Uncommon Doppler Echocardiographic Findings of Severe Pulmonic Insufficiency

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Background: Two-dimensional and Doppler echocardiography are standard methods to assess the severity of pulmonic insufficiency (PI). However, methods to define severity of PI – including the current published guidelines – remain qualitative and unvalidated.

Methods: We reviewed all the electronic reports of echocardiographic studies performed at our institution since the publication of the 2003 American Society of Echocardiography guidelines on native valvular regurgitation. There were 8,279 instances of severe valvular insufficiency among 100,167 echocardiographic studies (approximately 90% transthoracic and 10% transesophageal). We also searched for uncommon findings of severe PI.

Results: Of all forms of severe valvular insufficiency, PI was least common. There were 135 instances of severe PI as defined by the existing guidelines; they accounted for only 1.6% of all instances of severe valvular insufficiency. Premature closure of the tricuspid valve was seen in 6.6%, holodiastolic flow reversal in 3.7%, premature opening of the pulmonic valve in 1.5%, PI with laminar retrograde flow in 1.5%, very low peak velocity of the PI jet in 1.5% of patients with severe PI.

Conclusions: The published criteria do not include in detail the subtle signs of severe PI such as (1) holodiastolic flow reversal in the pulmonary artery, (2) PI with laminar retrograde flow, (3) premature opening of the pulmonic valve, (4) very low peak velocity of the PI jet, and (5) premature closure of the tricuspid valve. These signs should be considered in the grading of PI severity in addition to the existing guidelines criteria. (J Am Soc Echocardiogr 2010;23:1071-5.)

Keywords: Pulmonic insufficiency, Pulmonary artery, Doppler echocardiography, Transesophageal echocardiography

Existing guidelines for grading the severity of pulmonic insufficiency (PI) rely on anatomic assessment of the pulmonic valve and right ventricle, color Doppler to determine regurgitant jet size, continuous-wave Doppler to determine jet density and deceleration rate, and pulsed-wave Doppler to compare systolic pulmonic flow with systemic flow.[1] Several Doppler findings of severe PI are not well described in the current guidelines, and echocardiographers may not be familiar with these subtle findings. The purpose of this report is to describe uncommon, though clinically relevant, Doppler findings of severe PI that may help further define the severity of PI in addition to the existing guidelines. These echocardiographic findings also illustrate several hemodynamic principles related to severe PI.

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METHODS

We reviewed all the electronic reports of echocardiographic studies performed at our institution since the publication of the guidelines in 2003.[1] A total of 100,167 echocardiographic studies, of which approximately 10% were transesophageal echocardiographic studies, were performed over the subsequent 6-year period. There were 8,279 instances of severe valvular insufficiency among these studies. Individual patients may have had severe insufficiency of more than one valve. These instances were identified by a review of echocardiographic reports. Of all forms of severe valvular insufficiency, PI was least common. There were 135 instances of severe PI as defined by the existing guidelines; they accounted for only 1.6% of all instances of severe valvular insufficiency. Of the 135 patients with severe PI, transesophageal echocardiography (in addition to transthoracic echocardiography) was performed in 14 of them.

On transthoracic echocardiography, the evaluation of the pulmonic valve was performed primarily on the parasternal short-axis view; however, occasionally, evaluation of the right ventricular infundibulum and pulmonic valve was performed from the short-axis subcostal view. The findings are summarized in Table 1.

In addition to traditional echocardiographic findings of severe PI documented in the guidelines, we also searched for uncommon

Abbreviation
PI = Pulmonic insufficiency

findings of severe PI among our study patients. Examples of findings of severe PI found in this cohort are demonstrated below.

Table 1 Frequency of severe valvular regurgitation

Finding	Frequency
Mitral insufficiency	50.5%
Tricuspid insufficiency	33.8%
Aortic insufficiency	14.0%
Pulmonic insufficiency	1.6%

Percentages are based on 8,279 instances of severe valvular insufficiency found among 100,167 echocardiographic studies.

