Pulmonary valve echo motion in pulmonary regurgitation

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Four cases are presented to illustrate the echo patterns of pulmonary valve motion in patients with pulmonary regurgitation caused by pulmonary hypertension, idiopathic dilatation of the pulmonary artery, or congenital absence of the pulmonary valve or in association with pulmonary stenosis. Absence of the pulmonary ‘a’ wave, fluttering of the e-f slope, and mid systolic closure or ‘notching’ of the valve were noted with pulmonary hypertension. In the case with idiopathic dilatation of the pulmonary artery a normal echo pattern of pulmonary valve motion along with distinct dilatation of the pulmonary artery at the valvular level were present. Pronounced dilatation and systolic expansion of the pulmonary artery along with dilatation of the right ventricle were seen with congenital absence of the pulmonary valve. No pulmonary valve could be demonstrated on multiple scans from the right ventricle to pulmonary artery. In Case 4 large ‘a’ waves (14 mm) were noted, indicating a reversal of the normal end-diastolic gradient across the valve and suggesting that pulmonary regurgitation in this case was associated with right ventricular outflow obstruction. Study of the echo pattern of pulmonary valve motion may therefore provide useful information in establishing the cause of pulmonary regurgitation.

A number of recent reports have shown the clinical value of recording the echo pattern of pulmonary valve motion. Characteristic patterns of motion have been described in patients with pulmonary valvular stenosis (Weyman et al., 1975b), pulmonary hypertension (Nanda et al., 1974; Weyman et al., 1974b; Sakamoto et al., 1974), pulmonary infundibular stenosis (Weyman et al., 1975b), and with aorta to right atrial fistula secondary to rupture of a sinus of Valsalva aneurysm (Weyman et al., 1975a). The recording and localization of the pulmonary valve has also proved valuable in differentiating truncus arteriosus from tetralogy of Fallot (Chung et al., 1973) and in recognizing patients with transposition of the great vessels (Dillon et al., 1973).

Echocardiographic diagnosis of pulmonary regurgitation is based on evidence of right ventricular dilatation, paradoxical motion of the interventricular septum, and fluttering of the anterior tricuspid leaflet (Feigenbaum and Chang, 1972). But these findings, though helpful in determining the presence of pulmonary regurgitation, give no information as to its cause. Pulmonary regurgitation is most commonly caused by a raised pulmonary artery pressure resulting in dilatation of the pulmonary valve ring and regurgitation of the valve (Hurst, 1974). It may also occur as a congenital anomaly caused either by total absence or atresia of pulmonary valve tissue or by idiopathic dilatation of the pulmonary valve ring. In addition, pulmonary regurgitation may coexist with pulmonary valvular stenosis as a primary lesion or after pulmonary valvulotomy (Perloff, 1970). In this report we describe the echo pattern of pulmonary valve motion in four selected cases of pulmonary regurgitation. They illustrate the pattern of pulmonary valve motion in pulmonary regurgitation secondary to severe pulmonary hypertension, idiopathic dilatation of the pulmonary artery, congenital absence or atresia of the pulmonary valve, and pulmonary regurgitation in association with pulmonary stenosis. In each case the echo pattern of pulmonary valve motion was valuable in determining the etiology of the pulmonary regurgitation.

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Patients and methods

Echocardiographic tracings of the pulmonary valve were examined in four patients with pulmonary regurgitation who were chosen to represent the different clinical situations in which pulmonary regurgitation commonly occurs.

Case 1 This was a 31-year-old woman with severe pulmonary hypertension secondary to a persistent ductus arteriosus. Her pulmonary artery pressure was 120/54 mmHg, mean 84 mmHg (16/7.2, mean 11.2 kPa). A 2.8:1 pulmonary to systemic blood flow ratio with a 15.9 l/min pulmonary blood flow was present.

Case 2 This was a 30-year-old asymptomatic man who was referred for evaluation of a heart murmur noted during a routine examination. At catheterization the pulmonary artery pressure was 21/4 mmHg (2.8/0.5 kPa). Moderate pulmonary regurgitation along with pronounced dilatation of the pulmonary artery was noted.

Case 3 This was a 29-year-old man in whom pulmonary regurgitation was noted during infancy and was attributed to congenital absence of the pulmonary valve. There has been gradual dilatation of the pulmonary artery and right ventricle. Physical examination was consistent with massive pulmonary regurgitation.

