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AU:3, page 4	Pls confirm affiliation for the FM (Dr. Saric confirmed in ch 106): Kelly Axsom, MD Fellow, Cardiovascular Diseases Leon H. Charney Division of Cardiology New York University Langone Medical Cneter New York, New York	☐

s0065 ## Indirect Evaluation

p0075 Evaluation of the size and function of the right ventricle in the absence of pulmonary hypertension provides an indirect indicator of PR significance. In the presence of right ventricular volume overload from severe PR, right ventricular dilation, hypokinesis, and diastolic septal flattening may be seen.[4] In addition, there are several other supportive findings that can be used to substantiate the presence of severe PR: premature closure of the tricuspid valve, holodiastolic flow reversal in the pulmonary artery, premature opening of the pulmonic valve, PR jet with laminar retrograde flow, and low peak velocity of the PR jet.[12] A dedicated investigation carefully looking for these findings should be included in the evaluation of PR, especially if other echocardiographic evidence of severity is inconclusive.

s0070 ## SURGICAL TREATMENT OF SEVERE PULMONIC REGURGITATION

p0080 In patients with severe pulmonic regurgitation and NYHA Class II or III symptoms, pulmonic valve replacement is indicated.[6] In asymptomatic patients, surgical indications based on right ventricular size and function or regurgitant fraction have yet to be determined. Pulmonic valve replacement is usually performed with a homograft or xenograft and has been shown to minimize ventricular tachyarrhythmias and improve right heart hemodynamics.[6]

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sc0015 # 125 Pulmonic Regurgitation: Semiquantification

Au3 *Kelly Axsom, MD, Muhamed Saric, MD, PhD*

s0075 ## INTRODUCTION

p0085 Trivial or mild degrees of pulmonic regurgitation (PR) are common in the structurally normal heart, and the presence of pathologic PR is rare in adults.[1] Some degree of PR is present in between 5% and 78% of echocardiograms of structurally normal hearts.[2] Echocardiographic evaluation of the degree of PR is much less well defined than for the other heart valves and is either descriptive or semiquantitative. This is primarily due to the low prevalence of clinically severe PR. In one series, only 1.6% of all severe valvular regurgitation was due to PR.[3]

s0080 ## PULMONIC REGURGITATION EVALUATION OVERVIEW

p0090 As with other valve lesions, assessment of PR includes three basic elements: (1) establishing the mechanism of PR; (2) determining the severity of PR; and (3) assessing the impact of PR on cardiac chambers, primarily the right ventricle (RV) and the pulmonary artery (PA). Severity of PR is best assessed by Doppler echocardiography, whereas the mechanism of PR and its impact on cardiac chambers is evaluated by two-dimensional (2D) and three-dimensional (3D) echocardiography.

p0095 The American College of Cardiology and the American Heart Association guidelines on valvular heart disease address the issue of PR only briefly.[4] The American Society of Echocardiography recommendations for evaluation of severity of PR include evaluation of anatomical structure of the pulmonary valve, right ventricular size, color Doppler pulmonic regurgitant jet size, and spectral Doppler jet density, duration, and deceleration rate, as well as a comparison between pulmonic and systemic flow.[5] Other parameters of PR severity include timing of tricuspid valve closure and pulmonic valve opening, holodiastolic flow reversal in the pulmonary artery, low peak velocity of PR jet, and laminar retrograde flow.[3] Echocardiographic markers of PR severity are summarized in Table 125-1 and shown in Figures 125-1 and 125-2.

MECHANISM OF PULMONIC REGURGITATION s0085

A normal pulmonary valve is a semilunar, trileaflet valve located anterior, superior, and slightly to the left of the aortic valve. The valve leaflets are very thin and highly pliable, making the pulmonic valve the most challenging valve to image. Both congenital and acquired etiologies of PR have been discussed in detail in the preceding chapter, including the most common causes of severe PR, such as carcinoid disease and surgically repaired pulmonic stenosis of tetralogy of Fallot.[6] The primary mechanisms of PR include distorted or absent leaflets and/or annular dilatation. p0100

When assessing PR, it is best to evaluate the right ventricular outflow tract, pulmonic valve (PV), and PA together. Imaging of the pulmonic valve is often better on transthoracic echocardiography (TTE) than transesophageal echocardiography (TEE) given the anterior location of the valve. 2D TTE and TEE imaging typically allows for visualization of only long-axis views of the pulmonic valve. On TTE, PR is best visualized on the parasternal short axis at the level of the aortic valve and from the subxiphoid view. p0105

