Echocardiographic Evaluation of Pulmonary Hypertension

By NAVIN C. NANDA, M.D., RAYMOND GRAMIAK, M.D., TREVOR I. ROBINSON, M.D., AND PRAVIN M. SHAH, M.D.

SUMMARY

Echocardiographic studies of the pulmonary valve were obtained in 63 adults. Patients with normal pulmonary artery pressure (22 cases) showed an oblique position of the valve images in diastole, slow valve opening slopes (≤ 300 mm/sec), sizable (> 2 mm) posterior displacement of the cusp echoes with atrial systole (‘a’ dip), and a relatively short right ventricular pre-ejection period. In contrast, pulmonary valves in pulmonary hypertension (mean pressure > 20 mm Hg, 41 cases) appeared straight in diastole with rapid opening slopes (> 350 mm/sec) and prolonged pre-ejection periods (P < 0.001). In patients maintaining sinus rhythm (25 cases), mild to moderate pulmonary hypertension (mean pressure ≤ 40 mm Hg) was indicated by small ‘a’ dips (≤ 2 mm), while absence of ‘a’ dips denoted more severe pulmonary hypertension (mean pressure > 40 mm Hg) when uncomplicated by severe right heart failure. In the latter instance rapid valve opening slopes were associated with large ‘a’ dips. Echocardiography appears to be of value in the assessment of pulmonary hypertension.

Additional Indexing Words:
Ultrasound
Pulmonary stenosis
‘A’ dip Right ventricular pre-ejection period

THE DIAGNOSIS OF PULMONARY HYPERTENSION and evaluation of its severity represent a common challenging clinical problem which requires invasive techniques for accurate assessment. Ultrasound represents a noninvasive, non-traumatic, repeatable diagnostic technique whose role is well established in the evaluation of various conditions affecting the mitral valve,1,6 the aortic root,7,10 and the tricuspid valve.8,11 The recent development in our laboratory of a detection method for the pulmonic valve12 has allowed investigation of its function in normal subjects as well as in patients with pulmonary hypertension.13,14 The purpose of this report is to present our early experiences in the recognition of pulmonary hypertension and to describe criteria by which the severity of this condition can be estimated by echocardiographic examination of the pulmonary valve.

Material and Methods

The clinical material consisted of 63 adult patients in whom the pulmonic valve was recorded successfully by echocardiography 18–24 hr prior to cardiac catheterization. There were 38 males and 25 females. Their ages ranged from 16 to 66 years, the average being 42 years. Twenty-six patients had predominant mitral valve disease, 12 had ischemic heart disease, 11 had aortic valve disease, seven had congenital heart disease, six had cardiomyopathy, and one had Barlow’s syndrome. Patients with pulmonary stenosis were excluded from the study.

Standard techniques were employed to obtain right ventricular and pulmonary artery pressures during cardiac catheterization. Twenty-two patients had normal right-sided pressures (mean pulmonary artery pressure 20 mm Hg or less). All were in sinus rhythm. Forty-one patients had pulmonary hypertension, of whom 16 were in atrial fibrillation and the remainder in sinus rhythm. The diastolic gradient across the pulmonic valve was determined as the difference between the pulmonary artery diastolic pressure and the right ventricular end-diastolic pressure.

All ultrasonic examinations were carried out using a commercially available echograph (Picker) and a 2 MHz collimated transducer. Continuous records at high recording speed (equivalent to 125 mm/sec on a strip chart recorder) were made on 35 mm film by means of a Fairchild oscilloscope record camera and a dual beam oscilloscope operating as a slave.

In all patients pulmonary valve echocardiograms were obtained by a method previously described.12 The aortic root is identified either by angling the transducer medially and
Pulmonary valve echoes. An anterior and a posterior leaflet can occasionally be observed but usually only the posterior cusp is detected. Correlation with anatomic specimens indicates that the posteriorly located leaflet image is derived from the left pulmonary cusp.