Case 4 This was a 16-year-old boy with tetralogy of Fallot. When aged 3 he had a Blalock-Taussig anastomosis because of increasing cyanosis and tachypnoea. When aged 12 cardiac catheterization showed a large ventricular septal defect, right ventricular pressures at systemic levels (116/6 mmHg, 15.4/0.8 kPa; LV 116/8 mmHg, 15.4/1.1 kPa), overriding of the aorta, and both valvular and infundibular pulmonary stenosis. The pulmonary artery pressure was 16/6 mmHg (2.1/0.8 kPa). There was an 80-mmHg (10.6 kPa) gradient between the right ventricular and infundibular chamber and a 20-mmHg (2.7 kPa) gradient between the infundibular chamber and pulmonary artery. At the age of 15 total correction of the tetralogy of Fallot was performed. Pulmonary regurgitation was present postoperatively.

The echocardiographic examinations were made with an Ekoline 20A echograph combined with either a Honeywell 1856 Strip Chart recorder or an Electronics for Medicine (Model DR-8) recorder. A 2.25 MHz transducer focussed at 7.5 cm was used in each case. The technique for examining the pulmonary valve was similar to that previously described (Weyman et al., 1974a; Gramiak, Nanda, and Shah, 1972).

Results

Echoes from normal pulmonary valve

A normal posterior pulmonary leaflet recorded

FIG. 1 (A) Normal posterior pulmonary leaflet recorded throughout several cardiac cycles. The 'a' wave which follows the P wave of the electrocardiogram reflects the effect of atrial contraction on the pulmonary leaflet. Point b = position of leaflet at onset of ventricular ejection; b-c = rapid systolic opening of leaflet; c-d = leaflet in open position during systole; d-e = diastolic closure; e-f = leaflets in closed position during diastole; e-e' is a variable finding and probably represents transmitted pulsation from aorta. (B) More characteristic normal recording. The e-f segment, 'a' wave, and b point are clearly seen. However, as leaflet approaches fully open position (point c) it is lost in dense mass of echoes recorded from behind pulmonary artery. In these cases point c along with the c-d segment and diastolic closure (d-e) are not appreciated.
throughout several cardiac cycles is shown in Fig. 1. Owing to the position of the pulmonary valve in the chest and its plane of motion relative to the ultrasonic beam the echocardiogram usually records only one posterior leaflet. The lettering in this figure is similar to that previously described (Weyman et al., 1974a). The ‘a’ wave which follows the P wave of the electrocardiogram reflects the effect of atrial contraction on the pulmonary valve leaflet. That this wave is caused by atrial contraction has been confirmed by its constant relation to the P wave in complete heart block and its disappearance with atrial fibrillation. In normal patients we have found the ‘a’ wave depth recorded during quiet inspiration (‘A’ max.) to be 3.7 ± 1.2 mm (range 2–7 mm) (Weyman et al., 1974b).

After atrial contraction the leaflet normally returns to a baseline or closed position (point b) before the onset of ventricular ejection. From point b (the position of the valve at the onset of ventricular ejection) the leaflet moves rapidly 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 posterior to point f which precedes the onset of atrial systole. The anterior systolic (c–d) and posterior diastolic (c–f) slopes of the leaflet probably represent pulsatile movement of the pulmonary artery and valve apparatus. Fig. 1A shows further anterior motion from point e–e'. This is a variable finding and may represent transmitted pulsations from the aorta. While in Fig. 1A the leaflet is recorded throughout the cardiac cycle Fig. 1B represents a 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.

**Echoes from abnormal pulmonary valves**

Though in each of the four cases of pulmonary regurgitation the presence of pulmonary regurgitation was suspected or established by other means the echo study of pulmonary valve motion provided valuable confirmation of the cause of the valvular insufficiency.

In Case 1 the echo pattern of the pulmonary valve motion revealed the following (Fig. 2): Absence of any deflection of the posterior pulmonary leaflets after atrial systole (‘a’ wave), flattening of the c–f slope (8 mm/s versus normal mean of 37 mm/s) (Weyman et al., 1974b) and midsystolic closure or ‘notching’ and fluttering of the valve.

In Case 2 the echo pattern of pulmonary valve motion appeared normal (Fig. 3). The pulmonary artery at the level of the valve, however, was very dilated (4.2 cm): this compared with the aortic diameter of 3.1 cm. Fig. 4 is a two-dimensional sector scan showing the relative size of the aorta and pulmonary artery at the level of the pulmonary valve in this case. The presence of normal pulmonary valve echo motion along with distinct dilatation of the pulmonary artery at the valvular level suggested that the pulmonary regurgitation was caused by idiopathic dilatation of the pulmonary artery.

