Tricuspid Valve and Pulmonic Valve

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Objectives:
1. Review the structural anatomy of the normal tricuspid and pulmonic valve
2. Describe the most common mechanisms for tricuspid/pulmonic regurgitation and stenosis
3. Recognize the 2D TEE findings of tricuspid and pulmonic valvular disease
4. Assess and quantify tricuspid valve disease

Introduction:
Transesophageal echocardiography is an excellent tool for evaluating the tricuspid valve and allows limited evaluation of the pulmonic valve. TEE provides comprehensive and real time evaluation of a variety of disease processes that may affect the tricuspid or pulmonic valve.

TRICUSPID VALVE

Anatomy and Echocardiographic Imaging

The tricuspid valve complex consists of:
- Three leaflets (anterior, posterior, and septal)
- Chordae tendinae
- Two papillary muscles
- Fibrous tricuspid annulus
- Right atrial/ventricular myocardium

The tricuspid valve separates the right atrium from the right ventricle and serves to regulate blood flow from the atria to the ventricle. The tricuspid valve consists of three leaflets: anterior, posterior and septal (or medial). The free edge of each leaflet is connected to the right ventricle via chordae tendinae. The leaflet attaches to the RA-RV junction at the annulus. The three thin membranous leaflets are separated by indentations in a continuous sheet of leaflet tissue as opposed to true commissures. The tricuspid valve area is normally between 7 to 9 cm² making it the largest cardiac valve. The tricuspid annulus originates in a more apical position in relation to the mitral valve annulus. The annulus is non-planar, semi-elliptical shaped with the posteroseptal portion closest to the RV apex and the anteroseptal portion closest to the atrium (1). The inferior vena cava and the coronary sinus orifice are in close proximity to the tricuspid valve annulus. The shape of the tricuspid valve annulus is a non-planar, semi-elliptical, non-saddle shaped structure, which varies during the cardiac cycle (2). During RV contraction, the annulus plane along the lateral aspect moves towards the RV apex. The lateral aspect of the
annulus moves apically more than the septal aspect of the annulus and creates a hinge-like appearance echocardiographically. This apical movement of the lateral annulus during RV contraction has been validated as a measure of global RV function and is referred to as tricuspid annular plane systolic excursion (TAPSE). A TAPSE < 8.5 mm correlates with a RVEF < 25% and a TAPSE < 15 mm demonstrates a more significant reduced right ventricular ejection fraction (3).

The tricuspid valve annulus also expands and contracts being the largest in late diastole during atrial contraction and the smallest during early and mid-systole (4). Papillary muscles in the right ventricle, the largest being the anterior, which arises from the moderator band, support the tricuspid valve. The other two papillary muscles, the posterior and septal are much smaller than the anterior and occasionally are absent with tricuspid valve chordae inserting directly to the RV wall.

Echocardiographic evaluation of the tricuspid valve includes standard two-dimensional views, color flow Doppler, and spectral Doppler evaluation with both pulse wave and continuous wave Doppler. Recently three-dimensional evaluation of the tricuspid valve has become an additional evaluation technique. Many of the standard 20 views from the SCA/ASE TEE practice guidelines allow evaluation of the right-sided heart valves (5).

<table>
<thead>
<tr>
<th>View</th>
<th>Modality</th>
<th>Right sided Structures</th>
<th>Areas of interest</th>
<th>Other findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mid Esophageal 4 chamber (0-10°)</td>
<td>2-D, CFD, CW</td>
<td>RA, RV, LA, LV, IAS, IVS</td>
<td>Septal leaflet video right Anterior or Posterior leaflet video left</td>
<td>Tricuspid annular dimension in systole/diastole Annular motion TR</td>
</tr>
<tr>
<td>Mid Esophageal RV inflow-outflow view (60-90°)</td>
<td>2-D, CFD</td>
<td>RA, RV, LA, AoV, PV, PA</td>
<td>Anterior leaflet video right Posterior leaflet video left</td>
<td>TR PR PS Main PA</td>
</tr>
<tr>
<td>Mid Esophageal bicaval (90-150°)</td>
<td>2-D, CFD, CW</td>
<td>RA, IAS, SVC, IVC,CS, TV</td>
<td>Intraatrial septum <strong>Doppler evaluation of Tricuspid valve</strong></td>
<td>TR Insertion of coronary sinus catheters</td>
</tr>
<tr>
<td>Transgastric TV short axis (0-20°)</td>
<td>2-D</td>
<td>TV</td>
<td>Ant leaflet video left far field Post leaflet Video left near field Septal leaflet video right</td>
<td>All three TV leaflets in one view</td>
</tr>
</tbody>
</table>
Doppler evaluation of flow through the tricuspid valve is best performed in the midesophageal 4 chamber, midesophageal RV inflow-outflow view or the modified bicaval view. All three views should be obtained and the view providing the best alignment between the Doppler cursor and the area of interest (TV inflow vs. TR jet) should be utilized. For tricuspid inflow, the PW cursor should be placed at the tips of the tricuspid valve and early ventricular filling (E) wave and late diastolic filling from atrial contraction (A) wave should be recorded. Similar to mitral inflow, E wave velocity, A wave velocity, E/A velocity ratio and E-wave deceleration should be obtained. Overall E and A wave velocities are lower than mitral inflow velocities secondary to the larger cross sectional area of the tricuspid valve (2).

