showed abnormal geometrical configurations of the left ventricular cavity when viewed in short axis (Fig. 9). These changes ranged in appearance from the normally circular configuration to straightening of the ventricular septum or severe flattening of the ventricle in the anteroposterior dimension. The severity of these changes showed a loose association with the degree of elevation of the pulmonary arterial pressure. The clinical importance and the reasons for these changes are uncertain at the present time. It may be that progressive elevations in pulmonary arterial pressure that result in enlargement of the right ventricle, in turn, may alter the normal convexity of the interventricular septum and distort the appearance of the left ventricle.

In summary, it appears that two-dimensional echocardiography is a useful method for the evaluation of mitral valve orifice size in patients with mitral stenosis. Furthermore, two-dimensional echocardiographic assessment of mitral leaflet spatial motion explains many of the M-mode findings commonly observed in these patients. In the future, these baseline two-dimensional echocardiographic observations concerning stenotic mitral leaflet motion and morphology may provide a method for answering other important clinical questions such as determining the suitability for mitral valvulotomy.

References

Premature Pulmonary Valve Opening

L. S. WANN, M.D., ARTHUR E. WEYMAN, M.D., JAMES C. DILLON, M.D., AND HARVEY FEIGENBAUM, M.D.

SUMMARY Premature opening of the pulmonary valve (opening independent of atrial or ventricular systole) was originally described in a case of sinus of Valsalva rupture into the right atrium. Since that time we have observed five additional cases in which the pulmonary valve opened prematurely. Entities encountered included: 1) constrictive pericarditis; 2) Loeffler’s endocarditis; 3) Ebstein’s anomaly with tricuspid regurgitation; 4) tricuspid regurgitation following tricuspid valvulectomy, and 5) pulmonary regurgitation accompanied by atrial septal defect. In the first two cases, premature pulmonary valve opening is felt to be due to restriction of diastolic filling of the right ventricle with subsequent early diastolic rise in pressure equaling or exceeding pulmonary artery diastolic pressure. In the latter three cases, the increased volume of blood entering the right ventricle again appeared to result in a rapid rise in initial right ventricular diastolic pressure and to produce premature opening of the pulmonary valve. Premature pulmonary valve opening, therefore, does not appear specific for any particular clinical entity but reflects the relative pressures in the right ventricle and pulmonary artery during diastole.

A NUMBER OF RECENT REPORTS have demonstrated the clinical usefulness of recording the echocardiographic pattern of pulmonary valve motion. Characteristic echocardiographic patterns have been described in patients with pulmonary valvular stenosis,4 pulmonary infundibular stenosis,2 pulmonary regurgitation,9 Uhl’s anomaly,4 and pulmonary hypertension.5,6 We have previously reported a case of sinus of Valsalva aneurysm rupture into the right atrium in which one of the echocardiographic manifestations was early diastolic opening of the pulmonary valve.7 We felt that premature pulmonary valve opening (opening independent of atrial or ventricular systole) reflected a rapid early diastolic rise in right ventricular pressure which equaled or transiently exceeded pulmonary artery diastolic pressure, causing pulmonary valve opening. We postulated that this degree of early diastolic pressure elevation in the right ventricle might be specific for a lesion bringing systemic arterial pressure to the right side of the heart. Since that time we have observed five additional cases in which the pulmonary valve opened prematurely. In all, communication between the aorta and the right heart was absent. The purpose of this report is to describe these cases and to discuss the factors responsible for premature pulmonary valve opening.

Material and Methods

During the last 24 months, we have recorded technically adequate pulmonary valve echograms in approximately 750
patients. Six of these patients demonstrated premature opening of the pulmonary valve, defined as movement of the posterior pulmonary leaflet to a position adjacent to the posterior wall of the pulmonary artery prior to the onset of the P wave on the electrocardiogram, or prior to the onset of the QRS in cases of atrial fibrillation. The valve was considered to open prematurely only if the magnitude of posterior displacement during diastole was equal to that observed following ventricular systole, at which time the leaflets were assumed to be in the fully opened position.

