pathophysiological factor involved in the development of functional TR.

Unmet Needs and Future Research Directions

As TA dilation is the main player in the development of functional TR, it makes sense that the research in this area should focus on the quantitation of TA remodelling and function. However, leaflet size and tethering are the other key components that make the TV leak. In the presence of severe tethering of the leaflets, particularly if the leaflets are small, restrictive annuloplasty alone may not resolve functional TR and actually may worsen the regurgitation. In these cases, it may be safer to replace than to repair the TV [35].

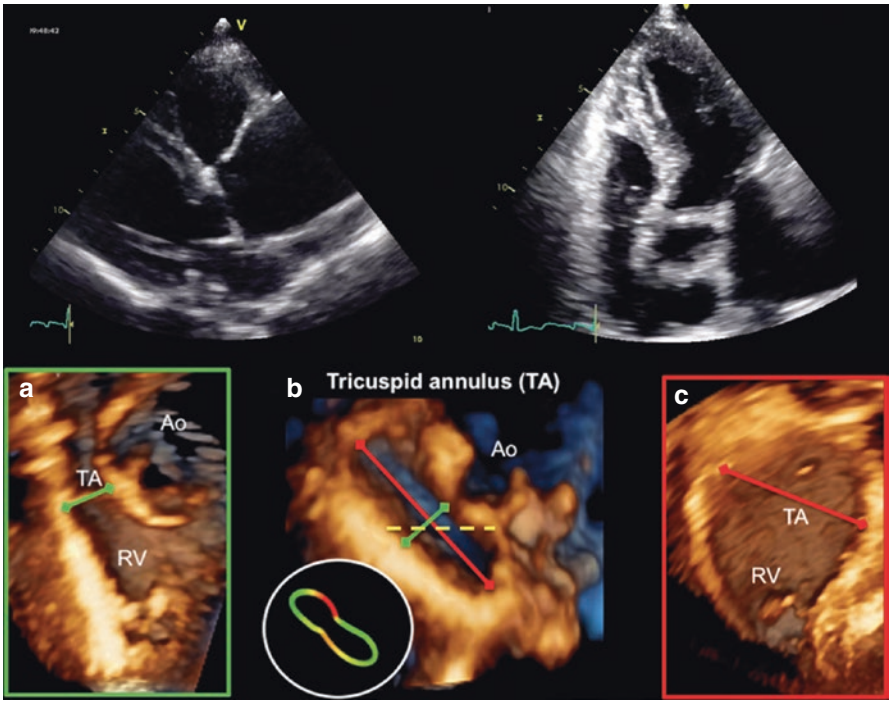

Fig. 10.8 Tricuspid annulus deformation due to extrinsic compression by dilated and tortuous ascending aorta demonstrated by 3D echocardiography, as the most likely cause of valve incompetency in a patient presenting with "idiopathic" tricuspid regurgitation. (**a**) minimal tricuspid annulus diameter (*green*); (**b**) en face view of tricuspid annulus, showing maximal (*red*) and minimal (*green*) diameters, as well as the 4-chamber diameter (*yellow*); (**c**) maximal tricuspid annulus diameter (*red*)

Further studies on the pathophysiology of functional TR, such as the relationships between TA area, extent of leaflets remodeling, right ventricular and atrial volumes and shape, and leaflet tethering are needed. The assessment of the relative contribution of annular dilations versus leaflet tethering to the occurrence of functional TR represents a challenge, which clarification is of critical importance for predicting which patients will benefit from annuloplasty-only repair techniques, and which patients require additional procedures (leaflet augmentation) or different treatment approaches (leaflet clipping, valve replacement, etc).

The various definitions of TA diameters used so far in tomographic imaging studies and the conflicting data regarding the correlation of TA size and TR severity highlights the need for standardization of TA linear measurements according to specific landmarks (mid-leaflet hinges, commissures, etc) which should be tested prospectively in future outcome studies.

Despite the lack of robust data and conflicting evidence, according to the 2012 ESC/EACTS [36] and 2014 AHA/ACC guidelines [37], we should still base our decision to indicate tricuspid annuloplasty on the 40 mm cut-off of TA diameter in apical 4-chamber view. When doing so, we have to be aware of its potential limitations and

that the current indications for the surgical treatment of functional TR are largely based on expert opinion. We definitely need prospective outcome studies and registries to specifically address the indications for functional TR treatment.

