Physiologic Correlates of Echocardiographic Pulmonary Valve Motion in Diastole

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SUMMARY It has been shown that the echocardiographic motion of the posterior aortic wall in diastole is closely related to the underlying left atrial events, possibly due to the anatomical proximity of the two structures. We observed that the pulmonary artery shares a similar close anatomical relationship with the left atrium. The present study in 55 consecutive patients with adequate echocardiographic recordings of the aortic root and the pulmonary valve demonstrates that the diastolic waveforms of the pulmonary valve and the posterior aortic wall are nearly identical in early diastole. The pulmonary valve e-f slope correlated with the posterior aortic wall O-R slope \( r = 0.95 \) and the S-f interval \( r = 0.94 \). No significant correlation was found between the pulmonary valve e-f slope and the pulmonary artery pressures.

The presence and amplitude of the maximum “a dip,” on the other hand, correlated with the presence of pulmonary hypertension, with some notable exceptions. In addition, the “a dip” on the pulmonary valve and the depth of the A wave on the posterior aortic wall were significantly correlated \( r = 0.85 \). It appears that the “a dip” on the pulmonary valve is influenced by dual mechanisms: pressure differences between the pulmonary artery and the right ventricle in late diastole and the left atrial events. Thus, the early diastolic waveform of the pulmonary valve, like the posterior aortic wall waveform, may primarily reflect underlying left atrial events and is not a measure of pulmonary artery pressure. The late diastolic waveform “a dip” has a dual mechanism, related in part to the underlying left atrial events, and also reflecting the instantaneous pressure differences across the pulmonary valve following atrial systole.

ECHOCARDIOGRAPHIC EVALUATION of the pulmonary valve was first described by Gramiak and associates.\(^1\) Subsequently, clinical and hemodynamic correlates of the valve motion have been reported.\(^2\)\(^,\)\(^4\)

The systolic opening of the valve is readily explained by the right ventricular-pulmonary arterial pressure events during ventricular systole. However, the diastolic motion pattern remains largely unexplained.

Strunk et al.\(^6\) have recently shown that the posterior aortic wall motion in diastole is closely related to left atrial volume changes. This was attributed to a close anatomic relationship between the two structures. We observed a qualitative similarity between the waveforms of pulmonary valve and the aortic root during diastole. We therefore reasoned that the pulmonary artery, also having a close anatomical relationship to the left atrium, may be influenced by the left atrial volume changes in diastole. This hypothesis was tested by quantitative measurements in a series of
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patients evaluated before cardiac catheterization in whom technically adequate aortic root and pulmonary valve echoes were obtained. After demonstration of a close similarity in waveforms, we undertook a critical analysis of the usefulness of diastolic motion in pulmonary hypertension.

Materials and Methods

Fifty-five patients admitted for cardiac catheterization were studied. A criterion for entrance into the study was the ability to record satisfactory echocardiograms of the pulmonic valve and aortic root. All patients were catheterized within 24 hours of the echocardiographic examination and the studies included measurements of right heart pressures (pulmonary arterial, right ventricular and right atrial pressures) and cardiac output in the basal resting state.

The echocardiographic examination was performed with a Unirad or a Picker Echoview Ultrasonoscope, using a 2.25 MHz transducer and a Cambridge Fiberoptic Strip Chart Recorder. A multichannel recording of the M-mode echocardiogram, electrocardiogram (lead II) and phonocardiogram was obtained at paper speeds of 50 and 100 mm/sec. The aortic root was visualized from a left parasternal position in the third or fourth interspace with 10-15° medial angulation. In order to visualize the pulmonary valve, the transducer was moved one interspace higher and again directed laterally and cephalad. Sequential aortic root and pulmonic valve echograms were obtained at the same examination in all cases.

The study population consisted of 20 females and 35 males ranging in age from 8-68 years. Thirteen patients had coronary arterial disease; 25 had valvular heart disease, including 15 with mitral stenosis; six had cardiomyopathy; six had congenital heart disease (five atrial septal defect and one pulmonary stenosis); three had primary pulmonary hypertension; and two with ill-defined chest pain syndromes were demonstrated to be free of heart disease.