M-mode echocardiographic recordings were obtained using an Ekoline 20A echograph coupled to a Honeywell 1856 fiberoptic strip chart recorder and a 2.25 MHz transducer focused at 7.5 cm. The technique of recording pulmonary valve echograms has been previously described.1-8

Cardiac catheterization, using a fluid-filled catheter manometer system and standard cineangiographic techniques, was performed as a part of the clinical evaluation in each of the six cases. Catheterization was not performed simultaneously with the echocardiographic examination.

Results

Case 1

A 22-year-old woman was admitted for evaluation of hepatomegaly and ascites of two years' duration. Physical examination revealed markedly distended jugular veins and a pericardial knock. Chest roentgenogram and electrocardiogram were normal. Cardiac catheterization was consistent with constrictive pericarditis and successful pericardial stripping was carried out.

Figure 1 illustrates the pulmonary valve echogram in this case. Panel A was obtained preoperatively, and Panel B after successful pericardial stripping. In Panel A, the posterior pulmonary valve echo can be seen to reach the posterior pulmonary artery wall soon after the onset of diastole (open position) and then to close gradually prior to the onset of ventricular systole, at which time normal opening occurs. This diastolic opening of the pulmonary valve corresponds in timing to the rapid rise in right ventricular pressure following the early diastolic dip noted in this patient's pressure tracings. Right ventricular pressure exceeds pulmonary artery pressure, presumably instantaneously, at the peak of an initial rapid ventricular filling phase and causes the pulmonary valve to open. Panel B shows normal pulmonary valve opening after relief of the pericardial constriction. The valve leaflet moves gradually posteriorly during diastole with increased posterior motion following atrial contraction, but the leaflet does not move to the completely opened position until ventricular systole occurs.

Case 2

A 35-year-old woman presented with severe dyspnea and murmurs of tricuspid and mitral stenosis. Migratory pulmonary infiltrates were present and her absolute eosinophil count was 25,000/mm.6 Catheterization revealed severe mitral stenosis (valve area 0.3 mm²) and tricuspid stenosis (valve area 0.9 mm). Mitral valve replacement and tricuspid valvulotomy were performed. At operation, a dense fibrous process was seen in the mitral valve and the left ventricular endocardium. The tricuspid valve leaflets were thickened and the commissures fused. Surgical specimens were consistent with Loeffler's endocarditis.

This patient's preoperative pulmonary valve echogram was consistent with pulmonary hypertension. Postoperatively, marked tricuspid regurgitation developed over a course of one month following surgery. The pulmonary valve echogram changed from a pattern consistent with pulmonary hypertension to normal followed by premature opening of the valve. Figure 2 is a recording made one month postoperatively.

![Figure 1](image_url)  
**Figure 1.** Panel A shows the preoperative pulmonary valve echogram of case 1 with constrictive pericarditis. The dark vertical line at the onset of the P wave shows the pulmonary valve to be fully opened at that time. Panel B shows a normal pulmonary valve echogram after pericardial stripping. e = valve position at beginning of diastole. f = valve position prior to atrial systole. a = contribution of atrial contraction.
Case 3

A 20-year-old man presented for outpatient follow-up of Ebstein's anomaly with severe tricuspid regurgitation. Cardiac catheterization had been performed 15 years earlier. He was asymptomatic. Physical examination revealed a large heart without palpable systolic movement. A protodiastolic gallop and a holosystolic murmur were present at the left sternal border. Chest roentgenogram showed a markedly enlarged cardiac silhouette. The electrocardiogram revealed right bundle branch block and atrial fibrillation.

The pulmonary valve echogram from this case is shown in figure 3. The posterior pulmonary valve leaflet begins to move posteriorly at the onset of diastole and gradually reaches the fully opened position just prior to the onset of the QRS complex. The valve opens completely only during inspiration and following relatively long R-R intervals.

Case 4

A 19-year-old man presented for outpatient follow-up without symptoms. He had undergone pulmonary valvulotomy seven years earlier for pulmonary stenosis, and postoperative catheterization revealed moderate pulmonary insufficiency. He had a small atrial septal defect which was not repaired. Physical examination revealed a normal-sized heart and the murmur of pulmonary insufficiency.

