

My Approach to Three-Dimensional Echocardiography for Pathophysiological Classification of Tricuspid Regurgitation

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Introduction

Although it was known as the “forgotten valve” in the past, there has been increasing interest in studying the tricuspid valve (TV) during the last two decades. Tricuspid regurgitation (TR) has been identified as a prognostic marker not only when associated with other cardiac diseases, like heart failure, mitral regurgitation, or aortic stenosis,¹⁻³ but also as an isolated entity.⁴ Understanding the pathophysiological mechanisms of this disease is of paramount importance, since new options of transcatheter devices and techniques are emerging, offering invasive treatment for high-risk patients unsuitable for surgical intervention.⁵

The TV is difficult to image in transesophageal echocardiography (TEE) for the following reasons: 1) the leaflets are much thinner compared to mitral leaflets, with greater anatomic variability; 2) it is an anterior structure, far away from the esophagus, with acoustic shadowing from the fibrous heart skeleton; and 3) it cannot be aligned to the esophageal probe in order to acquire en face views and requires use of lateral resolution. These restrictions may limit the ability of traditional TEE to evaluate the TV, making it necessary to complement the evaluation with special windows from the lower esophagus and transgastric views. Different from other cardiac valves, images of the TV obtained from transthoracic echocardiography (TTE) usually have better resolution than images obtained by TEE. Even though all protocols start with two-dimensional (2D) assessment of the TV, three-dimensional echocardiography (3DE) plays an important role in the evaluation of TV diseases, due not only to its ability to precisely depict the anatomy of the valve and the subvalvular apparatus, but also to its accuracy in quantitation of right ventricular (RV) and right atrial volumes and function and functional analysis of valvular dysfunction, especially for grading TR and evaluating dynamics of the tricuspid annulus

(TA) through dedicated software. All of this information is of unparalleled importance for patient management and pre-procedural planning in surgical and transcatheter approaches.

TV anatomy

The TV complex is a functional unity composed of leaflets, subvalvular apparatus, papillary muscles (PM), and TA (Figure 1), which interact during the cardiac cycle in a dynamic fashion in order to maintain valvular function. Although it is called traditionally “tricuspid”, this valve has a great anatomical variability. As a matter of fact, up to 46% of them can have two, four or even more cusps⁶ (Figure 2). TV leaflets are asymmetric; the anterior leaflet is typically the largest in radial diameter and area. The septal leaflet has the least mobility and is the shortest in radial diameter, while the posterior is the shortest circumferentially and often has multiple scallops. The attachment of the TV is more apically positioned than the mitral valve (normal distance ≤ 10 mm in relation to the anterior mitral leaflet).

The TV subvalvular apparatus is normally composed of two well differentiated PM: anterior and posterior, with a variable presence of a septal PM. The anterior PM is the most prominent and lends chordal support to anterior and posterior leaflets. It blends with the right end of the septal marginal trabeculae (moderator band) below the antero-posterior commissure. The posterior PM, which can be bifid or trifid, lends chordal support to the posterior and septal leaflets. The septal PM, when present, may be small or multiple, and is often indistinguishable from the ventricular wall. There are usually some small chords that emerge directly from the ventricular septum and attach to anterior and septal leaflets.⁶ Accessory chords may be directly attached to the RV free wall or to the moderator band (Figure 3). In the RV, the subvalvular chords are less distensible than those seen in the mitral valve, with dense collagen bundles, helping to explain why there is more extensive tethering when there is ventricular remodeling, with displacement of either the RV septal or lateral wall positions, affecting leaflet coaptation.

One of the most important structures for maintaining adequate TV function is the TA. It is a very dynamic structure, with varying size and geometry during the cardiac cycle, usually oval and saddle-shaped. It becomes more spherical and planar when dilated, extending towards the anterolateral and posterior RV walls, regions where there is no fibrous tissue around the valve, making it less resistant to remodelling.⁷ Based on current guidelines,^{8,9} a significant dilatation of the TA is present when there is a linear medial-lateral measurement > 40 mm or > 21 mm/m²

Keywords

Tricuspid Valve; Tricuspid Valve Insufficiency; Echocardiography, Three-Dimensional

