TRICUSPID AND PULMONARY VALVE DISEASE: NEW GUIDELINES

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University of Chicago

IMPACT OF TRICUSPID REGURGITATION

- KM survival curves for patients with TR
- Survival worse in moderate and severe TR

CLINICAL PRESENTATION

Highly variable

**Symptoms**

- vague

**Physical signs**

- often suggest advanced disease

**Asymptomatic, fatigue, RUQ discomfort, fluid retention, peripheral edema**

**Distention of JVP (C-v wave)**

**Auscultative findings of TR**

**Findings of PH, RV failure**

**Hepatic enlargement**

**Ascites, edema**

TRICUSPID VALVE DISEASE

In 2018

...echocardiography remains the mainstay for the assessment of tricuspid regurgitation

Accurate assessment is in YOUR hands
THE NORMAL TRICUSPID VALVE COMPLEX

1. Three leaflets
   - Anterior
   - Septal
   - Posterior
2. Fibrous annulus
3. Chordae tendinae
4. Papillary muscles
5. RA myocardium
6. RV myocardium

Courtesy Dr. Stephen P. Sanders, Professor of Pediatrics (Cardiology), Harvard Medical School

THE NORMAL TRICUSPID VALVE

Ra
↓
↓
Apex

Ton-Nu Circulation. 2006
THE TV ON 3D ECHO

RV perspective

RA perspective
AMBIGUITY OF LEAFLET IMAGED ON 2D

Apical 4-chamber view

MECHANISMS OF TRICUSPID REGURGITATION

Primary (or “Organic”)
Intrinsic abnormality of the valve apparatus
15-30%* of TR

Secondary (or “Functional”)
TV annular dilatation, RV dilatation and papillary muscle displacement
70-85%* of TR

Antunes MJ, Barlow JB, Heart 2007
PRIMARY “ORGANIC” TR

Intrinsic abnormality of TV leaflets and/or support apparatus

**Acquired**
- Degenerative, myxomatous
- Rheumatic disease
- Endocarditis
- Carcinoid
- Toxins
- Chest wall trauma
- Iatrogenic (leads, RV biopsy)
- Other (e.g. ischemic, PM rupture)

**Congenital**
- Ebsteins anomaly
- TV dysplasia
- TV tethering
- Perimembranous VSD
- Ventricular septal aneurysm
- Repaired tetralogy of Fallot
- Congenitally corrected TGA
- Other (giant RA)

FUNCTIONAL TRICUSPID REGURGITATION

- Pulmonary hypertension
- RV dysfunction
- Left heart disease
- Atrial fibrillation
- RA abnormalities

70-85%* of TR

Dreyfus G. J Am Coll Cardiol 2015;65:2331–6
MECHANISMS OF TRICUSPID REGURGITATION

<table>
<thead>
<tr>
<th>Group (N)</th>
<th>Controls (99)</th>
<th>Id FTR (141)</th>
<th>PHTN FTR (140)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TR sPAP</td>
<td>None</td>
<td>&lt;50 mmHg</td>
<td>≥ 50 mm Hg</td>
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<tr>
<td>Associations:</td>
<td>Controls</td>
<td>Aging, Afib</td>
<td></td>
</tr>
<tr>
<td>TA</td>
<td>Normal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tenting</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>RA Base</td>
<td>Normal</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Ta</td>
<td>Normal</td>
<td>↑↑↑↑↑</td>
<td></td>
</tr>
<tr>
<td>RV Base</td>
<td>Normal</td>
<td>↑↑↑↑↑</td>
<td></td>
</tr>
<tr>
<td>RV Length</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Remodeling</td>
<td>--</td>
<td>Conical</td>
<td>Elliptical</td>
</tr>
</tbody>
</table>


ECHOCARDIOGRAPHIC ASSESSMENT OF THE TV

1. Leaflets
   - Prolapse, flail
   - Thickening, restricted
   - Coaptation, tethering
2. Annulus diameter
3. RA size
4. RV size and function
5. Tricuspid valve dysfunction
   - Stenosis
   - Regurgitation: Jet area, VC, PISA, Jet density, Hepatic veins
6. Systolic PA pressure + IVC
7. Any associated left-sided heart disease
Echocardiographic Assessment of the TV

