Echocardiographic Patterns of Pulmonic Valve Motion with Pulmonary Hypertension

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SUMMARY

Echocardiographic tracings of the pulmonic valve were examined in two groups of patients. The first group contained 24 normal patients. The second group consisted of 32 patients with pulmonary hypertension (mean pulmonary artery pressure ≥20 mm Hg). Parameters considered included presence or absence and depth of the "a" wave, amplitude of valve opening (b-c separation), diastolic (e-f) slope, and presence or absence of mid-systolic closure or fluttering of the pulmonic leaflet. An "a" wave was present in all 24 normal subjects. The "a" wave varied with respiration and the maximum "a" wave depth (A Max) averaged 3.7 ± 1.2 mm (mean ± so, range 2-7 mm). A Max was ≥ 2 mm in all 24 normal patients. In 19 of 24 patients with pulmonary hypertension and sinus rhythm no "a" wave was present. In the other five patients small "a" waves (≤2 mm) occurred. In four of these five patients, right heart failure was present. The diastolic (e-f) slope averaged 36.9 ± 25.4 mm/sec (range 6-115 mm/sec) in normals. With pulmonary hypertension, this slope was significantly less than normal (average 5.2 ± 8.9 mm/sec, range -9 to +30 mm/sec, \( P < .001 \)). In six patients with pulmonary hypertension a negative e-f slope was seen; this never occurred in normals. Mid-systolic closure or notching of the systolic (c-d) segment occurred in 18 of 20 subjects with pulmonary hypertension in whom the leaflet was clearly recorded in mid-systole. This finding was not observed in normals. Likewise, mid-systolic fluttering was present in 22 of 24 patients with pulmonary hypertension. There was no difference in amplitude of leaflet opening between the two groups.

Additional Indexing Words:
Ultrasound Echocardiography

ECHOCARDIOGRAPHY has proven to be particularly valuable in the determination of cardiac chamber size and structural abnormalities. While intracardiac pressures cannot be appreciated directly by reflected ultrasound, changes in intracardiac pressure have been noted to alter the motion of the cardiac valves. Such observations have primarily involved alterations in left heart pressures and mitral valve echo motion. These changes include the decrease in the anterior mitral leaflet diastolic slope seen in patients with a pressure gradient across the mitral valve, such as with mitral stenosis\(^1\)\(^2\) or an atrial tumor.\(^3\) In addition abnormal mitral valve closure has been observed in the presence of an elevated left ventricular end diastolic pressure secondary to a high atrial contribution to the ventricular pressure.\(^4\) A decreased rate of mitral valve opening has also been noted with an elevated initial left ventricular diastolic pressure.\(^4\)

There have been preliminary observations that the echocardiographic recording of the pulmonic valve may reflect changes in pulmonary artery pressure.\(^5\)\(^6\) Since we have recently been interested in examining the pulmonic valve for the diagnosis of pulmonic stenosis\(^7\) and have noted that the valve could be recorded with greater frequency than previously anticipated, we decided to examine the pulmonic valve to see if we also could find a relationship between the pattern of pulmonic valve motion and the pressure in the pulmonary artery. This study presents the results of this effort.

Material and Methods

Echocardiographic tracings of the pulmonic valve were examined in two groups of patients. The first group contained 24 normal subjects. There were 18 males and six females, ranging in age from 15-55 years (average 21 years). Catheterization was not performed on the normal subjects.

The second group contained 32 patients with pulmonary hypertension proven by cardiac catheterization. There were 18 females and 14 males, ranging in age from 17-67 years.
(average 40 years). The mean pulmonary artery pressures in this group ranged from 20-85 mm Hg. There were four patients with mean pulmonary artery pressures in the 20-29 mm Hg range, nine in the 30-39 mm Hg range; eleven between 40-49 mm Hg; and two each in the 50-59, 60-69, 70-79 and 80-89 mm Hg ranges. In each of these cases, pulmonary hypertension was the result of heart disease. Valvular heart disease was the cause in 21 cases. Five patients had elevated pulmonary artery pressures secondary to large left-to-right shunts. There was one ventricular septal defect, one atrial septal defect, one ostium primum defect with both atrial and ventricular septal defects, one case with anomalous pulmonary venous return, and one patient with a patent ductus arteriosus. There were three cases of primary myopathies, one case of constrictive pericarditis, one case of discrete subvalvular aortic stenosis, and one case of coronary artery disease with left ventricular failure. Atrial fibrillation was present in eight of the patients with pulmonary hypertension. Complete right bundle branch block was present in four patients, incomplete right bundle branch block in two and left bundle branch block in one.