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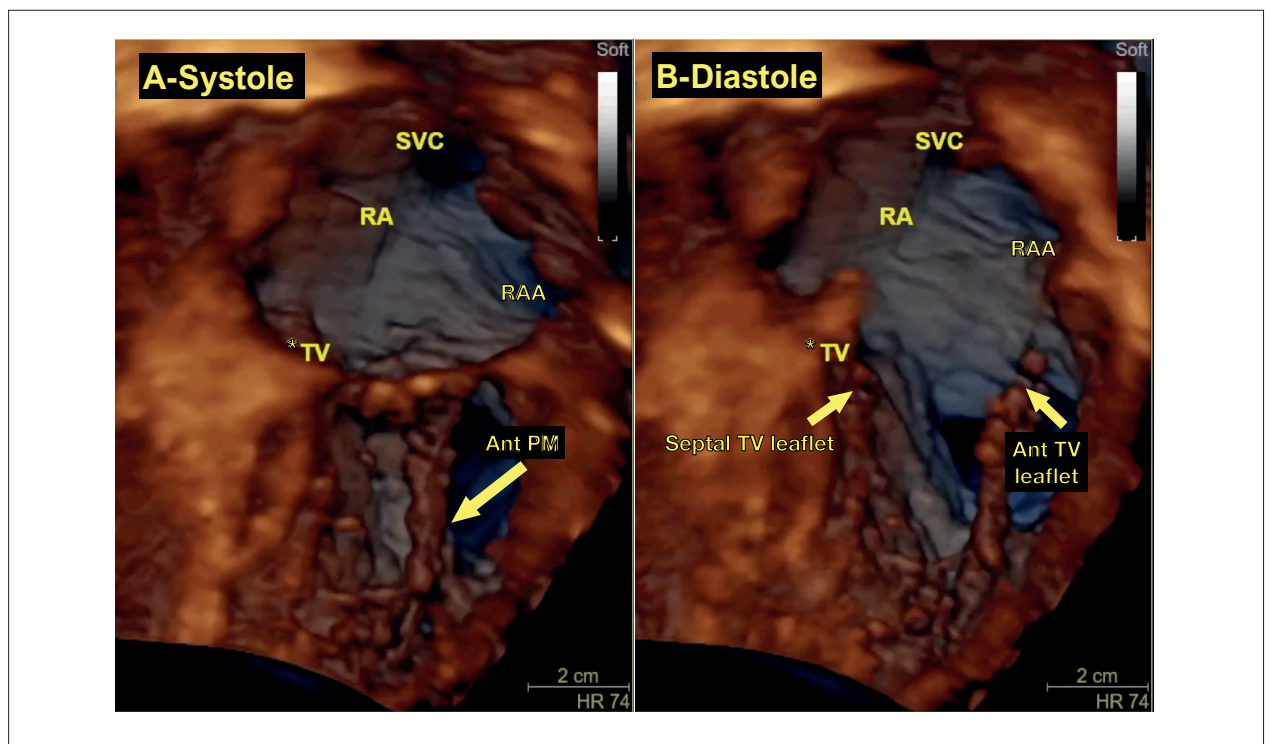


Figure 1 – TV complex anatomy viewed by 3DE. A functional unity composed of leaflets, subvalvular apparatus, PM, and TA. Ant PM: anterior papillary muscle; Ant TV leaflet: anterior tricuspid valve leaflet; RA: right atrium; RAA: right atrial appendage; SVC: superior vena cava; *TV: tricuspid valve annulus.

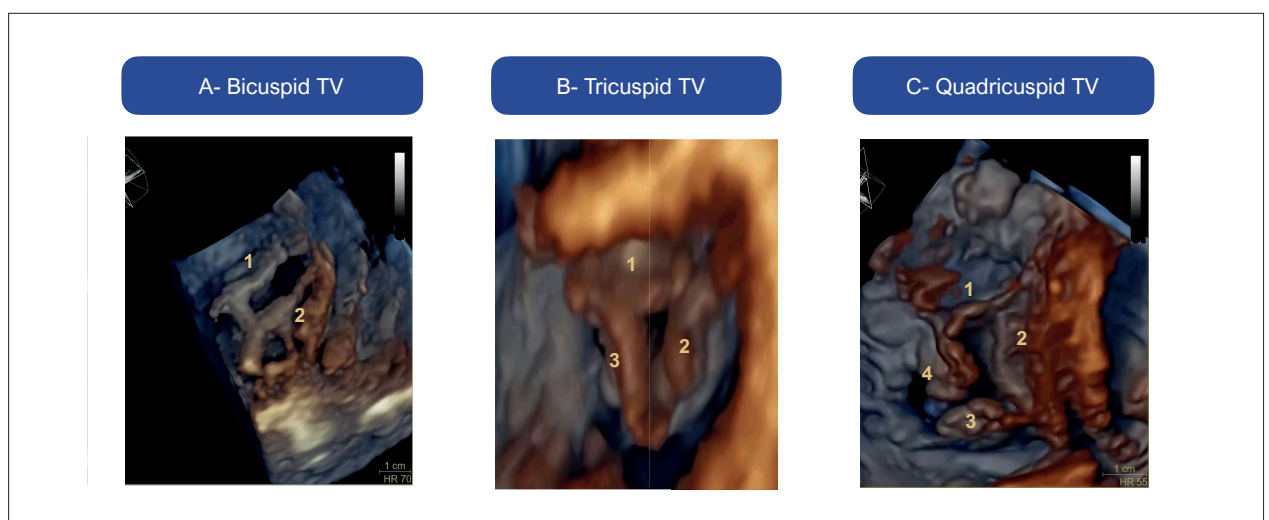


Figure 2 – Anatomical variations of TVs viewed by 3DE (ventricular perspective). In A we can see a bicuspid TV; in B a tricuspid TV; and in C a quadricuspid TV. TV: tricuspid valve

acquired by 2D echocardiography in apical 4-chamber view (during diastole). 3DE identifies numerous different linear dimensions for every TA in a cross section (Figure 4), as expected for an oval structure, and it is important to mention that 2D apical 4-chamber view almost always fails to demonstrate the largest TV diameter. Using 3DE, we can measure the largest dimensions and area of the TA with great

accuracy with multiplanar reconstruction tools. At the end of ventricular diastole, the normal value for the largest diameter of the TA is 40 ± 5 mm on 3DE (23 ± 3 mm/m²),¹⁰ always bearing in mind that this is a highly dynamic structure, whose area undergoes significant variations during the cardiac cycle (approximately 35%), being larger in the end of diastole and smaller in the middle and end of ventricular systole.¹¹