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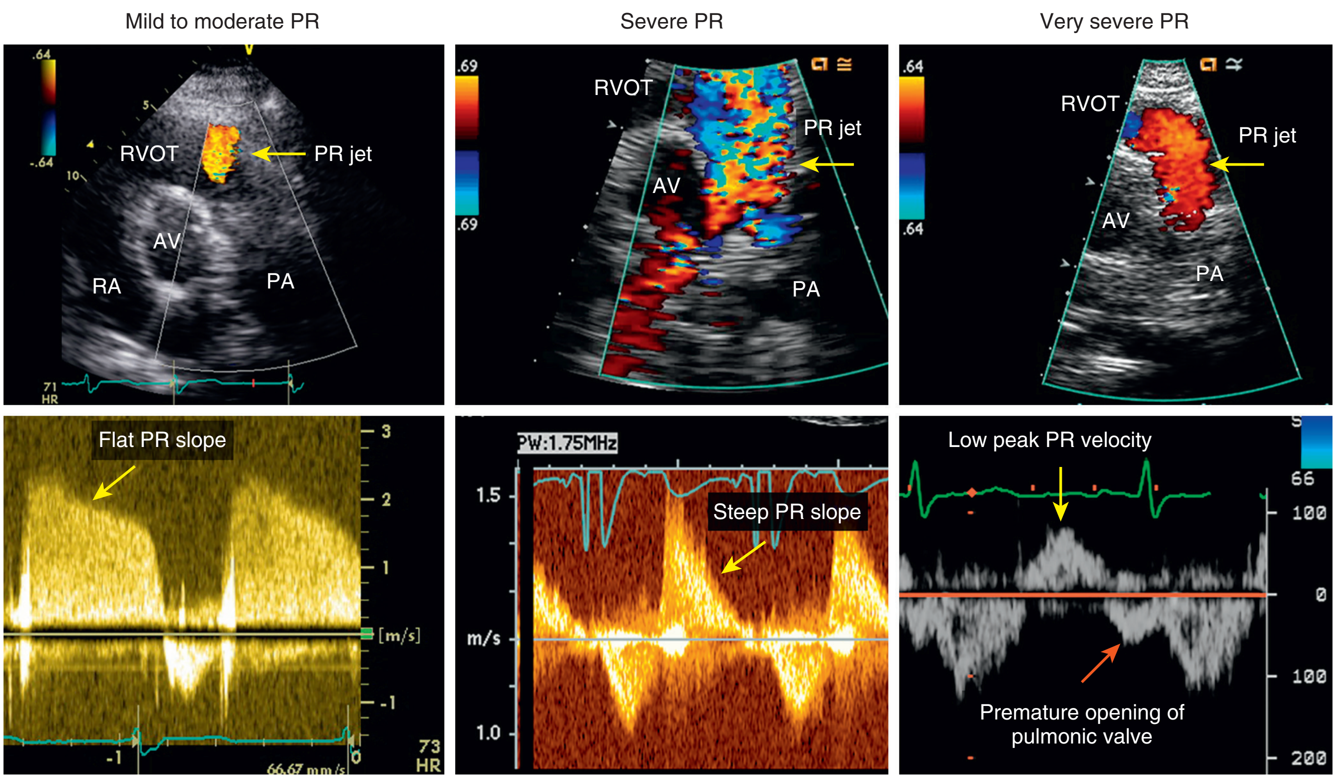
t0015 **TABLE 125-1** Markers of Severe Pulmonic Regurgitation

	Parameter	Moderate to Severe PR	Severe PR
Color Doppler	Jet length and area	Increased	May be short
	Jet turbulence	Turbulent jet	Laminar jet
	Vena contracta	Wide	Very wide
CW Doppler	Jet density	Dense	Very dense
	Deceleration slope	Short	Very short
	Premature cessation of retrograde flow	May be absent	Typically present
	To-and-fro flow	Absent	Present
	Peak PR velocity	Normal	Low
	Premature opening of pulmonic valve	Absent	Present
	Premature closure of tricuspid valve	Absent	Present
PW Doppler	Regurgitant volume	<60 mL/beat	≥60 mL/beat
	Regurgitant fraction	<50%	≥50%
	Holodiastolic flow reversal in PA	Absent	Present
Additional Signs	RV and PA size	Progressively increases with severity and chronicity of PR	

CW, Continuous-wave; *PA,* pulmonary artery; *PR,* pulmonic regurgitation; *PW,* pulsed-wave; *RV,* right ventricle.