Diastolic dysfunction of the right ventricle is characterized by impaired relaxation with A > E or restriction when E >>> A.

Hepatic vein flow analysis is also helpful when assessing the severity of tricuspid regurgitation. Similar to pulmonary venous inflow patterns, hepatic inflow patterns consist of an anterograde S and D wave and a retrograde A wave (atrial contraction). With elevation of right atrial pressure, S wave velocity and S/D ratio decreases and with severe tricuspid regurgitation, the S wave is reversed.

Doppler evaluation of the tricuspid valve can also estimate right ventricular systolic pressure and pulmonary artery systolic pressure as long as pulmonic stenosis is absent. Using the simplified Bernoulli equation, the peak velocity of the TR jet can be used to calculate the pressure gradient between the RV and RA. Addition of RAP pressure to this pressure gradient will yield an estimate of RV systolic pressure.

\[
RVSP \text{ or } PAP \text{ (systolic)} = 4 \, V^2 + CVP
\]

\[
V = \text{peak systolic velocity of TR jet}
\]
RVSP can also be calculated if the patient has a VSD by the following formula:
RVSP or PAP (systolic) = SBP – 4(V_{VSD})^2

**PULMONIC VALVE**

**Anatomy and Echocardiographic Imaging**

The pulmonic valve separates the right ventricle from the main pulmonary artery. It is a trileaflet valve and the leaflets are thinner than aortic valve leaflets. The three leaflets of the pulmonic valve are termed anterior, left and right. The pulmonic valve root anatomy is very similar to the aortic valve root anatomy and includes three leaflets or cusps, associated sinus of Valsalva and a sinotubular junction.

<table>
<thead>
<tr>
<th>View</th>
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<th>Right sided Structures</th>
<th>Areas of interest</th>
<th>Other findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transgastric RV outflow</td>
<td>2-D</td>
<td>RA, RV, TV, PVOT, PV, AoV</td>
<td>RVOT, PV Best view to measure TC annulus- correlates with surgical measurements</td>
<td>Right sided cardiac output (Qp) PR PS</td>
</tr>
<tr>
<td>(120-150°)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Modified Midesophageal</td>
<td>2-D, CFD, PW</td>
<td>Asc Ao, RPA, MPA, PV</td>
<td>Main PA, RPA</td>
<td>Right sided cardiac output (QP) PR PS</td>
</tr>
<tr>
<td>Ascending aortic SAX (60-90°)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Upper esophageal aortic arch</td>
<td>2-D, CFD, PW</td>
<td>PA, PV, Ao arch</td>
<td>Main PA</td>
<td>Right sided cardiac output (QP) PR PS</td>
</tr>
<tr>
<td>SAX (60-100°)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(Modified from Chapter 15, Assessment of the Tricuspid and Pulmonic Valves. Comprehensive Textbook of Perioperative TEE. 2nd eds. Savage, Aronson, Shernan. 2011)

Doppler evaluation of flow through the pulmonic valve is best performed in the upper esophageal aortic arch SAX or the transgastric RV outflow view.

Measurement of velocity across the pulmonic valve can be obtained from these views. With pulmonic regurgitation, estimation of pulmonary artery diastolic pressure can be performed. From the PR jet, the end diastolic jet velocity can be added to RVDP (or CVP) as RVDP equals CVP at end diastole.