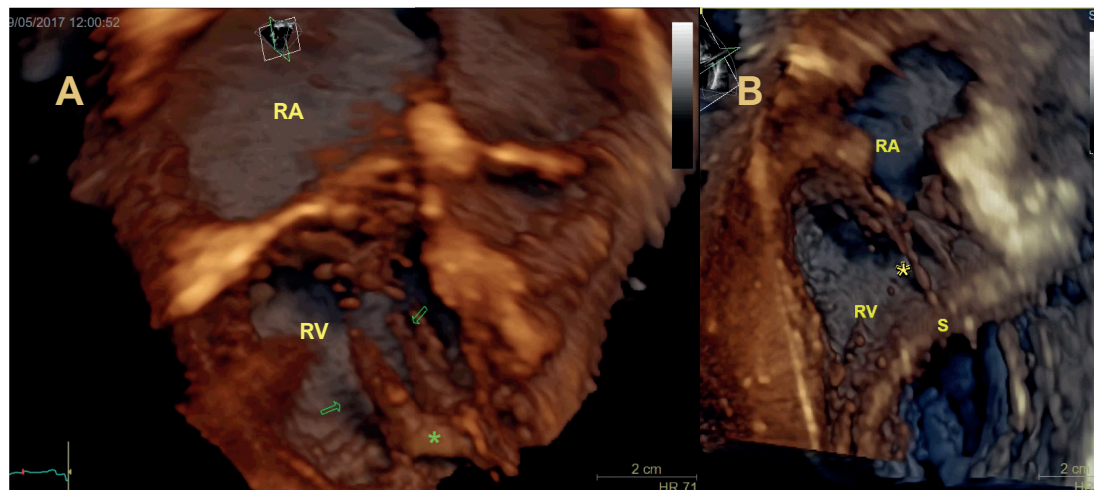


Figure 3 – Anatomical detail of a TV complex viewed by transthoracic 3DE. A) 3D rendered coronal view showing anterior papillary muscle (green arrow) rising from the septomarginal trabeculae (green asterisk) at the anterolateral wall. B) Oblique view, where we can see direct chordal attachment (yellow asterisk) to the interventricular septum. RA: right atrium; RV: right ventricle.

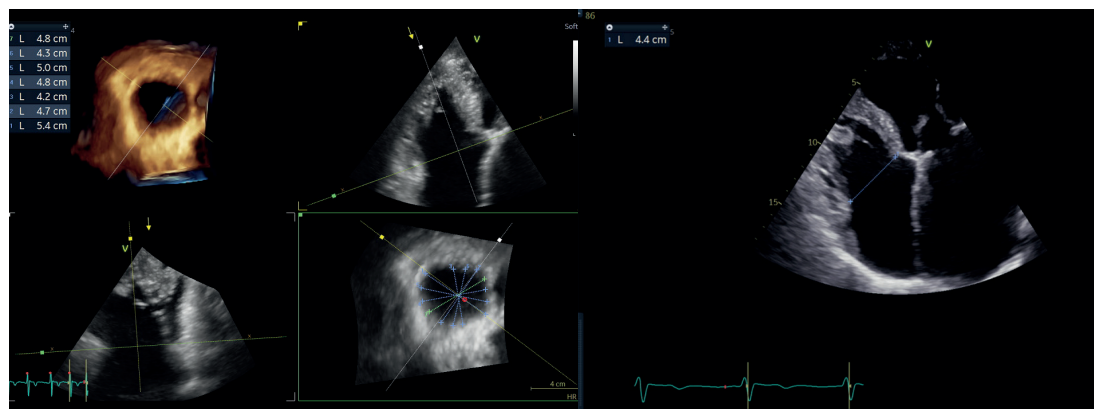


Figure 4 – Most TV annuli are oval-shaped, and 3DE is able to identify numerous different linear dimensions in a cross section with multiplanar reconstruction (A), and it is important to mention that 2D apical 4-chamber view almost always fails to demonstrate the largest TV diameter.

Pathophysiological classification of TR

It is of great importance to understand the pathophysiological mechanisms responsible for the development of TR, not only to define the best treatment modality and intervention timing for each specific patient, but also to prevent TR progression in some conditions, for example, in patients with atrial fibrillation that is amenable to cardioversion.

Traditionally, TR is stratified by the presence of leaflet tissue involvement into two broad categories: primary TR (organic) and secondary TR (functional). However, given the great complexity and superposition of different mechanisms in TR development, a new classification was made necessary to provide a better understanding, integrating more information, not only focusing on the tricuspid leaflet tethering, but also considering right

atrial, TA, and RV dilatation, as well as RV dysfunction. In this new approach, functional or secondary TR is divided into two categories: atrial functional TR (AFTR) and ventricular functional TR (VFTR). Furthermore, cardiac implantable electronic device (CIED)-related TR is also included as a new separate category.¹² Based on this new concept we propose a practical framework to differentiate between these pathophysiological phenotypes (Figure 5). This new classification provides further information to understand the great diversity of phenotypes of TR, assisting therapeutic and interventional planning.