RESULTS

Traditional Echocardiographic Findings of Severe PI

Two-dimensional transthoracic echocardiography of a patient with severe PI many years following surgical pulmonic valvulotomy demonstrated severe right ventricular dilation. A very wide vena contracta and a regurgitant jet that occupied a large area of the right ventricular outflow tract (approximately 70%) are demonstrated in Figure 1 and Video 1 (view video clip online).

In addition, continuous-wave Doppler (Figure 2) through the pulmonary valve demonstrated an intense PI flow signal, which was equal in intensity to the antegrade pulmonic valve flow signal. Figure 2 also demonstrates a steep deceleration of PI flow velocity. On transesophageal echocardiography, color M-mode imaging at the level of the pulmonic valve (Figure 3) demonstrated most of the PI confined to early diastole because of rapid equalization of right ventricular and pulmonary artery diastolic pressures, also consistent with severe PI. These findings meet the existing criteria for grading severe PI.

Continuous-wave Doppler in the parasternal short-axis view on transthoracic echocardiography in another patient with severe PI (Figure 4) demonstrated an intense signal with steep deceleration of PI flow velocity. In addition, there was increased antegrade velocity through the pulmonic valve (1.8 m/sec; normal < 1.3 m/sec). There was no evidence of pulmonic stenosis.

Less Common Findings of Severe PI

The following findings in severe PI are not well described in the current guidelines.

Holodiastolic Pulmonary Artery Flow Reversal. On transesophageal echocardiography in the first patient described with severe PI, pulsed-wave Doppler in the pulmonary artery with the sample volume placed 2 cm distal to the pulmonic valve demonstrated holodiastolic reversal of flow (Figure 5). The ratio of the velocity-time integrals of retrograde to antegrade flow at this position suggested that approximately 60% to 70% of antegrade flow was regurgitated. More precise quantification of antegrade and retrograde stroke volume was limited by a nonparallel intercept angle with the regurgitant jet and unequal distribution of distal flow within the pulmonary artery. Holodiastolic flow reversal in the pulmonary artery was observed in five of 135 instances (3.7%) of severe PI in our series.

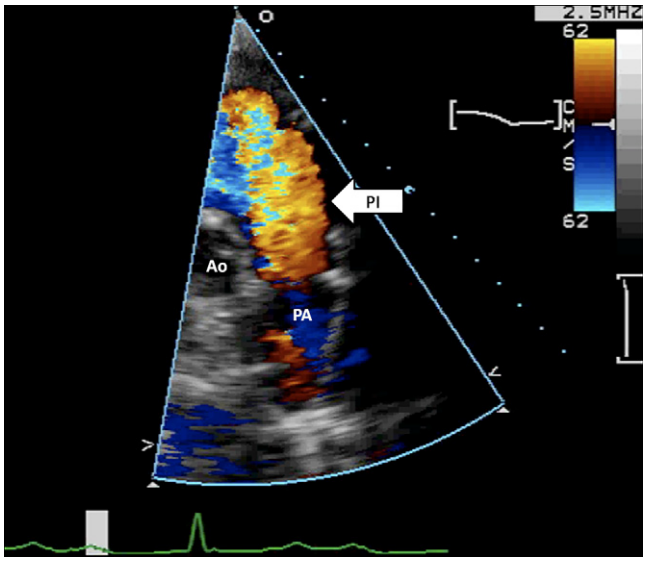

Figure 1 Transthoracic parasternal short-axis view at the basal level demonstrates severe PI by color Doppler. *Ao*, Aortic valve; *PA*, pulmonary artery.