PAP (diastolic) = 4 \( V^2 \) + RVDP or CVP
\[ V = \text{end diastolic velocity of PR jet} \]

PAP (mean) = 4 \( V^2 \) + RVDP or CVP

\[ V = \text{peak velocity of PR jet} \]

Cardiac output from the right heart can be calculated using 2-D imaging in conjunction with pulse wave Doppler recordings. The RV stroke volume can be estimated from measuring the PV diameter and obtaining the pulmonic valve velocity time integral.

RV stroke volume = 0.785 x (PV diameter)\(^2\) x VTI pv

Limitations to transesophageal echocardiographic evaluation of the pulmonic valve are secondary to the pulmonic valves anterior location in the thorax. Limited high-resolution views of the pulmonic valve makes comprehensive evaluation of pulmonic valve disease difficult utilizing TEE. **Epicardial** imaging of the pulmonic valve is feasible after sternotomy utilizing the **epicardial RV outflow tract view** and allows high-resolution 2D imaging of the pulmonic valve and standard Doppler quantification of the severity of pulmonic stenosis and regurgitation (6).

**Specific Pathologic Conditions**

**Tricuspid regurgitation (2,3)**

- Most common right heart valve abnormality
- Normal with aging (small CFD jet)
- Functional TR
  - Annular dilation
  - RV dilation
  - Subvalvular anatomy distortion
  - Secondary to:
    - Left sided lesions
    - Pulmonary hypertension
    - RV ischemia/infarction
- Pericarditis
- Pulmonic stenosis
- Trauma to TV
- Tricuspid regurgitation jet velocity directly related to RV systolic pressure, NOT TR severity
- Used to calculate RVSP (PASP)

### GRADING OF TRICUSPID REGURGITATION

<table>
<thead>
<tr>
<th>Severity</th>
<th>RA Area (%)</th>
<th>TR Jet Area (cm(^2)) *</th>
<th>Length of TR jet (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trace</td>
<td>&lt; 2</td>
<td>&lt; 1.5</td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>≤ 20</td>
<td>2-5</td>
<td>1.5-3.0</td>
</tr>
<tr>
<td>Moderate</td>
<td>21-33</td>
<td>5-10</td>
<td>3.0-4.5</td>
</tr>
<tr>
<td>Severe</td>
<td>≥ 33</td>
<td>&gt; 10</td>
<td>&gt; 4.5</td>
</tr>
</tbody>
</table>
(* Nyquist limit set 50-60 cm/s)  
(Table modified from References 7,8,9,10)

<table>
<thead>
<tr>
<th>Severity</th>
<th>Vena Contracta (mm)</th>
<th>TR PISA R radius (mm) **</th>
<th>EROA (mm²)</th>
<th>Regurgitant Volume (ml)</th>
<th>Hepatic Vein Flow</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>Not defined</td>
<td>&lt;5</td>
<td>-----</td>
<td>-----</td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>Not defined</td>
<td>5-9</td>
<td>-----</td>
<td>-----</td>
<td>blunting *</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt; 7</td>
<td>&gt; 9</td>
<td>≥ 40</td>
<td>≥ 45</td>
<td>Reversal</td>
</tr>
</tbody>
</table>

(* other common conditions can cause blunting including elevated RAP, decreased RV compliance, atrial fibrillation and is very non specific for severity of moderate TR  
** Nyquist limit set 28 cm/s with baseline shift)  
(Table modified from Reference 10, 11)

**TRICUSPID STENOSIS (12)**

- Most commonly rheumatic (≈ 90%)
- In patients with rheumatic mitral valve disease, TS occurs in only 3-5% of patients
- Other etiologies  
  - Congenital TS  
  - RA tumors  
  - Carcinoid heart disease  
  - Endomyocardial fibrosis  
  - Valvular vegetations  
  - Extra cardiac tumors
- 2-D findings  
  - Doming during diastole  
  - Thickening of leaflet tips  
  - Restricted leaflet movement  
  - Thickened and shortened chordae  
  - Commissural fusion
- Doppler findings  
  - Increased E wave velocity (> 1.5 m/s)