1. Leaflets
   - Prolapse, flail
   - Thickening, restricted
   - Adequate coaptation?
   - Tethering/tenting
   - Perforation/Trauma

Leaflets: Primary “Organic” TR

Prolapse – all 3 leafets
LEAFLETS: PRIMARY
“ORGANIC” TR

Traumatic Ruptured TV Leaflet

History of trauma - healed rib fracture

Iatrogenic: due to lead impingement

Pre-op

Post-op

LEAFLETS: PRIMARY “ORGANIC” TR

Carcinoid heart disease
ECHOCARDIOGRAPHIC ASSESSMENT OF THE TV

Annulus diameter

FUNCTIONAL TRICUSPID REGURGITATION

- TA dilatation occurs mostly along the RV free-wall
- Septal portion of the tricuspid annulus relatively fixed

Dreyfus et al. ATS 2005
TRICUSPID REGURGITATION IS LOAD DEPENDENT

Pre/Post Peritoneal Dialysis: Normal Annular Dimension

Annulus diameter may be a better indicator of TV dysfunction than presence/absence of TR

MECHANISMS OF TRICUSPID REGURGITATION

TR is highly dependent on annular dilatation, with significant TR occurring with only 40% dilatation, whereas it was seen at 75% dilatation in vitro MV studies. i.e. the TV leaks earlier than the MV

Spinner EM. Circulation 2011
**IMPORTANCE OF TRICUSPID ANNULUS SIZE IN SECONDARY TR**

- **N = 311** who had MV repair
- TV annuloplasty performed if TA diameter ≥ 70 mm
- Performing tricuspid annuloplasty based on TA dilatation rather than TR degree results in improved surgical outcome

<table>
<thead>
<tr>
<th></th>
<th>MV + TV repair</th>
<th>MV repair only</th>
</tr>
</thead>
<tbody>
<tr>
<td>Event-free survival @ 10 y</td>
<td>90.5%</td>
<td>93%</td>
</tr>
<tr>
<td>Grade III-IV TR</td>
<td>&lt;1%</td>
<td>34%</td>
</tr>
<tr>
<td>Class III-IV CHF</td>
<td>0%</td>
<td>14%</td>
</tr>
</tbody>
</table>


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**FUNCTIONAL TRICUSPID REGURGITATION**

A diastolic diameter >40 mm (or >21 mm/m²) indicates significant annular dilation
THE ACC/AHA 2014 VALVULAR HEART DISEASE GUIDELINES

TA dilated if >40 mm in apical 4-chamber view

Progressive functional TR (stage B)
Mild
Moderate
At time of indicated valve surgery

Asymptomatic severe TR (stage C)
Functional
Primary
Arrest of left-sided valve surgery

Symptomatic severe TR (stage D)
Resuspension
Functional
Primary
Progressive RV dysfunction

ECHOCARDIOGRAPHIC ASSESSMENT OF THE TV

RA size
RV size and function

ED
ES
LV
FAC 40%

ES
cmv
m/s

ESC/EACTS Guidelines for management of VHD EHJ 2012
ACC/AHA Guidelines for management of VHD JACC 2014
VOLUME OVERLOAD AND PRESSURE OVERLOAD

"D-shaped" septum


ECHOCARDIOGRAPHIC ASSESSMENT OF THE TV

1. Tricuspid valve dysfunction
   - Regurgitation: Jet area, VC, PISA, Jet density, Hepatic veins
   - Stenosis
2. Systolic PA pressure + IVC
3. Associated left-sided heart disease
ASE GUIDELINES AND STANDARDS

Recommendations for Noninvasive Evaluation of Native Valvular Regurgitation
A Report from the American Society of Echocardiography
Developed in Collaboration with the Society for Cardiovascular Magnetic Resonance

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Rebecca T. Hahn, MD, FASE, Yachi Han, MD, MMSc, Judy Hung, MD, FASE, Roberto M. Lang, MD, FASE,
Stephen H. Little, MD, FASE, Dipan J. Shah, MD, MMSc, Stanton Sherman, MD, FASE,
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Minnesota; San Francisco, California; New York, New York; Philadelphia, Pennsylvania; Boston, Massachusetts;
Toronto, Ontario, Canada; and Washington, DC