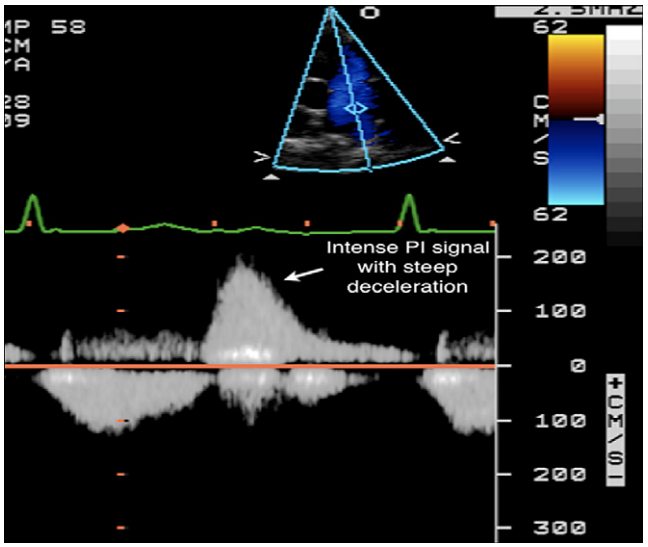

Figure 2 Continuous-wave Doppler through the pulmonic valve shows steep deceleration and intense signal of PI jet on transthoracic echocardiography.

PI With Laminar Retrograde Flow. The pulsed-wave Doppler recordings shown in Figure 5 demonstrate a narrow range of velocities within the sample volume. This is consistent with laminar (nonturbulent) flow. Laminar flow of PI can also be observed by color Doppler imaging in a patient with a congenitally absent pulmonic valve, as demonstrated in Figure 6. PI with laminar retrograde flow was documented in two of 135 instances (1.5%) of severe PI in our series.

Premature Opening of the Pulmonic Valve. Analysis with color Doppler of a patient with a congenitally absent pulmonic valve, as shown in Figure 6 and Video 2 (view video clip online), demonstrated premature antegrade flow in the pulmonary artery at end-diastole (Figure 7). Pulsed-wave Doppler with the sample volume

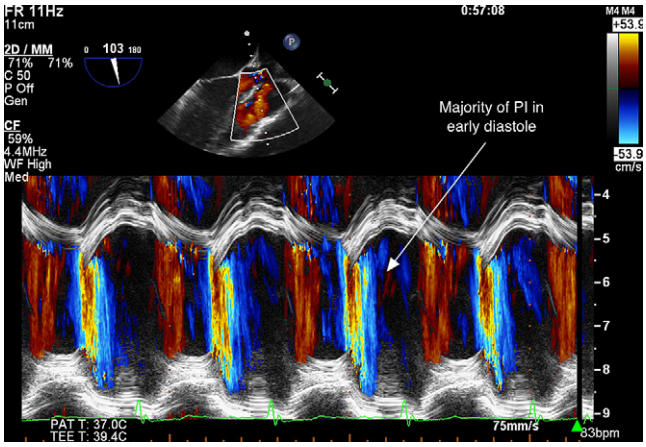

Figure 3 Color Doppler M-mode on transesophageal echocardiography reveals that the majority of PI occurs during early diastole.

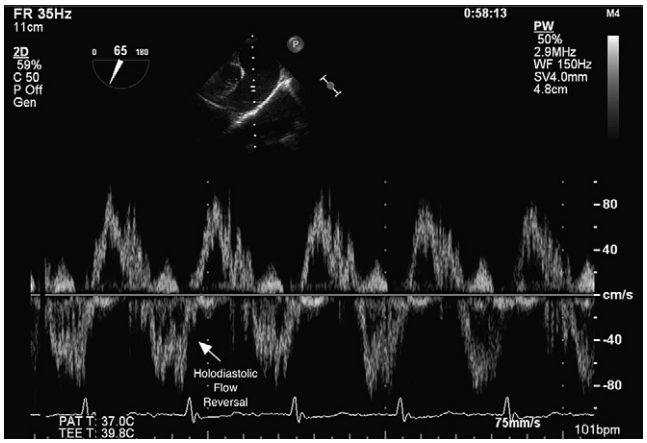

Figure 5 Pulsed-wave Doppler recorded in the pulmonary artery obtained by transesophageal echocardiography shows holodiastolic flow reversal.