In Case 3 obvious fluttering of the anterior tricuspid leaflet was present, indicating pulmonary regurgitation (Fig. 5). There was pronounced dilatation of the right ventricle and vigorous right ventricular contraction (Fig. 5, left). In addition, the pulmonary artery was massively dilated and very pulsatile (Fig. 5, right). No pulmonary valve could be demonstrated in multiple scans through the right ventricular outflow tract into the pulmonary artery. There was a suggestion of some rudimentary pulmonary valve tissue at the level where a normal pulmonary valve would be expected.

In Case 4 pulmonary regurgitation followed pulmonary valvulotomy. The presence of pulmonary

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**FIG. 2** Pulmonary valve echogram from Case 1 with pulmonary regurgitation secondary to severe pulmonary hypertension. The normal posterior deflection of pulmonic leaflet after atrial systole (‘a’ wave) is absent. There is relative flattening of diastolic slope and also partial midsystolic closure of leaflet (n) is present. PA=pulmonary artery; c=fully open systolic portion of valve; d=position of valve at the onset of diastolic closure.
Pulmonary valve echo motion in pulmonary regurgitation

FIG. 3 Pulmonary valve echogram from Case 2. Normal echo pattern of pulmonary valve motion but pulmonary artery is dilated (4.2 cm) at valvular level.

FIG. 4 Cross-sectional echogram from Case 2 showing relative size of aorta and pulmonary artery. This is a short axis recording with plane of transducer sweep oriented perpendicular to long axis of aorta. \( \text{AO} \) = aorta; \( \text{PA} \) = pulmonary artery; \( \text{PV} \) = pulmonary valve.

regurgitation was again established by the pattern of right ventricular volume overload and fluttering of the anterior tricuspid leaflet (Fig. 6). The pulmonary valve echo in this case was incompletely recorded. The presence of large ‘a’ waves (14 mm) however, confirmed the presence of the pulmonary valve, ruled out pulmonary hypertension as the cause of the valvular regurgitation, and indicated that right ventricular end-diastolic pressure following atrial systole exceeded simultaneous pulmonary artery pressure with resultant presystolic valve opening, a pattern seen in patients with valvular pulmonary stenosis.

It is interesting that in Case 2 fluttering of the anterior tricuspid leaflet was not present, though there was moderate pulmonary regurgitation. Though the volume of regurgitant flow was large the pressure in the pulmonary artery was low and may have failed to create sufficient turbulence to induce fluttering of the tricuspid leaflet.

Discussion

In each of these four cases of pulmonary regurgitation, resulting from differing pathophysiological mechanisms, the echo pattern of pulmonary valve motion provided information confirming the underlying cause. In examining the echo patterns of pulmonary valve motion the ‘starting point’ should be an examination of the ‘a’ wave. The ‘a’ wave reflects the relative pressures across the pulmonary
Composite scan from right ventricle (RV) through right ventricular outflow tract (RVOT) into pulmonary artery (PA) in Case 3. Distinct dilatation of right ventricle (8 cm) along with fluttering of anterior leaflet of the tricuspid valve (ATV), suggesting pulmonary regurgitation. Right ventricular outflow tract appears of normal size. There is massive dilatation of the pulmonary artery with pronounced systolic expansion consistent with severe pulmonary regurgitation. A structure in the right ventricular outflow tract at the level of the normal pulmonary valve (arrow, centre panel) with a pattern of motion somewhat similar to the pulmonary valve suggests the possibility of a rudimentary valve structure.