JASE 2017

ECHOCARDIOGRAPHIC ASSESSMENT OF THE TV

Color Doppler Imaging
1. Jet area
2. Vena contracta
3. Proximal flow convergence
**TR QUANTIFICATION: JET AREA**

<table>
<thead>
<tr>
<th>Color flow jet area (cm²)</th>
<th>Mild TR</th>
<th>Moderate TR</th>
<th>Severe TR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small, narrow</td>
<td>Central, moderate</td>
<td>Large jet, wall impinging, &gt;10 cm²</td>
<td></td>
</tr>
</tbody>
</table>

**Pitfalls:**
- Dependent on driving pressure, jet direction
- May over-estimate central jets and underestimate eccentric jets

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**TR QUANTIFICATION: VENA CONTRACTA**

**Pro**
- Independent of flow rate and driving pressure for a fixed orifice
- Less dependent on technical factors
- Good for severe TR

**Con**
- Problematic in multiple jets
- Convergence zone needs to be seen

< 0.3 cm = mild; ≥0.7 cm = severe

Nyquist 50-60 cm/s
TR QUANTIFICATION: 3D VENA CONTRACTA AREA

- 3D CD dataset
- Align orthogonal planes along jet
- Use mid-systole
- Limited spatial resolution may lead to overestimation

VCA > 0.4 cm² is a reasonable cutoff value for severe TR based on currently available data

Chen TE et al. JASE 2013
Zoghbi W et al. JASE 2017
TR QUANTIFICATION: PROXIMAL ISOVELOCITY FLOW CONVERGENCE

Peak TR velocity 386 cm/s
Aliasing velocity 32 cm/s
PISA radius 0.9 cm
Jet VTI 109 cm

EROA =
\[(2 \times 3.14 \times 0.9^2 \times 32) / 386\] = 0.4 cm²

RVol = 0.4 x 109 = 44 mL

**Pro**
- Rapid assessment
- Quantitative

**Con**
- Not commonly used
- Multiple jets are problematic
- Non-hemispheric shape can lead to underestimation
- Less experience with TR than MR
- Validated in only a few studies

Zoghbi W. et al. JASE 2017
The shape of the regurgitant orifice is not a

TR QUANTIFICATION: REGURGITANT VOLUME

- Similar ERO areas induce less RVol in TR than in MR because of the decreased driving force in TR
- The consequences with regards to venous flow reversal was the same

Tribouilloy CM, J. Am. Soc. Echocardiogr. 2002
# TR Quantification: Regurgitant Volume

## Table 1: Diagnostic value for severe regurgitation of various thresholds of ERO area and RVol

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ERO area</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥20 mm²</td>
<td>96</td>
<td>87</td>
<td>25</td>
<td>96</td>
<td>97</td>
<td>86</td>
<td>65</td>
<td>96</td>
</tr>
<tr>
<td>≥30 mm²</td>
<td>92</td>
<td>64</td>
<td>56</td>
<td>90</td>
<td>97</td>
<td>79</td>
<td>65</td>
<td>98</td>
</tr>
<tr>
<td>≥50 mm²</td>
<td>86</td>
<td>76</td>
<td>56</td>
<td>90</td>
<td>94</td>
<td>89</td>
<td>82</td>
<td>96</td>
</tr>
<tr>
<td>RVol</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥20 mL</td>
<td>68</td>
<td>67</td>
<td>49</td>
<td>94</td>
<td>81</td>
<td>100</td>
<td>100</td>
<td>75</td>
</tr>
<tr>
<td>≥45 mL</td>
<td>63</td>
<td>47</td>
<td>38</td>
<td>94</td>
<td>81</td>
<td>100</td>
<td>100</td>
<td>75</td>
</tr>
<tr>
<td>≥60 mL</td>
<td>38</td>
<td>67</td>
<td>49</td>
<td>94</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

ERO, Effective regurgitant orifice; RVO, regurgitant volume; PPV, positive predictive value; NPV, negative predictive value.

*Thresholds are recommended with the highest sum of sensitivity and specificity.