The echocardiographic examination was performed with an Ekoline 20A Echograph combined with either an Electronics for Medicine recorder or a Honeywell 1856 Fiberoptic Strip Chart recorder. A 2.25 MHz transducer with either a 0.5 or 0.25 inch outer diameter, focused at 7.5 cm was used. All patients were examined in the recumbent position with the transducer placed in the second, third or fourth intercostal space along the left sternal border. When looking for the pulmonic valve echo, the transducer generally was placed at least one interspace higher than that used for recording the mitral valve. Two standard techniques for examining the pulmonic valve have previously been described. The first approach described by Gramiak et al. is to locate the aortic valve, then either by angling the transducer cephalad or by moving the transducer one interspace higher, the supra valvular portion of the aorta is located. From this high, left parasternal position the beam is angled in a lateral and cephalad direction. As the image of the aortic wall disappears, the pulmonary artery becomes visible as an echo-free space anterior to the aorta with its anterior border within 1-2 cm of the lower margin of the chest. The posterior margin of the pulmonary artery is characteristically a wide band of echoes which may lie 2-4 cm behind the anterior margin of the artery. Pulmonary valve cusps appear as thin lines which move within the margins of the pulmonary artery. The second approach is to locate the mitral valve, then tilt the transducer superiorly and slightly toward the throat, but not enough to record the aorta. As the ultrasonic beam leaves the left ventricular cavity a dense mass of echoes will be recorded from the region of the right ventricular outflow tract and the potential space between the pulmonary artery and the left atrium. Continuing cephalad the echo-free pulmonary artery again will appear 1-2 cm below the anterior chest wall. The valve leaflets are then located by gentle scanning within the pulmonary artery. During this study we noted yet a third way of finding the pulmonic valve. In patients in whom a pulmonary artery impulse could be palpated along the left sternal border, placing the transducer directly over this impulse resulted in ready detection of the pulmonic valve.

All tracings were initially recorded at a paper speed of 25 mm/sec, which is the usual recording speed in our laboratory. In addition 20 tracings were also recorded at 50 mm/sec and 10 others at 100 mm/sec. In each case a minimum of ten pulmonic valve complexes were examined. Whenever possible these were consecutive complexes. Respiration was timed by a second independent observer and the tracing marked for the duration of inspiration.

Cardiac catheterization was performed in the standard manner. Echocardiograms were routinely recorded within 48 hr of cardiac catheterization.

Due to the position of the pulmonic valve in the chest and its plane of motion relative to the ultrasonic beam, the echocardiogram usually records only one posterior pulmonic leaflet. Figure 1A illustrates a normal posterior pulmonic leaflet recorded throughout several cardiac cycles. The lettering is similar to that previously described. The "a" wave which follows the P wave of the electrocardiogram reflects the effect of atrial contraction on the pulmonic leaflet. That this wave is due to atrial contraction has been confirmed by its constant relationship to the P wave in complete heart block and its disappearance with atrial fibrillation. From point b, which represents the position of the valve at the onset of ventricular ejection, the leaflet rapidly moves to a fully open position (point c). During systole there is a gradual anterior movement of the leaflet (c-d) followed by rapid diastolic closure of the valve (d-e). During diastole, the leaflet moves gradually posteriorly to point f which precedes the onset of atrial systole. The anterior systolic c-d and posterior diastolic e-f slopes of the leaflet probably represent pulsatile movement of the pulmonary artery and valve apparatus. In figure 1A there is further anterior motion from point e to e'. This is a variable finding and may represent transmitted pulsations from the aorta. While in figure 1A the leaflet is recorded throughout the cardiac cycle, figure 1B represents the more characteristic normal recording. In this tracing, the e-f slope, "a" wave and b point are clearly seen. However, as the leaflet approaches the fully open position (point c) it is lost in the dense mass of echoes recorded from behind the pulmonary artery. In these cases, point c along with the c-d slope and diastolic closure (d-e) are not appreciated.

Figure 2 diagrammatically illustrates the parameters of leaflet motion that were considered in this study. The first diagram (2A) indicates the standard measurement of "a" wave depth and amplitude of leaflet opening (b-c). Since the "a" wave depth normally increases with inspiration and decreases with expiration, the measurement taken was the maximum depth recorded during the inspiratory phase of quiet respiration or A Max. The second diagram (2B) illustrates the e-f slope measured from the most anterior position of the leaflet at the beginning of diastole (e) to the position of the leaflet at the onset of atrial contraction (f). Frequently in normal subjects, at slow heart rates (< 70/min) the e-f slope will end before atrial contraction occurs. In these cases a short, usually flat segment is interposed between point f and the onset of the "a" wave. The length of this segment is directly related to the cycle length. In this situation, the initial rapid slope was measured.