A schematic representation of the pulmonary valve cusp image recorded through a cardiac cycle is shown in figure 2 and illustrates the following determinations which were made from the pulmonary valve echograms: 1) maximal opening rate in mm/sec. This was obtained by averaging measurements (to the nearest 50 mm/sec) made from a number of pulmonary valve recordings; 2) maximal magnitude (in mm) of the posterior motion of the valve cusp following atrial systole (‘a’ dip); 3) pre-ejection period of the right ventricle as determined from the beginning of the QRS complex of the electrocardiogram to the onset of the valve opening measured in msec and corrected for heart rate. This measurement was not made in patients with bundle-branch block (5 cases) or those in atrial fibrillation; 4) amplitude of the valve opening movement (in mm). In addition, the position of the valve cusp in diastole relative to the walls of the right ventricular outflow-pulmonary artery complex was studied (fig. 3).

Results

Patients with normal right-sided pressures showed an obliquely posterior position of the pulmonary cusp echo in diastole resulting in nonparallelism with the margins of the right ventricular outflow-pulmonary artery complex in all cases but one. The pulmonary valve opened relatively slowly, the opening velocity in all equalling 300 mm/sec or less. A transient posterior displacement of the cusp signal (‘a’ dip) was observed following atrial systole in all cases. This posterior deflection was large (3–12 mm, average 4.4 mm) in 21 of 22 patients in this group. The pre-ejection period of the right ventricle was relatively short (mean 85
The amplitude of the opening movement was 9 mm or less in 15 of the 22 pulmonary normotensives (table 1 and fig. 3).

On the other hand, patients with pulmonary hypertension showed a general straightening of the diastolic configuration of the pulmonary cusp image in 38 of 41 cases. The diastolic echo was observed to be parallel to the walls of the right ventricular outflow-pulmonary artery segment. The pulmonary valve in this group showed rapid opening movements in contrast to the normotensives. Opening slopes of 350 mm/sec or more were observed in 22 of 25 patients in sinus rhythm and 13 of 16 patients in atrial fibrillation (fig. 4). With pulmonary hypertension, the effect of atrial systole on the pulmonary valve motion was minimized or absent in those maintaining sinus rhythm. Shallow 'a' dips (2 mm or less) were associated with mildly elevated pressures (mean pulmonary artery pressure 40 mm Hg or less). Patients with more severe pulmonary hypertension (mean pressure over 40 mm Hg) showed total absence of cusp motion following atrial systole (table 2 and fig. 5). The only exceptions to this were two patients with severe pulmonary hypertension complicated by severe right heart failure (right ventricular end-diastolic pressure 18 mm Hg and 28 mm Hg) who showed sizable 'a' dips (3 mm and 8 mm). However, they could be differentiated from normotensives by their rapid valve opening slopes (400 mm/sec and 600 mm/sec) and straight diastatic cusp positions (fig. 6). 'A' dips were not observed in patients with atrial fibrillation. As a rule, the opening movement of the pulmonary valve occurred late with pulmonary hypertension resulting in an increased right ventricular pressure. The correlation coefficient (r value) is 0.74. The closed circles represent patients with normal pulmonary artery pressure (mean pulmonary artery pressure <20 mm Hg), while the open circles denote pulmonary hypertensives.}

### Table 1

| Pulmonary Valve Echocardiographic Findings in Pulmonary Normotensive and Hypertensive Patients |
|---|---|---|---|---|
| Mean pulmonary artery pressure | Diastolic position | Opening slope mm/sec (mean) | 'A' dip mm (mean) (NSR only) | PEP/√R-R-time msec (mean) (NSR only) | Opening amplitude mm (mean) |
| ≤20 mm Hg (22 patients) | Oblique | 211 | 4.4 | 85.07 | 8.68 |
| >20 mm Hg (41 patients) | Straight | 420 | 0.92 | 110.3 | 11.15 |
| Level of significance of t value* | P < 0.001 | P < 0.001 | P < 0.001 | P < 0.05 |

Abbreviations: NSR = normal sinus rhythm; PEP = right ventricular pre-ejection period; √R-R-time = square root of the interval (in sec) between two consecutive QRS complexes of the electrocardiogram; se = standard error.

*Derived using unpaired t-test with unequal number of observations.

