Chapter 4
Hemodynamic Assessment of Tricuspid Valve Disease

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Introduction

Traditionally, the tricuspid valve (TV), and diseases thereof, have not garnered as much attention as other valves, relegating it as the "forgotten valve "of the heart [1, 2]. Much of the tricuspid valve's second-tier status can be attributed to the frequency with which TV disease results from other diseases, especially left-sided valvular disease [3]. As such, TV disorders have been largely addressed in the context of these other diseases, and therapies for TV disease have typically focused on treating those concurrent conditions with the assumption that the TV would itself improve accordingly [1, 3]. However, significant tricuspid valve disease itself can lead to worsening outcomes, including right heart failure [4]. In response, a number of exciting new techniques and technologies have emerged to help combat tricuspid valve disease. Successfully treating tricuspid valve disease is dependent on understanding TV anatomy, mechanics of blood flow through the valve, and appropriate ways to assess physiology.

Anatomy and Physiology of the Tricuspid Valve

Located between the right atrium and right ventricle, the tricuspid valve is the largest of the four valves in the heart and is the most apical. It can be divided into four parts: the annulus, the leaflets, the papillary muscles, and the chordal attachments. The tricuspid valve has three leaflets typically classified as septal, anterior, and posterior. The leaflets are unequal in size, with anterior leaflet typically having the largest area, and therefore the greatest range of motion. The annulus is a D-shaped,

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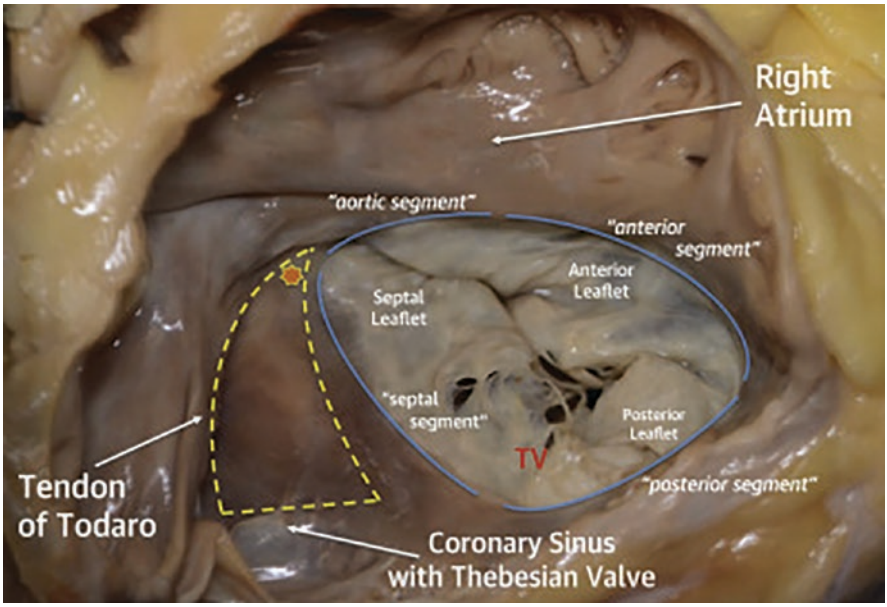

Fig. 4.1 Anatomical layout of the tricuspid valve showing the D-shape annulus and related structures. (With permission) (Asmarats et al. [30])

nonplanar structure made up of two separate parts: a large C-shape section that corresponds to the right atrium and the right ventricle, and a smaller, straighter section that corresponds to the septal leaflet and ventricular septum (Fig. 4.1). Of note, the annulus is a dynamic structure that can increase its area up to 30% during the cardiac cycle under normal loading conditions [5].

The atrioventricular node and the bundle of His cross the septal leaflet attachments 3–5 mm posterior to the antero-septal commissure [5]. Additionally, the noncoronary sinus of Valsalva is adjacent to the area between the septal and anterior leaflets. The septal leaflet of the tricuspid valve is also one of the landmarks used to identify the triangle of Koch (along with the tendon of Todaro and the coronary sinus) (see Fig. 4.1). From the exterior of the heart, the right coronary artery can be used to estimate the location of the tricuspid valve as it courses through the atrioventricular groove.