**GRADING of TRICUSPID STENOSIS SEVERITY**

<table>
<thead>
<tr>
<th>Specific Findings</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Pressure Gradient</td>
<td>≥ 5 mm Hg</td>
</tr>
<tr>
<td>Inflow time velocity integral</td>
<td>&gt; 60 cm</td>
</tr>
<tr>
<td>Pressure half time (T ½)</td>
<td>≥ 190 ms</td>
</tr>
<tr>
<td>Valve area by Continuity equation</td>
<td>≤ 1 cm²</td>
</tr>
<tr>
<td>Supportive Findings</td>
<td></td>
</tr>
<tr>
<td>Enlarged right atrium</td>
<td>&gt; Moderate</td>
</tr>
</tbody>
</table>
Inferior vena cava | Dilated
---|---
(Table modified from Reference 13)

**Pulmonic Regurgitation (14)**

- Commonly seen in adults (trivial or mild disease)
- Increased prevalence with aging
- Usually not clinically significant
- Clinically significant PS usually associated with
  - Pulmonary hypertension
  - Carcinoid
  - Rheumatic
  - Trauma
  - Endocarditis
  - Previous congenital cardiac surgery (TOF, corrected congenital PS)
- Used to calculate PADP and mean PA pressure

**GRADING of PULMONARY REGURGITATION**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonic Valve</td>
<td>Normal</td>
<td>Normal or abnormal</td>
<td>Abnormal</td>
</tr>
<tr>
<td>RV size</td>
<td>Normal</td>
<td>Normal or dilated</td>
<td>Dilated</td>
</tr>
<tr>
<td>Jet size by CFD</td>
<td>Thin (&lt; 10 mm)</td>
<td>Intermediate</td>
<td>Large, wide origin</td>
</tr>
<tr>
<td>Jet density and deceleration rate</td>
<td>Soft, slow deceleration</td>
<td>Dense, variable deceleration</td>
<td>Dense, steep deceleration, early termination of diastolic flow</td>
</tr>
<tr>
<td>CWD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonic systolic flow compared to systemic flow</td>
<td>Slightly increased</td>
<td>Intermediate</td>
<td>Greatly increased</td>
</tr>
</tbody>
</table>

(Table modified from reference 11)

**Pulmonic Stenosis (14)**

- 95% congenital or genetic
- 2-D findings
  - Commissural fusion
    - Acommissural
    - Unicommissural
    - Bicuspid
    - Dysplastic trileaflet valve
  - Leaflet thickening
  - Restricted systolic motion
  - RV hypertrophy
  - RA enlargement
  - Moderate to severe TR
- Carcinoid heart disease
**GRADING of PULMONARY STENOSIS**

<table>
<thead>
<tr>
<th>Severity</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak Velocity (m/s)</td>
<td>&lt; 3</td>
<td>3-4</td>
<td>&gt; 4</td>
</tr>
<tr>
<td>Peak Gradient (mm Hg)</td>
<td>&lt; 36</td>
<td>36-64</td>
<td>&gt; 64</td>
</tr>
</tbody>
</table>

(Table modified from Reference 13)

**DISEASES OF THE TRICUSPID AND PULMONIC VALVE**

**Functional or Secondary Tricuspid Regurgitation**

- Most common cause
  - Annular dilation
  - Dilation of the right ventricle
  - Secondary to
    - Pulmonary HTN
      - LV valvular abnormality
      - LV failure
      - Chronic RV ischemia
      - Cardiomyopathy
      - Volume overload
  - No intrinsic TV leaflet pathology
    - Asymmetrical dilation of annulus with a circular morphology
    - Loss of sphincter-like contraction of TC annulus

**Organic or Primary Tricuspid Regurgitation**

**Degenerative tricuspid regurgitation (3)**

- Billowing tricuspid valve
  - Associated with mitral valve prolapse
  - Coaptation line behind annular plane
  - Associated with diffuse myxomatous degeneration (Barlow’s disease)
- Flail tricuspid valve
  - Free edge of leaflet completely reversed into the right atrium
  - Consequence of ruptured chordae
    - Degenerative TR
    - Infective endocarditis
    - Trauma

**Ebstein’s Anomaly (15, 16)**

- Rare congenital condition (≈1 per 200,000 live births)
- Carpentier classification-anatomic at time of surgery
  - Type A-volume of true RV adequate
  - Type B- large “atrialized” component, anterior leaflet moves freely
- Type C: significant restriction of anterior leaflet, prone to RVOT obstruction
- Type D: near complete atrialization of RV with small infundibular component