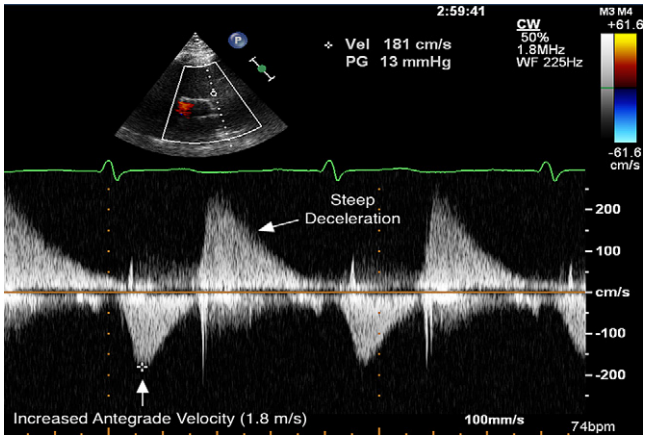

Figure 4 Continuous-wave Doppler through the pulmonic valve obtained by transthoracic echocardiography shows increased antegrade velocity through the pulmonic valve. Steep deceleration of PI signal is also noted.

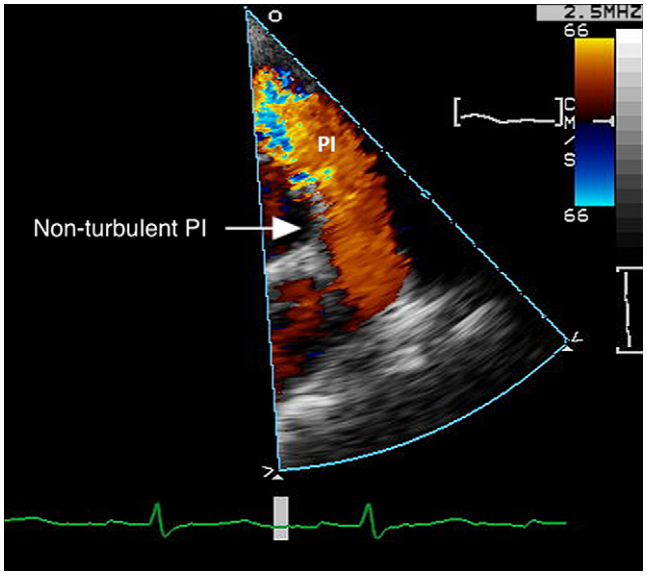

Figure 6 Color Doppler obtained on transthoracic parasternal short-axis view at the basal level demonstrates severe, hardly turbulent PI in early diastole. Note the timing of the frame on the electrocardiographic tracing.

in the proximal pulmonary artery demonstrated antegrade diastolic flow coinciding with atrial contraction (Figure 8). The findings in Figures 7 and 8 are indicative of premature opening of the pulmonic valve. PI with premature opening of the pulmonic valve was documented in two of 135 instances (1.5%) of severe PI in our series.

Low Peak Velocity of PI. Another feature of severe PI is the low peak velocity (≤0.8 m/sec) of the pulmonic regurgitant flow, as demonstrated in Figure 8. The low peak velocity is the result of a low-pressure gradient between the pulmonary artery and the right ventricle in early diastole. In this figure, the regurgitant flow also appears laminar (nonturbulent). PI with low peak velocity of the PI jet was documented in 2 of the 135 instances (1.5%) of severe PI in our series.

Premature Tricuspid Valve Closure. Continuous-wave Doppler through the tricuspid valve in Figure 9 demonstrates premature closure of the tricuspid valve in mid-diastole. Premature tricuspid valve closure was observed in nine of 135 instances (6.6%) of severe PI in our series. Among these nine instances, three were associated with first-degree atrioventricular block and another two with atrial flutter.