The diastolic waveforms of the pulmonary valve and posterior aortic wall from cardiac cycles of nearly identical length were traced for comparison (figs. 1A and B). To examine this relationship numerically and for statistical comparisons, several measurements were obtained. The pulmonary valve e-f slope was...
measured (mm/sec) from the most anterior position of the waveform (e point) to the point where the rapid posterior motion slows and the waveform became more horizontal (f point). The e point was clearly defined in all patients in this study. In cases where no discrete f point was discerned (as in patients with mitral stenosis), the slope of the continuous posterior diastolic motion was determined. The O-R slope of the posterior aortic wall was similarly measured from the most anterior position (O point) to the point where the rapid posterior motion slows and the waveform becomes more horizontal (R point). Three to five con-

Figure 2. Pulmonary valve (PV) e-f slope in mm/sec plotted against O-R slope of the posterior aortic wall (PAW) in mm/sec. The number of points does not represent the number of subjects, since some points represent more than one patient with identical values in this computer-generated plot.

Figure 3. The intervals $S_2$-$f$ on pulmonary valve (PV) and $S_2$-$R$ on posterior aortic wall (PAW) are plotted in patients with discernible $f$ and $R$ points. The number of points does not represent the number of subjects, for the same considerations as in figure 2.
secutive cardiac cycles were used for measurements, and average slopes and cycle lengths (R-R intervals) derived. The cycle lengths of the two recordings were required to be within 10% of each other.

Similarly, the time intervals between the onset of the second heart sound (S₂) and the pulmonary valve f point (S₂ − f), and the R point of the posterior aortic wall (S₂ − R) were measured from three to five consecutive cardiac cycles. These were averaged and compared. The left atrial size was measured in the standard manner at end-systole. The left atrial measurement was corrected for the body surface area and expressed as the left atrial index (LAI). Right atrial, right ventricular and pulmonary arterial pressures (PAP) were obtained during cardiac catheterization. Stroke volume index was calculated from cardiac outputs determined by the indicator dilution technique using indocyanine green.

In 42 patients in sinus rhythm, the maximal magnitude of the posterior motion of the pulmonary valve after atrial systole (“a dip max”) was measured during quiet respiration. The maximal depth of the posterior motion of the posterior aortic wall following atrial systole (A wave depth) was measured for comparison.

Results

The preliminary tracings of the pulmonary valve and the posterior aortic echograms showed that the two waveforms in diastole were nearly superimposable. The e-f slope of the pulmonary valve correlated with the O-R slope of the posterior aortic wall (r = 0.95) (fig. 2). Similarly, the S₂ − f interval correlated with the S₂ − R interval (r = 0.94), (fig. 3). All the 15 patients with mitral stenosis showed no discernible f or R points; instead, a gradual leisurely posterior decline in both the pulmonary valve and posterior aortic wall waveforms was recorded (fig. 4). These correlations confirmed the essential similarities of the two waveforms in early diastole. Similarly, the “a dip max” correlated with the A wave depth (r = 0.95), although with a wider scatter of the points (fig. 5).

Figure 6 shows the relationship between the pulmonary valve e-f slope and the mean PAP. No significant correlation was detected for the group as a whole. Considering patients with a flat e-f slope (e-f slope < 20 mm/sec)⁶ nine of 27, or 33%, had normal PAP (< 20 mm Hg). All patients with mitral stenosis had flat pulmonic valve and flat posterior aortic wall diastolic waveform regardless of the level of PAP. The mitral stenosis patients with low pressures tended to be hypovolemic, with reduced cardiac output.

Table 1 shows the relationship between the “a dip max” and mean PAP. All patients depicted were in normal sinus rhythm and none had clinical evidence of right ventricular failure. The PAP was normal in 72% of subjects with an “a dip max” greater than 2 mm and was elevated in 75% of patients (9–12) with absent “a daps.” Thus, we observed exceptions to the previously reported relationship between the “a dip” and the presence of pulmonary hypertension (fig. 7).

When left atrial size was considered, it was observed that the patients with enlarged left atria³ (> 2 cm/m²) demonstrated a flat pulmonary valve e-f slope regardless of PAPs (fig. 8). In patients with normal sized left atria, the e-f slope correlated with stroke volume index (r = 0.64). Patients with mitral regurgitation and atrial septal defect were excluded from this analysis of stroke volume index, since the forward stroke volume measured by the indicator dilution method does not represent the diastolic flow from the left atrium in these conditions. Since the e-f slope merely represents

![Figure 4](http://circ.ahajournals.org/)

**Figure 4. Echocardiogram of the aortic root (Ao) and pulmonary valve (PV) obtained from a patient with mitral stenosis. APS = aorto pulmonary sulcus; ECG = electrocardiogram; PCG = phonocardiogram; LA = left atrium; PAW = posterior aortic wall; PLAW = posterior left atrial wall. Note the absence of discernible R or f points.**
an early diastolic event, it does not accurately reflect the actual stroke volume index even in those with a normal sized left atrium.