Primary TR (organic)

TR may be the result of organic valvular disease (primary TR), when tissue damage or degeneration is the main reason

for valvular incompetence. Primary TR is far less common than secondary TR, with reported prevalence in literature of less than 10%, and generally more difficult to repair, demanding surgical TV replacement. In the new integrated classification of TR, the main pathophysiologic mechanisms for primary TR are leaflet changes leading to restricted or excessive leaflet mobility or leaflet perforation. Specific conditions have also been included in the Carpentier classification with type I including congenital heart diseases and endocarditis (both vegetations and perforation), type II referring to myxomatous valve disease with prolapse, traumatic TR (e.g., chest trauma) and TR after biopsy, and type IIIA including carcinoid, rheumatic dysfunction, radiotherapy, and tumors (Figure 6). Imaging plays a major role in the identification of these subgroups of valvopathies, because the echocardiographic criteria are definitive for diagnosis and easily detectable.

Ventricular secondary TR

Secondary TR is the most prevalent form. It is frequently the result of inappropriate leaflet coaptation as a secondary mechanism, in response to volume or pressure overload, due to RV remodeling, annular dilatations, displacement of the PM, and leaflet tethering (Figure 7). Left-sided heart diseases are the leading cause of secondary TR, either as a consequence of elevated pulmonary pressures due to left-sided valvular heart diseases or secondary to non-valvular heart failure, with high diastolic filling pressures, both in reduced or preserved ejection fraction. Secondary TR may also be frequently present in advanced isolated RV diseases, such as pulmonary hypertension (cor pulmonale), congenital heart diseases, or primary RV myocardial diseases (arrhythmogenic cardiomyopathy, RV myocardial infarction). For each patient with secondary TR, it is very important to have a full understanding of the mechanisms involved, to evaluate the possibility and best technical approach for a surgical management (when submitted to left-sided heart valve surgery), or in patients with high surgical risk, to explore transcatheter therapeutic options. Optimization of pharmacologic therapy for heart failure may change the degree of secondary TR, which is very sensitive to preload (volume) and afterload (elevated left ventricular filling pressures transmitted retrograde as elevation of capillary pressures and pulmonary artery systolic pressures), and, according to ACC and ESC guidelines, secondary TR should be treated percutaneously in patients under optimal medical treatment only.

Atrial secondary TR

What was previously known as “idiopathic” TR, today is recognized as AFTR, or “atriogenic” TR, a distinct pathophysiological phenotype, where TA dilatation and dysfunction is the main substrate of valvular incompetence. It is primarily caused by right atrial remodeling, as we often notice in patients with persistent atrial fibrillation,¹³ where right atrial volume is the key factor to determine alterations in the TV/TA complex¹⁴⁻¹⁶ (Figure 8). Differently from the classic form of functional TR (“ventricular” TR), patients with AFTR have normal-sized or only mildly dilated (conical-shaped) RV, usually without RV dysfunction.^{17,18}

For the diagnosis of AFTR, we have to exclude other conditions which may justify the development of TR, such as all causes of primary TR, the presence of left-sided valvular heart disease, left ventricular systolic dysfunction, pulmonary hypertension, or the presence of pacemaker wires. Distinguishing atrial from ventricular forms of secondary TR is very important, not only because they carry different prognostic implications (atrial TR develops more rapidly than ventricular TR, which is usually seen only in advanced stages of disease), but also to help guide specific interventions such as rhythm control or cardioversion in patients with persistent atrial fibrillation.

CIED-related TR

CIED-related TR is a multifactorial disease, comprising different possible types of pathophysiology, and it can share features of primary or secondary TR, depending on its etiological features. Primary CIED-related TR may be caused by lead impingement or adherence to TV leaflets (Figure 9), interference with subvalvular apparatus, leaflet perforation, laceration, or in some dramatic cases even leaflet avulsion (caused by lead extraction).

Approximately 25% to 29% of patients with permanent pacemaker have TR, which is more than double the frequency of TR compared to control groups.¹⁹ Defining the underlying mechanism is very important to explore the therapeutic options or even the need for lead extraction in some cases.¹² CIED-related TR is associated with poor outcomes²⁰ and must be avoided at any cost. By using 3DE it is feasible to guide lead positioning intraoperatively, avoiding complications and leaflet impingement, or evaluate the interference of the wire with the TV in the early postoperative period, when there is still the option to intervene and reposition the wire.

The pathophysiological link between the existence of device leads and the development of a significant TR or worsening of a pre-existent regurgitation is not always related to mechanical interference of the device with leaflet mobility or the subvalvular apparatus (primary TR); it can also be due to secondary mechanisms, for example, in cases where pacing causes chamber dilatation and/or heart failure, or when it causes significant RV dyssynchrony. In fact, secondary mechanisms may be responsible for more than 60% of cases of CIED-related TR,²¹ and even when primary TR is the initial mechanism, long-lasting volumetric overload may lead to RV dilatation and remodeling, generating a model of coexistent primary and secondary TR.

When considering lead extraction in an attempt to reduce TR, we must perform a detailed evaluation of the TV complex to determine if there is a significant secondary component (isolated or mixed); specifically looking for annular dilation, RV remodeling, RV dysfunction, and leaflet tethering, which can be determinants of worse treatment response.