DISCUSSION

Severe PI is usually related to either surgically treated or untreated congenital heart disease, for example, surgically corrected tetralogy of Fallot or congenital absence of the pulmonic valve. Other forms of acquired severe PI are usually related to pulmonary hypertension from intracardiac shunts, pulmonary artery dilation from connective tissue disease, myxomatous valve disease, endocarditis, rheumatic heart disease, carcinoid, syphilis, or trauma.[2] Determination of the severity of PI is often important during surgical evaluation and for serial observation for the conditions listed. Methods to define PI

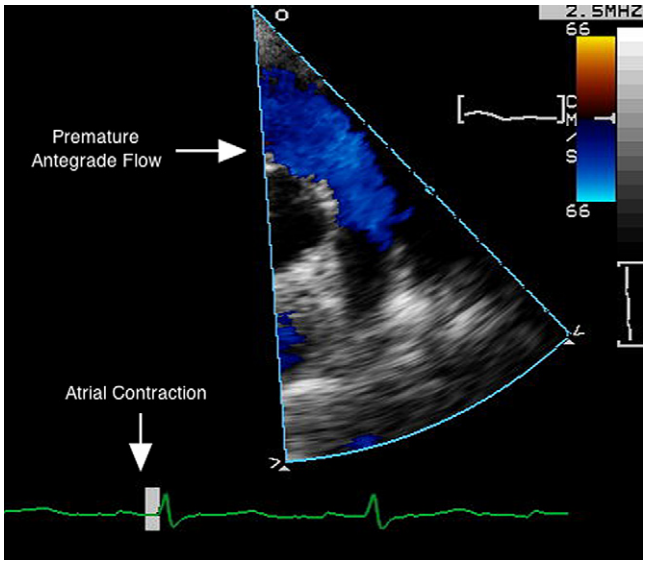

Figure 7 Color Doppler obtained by transthoracic parasternal short-axis view at the basal level reveals premature antegrade flow in the pulmonary artery coinciding with atrial contraction. Note the timing of the frame on the electrocardiographic tracing.

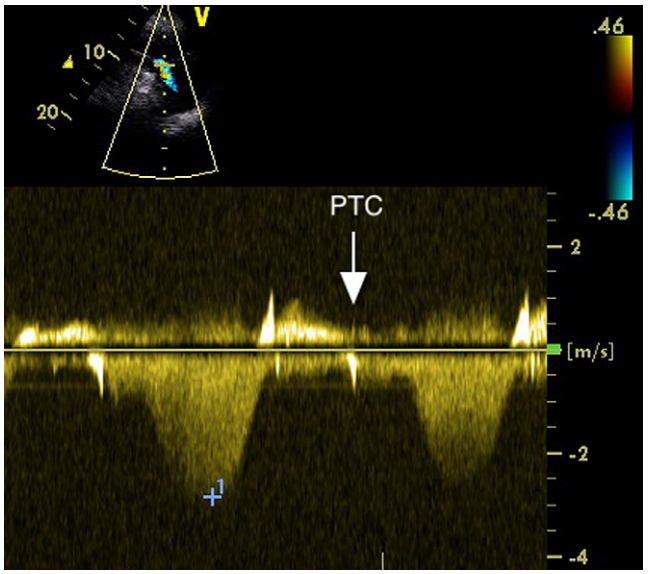

Figure 9 Continuous-wave Doppler through the tricuspid valve obtained by transthoracic echocardiography demonstrates premature closure of the tricuspid valve (PTC).