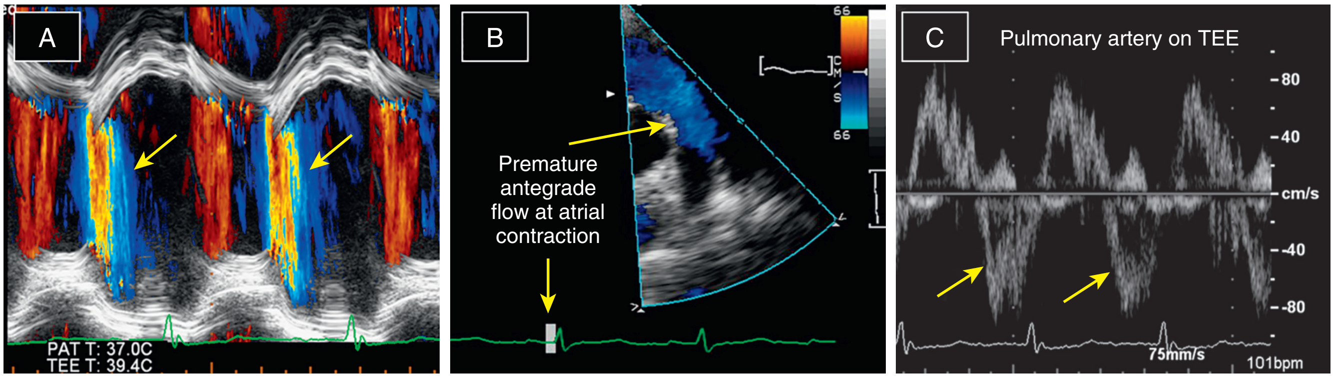
f0020 **Figure 125-1.** Color and spectral Doppler markers of pulmonic regurgitation. Transthoracic short-axis view at the level of the aortic valve demonstrates color Doppler (*top row*) and spectral Doppler *(bottom row)* findings in mild to moderate, severe, and very severe pulmonic regurgitation (PR). **Mild to moderate PR:** On color Doppler, jet is small *(arrow)* and vena contracta at jet origin is narrow. On spectral Doppler, the slope of PR jet is relatively flat *(arrow)*. (See accompanying Video 125-1.) **Severe PR:** On color Doppler, jet is large and turbulent *(arrow)*, whereas on spectral Doppler the PR slope is steep *(arrow)*. (See accompanying Video 125-2.) **Very severe PR:** On color Doppler, jet is laminar *(arrow)* and of short duration. On spectral Doppler there is low peak velocity of PR jet *(yellow arrow)* and there is premature opening of pulmonic valve with abnormal antegrade flow in late diastole. (See accompanying Video 125-3.) *AV,* Aortic valve; *PA,* pulmonary artery; *RA,* right atrium; *RVOT,* right ventricular outflow tract.

On TEE, the pulmonic valve and PA can be imaged at mid-esophageal and transgastric views using approximately a 60-degree acquisition angle. The short axis of the pulmonic valve can be obtained by 3D TTE and TEE imaging.[7]

s0090 SEMIQUANTITATIVE ASSESSMENT OF PULMONIC REGURGITATION SEVERITY

p0110 Color and spectral Doppler echocardiography are the primary means of quantifying the degree of PR. As with other valvular regurgitations, the severity of PR depends on the interplay among the regurgitant orifice size, the regurgitant volume, and the transvalvular pressure gradient (the diastolic pressure gradient between the PA and the RV in the case of PR).

To a certain point, as the regurgitant volume and the severity of PR increase, so do the size and turbulence of the regurgitant jet. However, when the regurgitant orifice becomes very large, a rapid equalization of pressures may lead to a smaller and more laminar regurgitant jet that terminates prematurely (before the end of diastole). It is important to bear in mind that PR is not the only p0115