Discussion

Recently Strunk et al. showed that the posterior aortic wall motion in diastole correlates with the angiographically-derived left atrial volume changes. It was postulated that the close anatomical proximity of the posterior aortic wall to the underlying left atrium was responsible for the posterior aortic wall motion. The echogram of the posterior left atrial wall shows little motion. The postulate is reasonable, since the posterior left atrial wall is a relatively fixed structure resting on the esophagus and the vertebrae bound by the pulmonary veins and mediastinal attachments. Changes in left atrial dimension through the cardiac cycle are likely to be best reflected in the anterior left atrial wall motion. At the level of the aortic root the anterior wall of the left atrium is in close anatomic proximity to the posterior aortic wall. Thus, changes in left atrial dimensions throughout diastole may be reflected in the posterior aortic wall motion.

The pulmonary artery shares a similar anatomical relationship to the left atrium, as illustrated in figure 9. Therefore, it might be expected that the pulmonary artery and its attached valve have a waveform similar to the posterior aortic wall. The results of this study show that the early diastolic waveforms of both the pulmonary valve and the posterior aortic wall are nearly identical, suggesting a common underlying mechanism. Comparing similar cycle lengths in the individual patient, the waveforms were nearly superimposable. The early diastolic slope (e-f slope) of the pulmonary valve and the O-R slope of the posterior aortic wall were closely correlated ($r = 0.95$). Both slopes may thus reflect the rate of early diastolic left atrial emptying.

If this hypothesis is correct, the disease states affecting left atrial wall motion (volume changes and size) must be reflected similarly in both waveforms. The early diastolic slope of both waveforms was found to be reduced in states where left atrial wall motion is likely to be reduced. This occurs 1) in mitral stenosis, which produces slowed left atrial emptying throughout diastole; 2) in the presence of a large left atrium, where a small change in wall motion is required to affect a given volume change, and 3) when the stroke volume is significantly reduced. The findings in this study support this hypothesis since: 1) In all patients with mitral stenosis, both the e-f slope of pulmonary valve and the O-R slope of the posterior aortic wall

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**Table 1. Relationship Between "a dip maximum" of the Pulmonary Valve and Mean Pulmonary Artery Pressures (PA) in Patients in Sinus Rhythm**

<table>
<thead>
<tr>
<th>PA mm Hg</th>
<th>&gt; 2 mm</th>
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<th>Absent</th>
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<tr>
<td>&lt;20</td>
<td>18</td>
<td>2</td>
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<tr>
<td>21–40</td>
<td>5</td>
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<td>&gt;40</td>
<td>2</td>
<td>0</td>
<td>7</td>
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FIGURE 6. Pulmonary valve (PV) e-f slope in mm/sec plotted against mean pulmonary arterial (PA) pressure in mm Hg. The dotted horizontal line divides normal from decreased e-f slopes and vertical line normal from elevated PA pressures. The closed circles represent patients with mitral stenosis. The correlation between the slopes and the pressures is poor, and all those with mitral stenosis have reduced slopes regardless of PA pressures. The number of points does not represent the number of subjects, for the same considerations as in figure 2.

FIGURE 7. The pulmonary valve (PV) echogram in one of the two patients with severe pulmonary hypertension (mean pulmonary artery pressure (PA) of 54 mm Hg) with "a dip maximum" > 2 mm. The right atrial pressure (RA) is normal and right ventricular end-diastolic pressure (RV) mildly elevated. The "a dip" is observed despite a 24 mm Hg end-diastolic pressure difference across the valve. APS = aortopulmonary sulcus; LA = left atrium; PCG = phonocardiogram; ECG = electrocardiogram.
were attenuated, and no discernible f or R points were present. Instead, a gradual posterior motion of both waveforms throughout diastole was recorded, reflecting the gradual decline in underlying left atrial volume. The pulmonary valve e-f slope was thus attenuated regardless of the level of the PAP. 2) All subjects with large left atria (defined as a LAI $>2$ cm/m$^2$) had flattened early diastolic waveforms. 3) The patients with reduced stroke volumes tended to have reduced diastolic slopes.

**Figure 8.** Pulmonary valve (PV) e-f slope in mm/sec plotted against the stroke volume index (SVI) in ml/m$^2$. The open circles represent subjects with a left atrial index (LAI) within the normal range (LAI $\leq 2$ cm/m$^2$) and the closed circles of those with large left atria. Patients with mitral regurgitation and atrial septal defect are not included.