Analysis of TV anatomy and mechanisms of TR with 3DE

3DE is a promising technique for accurate qualitative and quantitative analysis of the TV, obtaining important

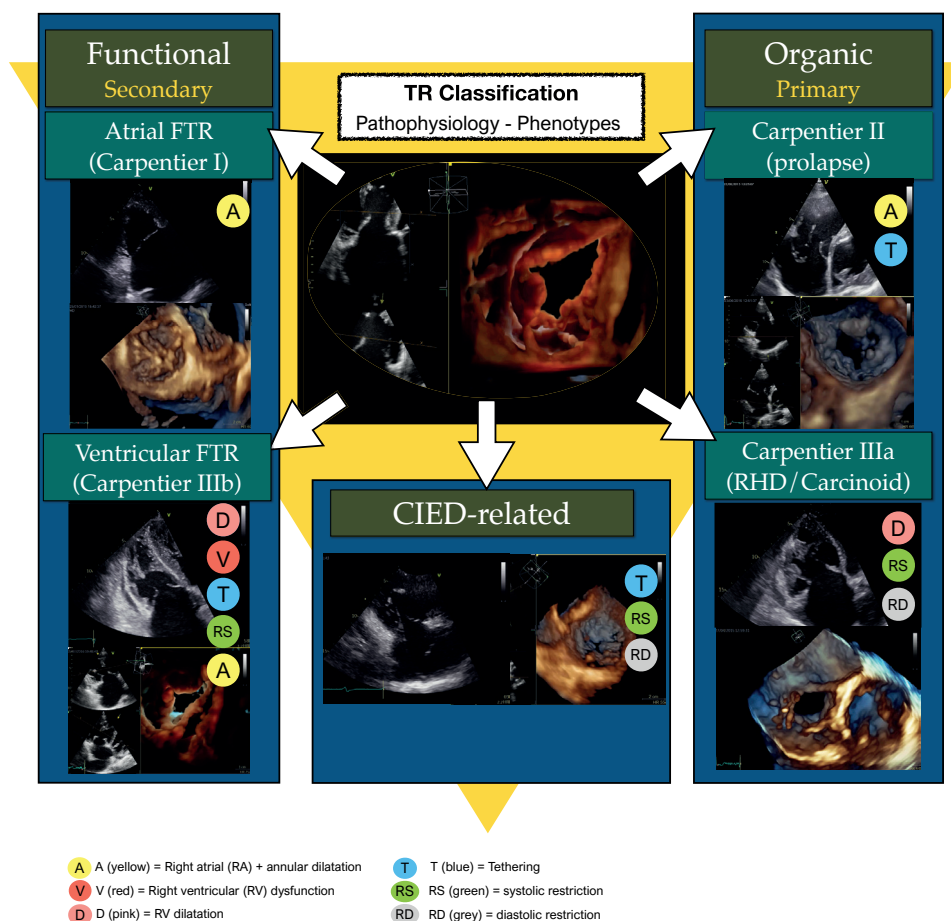


Figure 5 – Integrative classification of TR according to pathophysiology and predominant phenotypes. CIED: cardiac implantable electronic device; FTR: functional tricuspid regurgitation; RHD: rheumatic heart disease; TR: tricuspid regurgitation.

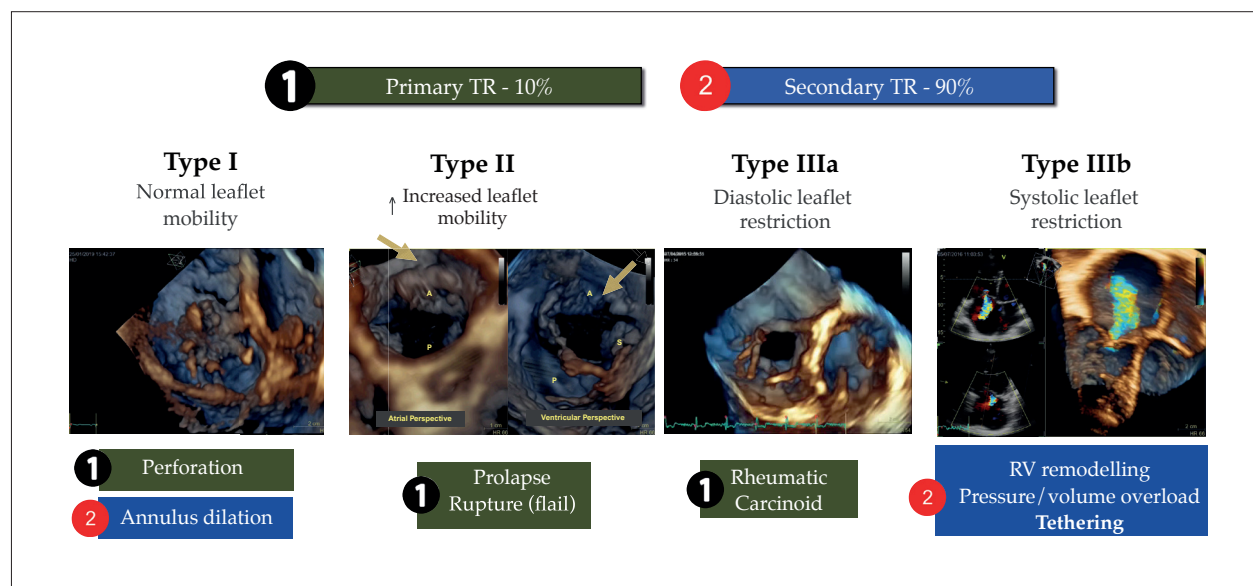
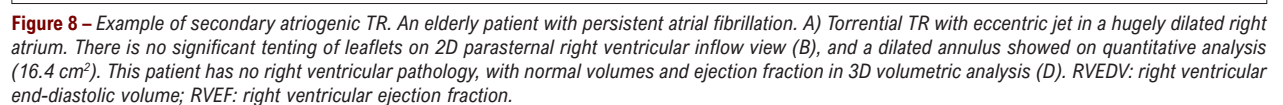
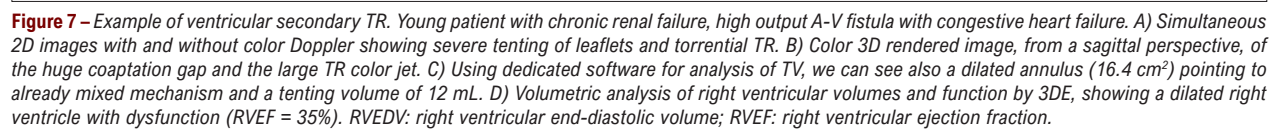