Pulmonary valve echogram from Case 4. Pulmonary valve recorded during only a limited segment of the cardiac cycle. This segment, which includes a large ‘a’ wave, confirms presence of the pulmonary valve, rules out pulmonary hypertension as a cause of pulmonary regurgitation, and demonstrates that right ventricular end-diastolic pressure at peak atrial contraction exceeds pulmonary artery pressure with resultant valve opening suggesting right ventricular outflow obstruction.
pulmonary valve echo was recorded and suggested the cause of the pulmonary regurgitation. In Case 1 the absence of an 'a' wave indicated the presence of pulmonary hypertension. This was confirmed by the flat e to f slope and midsystolic notch at the valve (Weyman et al., 1974b). In Case 2 the normal 'a' wave ruled out the presence of pulmonary hypertension and showed that pulmonary regurgitation was present in the face of normal pulmonary artery pressures, and therefore was congenital in nature. The finding of normal echo patterns of pulmonary valve motion together with marked dilatation of the pulmonary artery at the level of the pulmonary valve indicated that the pulmonary incompetence in this case was due to idiopathic dilatation of the pulmonary artery. In Case 4 pulmonary regurgitation followed pulmonary outflow tract revision and pulmonary valvulotomy. When pulmonary regurgitation occurs after valvulotomy the aetiology is obviously not in question. This case is presented, however, to illustrate further the type of information that can be gathered from an echo study of pulmonary valve motion and to suggest the pattern of valve motion that would be expected in the rare case where pulmonary regurgitation accompanies valvular pulmonary stenosis in the absence of surgical intervention. Though the pulmonary valve in this case was recorded during only a small portion of the cardiac cycle large 'a' waves (14 mm) were demonstrated. The presence of large 'a' waves ruled out pulmonary hypertension as a cause of the pulmonary regurgitation and confirmed the presence of a pulmonary valve. Since we have previously observed 'a' waves of this magnitude only in patients with moderate and severe pulmonary stenosis, this finding suggested persistent right ventricular outflow obstruction. This was supported clinically by the presence of a grade 3/4 late-peaking systolic ejection murmur with delayed pulmonary valve closure. We have observed three other cases in which mild pulmonary regurgitation was present after successful pulmonary valvulotomy. In each of these cases 'a' wave depth was normal postoperatively.

In Case 3 no pulmonary valve echo could be recorded on multiple scans from the right ventricular outflow tract to the pulmonary artery and back. In normal patients the technical difficulty of recording the echo of the pulmonary valve is such that, unless there is a particular interest in the valve, failure to record this structure may be the rule rather than the exception. Thus it is difficult to derive any information from a failure to record the pulmonary valve. In cases such as Case 3, however, in which the pulmonary artery is massively dilated and the right ventricular outflow tract and pulmonary infundibulum are readily available for study, it is to be expected that a pulmonary valve, if present, would be recorded with careful study of this region.

It is interesting that in Case 2 no fluttering of the anterior tricuspid leaflet was noted though moderate pulmonary regurgitation was present. Pulmonary regurgitation is most commonly associated with severe pulmonary hypertension. The large pressure gradients present in these cases generate high velocities of regurgitant flow throughout diastole. This high velocity of flow is clinically associated with the high frequency, decrescendo Graham Steell murmur. Fluttering of the anterior tricuspid leaflet has been in general associated with pulmonary regurgitation of this type. In patients with congenital incompetence of the pulmonary valve the pressure in the pulmonary artery is normally low and the rate of regurgitant flow also low. In these situations a low pitched, mid-diastolic murmur is present. It is not surprising that while the high velocity of flow in patients with pulmonary regurgitation and pulmonary hypertension creates obvious diastolic fluttering of the anterior tricuspid leaflet the low velocity of flow in patients with congenital pulmonary insufficiency may fail to do so.

Another problem in the interpretation of the echo patterns of pulmonary valve motion previously commented on is the difficulty in applying data derived from one leaflet to the function of the entire valve (Weyman et al., 1974a). This is particularly true in cases such as Case 2 where one normal pulmonary leaflet was recorded. Since developmental abnormalities of the pulmonary valve leaflets may be limited to one or two of the valve cusps recording a normal cusp does not exclude the possibility that one or the other of the remaining cusps may be abnormal. Conversely, in Case 3 the failure to record a posterior cusp or the suggestion of a rudimentary cusp on the posterior surface of the pulmonary artery does not exclude the presence of two other normal cusps. In these cases a consideration of the size or diameter of the pulmonary artery at the level of the valve may prove valuable. In patients with pulmonary regurgitation secondary to idiopathic dilatation of the pulmonary artery the diameter of the artery at the level of the valve is increased, as was the situation in Case 2. When pulmonary regurgitation is secondary to incomplete formation or absence of valve leaflet tissue the pulmonary artery at the level of the valve may be of normal size, as in Case 3. Therefore when a normal pulmonary valve is recorded in a patient with pulmonary regurgitation and a dilated artery the case may be presumed to be one of idiopathic dilatation of the pulmonary artery.

In this report we have presented four illustrative
cases of pulmonary regurgitation. In each case the echo pattern of pulmonary valve motion differed and helped to indicate the underlying pathophysiology. This suggests that in patients with pulmonary regurgitation examination of the pulmonary valve leaflet motion may yield valuable information concerning the underlying cause of the disorder.

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References


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