Tricuspid and Pulmonic Regurgitation: Echocardiographic assessment

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No Disclosures

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44 year old man with end stage liver disease

- Alcoholism
- Anemia, fatigue, jaundice
- Intermittant UGI bleeding
- Then 60 lb weight gain with ascites
- Hospitalized for stabilization and consideration of transplant
- Oliguria after dye procedure
- Creatinine 3.6 and grossly volume overloaded, pulsatile liver
**TR – clinical correlations**

- ≥ moderate usually “functional” – right ventricular (RV) dilatation, distortion of the subvalvular apparatus
- Tricuspid annular dilatation
- Symptoms and signs
  - Fatigue
  - Decreased exercise tolerance
  - Peripheral edema / anasarca
  - Hepatic congestion, enlargement in 90%
  - Pulsatile liver noted inconsistently
  - Decreased appetite
  - Abdominal fullness / ascites
  - JVD with V wave in 35%-75%
- Holosystolic murmur LSB ↑ with inspiration
- Noted in <20% severe TR due to equalization
- ↑ RA mean pressure

**TR - causes**

- Functional: due to RV dilatation from other cause
- Endocarditis
- Trauma
- Carcinoid heart disease
- Rheumatic heart disease
- TV prolapse
- Trauma/iatrogenic (radiation, drugs, biopsy, device lead)
- RV dysplasia
- Endomyocardial fibrosis
- Primary or secondary pulmonary hypertension
- Atrial septal defect / anomalous PV drainage

**TR natural history**

- Prolonged latent period, then:
  - RV and RA volume
  - AF common secondary to ↑ RA
  - R-CHF palliated with diuretics
  - Hepatic congestion + anorexia
  - Reduced exercise capacity
- Reduced longterm survival
- Symptoms improved in 88% operated patients
- ↑ death noted in unoperated group

Survival and TR severity
Nash J, JACC 2004;43:405-409
TR: AHA/ACC Class I and IIA indications
- Class I
  - TV repair is beneficial for severe TR in patients with MV disease requiring MV surgery
- Class IIa
  - TV replacement or annuloplasty is reasonable for severe primary TR when symptomatic
  - TV replacement is reasonable for severe TR secondary to diseased/abnormal TV leaflets not amenable to annuloplasty or repair

TR, AHA/ACC Guidelines continued
- Class IIb
  - "< severe TR in patients undergoing MV surgery if PHT and TV annular dilatation
  - Not indicated if RVSP ≥ 60 mm Hg in the presence of a normal MV
  - TV replacement or annuloplasty is not indicated in patients with mild TR

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

Echo in TR: 80-90% prevalence
- Severity, and cause of TR
- Impact on RV
  - RIMP
  - Displacement TV annulus with tissue Doppler imaging-TAPSE
  - dP/dt
  - Myocardial acceleration during isovolumic contraction
- Dense “dagger-shaped” early peak CW=severe TR
- Severe TR by vena contracta ≥0.7 cm
- PISA
- Others: ↑ IVC, HV systolic reversal – sensitive but also present in AF with < severe TR


ER and regurgitant volume in TR vs MR
Tribouilloy, CM, J Am Soc Echocardiogr 2002;15:958-
TR views, ± color

- RV inflow view
- PSAX
- Apical 4 chamber view
- SC 4 chamber view
- R sided chamber, IVC size
- Septal motion

PW, CW Doppler in TR

- Interrogate VC, PISA, forward and regurgitant spectra in ≥ 2 imaging planes
- Massive TR usually with CW velocity ≤ 2 m/sec due to equalization
- Dense, early peaking, triangular profile, with augmented diastolic flow
- TV E ≥ 1 m/sec = very severe TR
- Hepatic vein: normally dominant systolic flow becomes blunted, eventually reversed (80% sensitive), but affected by AF, ↑ RA pressure

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tricuspid valve</td>
<td>Usually normal</td>
<td>Normal or abnormal</td>
<td>Flail, poor coaptation, etc</td>
</tr>
<tr>
<td>RV/RA/IVC size</td>
<td>Normal</td>
<td>Normal or dilated</td>
<td>Usually dilated</td>
</tr>
<tr>
<td>Jet area, central jet (cm²)</td>
<td>&lt; 5</td>
<td>5-10</td>
<td>&gt;10</td>
</tr>
<tr>
<td>VC width (cm)</td>
<td>Not defined</td>
<td>Not defined, but &lt; 0.7</td>
<td>&gt; 0.7</td>
</tr>
<tr>
<td>PISA radius (cm)</td>
<td>≤ 0.5</td>
<td>0.6-0.9</td>
<td>&gt; 0.9</td>
</tr>
<tr>
<td>CW jet density, contour</td>
<td>Soft and parabolic</td>
<td>Dense, variable contour</td>
<td>Dense triangular, early peaking jet</td>
</tr>
<tr>
<td>Hepatic vein flow</td>
<td>Systolic dominance</td>
<td>Systolic blunting</td>
<td>Systolic reversal</td>
</tr>
</tbody>
</table>

Severe TR, nl RVSP, “mirror image” to and fro flow
67 year old woman

- Sudden onset class III dyspnea
- Found to be in new atrial fibrillation
- No prior echo studies