In this case, the posterior pulmonary leaflet gradually moved toward the posterior wall throughout diastole and reached the fully opened position just prior to atrial systole (fig. 4).

Case 5

A 25-year-old woman entered the hospital with acute staphylococcal tricuspid endocarditis. Physical findings included consolidation at the base of the left lung, a presystolic murmur, and a loud early systolic sound. Cineangiography with contrast material injected into the superior vena cava showed a mass attached to the tricuspid valve. Because of continued sepsis, heart failure, and recurrent septic
pulmonary emboli, tricuspid valvulectomy was performed. A large vegetation that had virtually replaced the tricuspid valve with abscesses in the papillary muscles and interventricular septum was noted at operation.

This patient's pulmonary valve echograms are illustrated in figure 5. Panel A, obtained prior to tricuspid valvulectomy, shows normal pulmonary valve motion. In panel B, after tricuspid valvulectomy, the posterior pulmonary leaflet has reached the fully opened position prior to the electrocardiographic P wave. This finding of premature pulmonary valve opening has persisted for the six months since operation.

![Figure 4](image.jpg)  
**Figure 4.** Pulmonary valve echogram of case 4 with pulmonary insufficiency and a small atrial septal defect. The dark vertical line is drawn at the onset of the electrocardiographic P wave and demonstrates pulmonary valve opening before this event.  

![Figure 5](image.jpg)  
**Figure 5.** Panel A is a normal pulmonary valve echogram from case 5 with tricuspid endocarditis. Panel B, obtained after tricuspid valvulectomy, shows premature pulmonary valve opening. The dark vertical lines are at the beginning of the electrocardiographic P wave.
Case 6

A 24-year-old male presented with bacterial endocarditis resulting in rupture of the right sinus of Valsalva into the right atrium. In this case as well, the posterior pulmonary leaflet was in full apposition with the posterior wall of the pulmonary artery prior to atrial systole. Details of this case have previously been reported.7

Table 1 summarizes the clinical findings and catheterization data of these six patients.

**Discussion**

Pulmonary valve opening normally occurs during ventricular systole when right ventricular pressure exceeds pulmonary artery pressure. In certain situations right atrial systole may increase right ventricular end-diastolic pressure to a level equalling or exceeding simultaneous pulmonary artery pressure, causing the pulmonary valve to open. This phenomenon occurs commonly in patients with moderate and severe valvular pulmonic stenosis and has been reported with Uhl's anomaly (congenital hypoplasia of the right ventricular myocardium). Since pulmonary artery pressure decreases continuously throughout diastole, relatively lower pulmonary artery end-diastolic pressures occur at longer cycle lengths. Thus, (at very slow heart rates) pulmonary artery end-diastolic pressure may fall to very low levels, and opening of the pulmonary valve may occur normally following atrial systole, particularly during inspiration. This is most commonly seen in young athletes but also may occur following premature ventricular contractions.

During initial diastole, pulmonary artery pressure is generally much higher than right ventricular pressure, and opening of the pulmonary valve prior to atrial or ventricular systole is very unusual. As noted in this report, we have observed this phenomenon to occur only six times in over 750 pulmonic valve echograms recorded over a two-year period. In the initial case, premature opening of the pulmonary valve was noted in a patient with a ruptured sinus of Valsalva aneurysm. Because this patient was in florid pulmonary edema, it was reasoned that the only source of pressure in the cardiovascular system in excess of pulmonary artery pressure at that particular portion of the cardiac cycle would be the aorta and that premature pulmonary valve opening might be specific for an aorta-to-right atrial or right-ventricular fistula. It appears now that any situation which produces rapid increase in right ventricular diastolic pressure to a level equalling or transiently exceeding pulmonary artery pressure will produce opening of the pulmonary valve.