Conclusions

The tricuspid annulus is the main player in the development and progression of functional tricuspid regurgitation. Due to its complex, three-dimensional shape and highly dynamic structure, linear measurements of tricuspid annular size using conventional echocardiography have significant limitations, and 3D echocardiography seems more suitable and accurate for this purpose. The design of the future outcome studies and registries should take into account these peculiarities of tricuspid valve quantification, for a better understanding of tricuspid regurgitation pathophysiology and management.

Key Points The most common cause of tricuspid regurgitation is tricuspid annular dilation.

- However, not all functional tricuspid regurgitations can be explained by annulus dilation only; leaflet tethering, pulmonary hypertension and, possibly, tricuspid annulus distortion and dysfunction can also contribute to the development of tricuspid regurgitation.
- Normal tricuspid annulus is a complex-shaped, elliptical and dynamic structure. With progression of functional tricuspid regurgitation, annulus becomes larger, rounder, flatter and less dynamic.
- 3D echocardiography will become the standard technique for accurately measuring the tricuspid annulus dimensions, as conventional echocardiography significantly underestimates its maximal size.
- Normal tricuspid annulus area by 3D echocardiography depends on gender, body size, as well as right atrial and right ventricular volumes.
- Current guidelines advocate for an aggressive early tricuspid annuloplasty at the time of left-sided valve surgery, if the enlarged annulus measures more than 40 mm (or 21 mm/m^2) in diastole in conventional apical four-chamber view. Lack of robust evidence and significant controversy exists around these cut-off values.

Review Questions

Select the Single Best Sentence

58. Which of the following statements about normal tricuspid annulus anatomy is true?

    (a) tricuspid annulus is larger than mitral annulus
    (b) similarly to mitral annulus, tricuspid annulus has two fibrous trigons
    (c) unlike the mitral annulus, tricuspid annulus has a circular shape
    (d) tricuspid annulus is less dynamic than mitral annulus

59. Which of the following statements about tricuspid annulus dilation is false?

    (a) is a more reliable sign of tricuspid valve dysfunction that the regurgitation itself
    (b) may explain the functional tricuspid regurgitation in atrial fibrillation patients
    (c) evolves in a symmetric fashion in all directions
    (d) may be associated with various degrees of leaflet tethering

60. Which is the most reliable sign of tricuspid valve dysfunction?

    (a) tricuspid regurgitation severity
    (b) tricuspid annulus dilation
    (c) tricuspid leaflet tethering
    (d) tricuspid coaptation distance

61. The largest tricuspid valve area is measured:

    (a) in early systole
    (b) in mid-systole
    (c) in early-diastole
    (d) in late diastole

62. Normal absolute tricuspid annulus area in late diastole is around:

    (a) 6 cm^2
    (b) 10 cm^2
    (c) 12 cm^2
    (d) 20 cm^2

63. Regarding the determinants of tricuspid annulus size by 3D echocardiography, which of the following statements is false?

    (a) tricuspid annulus size is smaller in women
    (b) tricuspid annulus size should be indexed by body surface area
    (c) tricuspid annulus size correlates with right ventricular volume, and not with right atrial volume
    (d) tricuspid annulus size correlates with both right atrial and ventricular volumes

64. With respect to 3D echocardiography, the measurements of tricuspid annulus by 2D echocardiography are:

    (a) more accurate
    (b) more reproducible
    (c) do not allow the assessment of tricuspid annulus area
    (d) may vary with the echocardiographic view and probe rotation

65. According to current guidelines, which is the cut-off of tricuspid annulus diameter in 4-chamber view for indicating surgical annuloplasty?

    (a) 35 mm
    (b) 40 mm
    (c) 42 mm
    (d) 70 mm

66. Which of the following imaging methods can be used to size the tricuspid annulus?

    (a) 2D/3D echocardiography
    (b) cardiac magnetic resonance
    (c) multi-detector computerized tomography
    (d) all of the above

67. Which of the following methods is the least accurate for assessing maximal TA diameter?

    (a) cardiac magnetic resonance
    (b) 3D transthoracic echocardiography
    (c) 2D transoesophageal echocardiography
    (d) 2D transthoracic echocardiography