## Zoghbi et al. Journal of the American Society of Echocardiography Volume 15 Number 9

## Structural

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>TV morphology</td>
<td>Normal or mildly abnormal leaflets</td>
<td>Moderately abnormal leaflets</td>
<td>Severe valve lesions (e.g., flail leaflet, severe retraction, large perforation)</td>
</tr>
<tr>
<td>RV and RA size</td>
<td>Usually normal</td>
<td>Normal or mild dilatation</td>
<td>Usually dilated</td>
</tr>
<tr>
<td>Inferior vena cava diameter</td>
<td>Normal &lt; 2 cm</td>
<td>Normal or mildly dilated 2.1–2.5 cm</td>
<td>Dilated ≥2.5 cm</td>
</tr>
<tr>
<td>Qualitative Doppler</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Color flow jet area</td>
<td>Small, narrow, central</td>
<td>Moderate central</td>
<td>Large central jet or eccentric wall-ipring jet of variable size</td>
</tr>
<tr>
<td>Flow convergence zone</td>
<td>Not visible, transient or small</td>
<td>Intermediate in size and duration</td>
<td>Large throughout systolic</td>
</tr>
<tr>
<td>CW Doppler</td>
<td>Feint/partial/prolateral</td>
<td>Dento, prolateral or triangular</td>
<td>Dento, often triangular</td>
</tr>
</tbody>
</table>

## Semiquantitative

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color flow jet area (cm²)</td>
<td>Not defined</td>
<td>Not defined</td>
<td>&gt;10</td>
</tr>
<tr>
<td>VCW (cm²)</td>
<td>0.3–0.69</td>
<td>≥0.7</td>
<td></td>
</tr>
<tr>
<td>PISA radius (cm²)</td>
<td>≤0.5</td>
<td>0.6–0.9</td>
<td>≥6.0</td>
</tr>
<tr>
<td>Hepatic vein flow</td>
<td>Systolic dominance</td>
<td>Systolic blunting</td>
<td>Systolic flow reversal</td>
</tr>
<tr>
<td>Tricuspid inflow</td>
<td>A-wave dominant</td>
<td>Variable</td>
<td>E-wave ≥1.0 m/sec</td>
</tr>
</tbody>
</table>

## Quantitative

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>EROA (cm²)</td>
<td>&lt;0.20</td>
<td>0.20–0.39²</td>
<td>≥0.40</td>
</tr>
<tr>
<td>RVO (GDP PISA) (mL)</td>
<td>&lt;30</td>
<td>30–45</td>
<td>≥45</td>
</tr>
</tbody>
</table>

Zoghbi W. et. al. JASE 2017
**TR QUANTIFICATION: CONTINUOUS WAVE DOPPLER**

**Pro**
- Simple
- Density is proportional to the number of RBCs reflecting the signal

**Con**
- Overlap between moderate and severe
- Pattern seen in severe TR may be present in patients with severely elevated RA pressure

**Severe TR**
- Faint
- Partial
- Parabolic

**Mild TR**
- Dense
- Triangular jet
- Early peaking (↑RA pressure)

**TR QUANTIFICATION: HEPATIC VEIN PULSE WAVE DOPPLER**

**Pro**
- Simple
- Can be obtained with both TTE and TEE

**Con**
- Depends on compliance of the RA and RV
- Affected by respiration, preload, pacemaker rhythm, CHB and atrial fibrillation/flutter

**Severe TR**
- Align insonation beam with the hepatic vein

**Reversal of flow in the hepatic vein with severe TR**

Feigenbaum's Echocardiography and Zoghbi W. et al. JASE 2017
Systolic PA pressure + IVC

\[ P_1 - P_2 = 4v^2 \]
\[ P_1 = 4v^2 \times P_2 \]
\[ RVSP = 4v^2 + P_{RA} \]

\( V \) = Peak velocity of TR jet
\( P_{RA} \) = Jugular venous pulse (estimated using IVC collapsibility)
ECHOCARDIOGRAPHIC ASSESSMENT OF THE TV

\[ RVSP = 4v^2 + P_{RA} \]

<table>
<thead>
<tr>
<th>IVC Diameter</th>
<th>Collapse</th>
<th>RAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 2.1 cm</td>
<td>&gt;50%</td>
<td>3 mmHg</td>
</tr>
<tr>
<td>&gt;2.1 cm</td>
<td>&lt;50%</td>
<td>15 mmHg</td>
</tr>
<tr>
<td>IVC does not conform</td>
<td></td>
<td>8 mmHg</td>
</tr>
</tbody>
</table>

Feigenbaum’s Echocardiography 7th Edition

CMR IMAGING OF THE TRICUSPID VALVE: WHEN AND HOW?