- Echocardiographic grading for neonates (17)
  - Grades I-IV
    - Ratio of
      \[
      \frac{\text{area RA + atrialized right ventricle}}{\text{area of functional right ventricle + left atrium + left ventricle}}
      \]
    - Grade 1 ratio < 0.5
    - Grade 2 ratio 0.5-0.99
    - Grade 3 ratio 1.0-1.49
    - Grade 4 ratio > 1.5

- Echocardiographic Findings
  - Enlarged and fenestrated anterior TCV leaflet with hypoplastic posterior and septal leaflets
  - Posterior and septal leaflet thickened and adherent to RV wall (failure of delamination)
  - Apical displacement of the leaflets (septal leaflet > anterior or posterior leaflet)
  - Associated TR and RV dysfunction
  - Septal leaflet insertion > 8 mm/m² apically inserted in comparison to the insertion of the anterior mitral leaflet (ME 4-C view)
  - RV divided into two sections
    - “Atrialized” portion of the RV (thin RV wall superior to TC annulus)
    - Ventricular portion of the RV (trabecular and RVOT region)
  - TC annular dilation
  - Dilation of both portions of the RV
  - Marked enlargement of Right atrium
  - Patent foramen ovale (80-94% of cases)

- Other congenital abnormalities
  - MV prolapse
  - Bicuspid Aortic Valve
  - Coarctation of the aorta
  - Ventricular septal defects
  - Pulmonary stenosis
  - Hypoplastic pulmonary artery

- Physiology
  - RV failure
  - Tricuspid regurgitation
  - Atrialized portion of RV expands with atrial contraction
  - Arrhythmias
Carcinoid Heart Disease (18)

- Enterochromaffin cells in GI tract
- Primary carcinoid tumors which drain into portal circulation for liver metabolism
- Metastatic carcinoid tumors to liver with vasoactive substances released into systemic venous circulation with resultant clinical signs (carcinoid syndrome and carcinoid heart disease)
- Vasoactive amines
  - Serotonin
  - Bradykinin
- Vasoactive amines degraded in pulmonary circulation and therefore right heart valves affected and sparing of left heart valves
- Right heart valves (TV and PV)
  - Fibrosis
  - Chronic inflammation
  - Neovascularization
  - Carcinoid plaque deposition on “downstream” endocardial surfaces (Ventricular surface of posterior and septal leaflets of TV, PA surface of PV)
  - Carcinoid plaques on both sides of anterior leaflet of TV
  - Short, thickened, retracted and immobile TCV leaflets leading to severe TR.
  - Leads to mixed physiologic picture of primarily tricuspid regurgitation and pulmonic stenosis
- Echocardiographically
  - Shortened and thickened tricuspid leaflets
  - Moderate to severe TR
  - Associated tricuspid stenosis
  - Thickened, retracted pulmonic valve with doppler gradient (PS)
  - Pulmonic regurgitation
  - Left sided valvular lesions in 7% (associated with PFO or primary carcinoid of the lung)

Drug-induced Cardiac Valvulopathy (18)

- Ergot alkaloids
  - Methysergide—similar to serotonin
    - Left and right heart valvulopathy
  - Ergotamine
- Phenteramine—MAO inhibitor
  - Inhibits serotonin degradation-appetite suppressant
  - Still available, not directly implicated in causing valvulopathy
- **Fenfluramine**
  - Serotonin releaser and reuptake inhibitor
- **Dexfenfluramine**
  - Dextroisomer of Fenfluramine
- **1990s—linked drugs to PA HTN and left sided heart disease**
  - 24 women
    - All with aortic and/or mitral regurgitation
    - 5 (28%) required valve replacement surgery
  - Histopathology findings of plaque like encasement of valves similar to carcinoid heart disease

**Rheumatic Heart Disease (3)**

- Less common than mitral rheumatic heart disease
- Leaflet doming, thickening, restriction
- Chordal contracture, commissural fusion
- Papillary to chordal fusion
- Development of TR more common than TS
- Improved visualization with 3D TEE
- Secondary to PA HTN related to left sided valvular disease