### Table 2

<table>
<thead>
<tr>
<th>Mean Pulmonary Artery Pressure (mm Hg)</th>
<th>&gt; 2 mm</th>
<th>≤2 mm</th>
<th>Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤20 (22 pts)</td>
<td>21</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>21–40 (10 pts)</td>
<td>1</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>&gt;40 (15 pts)</td>
<td>2*</td>
<td>0</td>
<td>13</td>
</tr>
</tbody>
</table>

*Severe right heart failure.
tricular pre-ejection period. The amplitude of the opening movement of the pulmonary valve was generally greater in this group when compared to the normal pressure category, being more than 9 mm in 29 of 41 cases.

The diastolic gradient across the pulmonary valve showed correlation with the magnitude of the 'a' dip. With low gradients of 15 mm or less, 'a' dips were regularly noted while higher gradients (over 20 mm) resulted in complete absence of any deflection attributable to atrial systole on the pulmonary valve image.

Statistical comparison of the pulmonary valve opening slopes, the magnitude of the 'a' dip and the pre-ejection period of the right ventricle in the pulmonary normotensive and hypertensive groups revealed a highly significant difference ($P < 0.001$). Less significant ($P < 0.05$) was the comparison of the amplitudes of the opening movements which, standing alone, failed to separate the pulmonary normotensives from those with hypertension.

**Discussion**

Detection of the pulmonary valve using ultrasound is a relatively new technique. A previous study from this laboratory described the detection method and obtained anatomic validation of cusp echoes using indocyanine green injection during cardiac catheteriza-
Pulmonary valve detection has already proved useful in the identification of patients with dextro-transposition of the great vessels and in the differential diagnosis of truncus arteriosus.

The initial over-all detection rate of the pulmonic valve was approximately 35%. With additional experience this has improved and a recent review shows that it is now possible to obtain satisfactory echograms of the pulmonary valve in about 55% of the adults referred to our service. Detection rate in patients with pulmonary hypertension is even higher (70%), probably a result of pulmonary artery dilatation and possibly a more vertical orientation to the ultrasonic beam (fig. 1). Although it is difficult to obtain complete tracings throughout the cardiac cycle, it is relatively easy to record the mid and late diastolic positions together with the opening movements of the pulmonary valve. Clinically useful echograms can thus be obtained in a high proportion of patients.

The 'a' dip of the pulmonary valve echogram represents a movement of the cusp towards the open position and has been noted to follow the P wave of the electrocardiogram in normal patients as well as in those with various degrees of atioventricular block. Its disappearance in the presence of atrial fibrillation further identifies it as a response to the low pressure events in the right ventricle associated with atrial systole. A non-tense pulmonic valve cusp, therefore, can respond to this stimulus. On the other hand, when the valve cusp is tense, as occurs in pulmonary hypertension, deflection due to atrial systole will be reduced or eliminated depending on the degree of hypertension. Furthermore, abnormally elevated right ventricular pressures, as were recorded in two of our patients, can result in the production of 'a' dips despite the presence of significant pulmonary hypertension. Thus, it is clear that the diastolic gradient across the pulmonary valve is the single most significant parameter which governs the generation of the 'a' dip. Transient decrease of this gradient can occur with normal pulmonary artery pressures during peak inspiration and produces very large 'a' dips which move the valve to a position close to that seen during opening. This is also a well-documented phenomenon in pulmonic stenosis and produces a similar echocardiographic pattern (fig. 7). Recently, echocardiographic assessment of the effect of atrial systole on the pulmonary valve motion in diastole has been found helpful in the clinical evaluation of pulmonic stenosis.

In pulmonary hypertensives, the increased pulmonary artery pressure results in pulmonary valve opening in a relatively high portion of the right ventricular pressure curve where the pressure is rising.