<table>
<thead>
<tr>
<th>Approach</th>
<th>TR</th>
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</thead>
<tbody>
<tr>
<td>Preferred method for quantitation(^a)</td>
<td>(RV SV) – (PA total forward SV)</td>
</tr>
<tr>
<td>Secondary methods for quantitation(^b)</td>
<td>(RV SV) – (AO total forward SV)</td>
</tr>
<tr>
<td></td>
<td>(RV SV) – (LV SV)</td>
</tr>
<tr>
<td>Corroborating signs of significant regurgitation</td>
<td>RV dilation, right atrium dilation</td>
</tr>
</tbody>
</table>

The strength of CMR is its ability to quantitatively assess RVol, fraction, and ventricular and atrial remodeling.

Zoghbi W. et. al. JASE 2017
GRADING OF TRICUSPID REGURGITATION SEVERITY

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Structural</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TV morphology</td>
<td>Normal or mildly abnormal leaves</td>
<td>Moderately abnormal leaves</td>
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</tr>
<tr>
<td>CW Doppler</td>
<td>Flat/partial/parabolic</td>
<td>Dense, parabolic or triangular</td>
<td>Dense, often triangular</td>
</tr>
<tr>
<td><strong>Semi- Quantitative</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Color flow jet area (cm²)</td>
<td>Not defined</td>
<td>Not defined</td>
<td>&gt;10</td>
</tr>
<tr>
<td>VTI (cm)</td>
<td>0.3</td>
<td>0.3-0.6</td>
<td>&gt;0.7</td>
</tr>
<tr>
<td>PISA radius (cm²)</td>
<td>0.5</td>
<td>0.6-0.9</td>
<td>&gt;0.9</td>
</tr>
<tr>
<td>Hepatic vein flow</td>
<td>Systolic dominance</td>
<td>Systolic blunting</td>
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</tr>
<tr>
<td><strong>Quantitative</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ERDA (cm²)</td>
<td>&lt;0.20</td>
<td>0.20-0.39</td>
<td>&lt;0.40</td>
</tr>
<tr>
<td>RVH (GPI/PIA) (ml)</td>
<td>&lt;30</td>
<td>30-44</td>
<td>&gt;45</td>
</tr>
</tbody>
</table>

Zoghbi W. et al. JASE 2017

Chronic Tricuspid Regurgitation by Doppler Echocardiography

Specific Criteria for Mild TR
- Thin, small central color jet
- VTI <0.3 cm
- PISA radius <0.4 cm at Nyquist 40-40 cm/s
- Incomplete or fast CW jet
- Systolic dominant hepatic ven flow
- Tricuspid A-wave dominant inflow
- Normal RVRA

Specific Criteria for Severe TR
- Dilated annulus with no valve coaptation or flail leaflet
- Large central jet >50% of RA
- VTI >0.7 cm
- PISA radius >0.9 cm at Nyquist 30-40 cm/s
- Dense, triangular CW jet or sine wave pattern.
- Systolic reversal of hepatic ven flow
- Dilated RV with preserved function

Perform VTI measurement, and may perform quantitative PISA method, whenever possible

Indeterminate TR
Consider further testing:
TEE or CMR for quantification

* Clinical experience in quantitation of TR is much less than that with mitral and aortic regurgitation

JASE 2017
NEW DIRECTIONS: EVALUATION OF FTR
A MORE COMPREHENSIVE APPROACH

TABLE 1  Stages of Functional Tricuspid Regurgitation

<table>
<thead>
<tr>
<th></th>
<th>Stage 1</th>
<th>Stage 2</th>
<th>Stage 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>TR severity</td>
<td>None or mild</td>
<td>Mild or moderate</td>
<td>Severe</td>
</tr>
<tr>
<td>Annular diameter, mm</td>
<td>&lt;40</td>
<td>&gt;40</td>
<td>&gt;40</td>
</tr>
<tr>
<td>Leaflet coaptation mode</td>
<td>Normal*</td>
<td>Edge-to-edge*</td>
<td>Absent†</td>
</tr>
</tbody>
</table>

*No leaflet tethering (<8 mm). †Leaflet tethering may be present (≥8 mm). †If leaflet tethering is present.