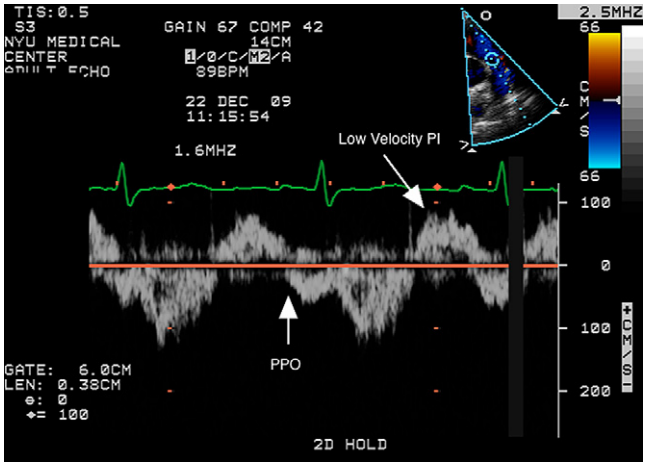

Figure 8 Pulsed-wave Doppler obtained by transthoracic imaging at the pulmonic valve demonstrates low-velocity nonturbulent PI and premature pulmonic valve opening (PPO).

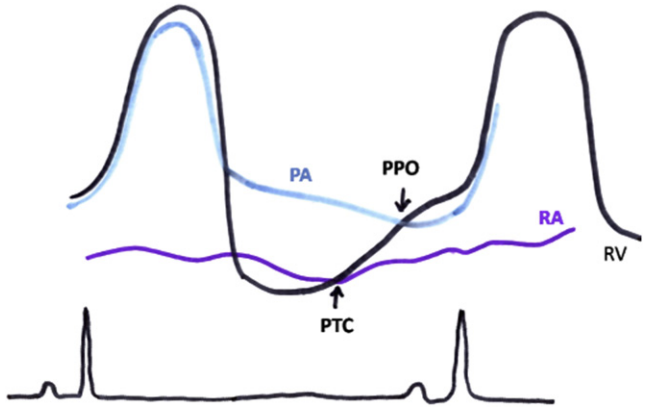

Figure 10 Illustration of the right-heart hemodynamics in severe PI. Premature tricuspid valve closure (PTC) occurs in mid-diastole, when right ventricular diastolic pressure (RV; *black line*) exceeds right atrial pressure (RA; *purple line*). Premature pulmonic valve opening (PPO) occurs when right ventricular end-diastolic pressure rises above pulmonary artery diastolic pressure (PA; *blue line*).

severity, including the current published guidelines, remain qualitative and unvalidated.[1]

The published criteria to measure PI severity do not include in detail the subtle signs presented above, namely, (1) holodiastolic flow reversal in the pulmonary artery, (2) PI with laminar retrograde flow, (3) premature opening of the pulmonic valve, (4) low peak velocity of the PI jet, and (5) premature closure of the tricuspid valve. The right-heart hemodynamics of severe PI are summarized in Figure 10. The frequency of four of these uncommon findings among the cases of severe PI is presented in Table 2. All the findings described may be seen on transthoracic echocardiography. Transesophageal echocardiography, however, may provide better visualization of flow reversal and a more parallel intercept angle for pulsed-wave Doppler in the pulmonary artery.

Holodiastolic flow reversal in the pulmonary artery, demonstrated in Figure 3, may represent a more specific finding of severe PI than the other findings discussed. Rapid blood runoff during diastole into the right ventricle creates pulmonary artery flow reversal. This runoff is caused by the pressure gradient that exists at the onset of diastole between the pulmonary artery and the right ventricle and the absence of a competent pulmonic valve. As the pressure gradient diminishes, the velocity of retrograde pulmonary artery flow declines, and eventually this flow ceases. This is analogous to flow reversal in the descending thoracic aorta in severe aortic insufficiency. Diastolic flow reversal in the pulmonary artery is also present in an uncomplicated patent ductus arteriosus. However, the retrograde ductal flow persists throughout systole as well and is usually much faster. Furthermore,

Table 2 Frequency of uncommon Doppler echocardiographic findings in severe PI

Finding	Frequency
Premature closure of the tricuspid valve	6.6%
Holodiastolic flow reversal	3.7%
Premature opening of the pulmonic valve	1.5%
PI with laminar retrograde flow	1.5%
PI with low peak velocity of the PI jet	1.5%