f0025 **Figure 125-2.** Additional markers of severe pulmonic regurgitation. **A,** Color M mode demonstrates that pulmonic regurgitation (PR) jet terminates abnormally in early diastole *(arrows)*. This is in contrast to less severe forms of PR, where the PR jet lasts throughout diastole. **B,** Color Doppler on transthoracic short-axis view at the level of the aortic valve demonstrates abnormal antegrade flow across the pulmonic valve at the time of atrial contraction indicative of premature pulmonic valve opening resulting from elevated right ventricular pressures in severe PR. **C,** Transesophageal echocardiography (TEE) spectral Doppler tracing demonstrates abnormal holodiastolic flow reversal *(arrows)* in the pulmonary artery indicative of severe PR. Au1

determinant of the rate of pressure equalization; rapid equalization of pressures may also be mediated by a low diastolic PA pressure and/or high RV diastolic pressure. Collectively, all these phenomena form the basis of semiquantitative PR assessment by both color and spectral Doppler imaging.

p0120 Historically, angiography has been the primary means of validating echocardiographic methods for PR. However, angiography has recently been supplanted by cardiac magnetic resonance imaging (MRI).

s0095 Color Doppler

p0125 Color Doppler is the most widely accepted method for assessing PR. Typical PR jet is seen in the right ventricular outflow tract during diastole. PR jets in the normal heart are usually narrow or “spindle-like” and originate centrally from the pulmonary leaflet cooptation site.

s0100 Jet Length

p0130 Original studies suggested that a jet length less than 10 mm implies trivial regurgitation.[1] Theoretically, a more severe PR should be associated with longer jets. However, jet length is not considered a reliable index of PR severity because abrupt cessation of diastolic flow in severe PR may lead to relatively short PR jets. Furthermore, the more severe the PR, the less likely it is that a full jet length can be appreciated on the parasternal TTE window.

s0105 Jet Area

p0135 Planimetry of the jet area indexed for body surface area has been shown to correlate well with angiographic grades of PR severity in patients with tetralogy of Fallot repair.[6] However, there are no well-validated criteria on what jet area defines severe PR. Again it is important to emphasize that with very severe PR, when equalization of PA to RV gradient take place early in diastole, the color Doppler jet area can be small and brief; this seemingly paradoxical diminution of jet area size should not be misinterpreted as less than severe PR.

s0110 Turbulent Versus Laminar Regurgitant Jet

p0140 Lesser degrees of PR are typically characterized by turbulent jets. When PR is very severe and when there is rapid equalization of pressure across the pulmonic valve in diastole, the PR jet becomes more laminar. This seemingly paradoxical finding is an important semiquantitative marker of PR severity.

Vena Contracta s0115

The use of vena contracta width for determining severity of regurgitation is generally reserved for the other valves and has not been validated for PR. Semiquantitatively, a wide vena contracta in the setting of brief and small color Doppler jet area supports the diagnosis of severe PR. p0145

Continuous-Wave Spectral Doppler s0120

Continuous-wave (CW) spectral Doppler of the PR jet should be attempted on all echocardiographic exams. Semiquantitative measures of PR severity include signal density, deceleration slope, and the timing of jet cessation. p0150

Jet Density s0125

Theoretically, a more severe PR should be associated with more dense spectral tracings. However, there is no accepted method for fully quantifying PR using spectral density. p0155

Deceleration Slope s0130

Rapid deceleration slope of the diastolic Doppler signal is only a rough estimate of PR severity because there are no well-validated deceleration cutoff values for various degrees of PR. Although severe PR leads to a rapid deceleration slope of the PR jet, not all instances of rapid deceleration slope are indicative of severe PR. This is because the deceleration slope depends on both the size of the regurgitant orifice and the diastolic PA-to-RA pressure gradient. Thus in patients with low PA diastolic pressures and/or elevated RV diastolic pressures, a rapid slope may be seen in the absence of severe PR. p0160

Premature Cessation of Retrograde Flow s0135

Severe PR is characterized by premature cessation of retrograde spectral Doppler flow; instead of end diastole, the flow ends in mid to late diastole. However, not all instances of premature PR jet cessation are indicative of severe PR, as the phenomenon may also be seen in patients with low PA diastolic pressures and/or elevated RV diastolic pressures in the absence of severe PR. p0165

To-and-Fro Flow s0140

In patients with very severe PR, there may be little net antegrade flow across the PV; the bulk of flow simply recirculates across the PV (retrograde in diastole, then antegrade in systole). This gives p0170 rise to a characteristic sinusoid "to-and-fro" velocity pattern on CW Doppler in patients with severe PR.

s0145 Low Peak Pulmonic Regurgitation Velocity

p0175 Another marker of severe PR is a low peak PR velocity. It is typically seen with the to-and-fro flow pattern just described.