63 year old woman

- St Jude AVR 10 yr prior
- Edema, fatigue
- Outside workup raised? Constrictive pericarditis
- JVD, hepatomegaly, pulsatil liver

Therapeutic / surgical considerations

- Correctable causes:
  - Pacer lead impingement
  - Pulmonary hypertension (PHT) 2° OSA
- Non-correctable: TR due to primary pulmonary hypertension, or secondary to pulmonary thromboemboli
- Symptom Rx: diuretics, fluid Na+ restriction
- Bowel edema renders oral diuretics ineffective
- Diuretics improve edema but may worsen fatigue and dyspnea.
- Surgical Indications:
  - Severe TR and symptoms
  - Mitral valve disease or other cardiac disease requiring surgery
  - Progressive RV enlargement or dysfunction
  - Traumatic TV flail with severe TR
- Carcinoid patients high-risk - perioperative profound peripheral vasodilatation and hypotension – Rx with octreotide
Color Quantitation

- PISA method more challenging than MR
- Vena contracta > 0.7 cm 0% sensitive and specific
- Eccentric jets problematic
- Record in multiple imaging planes
- Look for congruency of multiple indicators

Surgery discussed

“The outlook for patients who have previously undergone left-sided valvular surgery who subsequently present with symptomatic severe TR is less optimistic. Repeat surgery to specifically address TR in these patients can be performed with acceptable early mortality (8.8%)…”

- Trial of medical therapy offered
- Returns two months later, asymptomatic

Bruce CJ, Connolly HM (Circulation. 2009;119:2726-2734.)
**Pulmonic Regurgitation** - 40-78% of echos
- Pathology rare, usually with other structural disease
- PR severity evaluation is generally qualitative, since quantitative standards lacking
- Think:
  - Pulmonary hypertension
  - Carcinoid
  - Post valvotomy PS
  - Congenital, ie tetralogy post patch PA repair

**PR: 2D views**
- Parasternal short axis RVOT view
- Subcostal short axis
- Primary abn: bicuspid, quadricuspid, doming, prolapse, hypoplasia
- Pulmonary artery size and number
- RV size and function
- Septal motion, LV shape

**Doppler in PR**
- Jet size, extent, spatial orientation largely dictated by regurgitant volume and PA to RV gradient – ie when severe JET IS BRIEF
- VC less well validated than other valves, but likely more reliable than jet area. Cutoffs not established

**CW Doppler in PR**
- End diastolic velocity of PR allows calculation of PA end diastolic pressure = \(4v^2\)
- Dense jet correlates with more severe PR
- Rapid deceleration rate consistent with severe PR but affected by RV status, filling pressures

**Consequences of severe PR**
- Progressive RV dilation
- Reduced RV function
- CHF
- QRS prolongation, ventricular arrhythmias, and sudden death.
Subject to significant errors due to difficulties of measurement of pulmonic annulus and a dynamic RVOT; not well validated.

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<thead>
<tr>
<th>Parameter</th>
<th>Utility, advantage</th>
<th>Disadvantages</th>
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</thead>
<tbody>
<tr>
<td>RV size</td>
<td>RV enlargement sensitive for chronic</td>
<td>Enlargement seen in other conditions</td>
</tr>
<tr>
<td></td>
<td>significant PR. Normal size virtually</td>
<td></td>
</tr>
<tr>
<td></td>
<td>excludes</td>
<td></td>
</tr>
<tr>
<td>Paradoxical septal motion</td>
<td>Simple sign of severe PR</td>
<td>Not specific for PR</td>
</tr>
<tr>
<td>(volume overload pattern)</td>
<td></td>
<td></td>
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<tr>
<td>Jet length-color flow</td>
<td>Simple</td>
<td>Poor correlation with severity of PR</td>
</tr>
<tr>
<td>Vena contracta width</td>
<td>Simple, quantitative method that works well</td>
<td>More difficult to perform; requires good images</td>
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<tr>
<td></td>
<td>for other valves</td>
<td>of pulmonary valve; lacks published validation</td>
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<tr>
<td>Jet deceleration rate-CW</td>
<td>Simple</td>
<td>Sleep deceleration not specific for severe PR</td>
</tr>
<tr>
<td>Flow quantitation-PW</td>
<td>Quantitates regurgitant flow and Fraction</td>
<td>Subject to significant errors due to difficulties</td>
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<td></td>
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Disadvantages:

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PR Therapy

- No medical therapy has been demonstrated to be effective in reducing the degree of PR
- PV replacement for Symptoms
  - arrhythmias
  - decreased RV systolic function (ejection fraction 40%)
  - progressive RV dilation (MR RV EDV ≥ 160 mL/m² or ≥ 82 mL/m² for RV ESV)
  - decline in functional aerobic capacity
  - moderate TR due to progressive annular dilatation
  - Severe PR in pt requiring another cardiac operation
  - Prolonged or ↑ QRS duration (total QRS duration 180 milliseconds or QRS duration increase > 3.5 ms/y)

Results of PV replacement for severe PR

- RV size tends to normalize and functional status improves after PV replacement for PR late after tetralogy of Fallot
- RV function may not fully recover once marked enlargement and systolic dysfunction are evident.