TR = tricuspid regurgitation.

Dreyfus, GD. et. al. J Am Coll Cardiol 2015

MECHANISMS OF TRICUSPID STENOSIS

- Rheumatic
- Infiltration: Carcinoid
- Rare: Congenital, valvular or pacemaker IE, mechanical obstruction, Lupus valvulitis

Consequence of TS

- Tricuspid stenosis
- Elevation of RA pressure
- Right-sided heart failure
TRICUSPID STENOSIS

• Hallmark of stenotic valve: ↑ transvalvular CWD velocity
• Peak inflow (normal TV) < 0.7 m/s
• TV inflow ↑ with inspiration
• In TS peak velocities >1.0 m/s up to 2 m/s with inspiration

Mean gradient = 4v²

TVI=60 cm; mean grad = 9 mmHg
P1/21 = 173 ms

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TRICUSPID STENOSIS

Findings indicative of hemodynamically significant TS*

Specific findings
- Mean pressure gradient ≥ 5 mmHg
- Inflow time-velocity integral > 60 cm
- $T_{1/2}$ ≥ 190 ms
- Valve area by continuity equation$^a$ ≤ 1 cm$^2$

Supportive findings
- Enlarged right atrium > moderate
- Dilated inferior vena cava

$^a$Stroke volume derived from left or right ventricular outflow. In the presence of more than mild TR, the derived valve area will be underestimated. Nevertheless, a value ≤ 1 cm$^2$ implies a significant haemodynamic burden imposed by the combined lesion.

*with or without regurgitation

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THE NORMAL PULMONIC VALVE

The PV is a semilunar valve with 3 cusps

Aims of imaging
1. Inspection of valve and leaflets
2. Quantify stenosis/regurgitation
3. Assess the RVOT
4. Pulmonary annulus
5. Main PA
6. Proximal PA branches
7. RV size and function
MECHANISMS OF PULMONARY REGURGITATION

Primary (or “Organic”)

Secondary (or “Functional”)

Elevated PA pressure (volume of regurgitation usually small)

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PRIMARY “ORGANIC” PR

Intrinsic abnormality of PV leaflets and/or support apparatus

Acquired
- Rare (<1% of patients)
- Associated with rheumatic heart disease, endocarditis, carcinoid valve disease, pergolide-induced disease
- Rare: blunt chest trauma

Congenital heart disease

Status post balloon valvuloplasty for PS
**PR QUANTIFICATION: VENA CONTRACTA AND JET WIDTH: PV ANNULUS RATIO**

**Pro**
- VC is a surrogate for ERO, is independent of flow rate and driving pressure for a fixed orifice
- Less dependent on technical factors

![Vena contracta width](vena_contracta.png)  ![Jet:Annulus ratio](jet_annulus_ratio.png)

1. Vena contracta width
2. Jet : PV annulus ratio $>$0.5 correlates with severe PR on CMR
3. Jet length ($<$10 mm = mild PR)
4. Jet area
   Use: Parasternal SAX or subcostal views, zoomed in diastole

**Con**
- Problematic in multiple jets
- No cut-offs

**PR QUANTIFICATION: PULSE WAVE DOPPLER**

**Pro**
- Simple supportive sign of severe PR

**Con**
- Depends on compliance of the PA
- Brief velocity reversal is normal

Align ultrasound beam with the flow in the RPA and LPA. Obtain PWD from both branch PAs

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**PR QUANTIFICATION: CONTINUOUS WAVE DOPPLER**

**Pro**
- Simple
- Density is proportional to the number of red blood cell reflecting the signal
- Faint/incomplete jet is compatible with mild PR
- Values of PHT <100 msec are consistent with severe PR

**Con**
- Poor alignment of Doppler may occur in eccentric jets
- Affected by RV and PA pressure

\[ \text{Pressure half-time} < 100 \text{ msec} \]

Mild PR

Severe PR

**RF = Regurgitant fraction measured on CMR. RV end-diastolic volumes also measured on CMR**

PR QUANTIFICATION: REGURGITANT VOLUME AND FRACTION

**Pro**
- Simple
- Density is proportional to the number of red blood cell reflecting the signal
- Faint/incomplete jet is compatible with mild PR
- Values of PHT <100 msec are consistent with severe PR