s0150 Premature Tricuspid and Pulmonic Valve Events

p0180 Premature closure of the tricuspid valve and premature opening of the pulmonic valve are markers of severe PR seen on Doppler echocardiography and reflect elevated right ventricular pressures.

s0155 Pulsed-Wave (PW) Spectral Doppler

p0185 When the PR jet is not aliased, spectral PW Doppler recordings can be used to assess the severity of PR in the same manner as described for CW.

s0160 Regurgitant Fraction by Pulmonic Valve Pulsed-Wave

p0190 Spectral PW Doppler recordings can be used to roughly estimate the regurgitant fraction of PR by tracing retrograde and antegrade flow velocity profiles individually to obtain respective velocity time integrals (VTIs). Assuming a constant PV diameter in systole and diastole, a ratio of retrograde VTI (a measure of regurgitant volume) to antegrade VTI (a measure of total stroke volume) represents the regurgitant fraction.[8] Theoretically, a ratio of 0.5 or more would indicate severe PR (i.e., a regurgitant fraction of $\geq 50\%$). However, there are no empirically validated cutoff values for grading PR severity using this method. Furthermore, this method cannot be used in patients with concomitant pulmonic stenosis because of poststenotic turbulence.

s0165 Regurgitant Volume and Fraction by Comparing Pulmonic to Systemic Flow

p0195 Systolic antegrade flow across the right ventricular outflow tract (RVOT) in a patient with PR represents the total stroke volume (TSV). If the patient has no significant aortic regurgitation and no shunt, then systolic antegrade flow across the left ventricular outflow tract (LVOT) represents the net stroke volume (NSV). The difference between TSV and NSV represents the regurgitant volume.

p0200 RVOT and LVOT stroke volumes (TSV and NSV, respectively) can be calculated using the standard echocardiographic method of measuring the outflow tract diameter (d) in systole, calculating the cross-sectional outflow tract area (A) assuming a circular shape $[A = (0.5d)^2 \times \pi]$ and then multiplying the area by the systolic VTI. Thereafter, regurgitant volume and fraction can be calculated as:

$$\text{Regurgitant volume} = \text{TSV} - \text{NSV}$$
$$\text{Regurgitant fraction} = \text{Regurgitant volume}/\text{TSV}$$

p0205 Although these calculations are feasible, the cutoff values have not been validated for PR. The major limitation to this method is the frequent inability to measure the RVOT diameter accurately. It has been shown that regurgitant fraction calculation by echocardiography only moderately correlates with measurements obtained with cardiac MRI.[9]

s0170 Holodiastolic Flow Reversal in Pulmonary Artery

p0210 Normally, the PW spectral Doppler profile in the pulmonary artery contains a large antegrade component in systole and a very small retrograde component confined to early diastole. As the severity of PR increases, so does the duration of retrograde flow in the pulmonary artery. In severe PR there is typically a holodiastolic flow reversal in the pulmonary artery spectral Doppler tracings. This is analogous to holodiastolic flow reversal in the thoracic and abdominal aorta in the setting of severe aortic regurgitation.

M-Mode Echocardiography s0175

Many aspects of PR assessment by M-mode echocardiography have only historic value. However, with its excellent temporal resolution, M mode allows for very precise timing of cardiac events. For instance, one can easily demonstrate by color M mode the short, early diastolic nature of the jet in severe PR. p0215

Moreover, recently a PR index by M-mode echocardiography (PRIME) to estimate the pulmonary regurgitation fraction (PRF) using a nonlinear regression was developed. M-mode tracings of the right pulmonary artery from the suprasternal notch are used to measure the maximal systolic and minimum diastolic dimensions. PRIME values of 1.21 or greater identified patients with a PRF of at least 25%. When compared with PRF calculated by cardiac MRI, this measurement was accurate. It remains unknown if this method can be replicated by other investigators.[10] p0220

IMPACT OF PULMONIC REGURGITATION ON CARDIAC CHAMBERS s0180

Chronic PR is associated with PA and RV dilatation. PA dilatation may both lead to and be the result of severe PR. RV dilatation and interventricular septal flattening in diastole (a marker of RV volume overload) are nonspecific signs of severe chronic PR. Conversely, a normal RV size excludes severe chronic PR. p0225

Please access ExpertConsult to view the corresponding videos for this chapter. p0230

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