Figure 6 – TR mechanisms, adapted from classical Carpentier classification (Carpentier A., et al. J Thorac Cardiovasc Surg 1983). RV: right ventricle.



parameters which can guide the best therapeutic option for each patient. 3DE is more accurate than 2D in severity assessment with vena contracta area and anatomic evaluation of the valve. With 3DE, it is possible to study any of the components of the TV complex in a dynamic way, with good temporal and spatial resolution.

Step by Step approach for acquisition and analysis of an optimized TV 3DE Dataset:

- The main key to a reliable 3DE analysis is to obtain a good echocardiographic dataset. Specifically, focusing on the RV is important, along with good patient positioning at left lateral decubitus, preferentially with a proper examination

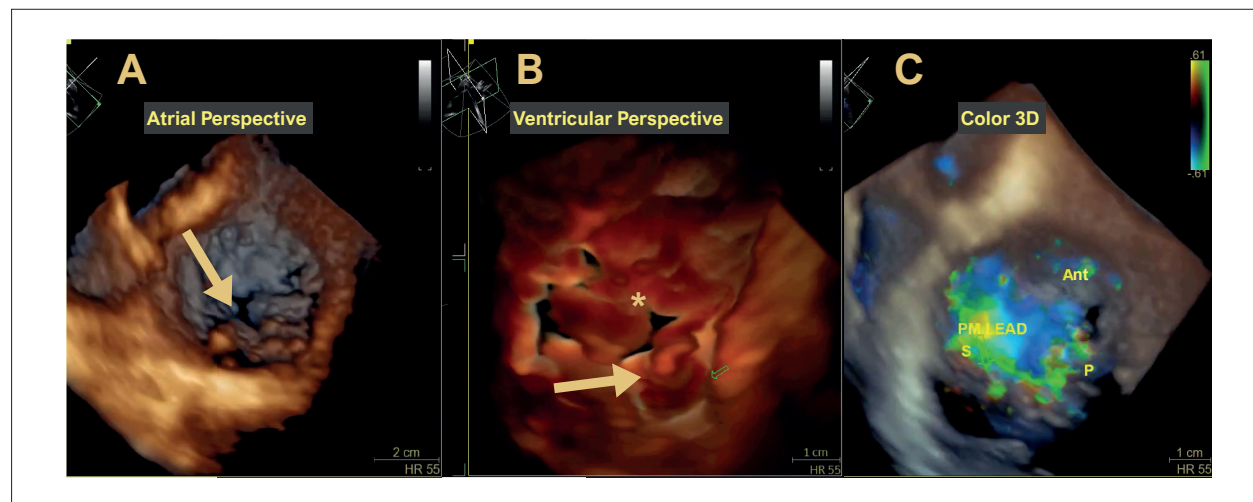


Figure 9 – CIED-related TR caused by pacemaker lead impingement (arrows). Transthoracic acquisition, with 3D rendered views of the TV from atrial perspective (A), ventricular perspective showing lack of coaptation of leaflets (asterisk) caused by septal leaflet impingement (B). In C, 3D color image from atrial perspective showing torrential TR. Ant: anterior leaflet; P: posterior leaflet; PM: pacemaker; S: septal leaflet.