We initially attempted to measure a right ventricular pre-ejection period (PEP) between the onset of the Q wave of the electrocardiogram to the b point of the pulmonic valve echogram and the rate of valve opening or b-c slope of the pulmonic valve. Unfortunately, most of our pulmonary valve echoes were recorded at less than 100 mm/sec, and it became obvious that these measurements were inaccurate at these paper speeds. Both of these measurements were noted to be useful in a previous preliminary study which examined the pulmonic valve motion in patients with pulmonary hypertension. We did not record more patients.
at 100 mm/sec because we found technical difficulties in recording satisfactory pulmonic valve echograms at such rapid paper speed. This problem was more frequent in the normal control subjects rather than in those with pulmonary hypertension. The pulmonic valve echoes were frequently incomplete and when spread out it was usually difficult to connect the series of dots and broken lines. This problem was particularly troublesome when analyzing the rapid, terminal segment of the b-c slope. Therefore we could not use the right ventricular PEP and the b-c slope measurements. As a result, this study primarily represents an attempt to see whether a pulmonic valve recorded at the usual 25 or 50 mm/sec speeds could provide information concerning pulmonary hypertension.

Results

``a`` Wave

A clearly discernible ``a`` wave was present in each of the 24 normal subjects. In these patients A Max (the maximum depth of the ``a`` wave recorded during quiet inspiration) averaged 3.7 ± 1.2 mm (range 2-7 mm). Sinus rhythm was present in 24 of the 32 patients with pulmonary hypertension. In these 24 patients A Max averaged 0.4 ± 0.8 mm (range 0-2 mm). No ``a`` wave was recorded in 19 of these 24 patients; ``a`` waves were present in the other five but were small (≤ 2 mm). In no patient with pulmonary hypertension was an ``a`` wave over 2 mm recorded, whereas A Max was ≥ 2 mm in each of the 24 normal patients (P < .001).

Leaflet Opening

The amplitude of leaflet opening averaged 13.9 ± 1.8 mm in normal subjects. In patients with pulmonary hypertension leaflet opening averaged 14.4 ± 3.0 mm. Thus there was no significant difference between the means of the two groups.

E-F Slope

The e-f slope in 24 normal subjects averaged 36.9 ± 25.4 mm/sec (range 6-115 mm/sec). In the 32 patients with pulmonary hypertension this slope averaged 5.2 ± 8.9 mm/sec (range −9 to +30 mm/sec, P < .001). In six patients with pulmonary hypertension a negative e-f slope occurred which was never seen in the normal subjects.

Fluttering and Mid-Systolic Closure

Normally when the leaflet is in the fully open position during systole (c-d), it is lost in the dense mass of echoes behind the pulmonary artery. When it is recorded in normal subjects, a straight c-d pattern followed by diastolic closure, d-e, is seen. In patients with pulmonary hypertension, we have noted systolic fluttering and mid-systolic closure of this c-d segment as illustrated in figures 3 and 4. Fluttering was present in 22 of 24 patients with pulmonary hypertension. In the other eight patients, the c-d portion of the cycle was not clearly recorded. Likewise, mid-systolic
closure or notching of the leaflet was present in 18 of 20 subjects in whom the leaflet could be seen clearly enough in mid-systole to determine its presence or absence. Both mid-systolic closure and fluttering were clearly observed in all six patients with mean pulmonary artery pressures of > 60 mm Hg. Very fine fluttering of the c-d slope was seen in two normal subjects, but mid-systolic closure was never observed.

Figure 3 illustrates the absence of the "a" wave, negative e-f slope and mid-systolic notching that are characteristic of pulmonary hypertension. Figure 4 is a recording from a patient with atrial fibrillation and pulmonary hypertension. In this case, although no "a" wave is present, the diagnosis is apparent from the flat e-f slope (negative with shorter cycles) and mid-systolic notching and fluttering.

Figure 5 illustrates both pulmonic leaflets in another patient with pulmonary hypertension. Again, the absence of an "a" wave, together with a flat e-f slope and mid-systolic notching and fluttering are characteristic.

Discussion

From this study, we conclude that the presence or absence and the amplitude of the "a" wave, magnitude of the e-f slope and presence of mid-systolic closure or notching and fluttering of the posterior pulmonic leaflet appear to be of value in the diagnosis of pulmonary hypertension. There was no difference between the two groups in the magnitude of leaflet opening. These findings with regard to "a" wave magnitude and e-f slope are consistent with the observations previously reported. The presence or absence of an "a" wave appears to be a reliable first step in the echocardiographic diagnosis of pulmonary hypertension. The "a" wave has been shown to possibly reflect the relative pressures existent across the pulmonic valve at the time of atrial systole. In moderate and severe pulmonic stenosis, when the increased force of right atrial contraction and elevated right ventricular end diastolic pressure, together with a normal or low pulmonary artery pressure, produce a positive gradient across the valve, a marked increase

attachment
Tracing from a patient with pulmonary hypertension and atrial fibrillation. In this case the “a” wave is absent due to atrial fibrillation. However, the flat e-f slope (negative with shorter cycles) and mid-systolic notching and fluttering are consistent with pulmonary hypertension.