References

1. Tei C, Pilgrim JP, Shah PM, Ormiston JA, Wong M. The tricuspid valve annulus: study of size and motion in normal subjects and in patients with tricuspid regurgitation. Circulation. 1982;66:665–71.
2. Sadeghi HM, Kimura BJ, Raisinghani A, Blanchard DG, Mahmud E, Fedullo PF, et al. Does lowering pulmonary arterial pressure eliminate severe functional tricuspid regurgitation? Insights from pulmonary thromboendarterectomy. J Am Coll Cardiol. 2004;44:126–32.
3. Muraru D, Surkova E, Badano LP. Revisit of functional tricuspid regurgitation; current trends in the diagnosis and management. Korean Circ J. 2016;46:443–55.
4. Taramasso M, Pozzoli A, Guidotti A, Nietlispach F, Inderbitzin DT, Benussi S, et al. Percutaneous tricuspid valve therapies: the new frontier. Eur Heart J. 2017;38(9):639–647.
5. Ton-Nu TT, Levine RA, Handschumacher MD, Dorer DJ, Yosefy C, Fan D, et al. Geometric determinants of functional tricuspid regurgitation: insights from 3-dimensional echocardiography. Circulation. 2006;114:143–9.
6. Mihaila S, Muraru D, Piasentini E, Miglioranza MH, Peluso D, Cucchini U, et al. Quantitative analysis of mitral annular geometry and function in healthy volunteers using transthoracic three-dimensional echocardiography. J Am Soc Echocardiogr. 2014;27:846–57.
7. Anwar AM, Soliman OI, Nemes A, van Geuns RJ, Geleijnse ML, Ten Cate FJ. Value of assessment of tricuspid annulus: real-time three-dimensional echocardiography and magnetic resonance imaging. Int J Cardiovasc Imaging. 2007;23:701–5.
8. Anwar AM, Geleijnse ML, Ten Cate FJ, Meijboom FJ. Assessment of tricuspid valve annulus size, shape and function using real-time three-dimensional echocardiography. Interact Cardiovasc Thorac Surg. 2006;5:683–7.
9. van Rosendael PJ, Joyce E, Katsanos S, Debonnaire P, Kamperidis V, van der Kley F, et al. Tricuspid valve remodelling in functional tricuspid regurgitation: multidetector row computed tomography insights. Eur Heart J Cardiovasc Imaging. 2016;17:96–105.
10. Fukuda S, Saracino G, Matsumura Y, Daimon M, Tran H, Greenberg NL, et al. Three-dimensional geometry of the tricuspid annulus in healthy subjects and in patients with functional tricuspid regurgitation: a real-time, 3-dimensional echocardiographic study. Circulation. 2006;114:I492–8.
11. Jenei C, Muraru D, Addetia K, Veronesi F, Cavalli G, Aruta P, et al. Determinants of normal tricuspid annulus area in healthy volunteers: a three-dimensional echocardiographic study [abstr]. Eur Heart J. 2015;Abstract Supplement:P85197.