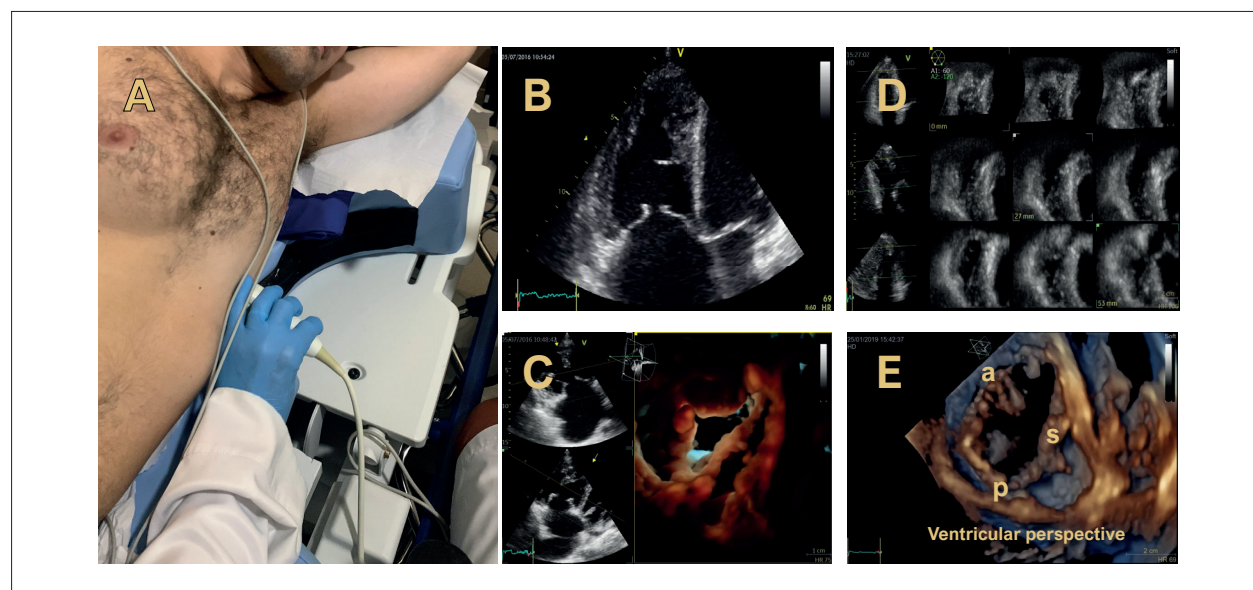


Figure 10 – Acquiring an optimized TV 3DE dataset. A) Acquiring a good apical right ventricular focused view is very important. Ensure good patient positioning at left lateral decubitus, preferentially with a proper examination bed, with a removable lateral part to properly expose the chest to the transducer. B) To obtain good 2D images, it is essential to have a good 3D image, including all the TV in the dataset, in the most central position possible, with some surrounding structures, such as the interventricular septum and aorta for spatial orientation (C). Before storing the acquired dataset, we should always check the quality of the images obtained, verifying whether all right ventricular walls are included and looking for artifacts. We can do this on rendered images or with a multislice tool (D). The suggested anatomic view of the TV is acquired from the ventricular side, by placing the septum on our right side (3 o'clock) and the aortic valve on top of it (1 o'clock). Thus, the septal leaflet (s) is shown on the right side, next to the septum, the anterior leaflet (a) on top, next to the aortic valve, and the posterior leaflet (p) on the bottom side opposite the aortic valve (E).

bed, with a removable lateral part to properly expose the chest to the transducer (Figure 10A).

- The first important rule in 3DE is that optimal 2D images are necessary in order to achieve adequate analysis of 3DE views. In case of poor acoustic window, the acquisition of 3DE will be difficult and data obtained may not be interpretable or reliable (Figure 10B).

- Asking for breath-hold from the patient, if possible, to enable a multi-beat acquisition without significant artifacts (stitching), can help us achieve decent temporal and spatial resolution at the same time. This requires a patient on sinus rhythm or at least not on a significant arrhythmia with great R-R variation between the cardiac cycles. Even with atrial fibrillation, it may be possible to acquire multi-beat images, but never for example with a bigeminy rhythm.

- In order to achieve a good temporal resolution, ensuring a satisfactory volume rate, we have to use the maximum number of beats possible for each patient (difficult when the patient cannot or is not cooperative or in the setting of rhythm disturbances), while limiting lateral and elevation widths and focusing on the TV and subvalvular apparatus only. Surrounding structures such as the interventricular septum and the aortic valve should also be included and serve as markers during 3D analysis (Figure 10C).

- Before storing the acquired dataset, we should always check the quality of the images obtained, looking for artifacts (significant stitching) and confirm if the entire structure of interest was adequately included. This can be done either in volume rendering mode or in the 2D multi-slice/multiplanar mode (Figure 10D).

- For post-processing we can navigate freely into the dataset looking for anatomic abnormalities and the relation to surrounding structures. The suggested anatomic view of the TV is acquired from the ventricular side, by placing the septum on our right side (3 o'clock) and the aortic valve on top of it (1 o'clock). That way the septal leaflet

is demonstrated on the right side, next to the septum, the anterior leaflet on the top, next to the aortic valve, and the posterior on the bottom side opposite to the aortic valve (Figure 10E). Easy cropping features are also available, embedded in most machines, and they are an important tool to understand the etiology and pathophysiology of TR.

- It is possible to analyze dimensions of the TV annulus using multiplanar slicing or multiplanar reconstruction tools. Both modalities enable the alignment of planes to obtain accurate annular dimensions and area (Figure 11).

- To analyze tenting volume, coaptation height, annulus area, and other quantitative parameters, we can adapt the mitral valve software (available from many vendors), or use a specific software dedicated to the TV developed by GE Healthcare (AutoTVQ) (Figure 12). These applications are easy to use and have a good correlation of measurements, when compared to cardiac computed tomography.