The majority of right ventricular filling, and therefore flow across the tricuspid valve, occurs during the passive phase of diastole and is driven by the pressure gradient between the right atrium in the right ventricle. Given the large size and relatively low pressures of the right atrium and right ventricle, diastolic velocities across the tricuspid valve are typically low, with a peak transannular velocity of less than 1 m/s and a mean velocity gradient of less than 2 m/s [3]. Active atrial contraction, and the resultant increase in right atrial pressure, results in additional blood flow into the right ventricle. Normal pressure waveforms of the right atrium and right ventricle including the "c-wave," which represents the bulging of the tricuspid valve

back into the right atrium following valve closure [6]. Disturbances to valve anatomy and function will significantly alter the normal pressure relationship between the right atrium and right ventricle, resulting in abnormal pressures, gradients, and flow across the TV.

Diseases of the Tricuspid Valve

Tricuspid Stenosis

Tricuspid stenosis (TS) results from a narrowing of the valvular opening and is a rare disease, most commonly resulting from rheumatoid fever. TS is almost always found in conjunction with mitral stenosis [7, 8]. Using first-pass principles, narrowing of the tricuspid valve causes an increased impedance to blood flow from the right atrium to the right ventricle, particularly during active atrial contraction. The result is an enlarged a-wave seen on right atrial tracings [4, 9]. More specifically, the change in right atrial pressure is governed by Poiseuille's law [$Q = \frac{\pi P r^4}{8 \mu l}$, where Q = flow rate, P = pressure, r = radius, μ = fluid viscosity, and l = length of tubing]. Rearranging the equation shows that pressure is inversely proportional to the size of the opening [$P \propto \frac{1}{r^4}$] and reveals why pressures increase markedly with reduction in the valve area. Clinical assessment of TS can be done either invasively with cardiac catheterization or noninvasively with Doppler echocardiography.

Cardiac Catheterization

Tricuspid stenosis can be evaluated invasively through a right heart catheterization. Simultaneous recordings of the right atrial and right ventricular pressures using a dual lumen catheter will yield pressure differences between the two chambers [10]. Due to the smaller orifice of a stenotic TV, passive blood flow during early diastole will be reduced, resulting in a larger blood volume in the right atrium during atrial systole, ultimately resulting in higher atrial systolic pressures. Concurrently, the stenotic obstruction will result in the right ventricular diastolic pressure being lower than normal conditions. These changes result in a significantly increased A-wave and a more gradual Y-descent in right atrial pressure tracings [11]. Right ventricular waveforms are normal except for reduced end-diastolic pressures due to incomplete filling of the right ventricle [12] (Fig. 4.2).

As previously noted, an elevated pressure gradient will occur across the stenotic tricuspid valve proportional to the inverse of the valve area. Severity of tricuspid stenosis, therefore, can be evaluated based on the resultant gradient. Because the gradient across the TV is dynamic throughout the cardiac cycle, the mean gradient during ventricular diastole is used to evaluate the TV [13]. Severe tricuspid stenosis

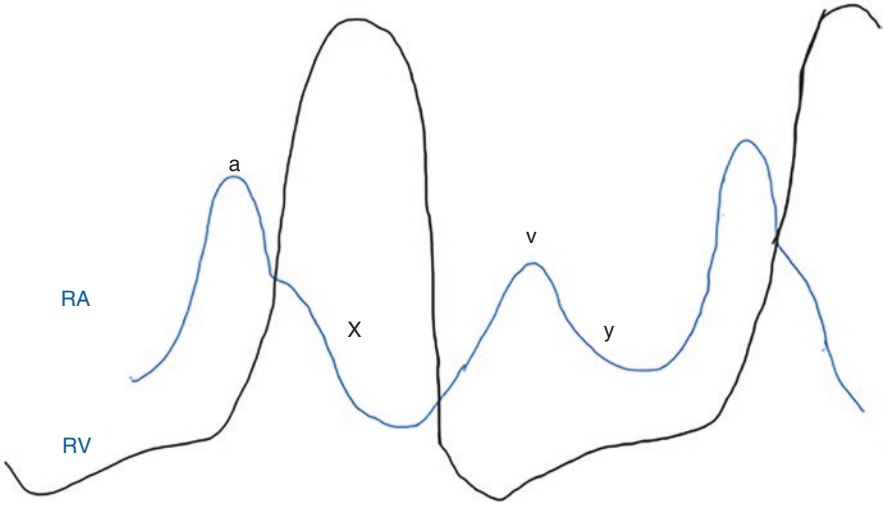

Fig. 4.2 Simultaneous right atrial and right ventricular waveforms in tricuspid stenosis. The "a-wave" is increased, the "y-descent" is more gradual, and the right ventricular end-diastolic pressure is reduced

correlates with a mean gradient of ≥5 mmHg; however, calculation of the gradient is affected by heart rate, with higher gradients occurring with faster heart rates and/or the simultaneous presence of tricuspid regurgitation [4, 13].