**Endocarditis**

- Diagnostic procedure of choice
  - Vegetations
  - Perivalvular abscesses
- Less common than mitral or aortic valve endocarditis
  - More common involvement of TCV with IVDA
- Comprehensive evaluation of all valves in patients with presumed endocarditis
- Risk factors
  - Immunosuppression
  - IV drug abusers
  - Indwelling catheters/pacing wires

**Pacemaker/Defibrillator Leads (1, 23)**

- Underappreciated
- TR worsened by one grade or more after implantation in 24.2% of patients
- Worsening more common in patients undergoing AICD
- 17.8% of patients with mild TR increased to moderate – severe TR after lead implantation
- Persistent problem after tricuspid annuloplasty therefore recommend removal of trans-tricuspid lead and replacement with epicardial lead
SURGICAL PROCEDURES RELATED TO THE TRICUSPID AND PULMONIC VALVE

Tricuspid Regurgitation—when to repair

Tricuspid valve annuloplasty is the treatment of choice for functional tricuspid regurgitation secondary to pulmonary hypertension from left sided valvular heart disease. Indications for surgical repair of the tricuspid insufficiency include right heart failure and moderate to severe tricuspid regurgitation. Some recommend repair based upon the degree of dilation of the tricuspid annulus regardless of the severity of tricuspid regurgitation given the dynamic nature of tricuspid regurgitation relating to changes in RV preload, afterload and RV systolic function. The annulus in patients with functional TR becomes more planar and circular with dilation in the septal-lateral direction (1).

When to repair the tricuspid valve when doing an associated procedure on the mitral valve has been a controversy in the literature. Recent publications suggest that the TC valve should be repaired when the tricuspid annulus diameter (anteroseptal commisure-anteroposterior commisure distance) was greater than twice the normal size (≥ 70 mm). In follow up, TR grade and NYHA class were significantly improved in patients undergoing TV annuloplasty plus mitral valve repair in patients when using this criteria. In-hospital mortality and survival rates were also improved in the TV annuloplasty group (19). Concomitant surgical repair of the tricuspid valve at the time of mitral valve surgery has demonstrated improved outcomes, functional class and survival and should be considered standard of care (1). Surgical repair of the tricuspid valve includes ring annuloplasty, purse-string annuloplasty and tricuspid valve replacement. Ring annuloplasty is associated with improved event free survival, long-term survival and freedom from recurrent TR when compared with purse-string annuloplasty (20). Patients requiring repeat sternotomy to specifically address symptomatic severe TR after previous left-sided valvular surgery have increased perioperative and postoperative risk and poorer outcomes when compared to patients undergoing primary operations to concomitantly surgically correct both left and right-sided valvular processes (12).

Tricuspid valve annuloplasty is also the procedure of choice for organic TV disease when feasible as it is associated with improved perioperative, midterm and event free survival than TV replacement (21). Comparing outcomes for tricuspid valve replacement vs. repair for organic disease, predictors for worse outcomes included TV replacement, male sex, age, left ventricular dysfunction, endocarditis, previous stroke or renal failure and associated mitral valve surgery (12). Surgical correction of Ebstein’s anomaly includes tricuspid valve repair with movement of anterior papillary muscle toward the ventricular septum to facilitate anterior to septal leaflet coaptation with a purse string annuloplasty. In specific cases, surgical plication or excision of the atrialized right ventricle will be performed (16). Tricuspid valve replacement is the procedure of choice for carcinoid heart disease (12). Perioperative treatment may include the use of octreotide to treat carcinoid crisis.
2006 ACC/AHA Guidelines Pertaining to the Surgical Management of Tricuspid Valve Disease/Regurgitation (22)

Class I
Tricuspid valve repair is beneficial for severe TR in patients with MV disease requiring MV surgery. (Level of Evidence: B)

Class IIa
1. Tricuspid valve replacement or annuloplasty is reasonable for severe primary TR when symptomatic. (Level of Evidence: C)
2. Tricuspid valve replacement is reasonable for severe TR secondary to disease/abnormal tricuspid leaflets not amenable to annuloplasty or repair. (Level of Evidence: C)

Class IIb
Tricuspid annuloplasty may be considered for less than severe TR in patients undergoing MV surgery when there is pulmonary hypertension or tricuspid annular dilatation. (Level of Evidence: C)

Class III
1. Tricuspid valve replacement or annuloplasty is not indicated in asymptomatic patients with TR whose pulmonary artery systolic pressure is less than 60 mm Hg in the presence of a normal MV. (Level of Evidence: C)
2. Tricuspid valve replacement or annuloplasty is not indicated in patients with mild primary TR. (Level of Evidence: C)

Tricuspid Stenosis
For patients with symptomatic TS or a valve area less than 1 cm², medical therapy is ineffective and tricuspid valve replacement is the procedure of choice.