12. Miglioranza MH, Mihaila S, Muraru D, Cucchini U, Iliceto S, Badano LP. Dynamic changes in tricuspid annular diameter measurement in relation to the echocardiographic view and timing during the cardiac cycle. J Am Soc Echocardiogr. 2015;28:226–35.
13. Anyanwu AC, Adams DH. Functional tricuspid regurgitation in mitral valve disease: epidemiology and prognostic implications. Semin Thorac Cardiovasc Surg. 2010;22:69–75.
14. Dreyfus GD, Corbi PJ, Chan KM, Bahrami T. Secondary tricuspid regurgitation or dilatation: which should be the criteria for surgical repair? Ann Thorac Surg. 2005;79:127–32.
15. Badano LP, Muraru D, Enriquez-Sarano M. Assessment of functional tricuspid regurgitation. Eur Heart J. 2013;34:1875–85.
16. Dreyfus GD, Chan KM. Functional tricuspid regurgitation: a more complex entity than it appears. Heart. 2009;95:868–9.
17. Rogers JH, Bolling SF. The tricuspid valve: current perspective and evolving management of tricuspid regurgitation. Circulation. 2009;119:2718–25.
18. Kobayashi J, Kawashima Y, Matsuda H, Nakano S, Miura T, Tokuan Y, et al. Prevalence and risk factors of tricuspid regurgitation after correction of tetralogy of fallot. J Thorac Cardiovasc Surg. 1991;102:611–6.
19. Surkova E, Bidviene J, Brunello G, Veronesi F, Cavalli G, Sokalskis V, et al. Tricuspid annulus area correlates more with right atrial than right ventricular volumes in patients with different mechanisms of functional tricuspid regurgitation. A 3-dimensional echocardiography study [abstr]. Eur Heart J Cardiovasc Imaging. 2016;Supplement.
20. Dreyfus J, Durand-Viel G, Raffoul R, Alkhoder S, Hvass U, Radu C, et al. Comparison of 2-dimensional, 3-dimensional, and surgical measurements of the tricuspid annulus size: clinical implications. Circ Cardiovasc Imaging. 2015;8:e003241.
21. Miglioranza MH, Mihaila S, Muraru D, Cucchini U, Iliceto S, Badano LP. Variability of tricuspid annulus diameter measurement in healthy volunteers. JACC Cardiovasc Imaging. 2015;8:864–6.
22. Dreyfus GD, Martin RP, Chan KM, Dulguerov F, Alexandrescu C. Functional tricuspid regurgitation: a need to revise our understanding. J Am Coll Cardiol. 2015;65:2331–6.
23. Addetia K, Yamat M, Mediratta A, Medvedofsky D, Patel M, Ferrara P, et al. Comprehensive two-dimensional interrogation of the tricuspid valve using knowledge derived from three-dimensional echocardiography. J Am Soc Echocardiogr. 2016;29:74–82.
24. Stankovic I, Daraban AM, Jasaityte R, Neskovic AN, Claus P, Voigt JU. Incremental value of the en face view of the tricuspid valve by two-dimensional and three-dimensional echocardiography for accurate identification of tricuspid valve leaflets. J Am Soc Echocardiogr. 2014;27:376–84.
25. Muraru D, Badano LP, Sarais C, Solda E, Iliceto S. Evaluation of tricuspid valve morphology and function by transthoracic three-dimensional echocardiography. Curr Cardiol Rep. 2011;13:242–9.
26. Muraru D, Veronesi F, Maddalozzo A, Dequal D, Frajhof L, Rabischoffsky A, et al. 3d printing of normal and pathologic tricuspid valves fromtransthoracic 3d echocardiography data sets. Eur Heart J Cardiovasc Imaging 2016;in press.
27. Veronesi F, Caiani EG, Sugeng L, Fusini L, Tamborini G, Alamanni F, et al. Effect of mitral valve repair on mitral-aortic coupling: a real-time three-dimensional transesophageal echocardiography study. J Am Soc Echocardiogr. 2012;25:524–31.
28. Tsang W, Meineri M, Hahn RT, Veronesi F, Shah AP, Osten M, et al. A three-dimensional echocardiographic study on aortic-mitral coupling in transcatheter aortic valve replacement. Eur Heart J Cardiovasc Imaging. 2013;14:950–6.
29. Anderson RH. Clinical anatomy of the aortic root. Heart. 2000;84:670–3.
30. Kurisu K, Baba H, Nakashima H, Kajiwara T, Hisahara M, Joo K, et al. Tricuspid regurgitation resulting from acute type a aortic dissection. Ann Thorac Surg. 2014;98:e5–6.
31. Mori S, Yagi T, Otomo K, Meguro T. Severe deformation of right atrium and tricuspid annulus due to compression by tortuous aorta. J Cardiovasc Electrophysiol. 2012;23:881.
32. Gibbs KL, Reardon MJ, Strickman NE, de Castro CM, Gerard JA, Rycyna JL, et al. Hemodynamic compromise (tricuspid stenosis and insufficiency) caused by an unruptured aneurysm of the sinus of valsalva. J Am Coll Cardiol. 1986;7:1177–81.

33. Bulkley BH, Hutchins GM, Ross RS. Aortic sinus of valsalva aneurysms simulating primary right-sided valvular heart disease. Circulation. 1975;52:696–9.
34. Muraru D, Bidviene J, Cavalli G, Cavaliere A, Badano LP. Tricuspid regurgitation in a patient with ascending aorta aneurysm. Eur Heart J Cardiovasc Imaging 2016;17 (12), 1435.
35. David TE. Functional tricuspid regurgitation: a perplexing problem. J Am Soc Echocardiogr. 2009;22:904–6.
36. Joint Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology, European Association for Cardio-Thoracic Surgery, Vahanian A, Alfieri O, Andreotti F, Antunes MJ, et al. Guidelines on the management of valvular heart disease (version 2012). Eur Heart J. 2012;33:2451–96.
37. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP, 3rd, Guyton RA, et al. 2014 aha/acc guideline for the management of patients with valvular heart disease: a report of the american college of cardiology/american heart association task force on practice guidelines. J Am Coll Cardiol 2014;63:e57–185.