Historically, severe tricuspid stenosis is defined as a TV area of ≤1.0 cm^2 [4]. Although Poiseuille's law describes the general relationship between valve area and pressure gradients, the more accepted formula to calculate TV is the equation originally described by Gorlin and Gorlin,

$$\text{TV Area} = \frac{\text{Cardiac Output}}{(\text{Diastolic Filling Period})(44.5)(\sqrt{\text{mean RAP} - \text{mean RV diastolic pressure}})}$$ [14].

Utilization of this equation requires measurement of cardiac output and definition of the diastolic filling period across the TV. Current practice typically employs the use of catheterization lab software to calculate the valve area using this formula.

Doppler Echocardiography

Doppler echocardiography provides a noninvasive method for assessing tricuspid valve disease. Although echocardiographic analysis does not provide direct measurements of pressures, blood flow velocities can be assessed using Doppler signals. Translating velocities into pressure estimates can then be achieved using the law of conservation of energy, and namely the Bernoulli equation. The Bernoulli equation defines the relationship between potential energy and kinetic energy within a closed system, stating that the total energy remains constant [15]. In its full version, the Bernoulli equation is expressed as $P_{0_1} + \rho g h_1 + \frac{1}{2}\rho v_1^2 = P_{0_2} + \rho g h_2 + \frac{1}{2}\rho v_2^2$. In this

equation, potential energy is expressed in the term ρgh, where ρ is the density of the fluid, g is gravitational acceleration, and h is the height of the fluid column. Within the heart, it is assumed that differences in height are negligible, and therefore this potential energy term mathematically cancels out. This assumption leads to the simplified Bernoulli equation, $\Delta P = \frac{1}{2}\rho\left(v_2^2 - v_1^2\right)$, where v is velocity and ΔP represents the pressure gradient of interest. Of note, due to the relatively small values of v_1 compared to v_2, the term v_1^2 is assumed to approach zero and is ignored [15]. Using the density of blood ($1060\frac{\text{kg}}{\text{m}^3}$) and velocity ($\frac{\text{m}}{\text{s}}$) results in pressures measured by Paschal units. Converting to Paschal units into standard mmHg units reveals the commonly used Doppler derived pressure gradient formula $\Delta P = 4v^2$, with v being the maximal velocity measured across the valve [16, 17]. Mean gradient values of ≥5 mmHg generally signify severe stenosis [7].

In significant tricuspid stenosis, resting cardiac output is reduced and does not increase with exercise due to the fixed obstruction to flow [4, 9]. Physiologically, the decrease in cardiac output translates to a longer period of time for pressures to equalize across the tricuspid valve, with longer times being resulting from more severe stenosis. This concept can be quantified using the pressure half time (PHT), which is the time needed for maximal velocity to reduce by 50%. PHT values of ≥190 ms correspond to severe TS [18].

The use of noninvasive techniques to evaluate tricuspid stenosis is often sufficient to establish severity, and the use of invasive measures is generally recommended only when noninvasive measurements are inconclusive or inadequate [4].

Treatment of Tricuspid Stenosis

Therapeutic interventions upon a stenotic tricuspid valve are generally reserved for cases of severe TS. Because most cases of TS occur in the setting of left-sided valve disease, current guideline recommendations suggest surgical intervention on a severely stenotic TV at the time of left-sided valve surgery. Isolated tricuspid stenosis warrants surgical intervention when patients are considered significantly symptomatic, but outcomes are dependent on postoperative right ventricular function [4, 19]. For patients with severe, symptomatic TS without associated tricuspid regurgitation, percutaneous balloon commissurotomy can be considered [4].

Tricuspid Regurgitation

Tricuspid regurgitation (TR) is characterized by the backflow of blood from the right ventricle into the right atrium during ventricular systole. Tricuspid regurgitation is generally categorized as either primary or secondary. Primary TR results from a structural malfunction of the tricuspid valve, such as perforation (seen with

rheumatoid heart disease or infectious endocarditis), Ebstein's abnormality, the presence of wires for cardiac rhythm devices, and several other causes [1, 3, 4]. Secondary TR results from loss of TV functionality related to changes in the TV annular geometry, but not the valve leaflets themselves. Although quantification methods do exist to characterize TR severity, the clinical presence of right heart failure symptoms alone signals poor outcomes regardless of TR quantification. In cases of severe TR, outcomes also tend to be poor and are independent of other cardiac performance measures [20].