2007 ESC Guidelines Pertaining to the Surgical Management of Tricuspid Valve Disease (14)

Class I
Severe TR in a patient undergoing left-sided valve surgery (Level of Evidence: C)
Severe primary TR and symptoms despite medical therapy without severe RV dysfunction (Level of Evidence: C)
Severe TS (with or without TR) with symptoms despite medical therapy (Level of Evidence: C)
Severe TS (with or without TR) in a patient undergoing a left-side valve intervention (Level of Evidence: C)
Class IIa
Moderate organic TR in a patient undergoing left-sided valve surgery (Level of Evidence: C)
Moderate secondary TR with dilated annulus (>40 mm) in a patient undergoing left-sided valve surgery (Level of Evidence: C)
Severe TR and symptoms; after left-sided valve surgery; in the absence of left-sided myocardial, valve, or RV dysfunction; and without severe PHT (systolic pulmonary artery pressure > 60 mm Hg) (Level of Evidence: C)

Class IIb
Severe isolated TR with mild or no symptoms and progressive dilation or deterioration of RV function (Level of Evidence: C)

Pulmonic Regurgitation (14)

Patients with significant pulmonic regurgitation present with signs of RV failure secondary to RV dilation and decreased RV function and are prone to ventricular arrhythmias and sudden death. Unfortunately chronic severe PR is well tolerated for years allowing for the RV to dilate and fail without clinical symptoms.

Procedural intervention is indicated for severe PR when any of the following conditions are present:
Arrhythmias or prolongation of the QRS (≥ 180 ms)
Decrease RVEF (<40%) by cardiac MRI
Increase RV chamber size (RVEDV ≥ 160 ml/m² or RVESV ≥ 82 ml/m²) by cardiac MRI
Decline in aerobic capacity
Progressive annular dilation
Severe PR in a patient undergoing another cardiac operation.

In patients undergoing pulmonary valve replacements, bioprosthetic valves are preferred as homografts have unpredictable durability and mechanical valves are associated with thrombosis due to the low-pressure conditions in the right heart. Percutaneous valve replacements techniques are now clinically available and have been used successfully for patients with pulmonic valve regurgitation.

Pulmonic Stenosis (14)

Patients with clinically significant pulmonic stenosis typically present with fatigue and dyspnea. With progression of disease, systemic or suprasystemic RV pressure leads to exertional lightheadedness or syncope and right heart failure. Intervention is indicated with peak gradient across the pulmonic valve ≥ 50 mm Hg and percutaneous balloon valvuloplasty is the procedure of choice for congenital pulmonic stenosis. Post procedure pulmonic regurgitation is common following valvuloplasty. For patients with acquired pulmonic stenosis from carcinoid or rheumatic disease, pulmonic valve replacement is preferable over balloon valvuloplasty secondary due to leaflet morphology, and concomitant pulmonic regurgitation. Patients with pulmonic stenosis typically have
RVOT obstruction from hypertrophy of the RV myocardium. After relief of pulmonic valve stenosis, the RVOT obstruction may worsen and create a “suicide RV” with suprasystemic RV pressures and systemic hypotension and cyanosis. Preprocedure beta blockade therapy is indicated and should be continued for 3-6 months post procedure as the ventricle remodels and the RVOT obstruction resolves.

In patients undergoing surgery for pulmonic stenosis, similar concerns regarding the choice of prosthesis exists as for pulmonic regurgitation.

**Conclusion**

Transesophageal echocardiography provides an accurate and comprehensive evaluation of disease processes that affect the tricuspid and pulmonic valve. Utilization of perioperative transesophageal echocardiography can lead to alterations in surgical and procedural management. Recent guidelines and consensus statements regarding the management of patients with right sided valve issues have been published and serve as an excellent clinical reference for patient management.

References: