Complete Tricuspid Valve Evaluation
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Anatomy

The tricuspid valve is a complex, continuous veil of thin fibrous tissue, with a considerably larger and irregular orifice than the mitral valve. The annulus is ovoid in shape and the fibrous tissue is separated into three leaflets of unequal size. The largest leaflet is the anterior leaflet, which is semicircular is shape and stretches from the infundibulum anteriorly to the inferolateral wall posteriorly. The septal leaflet is semi-oval, stretches from the infundibulum posteriorly along the interventricular septum to the posterior right ventricular wall, and it is the least mobile of the leaflets. The insertion of the septal leaflet is characteristically ≤ 10mm inferior or apical relative to the septal insertion of the anterior mitral valve leaflet. The posterior leaflet is the smallest leaflet and it stretches from the posterior interventricular septum to the inferolateral wall of the ventricle. The three papillary muscles supporting the tricuspid valve lie beneath the three corresponding commissures. The anterior papillary muscle is the largest, lies beneath the commissure between the anterior and posterior leaflets, and arises from the moderator band and the anterolateral free wall of the ventricle. The posterior papillary muscle lies beneath the commissure between the posterior and septal leaflets. The smallest (sometimes absent) septal papillary muscle anchors the septal and anterior leaflets high in the infundibulum. Chordae arising from the papillary muscles attach to the free edges of the corresponding leaflets which they support. Knowledge of tricuspid valve anatomy (number of leaflets & papillary muscles, inferior and apical insertion of the septal leaflet, origin of the anterior papillary muscle from the moderator band) is frequently valuable for intraoperative echocardiography, particularly congenital ventricular situs.

Flow Dynamics

There are several differences in flow characteristics of the tricuspid valve from the mitral valve. Tricuspid valve opening normally precedes mitral valve opening due to the lower pressure in the right ventricle, the time required for the right ventricular pressure to fall to the level of the right atrium is less. Also, because the left ventricle depolarizes first, mitral valve closure precedes tricuspid valve closure. This results in a tricuspid diastolic flow period which is slightly longer than that of the mitral inflow. Additionally, the right atrium contracts prior to the left atrium and therefore the tricuspid A wave precedes the mitral A wave. The impetus for AV
valvular flow is the diastolic atrioventricular pressure gradient, which is governed by the net stiffness or compliance of the two chambers and the inherent resistance of the valve. The absolute magnitude of tricuspid E-wave velocity and the E-wave deceleration rate is lower than that of the mitral valve, due to the lower right atrial pressure and the greater compliance of the right ventricle respectively. Despite the lower tricuspid inflow velocities, flow remains constant across the tricuspid and mitral valve due to the larger tricuspid valve orifice. Respiratory effects are more predominant on normal tricuspid flow, with inspiration causing an approximate 14% increase in E and A wave velocities, yet the E/A ratio remains unchanged.

Interrogation

Typically the tricuspid valve is examined in the imaging planes in which the right ventricle is visualized, most commonly the mid-esophageal four-chamber view at a zero degree imaging plane. Here, adjacent to the interventricular septum the septal leaflet can be seen with the anterior leaflet of the mitral valve bordering the opposing side of the septum. This imaging plane places the anterior leaflet of the tricuspid valve to the far left of the image attached to the right ventricular free wall. The tricuspid valve can be sequentially scanned from inferior to superior by slowly withdrawing the probe, starting from an inferior position with the coronary sinus in view. If mid-esophageal four-chamber view provides a perpendicular view of the annulus, then tricuspid annular peak systolic excursion (TAPSE) via tissue Doppler exam can provide an objective measure of right ventricular function. Otherwise, TAPSE can be assessed via a deep transgastric view with the probe rotated to the patient’s right. This provides an image similar to a transthoracic apical four-chamber view. Next, while keeping the tricuspid valve in the center of the image, the multiplane angle is rotated forward approximately 30-40 degrees. This is often the view which places tricuspid valve inflow parallel to the axis of spectral and color-flow Doppler, to allow for accurate assessment of pressure gradient and flow patterns. Doppler interrogation is also commonly performed from a mid-esophageal four-chamber view. Rotating the multiplane image further forward to 60-90 degrees yields a mid-esophageal inflow/outflow view by adding in the infundibulum, pulmonic valve, and the proximal pulmonary artery. With the pulmonic valve now in view the posterior leaflet is seen on the left side of the image attached to the RV free wall and the leaflet on the right is typically the anterior leaflet, although retroflexion of the probe can allow visualization of the septal leaflet on the right side of the image. From a transgastric view at zero degrees, the tricuspid valve, papillary muscles, and chordae can be seen en-fosse by rotating the image to the right and withdrawing the probe to the base of the heart. Slow retroflexion and anteflexion of the probe can optimize structure visualization. In abnormals, particularly adult congenital cases, an alternative to the mid-esophageal inflow-outflow view can be obtained from this transgastric view by rotating the image plane to 90-120 degrees. This places the image more perpendicular to tricuspid valve inflow, yet provides visualization of the infundibulum and subvalvular
pulmonic valve anatomy. Real-time three-dimensional imaging has been described as useful for characterization of tricuspid valve anatomy and pathology. Although tricuspid valve examination is described in the SCA/ASE statement document on intraoperative examination, there has not been formulated a systematic exam of the tricuspid valve with two-dimensional and corresponding three dimensional imaging.

**Right Ventricular Inflow Obstruction-Tricuspid Stenosis**

Acquired tricuspid stenosis is most commonly seen in rheumatic heart disease, but may be present in malignant carcinoid or endocardial/endomyocardial fibrotic diseases. Congenital tricuspid stenosis is rare and tricuspid atresia is commonly found in association with right ventricular hypoplasia and pulmonic atresia. Thrombi or tumors may also cause leaflet or orifice obstruction. In rheumatic heart disease the tricuspid leaflets appear echo dense due fibrosis, most commonly demonstrated at the leaflet edges. Chordal thickening is also seen, but both the leaflets and chordal changes are less pronounced than in mitral stenosis. Calcifications are rarely noted in rheumatic tricuspid stenosis. Fibrosis and fusion of the commissures leads to leaflet doming typical of rheumatic disease, with anterior tricuspid valve leaflet doming being most prominent. In general, only patients with anterior leaflet doming have a significant reduction in tricuspid valve orifice sufficient to produce a hemodynamically detectable gradient. Transvalvular gradients are obtained using a simplified Bernoulli equation, with mean gradients correlating closely with those obtained at catheterization. Calculating tricuspid valve area is problematic in that stenosis is rarely seen without tricuspid valve regurgitation, thereby invalidating the continuity equation. Use of the pressure half-time equation is not commonly used since this method was originally validated with a constant for the mitral valve. A constant of 190 ms. has been tested with varying results compared to catheterization data. Therefore, 2-D pathognomonic features of the tricuspid valve with a mean inflow gradient of ≥ 5 mm.Hg. and a T½ ≥ 190ms are indicative of significant tricuspid stenosis.

**Right Ventricular Inflow Incompetence-Tricuspid Regurgitation**

Tricuspid insufficiency is most commonly functional due to right ventricular dilation with associated increase in tricuspid annular dimension, a decrease in normal systolic diminution of annular area, or displacement and abnormal contraction of the papillary muscles. Functional tricuspid regurgitation is usually due to left-sided valvular or myocardial disease leading to pulmonary hypertension. Acquired causes of tricuspid regurgitation include rheumatic disease, tricuspid valve prolapse, endocarditis, carcinoid syndrome, trauma, or rarely congenital syndromes. Transvenous pacing or defibrillator leads as well as prior trauma due to myocardial biopsy following cardiac transplantation can impair normal tricuspid valve function. Carcinoid tumors are seen in patients with hepatic metastases and frequently present with a syndrome of
episodic bronchospasm, flushing, and diarrhea. The fibrosis of the valve leads to both tricuspid stenosis, but most prominently with regurgitation. Isolated tricuspid regurgitation is rare; instead it is seen more frequently in those with mitral valve prolapse +/- connective tissue disorders (e.g. Marfan’s syndrome). Ebstein’s anomaly is a congenital disorder characterized by an apically displaced and deformed tricuspid valve, with the atrialized portion of the right ventricle leading to right ventricular systolic dysfunction. Diastolic regurgitation can occur in patients with arrhythmias. Minor tricuspid regurgitation can be seen in normal subjects, with an increasing incidence of 30% after the age of 60 years. Tricuspid regurgitation is also normally seen in physically conditioned athletes. Generally, normal tricuspid regurgitation does not extend greater than 1 cm. into the right atrium and it does not comprise greater than 18% of the right atrium. Due to the lack of extensive data on quantification of tricuspid regurgitation, integration of parameters and findings are required to substantiate significant regurgitation. Specific 2-D pathologic findings, a vena-contracta of >0.7 cm, a regurgitant jet area >10 cm. $^2$, and hepatic vein flow reversal support significant tricuspid regurgitation.

Workshop: Anatomy, imaging techniques, echocardiographic windows, and case studies will be discussed.

References