Cardiac Catheterization

Catheter evaluation of tricuspid regurgitation is often confounded by the presence of atrial fibrillation. The presence of atrial fibrillation will significantly alter right atrial pressure tracings and greatly reduce the interpretability of waveform analysis, particularly when trying to evaluate A-wave and x-descent contours [21]. Because the major flow abnormality in tricuspid regurgitation occurs during ventricular systole, contraction of the right ventricle against an incompetent valve will result in increased flow back into the right atrium. This pattern manifests as tall V-waves on right atrial pressure tracings and results in increased volume within the right atrium at the end of atrial filling. The excess volume then rapidly flows back into the ventricle during early diastolic filling and can be seen in the form of a rapid y-descent. The combination of large V-waves ("ventricularization" pattern) and an elevated mean right atrial pressure does correlate, however, with significant TR [21]. Overall, the utility of isolated invasive hemodynamics to diagnose severe TR is limited and generally not used as a primary tool.

Doppler Echocardiography

Doppler echocardiography is the primary tool used to evaluate tricuspid regurgitation. An extensive and detailed review of the many parameters used to quantify TR is beyond the scope of this text but can be found elsewhere [22]. Core principles are reviewed here, however, with a focus on severe TR given the general recommendations for intervention at that level of valve dysfunction [4, 22].

In severe TR, the additional volume within the right atrium should result in increased flow across the TV during ventricular filling. Because diastolic filling time should not be affected by TR, the velocity of flow across the TV should increase with larger volumes (i.e., worsening TR). Criteria define tricuspid valve inflow velocities of $\geq$1 m/s as correlating with severe TR [23].

Quantification of regurgitant blood volume across the tricuspid valve can be used to identify severe TR. Operating from the conservation of mass principle, the volume entering the right ventricle during diastolic filling will equal the forward flow volume plus the regurgitant volume during systole [TV inflow volume = Right ventricular forward flow volume + TV regurgitant volume] [23]. Using the principle of

time-velocity integral (TVI), both the TV inflow volume and the right ventricular outflow tract volume (right ventricular forward flow volume) can be estimated (detailed methodology of TVI calculation described elsewhere) [24, 25]. Therefore, TV regurgitant volume = TV inflow volume − Right ventircular outflow volume. Regurgitant volumes of ≥45 mL are indicative of severe TR; however, technical challenges with signal acquisition can affect the accuracy of this calculation [23].

Adopting the concept of proximal isovelocity surface area (PISA) measurements from mitral valve evaluations provides another method of TR quantification [26]. Although more thorough descriptions of PISA measurements are available [23], a brief description is provided here. Again, derived from the basis of conservation of mass, the volume of blood per time unit (known as flux) flowing through two points should be equal. Fluid volume is represented by area × velocty; as such, the continuity equation can be represented as $A_1v_1 = A_2v_2$. By allowing A_1 to be the regurgitant area of the TV, v_1 becomes the velocity through the valve, which is acquired by Doppler velocity measurement at the regurgitant orifice. The second area of flux is then identified as the hemisphere through which regurgitant flow passes at an identified velocity. This second velocity is identified using the Nyquist limit, which represents the maximal frequency shift detectable by Doppler based on acquisition settings [15, 26]. Visually, adjusting the Nyquist limit allows a hemisphere to be defined on color Doppler images, the radius of which can be measured and used to calculate surface area [23, 26]. Mathematical rearrangement then allows the regurgitant area to be calculated ($A_1 = \frac{A_2 v_2}{v_1}$). A regurgitant area of ≥0.4 cm^2 is used to define severe TR, although PISA quantification may underestimate severity of TR [23, 27].

Treatment of Tricuspid Regurgitation

Guideline recommendations suggest that severe tricuspid regurgitation be addressed in the presence of symptoms or in the event of right ventricular dysfunction [4]. Similar to tricuspid stenosis, the presence of severe TR typically occurs in the context of left-sided valve disease [3]; therefore, the surgical therapies upon a regurgitant tricuspid valve (repair or replacement) are often tied to operative plans for the left-sided valve lesion, if at all [4]. Traditionally, treatment of the left-sided valve lesion was thought to be sufficient to allow improvement in TR [1]. For isolated, severe secondary TR, surgical valve repair or replacement is not commonly performed and may be due to the operative risk associated with open valve procedures in the presence of right ventricular dysfunction [1, 19]. For this latter scenario, newer percutaneous approaches may mitigate operative risks and provide therapeutic strategies in generally untreated cohort by using novel technologies and approaches [28, 29]. These newer methods are currently investigational and are undergoing clinical testing to prove safety and efficacy.

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