Conclusion

Echocardiography is the most important noninvasive diagnostic method for evaluation of TV diseases, and 3DE has been proven to have additional value, not only for evaluation of valve anatomy and quantitation of TR, but also for a complete understanding of its pathophysiological mechanisms, playing a decisive role in planning the best therapeutic strategy. Quantitative data obtained from 3D TV analysis by specific software can provide valuable information to tailor the best surgical or percutaneous technique for each patient and to help develop new treatment strategies for valve intervention in the near future.

Author Contributions

Conception and design of the research and writing of the manuscript: Félix AS, Alcantara ML, Papadopoulos K; critical revision of the manuscript for intellectual content: Félix AS.

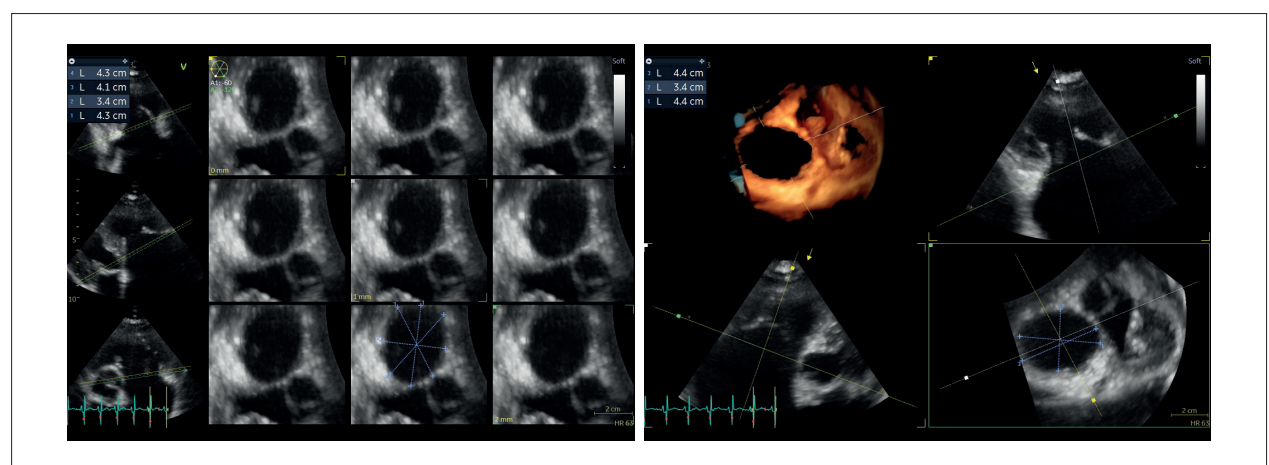


Figure 11 – Tricuspid annular dimensions and area analysis by 3DE post-processing. A) multi-slice tool with appropriate alignment for TV annulus (hinge point of leaflets) in 3 different projections (2D on the left image) showing an oval annulus with different dimensions depending on the axis chosen. B) The same aspect is showed by multiplanar reconstruction.

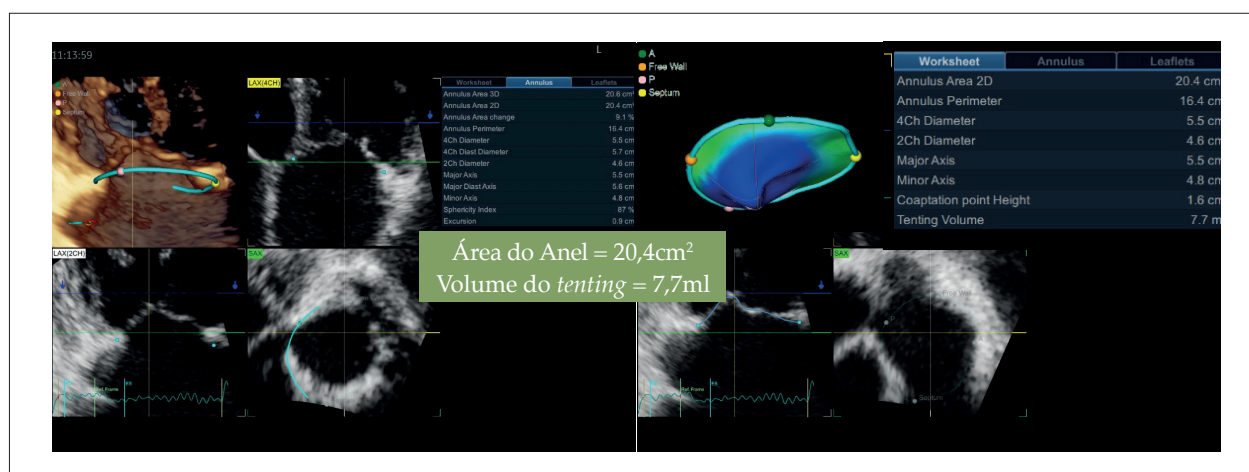


Figure 12 – TV analysis using dedicated software (GE healthcare) in a patient with pulmonary arterial hypertension. In this case, we can see not only an important ventricular mechanism of secondary TR, with large tenting volume of 7.7 mL (normal volume = 1.1 ± 0.6 [Sukmawan, R, JASE 2007]), but also annular dilatation = 20.4 cm^2 (normal = $8.6 \pm 2.0 \text{ cm}^2$ [Addetia et al., JACC 2017]), secondary to a chronic associated volumetric overload due to torrential TR.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This article does not contain any studies with human participants or animals performed by any of the authors.

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