Figure 6 is a pulmonary valve echochogram superimposed on simultaneous right ventricular and pulmonary artery pressure recordings from a patient with congenital complete heart block, atrial septal defect, pulmonic stenosis, and mild pulmonary hypertension. This figure illustrates the relationship between right ventricular and pulmonary artery pressure and pulmonary valve echo motion. In this recording, the atrial and ventricular contractions are so related that atrial systole initially elevates right ventricular diastolic pressure to a point which equals or slightly exceeds pulmonary artery diastolic pressure and produces opening of the pulmonary valve. Following atrial contraction, the right ventricular diastolic pressure gradually falls below simultaneous pulmonary artery pressure and the valve returns to the closed position. The pressure then rises again in the absence of either atrial or ventricular contraction to equal pulmonary artery diastolic pressure and the valve again begins to reopen. This is followed by full opening of the valve at the time of ventricular systole.

In this report, we have reviewed our original case in which the pulmonary valve opened prior to atrial or ventricular systole and have described five additional cases. Although the underlying pathophysiology was different in each of these cases, the net effect on the instantaneous pressures across the pulmonic valve was similar, resulting in premature valve opening.

**Table 1. Clinical Data**

<table>
<thead>
<tr>
<th>Pt/age/sex</th>
<th>Diag</th>
<th>Right atrium</th>
<th>Pressures*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean s v</td>
<td>RV PA PCW</td>
</tr>
<tr>
<td>1/22/F</td>
<td>Constrictive pericarditis</td>
<td>20 20 20</td>
<td>40 38 21</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2/35/F</td>
<td>Loeffler's endocarditis</td>
<td>4 9 5</td>
<td>62 62/23</td>
</tr>
<tr>
<td>3/20/M</td>
<td>Ebstein's anomaly</td>
<td>5 4 5</td>
<td>24 26/7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4/19/M</td>
<td>Pulmonary regurgitation and ASD. Post pulmonary valvulotomy</td>
<td>— — —</td>
<td>26/3 20/3</td>
</tr>
<tr>
<td>5/24/F</td>
<td>Tricuspid regurgitation. Post tricuspid valvulotomy for endocarditis</td>
<td>12 12 15</td>
<td>— — —</td>
</tr>
<tr>
<td>6/24/M</td>
<td>Sinus of Valsalva aneurysm ruptured into right atrium</td>
<td>29 39 31</td>
<td>57/26 56/26</td>
</tr>
</tbody>
</table>

*Pressure given in mm Hg

Abbreviations: s = pressure at time of s wave; v = pressure at time of v wave; RV = right ventricular; PA = pulmonary artery; PCW = pulmonary capillary wedge; LV = left ventricle.
diastolic pressure and subsequent opening of the pulmonary valve.

In cases 3, 4 and 5, patients with Ebstein’s anomaly with tricuspid regurgitation, free tricuspid regurgitation, and atrial septal defect with pulmonary regurgitation, respectively, the basic hemodynamic alteration was a large volume flow into the right ventricle during diastole. In each case, the ability of the right ventricle to accept this large volume appears to have been impaired to the extent that there was a rapid rise in right ventricular initial diastolic pressure.

In case 6, aortic diastolic pressure was transmitted to the right ventricle and resulted in elevation of right ventricular pressure with opening of the pulmonary valve.

Opening of the pulmonary valve is governed, therefore, by the pressure dynamics and compliance of the right ventricle and pulmonary artery. In each case of premature pulmonary valve opening presented, alterations in right ventricular diastolic pressure relative to pulmonary artery diastolic pressure account for opening of the pulmonary valve without the contribution of atrial or ventricular systole.

With the exception of the patient with Loeffler’s endocarditis, patients with disorders similar to each of the cases reported here have been examined in whom the pressure alterations were not as striking and in whom premature opening of the pulmonary valve did not occur.

We conclude that the echocardiographic observation of premature pulmonary valve opening does not indicate a specific disease entity, but may be an integral part of the altered hemodynamics of one of several processes which result in elevation of right ventricular pressure early in diastole.

References

Premature pulmonary valve opening.
L S Wann, A E Weyman, J C Dillon and H Feigenbaum

doi: 10.1161/01.CIR.55.1.128
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1977 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/55/1/128

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org//subscriptions/