**Figure 9.** Anatomical drawing of the relationship between the pulmonary artery and posterior aortic wall (PW) to the left atrium (LA). The echocardiograms of the pulmonary valve and the aortic root are shown in panels A and B respectively. The transducer position A directs the beam through the posterior pulmonary cusp (PV), the aortopulmonary sulcus (APS) and the left atrium (LA). The transducer position B directs the beam through the walls of the aortic root, the right (R) and noncoronary (N) cups of aortic valve and exits through the left atrium (LA). AoV = aortic valve; AW = anterior aortic wall, LPV = left pulmonary vein; RPV = right pulmonary vein, RV = right ventricle; R, A, P = right, anterior and posterior leaflets of the pulmonary valve; R, L, N = right, left and noncoronary leaflets of the aortic valve.
No significant correlation was found between the pulmonary valve e-f slope and PAP. Considering patients with flat e-f slopes, 18 of 27 did have pulmonary hypertension; however, all 18 had either large LAIs or low stroke volume index. These considerations suggest that flattening of the pulmonary valve e-f slope may have little to do with the presence or severity of pulmonary hypertension. Earlier observations relating the two may have been fortuitous, since a substantial number of adult patients with pulmonary hypertension will have underlying mitral stenosis or chronic congestive heart failure with reduced stroke volume index and enlarged left atria. It is difficult to find an adequate physiologic explanation as to why pulmonary hypertension per se would influence the early diastolic waveforms.

The “a dip” of the pulmonary valve waveforms appears to be a complex event. While the A wave of the posterior aortic wall may be principally related to a decrease in left atrial volume produced by left atrial systole, the “a dip” is also influenced by the right-sided events. In a normal subject, right atrial systole produces an A wave of 5–7 mm Hg which is transmitted to the right ventricle. The pressure in the pulmonary artery in late diastole is low. Thus, at end-diastole the right ventricular pressure is normally only slightly lower than the PAP. With inspiration there is an increase in venous return which raises the right ventricular end-diastolic pressure. Inspiration also produces a fall in pulmonary artery diastolic pressure. This may produce a presystolic gradient and an opening of the pulmonary valve — the “a dip.” Thus, changes in “a dip” depth through the respiratory cycle can be readily explained. When the pulmonary artery diastolic pressure is raised above that in the right ventricle, the maximum “a dip” will be attenuated or abolished.

A fair correlation of the maximum “a dip” and presence and severity of the pulmonary hypertension was found in this study. Seventy-two percent of subjects with normal PAPs had “a dips” greater than 2 mm, while 28% had reduced “a dips.” The exceptions therefore suggest other influences. Recently, Hada et al. emphasized the importance of transducer position and angulation in recording the “a dip.” Using an unusual transducer position one interspace higher with angulation downward and medial they were able to demonstrate flattening or absence of the “a dip” in four of 19 subjects who had a normal “a dip” using the standard transducer position. The spatial relationship between the ultrasonic beam and the valve may influence the “a dip” and may account for the attenuated “a dips” recorded in subjects with normal PAP in this study. The alternative explanations may be related to left atrial size and stroke volume, as discussed earlier.

Hada et al. have also reported that in 31 normal subjects the A waves of both semilunar valves correlated closely during expiration; 129 vs 132 msec in duration and 3.7 vs 3.6 in depth. We found a correlation between the “a dip” of the pulmonary valve and the A wave of the posterior aortic wall (r = 0.85). This suggests that motion of the great vessels related to underlying left atrial events may be a factor influencing the “a dip,” independent of the pulmonary arterial hypertension.

The results of this study demonstrate a close relationship between early diastolic waveforms of the pulmonary valve and the posterior aortic wall, possibly reflecting a common influence of the left atrial volume changes in early diastole. The pulmonary valve e-f slope is not a measure of PAP. The factors in late diastole, when atrial contraction results in different hemodynamic considerations on the right and left sides, are complex. The aortic root motion (A wave) is related mostly to left atrial volume changes, whereas the pulmonary valve “a dip” is influenced both by instantaneous pressure differences between the pulmonary artery and the right ventricle in late diastole, as well as by the left atrial events. Future studies with acute interventions examining the effects of changes in PAPs on the pulmonary valve motion will further clarify the relative importance of these diverse factors.

References

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_Circulation_. 1978;58:1064-1071
doi: 10.1161/01.CIR.58.6.1064

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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:
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