![Figure 7](image-url)

*Figure 7*

*Pulmonary valve opening with atrial systole. In both illustrations the pulmonary valve makes a strong movement toward opening following atrial systole and before the onset of the QRS complex. The cusp rebounds toward closure and then reopens with ventricular systole. Left panel. This was obtained from a patient with valvular pulmonary stenosis (gradient 40 mm Hg). Right panel. This was obtained from a pulmonary normotensive subject (mean pulmonary artery pressure 20 mm Hg) and at peak inspiration. PV = pulmonary valve echo tracing; PHONO = phonocardiogram; ECG = electrocardiogram.*
rapidly and this may explain the accelerated opening rate of the pulmonic valve observed in this group. Rapid valve opening slopes were also found in the two patients with severe right ventricular failure and pulmonary hypertension, providing a diagnostic clue to the nature of the underlying problem.

For similar hemodynamic reasons, the isovolumic contraction time of the right ventricle is increased with pulmonary hypertension and explains the delay in the onset of the opening movement of the pulmonary valve observed in this group in the present study.

An upwardly oblique direction of the ultrasonic beam is required for recording movements of the pulmonary valve because of the proximity of lung tissue. This angling technique views the right ventricular outflow-pulmonary artery junction obliquely and examines the left cusp of the pulmonic valve from its right ventricular surface (fig. 1). Thus, upward or opening movements of the pulmonic valve are recorded as posterior deflections. Those which follow atrial and ventricular systole produce transient, circumscribed displacements of the cusp signal which can be correlated with a simultaneously recorded electrocardiogram. The generalized posterior diastolic movement of the echo trace, characteristic of normotensive subjects, is more difficult to explain. It is possible that the pulmonic valve is displaced upward as the right ventricle fills during diastole and results in a diastolic echogram which traces an obliquely posterior path. In the present study, three patients with large atrial septal defects, who had roentgenologic evidence of pulmonary artery enlargement but normal mean pulmonary artery pressures, did not show straightening of the pulmonic valve images in early and mid-diastole. Thus, the absence of posterior movement of the pulmonary valve prior to atrial systole, observed with pulmonary hypertension, cannot be adequately explained on the basis of dilatation of the pulmonary artery alone.

The most useful information regarding the severity of pulmonary hypertension is derived from the pulmonary valve echogram when both the ‘a’ dip and the pulmonary valve opening slope are taken into account. Absence of pulmonary hypertension is indicated by the presence of a large ‘a’ dip and a slow opening slope of the pulmonary valve. A rapid opening slope generally indicates pulmonary hypertension. Associated persistence of minimal posterior displacement of the cusp echo during atrial systole is consistent with mild to moderate elevation of pulmonary artery pressure (mean pressure 40 mm Hg or less), while absence of any influence of atrial systole on the pulmonic cusp motion indicates a more severe degree of pulmonary hypertension (mean pressure > 40 mm Hg). A large ‘a’ dip in the presence of a rapid valve opening slope may indicate co-existent severe right heart failure. With atrial fibrillation it is less easy to assess the degree of pulmonary hypertension since these patients do not show ‘a’ dips and reliance has to be placed on other criteria, chiefly the valve opening slope determinations.

Other criteria are also of value in detecting pulmonary hypertension, but are less useful in the assessment of its severity. Determination of the right ventricular pre-ejection period is of limited value. It requires correction for heart rate and is not useful in patients with intraventricular conduction defects. The diastolic position of the pulmonary valve is a qualitative determination which, although very useful in identifying pulmonary hypertensives, does not help in the evaluation of its severity. The amplitude of valve opening movement is the least useful criterion in separating pulmonary normotensives from hypertensives, as there is a considerable overlap of values in the two groups.

Recognition and quantitation of the severity of pulmonary hypertension by a noninvasive, nonionizing technique like echocardiography has potential advantages. It can be used to carry out repeated examinations without any side-effects, as well as follow stages in the development of pulmonary hypertension complicating certain disease processes. Also, it would make it feasible to undertake longitudinal studies to evaluate the long-term effects of surgery where reduction of pulmonary hypertension is a desired goal.

The present study indicates that ultrasound is a useful tool in the diagnosis and evaluation of pulmonary hypertension. In addition to differentiating patients with elevated pulmonary artery pressures from those with normal right heart pressures, an estimate of the severity of pulmonary hypertension is obtained. Use of this technique in the evaluation of pulmonary hypertension represents another extension of the capability of echocardiography.

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References


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