Both anterior (APV) and posterior (PPV) leaflets from another patient with pulmonary hypertension. Again, the flat e-f slope, absence of the “a” wave and mid-systolic notching and fluttering are apparent.

in “a” wave depth has been observed. In normal patients, there is a small difference between the pulmonary artery end-diastolic and right ventricular end-diastolic pressures at the peak of atrial contraction. In this case the change in pressure produced by atrial systole results in some leaflet motion. With pulmonary hypertension, the pulmonary artery end-diastolic pressure is usually well above the simultaneous right ventricular end-diastolic pressure and an absent or markedly damped “a” wave would be expected.

A clearly discernible “a” wave was present in all 24 normal subjects. A Max averaged 3.7 mm (range 2-7 mm). In 19 of 24 patients with sinus rhythm and pulmonary hypertension no “a” wave was present. In the remaining five patients in whom an “a” wave was present, it was small (≤ 2 mm). In four of these five patients, right ventricular failure with elevated right ventricular end-diastolic and right atrial pressure was present. It is possible that with failure, the elevation in right ventricular end-diastolic pressure may narrow the gradient across the valve and allow for valve motion. However, in five other patients with right ventricular failure and similar gradients, no “a” wave was recorded. When atrial fibrillation is present, as is commonly the case, no “a” wave will occur and other criteria for pulmonary hypertension must be sought.
The e-f slope probably represents diastolic motion of the pulmonary artery. All right-sided slopes are subject to respiratory and cycle length variation. Therefore, numerical values represent averages of a number of complexes or measurement of representative complexes. In either case, the numbers are not precise, and we prefer to consider the e-f slope in terms of negative (e point below the f point), flat (0-20 mm/sec), normal (20-70 mm/sec), or steep (> 70 mm/sec). In pulmonary hypertension, the e-f slope tends to be flat. The average e-f slope in these patients was 5.2 mm/sec, compared to an average of 36.9 mm/sec in normal patients. While a flat e-f slope is generally seen in pulmonary hypertension, there is enough overlap with normal patients to limit the usefulness of this measurement by itself.

On the other hand, a negative e-f slope, recorded in six patients with pulmonary hypertension, was never seen in normals and appears by itself to indicate pulmonary hypertension. An e-f slope of > 20 mm/sec was seen in only four patients with pulmonary hypertension; in two of these cases increased pulmonary flow secondary to left-to-right shunting was present. Therefore, when a normal slope occurs in the presence of pulmonary hypertension, it may suggest the possibility of increased pulmonic flow.

Mid-systolic fluttering and closure or notching of the valve were seen in all but two cases of pulmonary hypertension in which the leaflet could be seen clearly enough during systole to determine their presence or absence. In normals, the leaflet is usually not recorded during systole. When we were able to record this segment it was straight; and although fine fluttering was seen in two patients, coarse fluttering and notching were never seen. It is not clear why this segment is recorded well in pulmonary hypertension and rarely seen in normals. One possibility is that decreased pulmonary blood flow in patients with pulmonary hypertension might allow for early leaflet closure. This same phenomenon, however, was seen in patients with left-to-right shunts and increased pulmonic flow. Another possibility is that pulmonary artery dilatation which usually accompanies pulmonary hypertension permits the fully open leaflet to remain in the turbulent stream of blood rather than against the arterial wall as in normals. This possible explanation is supported by the fact that the degree of leaflet opening is the same in both groups.

It has been suggested that normal patients could be differentiated from those with pulmonary hypertension by the rate of valve opening or b-c slope. At our standard recording speeds of 25 and 50 mm/sec we were unable to confirm these findings. It is very probable that these recording speeds were too slow to permit accurate measurement of these slopes. However, in a small group recorded at 100 mm/sec we were still unable to document any separation of these two groups using this measurement. Another possible reason for the difference in results may be the fact that in normals the leaflet does not open at a constant rate: there is an obvious acceleration as leaflet opening progresses. The initial or slow portion of this slope is usually well recorded, whereas the later or rapid portion is frequently lost in the dense band of echoes recorded from behind the pulmonary artery. If one were to measure the initial or slow slope in normals and the more prominent rapid plane in pulmonary hypertension, then an obvious difference would occur. In this case, however, the amplitude of valve opening in the normal group would appear decreased since only a portion of the opening slope would be appreciated.

A noninvasive assessment of pulmonary artery pressure would be of obvious clinical value. This study confirms the previous observation that the echocardiographic recording of pulmonic valve motion may be a useful indicator of pulmonary hypertension.

References


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