Percentages are based on 135 instances of severe PI among 8,279 instances of severe valvular insufficiency.

aneurysmal dilation of the pulmonary artery is also sometimes associated with diastolic flow reversal.[3] However, neither ductal flow nor aneurysmal dilatation of the pulmonary artery is usually associated with severe PI. An attempt to quantify PI severity using this finding has been made. A study by Goldberg and Allen[4] demonstrated the successful use of pulsed-wave Doppler to calculate regurgitant volume and fraction across regurgitant semilunar valves. Diastolic flow reversal in the pulmonary artery is therefore a quantifiable marker of PI severity that requires further validation.

PI with laminar retrograde flow as suggested by a relatively laminar pulsed-wave spectral Doppler pattern and minimal aliasing on color-Doppler imaging (Figures 5 and 6) is another feature of severe PI. The large effective regurgitant orifice area associated with severe PI often produces nonturbulent regurgitant flow. In contrast, turbulent regurgitant flow implies a broad distribution of flow velocities, which arises from a relatively small regurgitant orifice area.

Premature opening of the pulmonic valve at end-diastole arises from a transient rise in right ventricular pressure above pulmonary artery pressure during atrial contraction (Figures 7, 8, and 10). Severe PI leads to higher right ventricular end-diastolic pressure and lower pulmonary artery end-diastolic pressure. Depending on right ventricular compliance and in the absence of pulmonary hypertension, premature opening of the pulmonic valve can occur, resulting in antegrade flow from the right ventricle to the pulmonary artery at end-diastole. Other conditions associated with premature opening of the pulmonic valve include sinus of Valsalva rupture into the right atrium, constrictive pericarditis, Loeffler's endocarditis, Ebstein's anomaly with tricuspid regurgitation, and right ventricular infarction.[5,6] Although this finding lacks specificity for severe PI, its presence can reflect the effect of PI on right ventricular hemodynamics.[2]

PI causes an increase in right ventricular diastolic pressure and a decrease in pulmonary artery diastolic pressure. A low peak velocity of PI (Figure 8) implies that a significant portion of the antegrade pulmonic flow is regurgitated, and this diminishes the pressure gradient between the pulmonary artery and the right ventricle during diastole. Severe PI, especially in the absence of pulmonary hypertension, can produce such a small gradient.

Premature closure of the tricuspid valve is caused by an elevation of right ventricular pressure above right atrial pressure in diastole, driven by the increased volume of blood returned to the right ventricle in diastole by severe PI (Figures 9 and 10). This finding also depends on the compliance of the right ventricle. The physiology of this finding is similar to that of diastolic mitral valve closure in acute, severe aortic insufficiency.[7] Furthermore, premature closure of the tricuspid valve can be seen in patients with the first-degree or greater atrioventricular block, premature atrial contraction, or atrial flutter.

Study Limitations

The prevalence of severe PI among the population studied was lower than that of any other type of valvular insufficiency (Table 1). This is likely related to the general population studied in an adult noninvasive cardiology laboratory. The prevalence of severe PI is likely to be significantly higher in laboratories with large populations of adults with congenital heart disease, specifically those with surgically corrected tetralogy of Fallot and pulmonic stenosis.

Our lab routinely checks for PI, and we believe that we find severe PI, as defined by the existing guidelines, with high frequency. Although this was a retrospective analysis, the incidences of the unusual findings described are likely not significant underestimations of their true prevalence in our case series of severe PI.

Although we do perform routine M-mode and two-dimensional echocardiography of the right ventricle to judge the severity of right ventricular overload in these patients, we do not routinely perform quantitative assessment of right ventricular volumes. Quantitative data on right ventricular volumes were therefore not available.

The criteria described should be considered in the grading of PI severity in addition to the criteria discussed in the existing guidelines.

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