**Con**
- Poor alignment of Doppler may occur in eccentric jets
- Affected by RV and PA pressure

RVol = SV\text{RVOT} - SV\text{LVOT}

SV\text{LVOT} = CSA\text{LVOT} \times \text{VTI}_{\text{LVOT}}
SV\text{RVOT} = CSA\text{RVOT} \times \text{VTI}_{\text{RVOT}}

CSA\text{LVOT} = 0.785 \times d^2_{\text{LVOT}} \times \text{VTI}_{\text{LVOT}}
CSA\text{RVOT} = 0.785 \times d^2_{\text{RVOT}} \times \text{VTI}_{\text{RVOT}}

RVoL = (0.785 \times 3^2 \times 18.5) - 0.785 \times 2.1^2 \times 20.59
RVoL = 131 - 71
RVoL = 60 mL

RF = RVoL/SV\text{RVOT}
RF = 60/131 = 46%
PR QUANTIFICATION: REGURGITANT VOLUME AND FRACTION

Pro
- Valid with multiple jets
- Quantitative

Con
- RVOT probably most difficult site to measure SV
- In case of AR would need to use mitral annulus site
- Scant experience

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PR QUANTIFICATION: REGURGITANT VOLUME AND FRACTION BY CMR

Forward SV by phase contrast was 129 mL, and reverse (regurgitant) volume was 78 mL, yielding an RF of 60%

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PR QUANTIFICATION: SUMMARY

Table 16: Echocardiographic and Doppler parameters useful in grading PR severity

<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 and may not be visible</td>
</tr>
<tr>
<td>RV size, color Doppler²</td>
<td>Normal</td>
<td>Normal or dilated</td>
<td>Dilated¹</td>
</tr>
<tr>
<td>Jet size, color Doppler</td>
<td>Thin (usually &lt;10 mm in length) with a narrow origin</td>
<td>Intermediate</td>
<td>Broad origin; variable depth of penetration</td>
</tr>
<tr>
<td>Ratio of PR jet width/pulmonary annulus</td>
<td>&gt;0.75</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jet density and contour</td>
<td>Soft</td>
<td>Dense</td>
<td>Dense; early termination of diastolic flow</td>
</tr>
<tr>
<td>Deceleration time of the PR spectral Doppler signal</td>
<td>Short, &lt;260 msec</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pressure half-time of PR jet</td>
<td>&lt;0.77</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic flow reversal in the main or branch PA (P)</td>
<td>Prominent</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonic systolic flow (VTI)</td>
<td>Slightly increased</td>
<td>Intermediate</td>
<td>Greatly increased</td>
</tr>
<tr>
<td>Compared to systemic flow (LVOT VTI) by PW³</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RF⁴</td>
<td>&lt;20%</td>
<td>20%-40%</td>
<td>&gt;40%</td>
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MECHANISMS OF PULMONARY STENOSIS

Congenital
The valve can be tri-leaflet, bicuspid, unicuspid, dysplastic
Associated with TOF, DORV, complete AV canal defect
Peripheral PS may co-exist with PS (Noonanss, Williams)

Acquired
Rheumatic, Carcinoid (combined stenosis and regurgitation)
Functional pulmonary stenosis (external compression of RVOT)
Proximal (RVOT) stenosis
Supra-valvular stenosis

Most common
### PS QUANTIFICATION

#### Feigenbaum’s Echocardiography and Hung J. et. al. JASE 2009

**Bernoulli equation** \( P = 4v^2 \)

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<td>Peak velocity (m/s)</td>
<td>&lt;3</td>
<td>3–4</td>
</tr>
<tr>
<td>Peak gradient (mmHg)</td>
<td>&lt;36</td>
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#### Cuypers JAAE, et al Heart 2013

**Bernoulli equation** \( P = 4v^2 \)

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*Feigenbaum’s Echocardiography and Hung J. et. al. JASE 2009*

*Cuypers JAAE, et al Heart 2013*
PS QUANTIFICATION

sPAP = RVSP – PV
pressure gradient

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Bemoulli equation \( P = 4v^2 \)

Cuypers JAAE, et al Heart 2013
Feigenbaum's Echocardiography and Hung J. et. al JASE 2009