Diastolic Forward Blood Flow in the Pulmonary Artery Detected by Doppler Echocardiography

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The etiology of diastolic motion of the pulmonary valve seen on the M-mode echocardiogram has been the subject of much debate. To further investigate diastolic events in the pulmonary artery, the patterns of diastolic pulmonary artery blood flow velocity were studied using pulsed Doppler echocardiography in patients with a normal heart. Two diastolic waveforms were found, one in early diastole related to passive filling of the right ventricle and one in late diastole related to atrial contraction. These waveforms were also related to the two recognized phases of diastolic pulmonary valve motion detected by M-mode echocardiography.

Diastolic opening motion of the pulmonary valve detected by M-mode echocardiography was first described by Graimiak et al. (1) in 1972. This finding was confirmed by other workers (2,3) and attributed to right ventricular diastolic pressure exceeding pulmonary artery pressure at the time of atrial contraction. A further phase of pulmonary valve motion occurring in early diastole was reported by Weyman et al. (4) in 1974, and is considered to be due to transmitted cardiac motion (4,5).

Diastolic velocity in the pulmonary artery may be detected by pulsed Doppler echocardiography and has also been attributed to atrial contraction, although this has not been formally investigated. We recently studied this phenomenon in a series of young patients.

Methods

Study population. The study group consisted of 50 normal infants and adults aged 2 months to 42 years (mean 14.3 years), who either were volunteers or were referred to the cardiac department and found to have an innocent heart murmur. An additional 11 patients with differing cardiac status were subsequently studied to help confirm our findings and conclusions.

Echocardiographic technique. All subjects were investigated with pulsed Doppler and two-dimensional echocardiography using a real time imager with a range-gated velocimeter (Honeywell). Examinations were performed with the subject in the lateral decubitus position, using a 3.5 MHz transducer positioned in short axis at the left parasternal edge or in the four chamber plane at the apex. When measuring velocities in the pulmonary artery, the sample volume was positioned distal to the pulmonary valve and proximal to the pulmonary artery bifurcation (Fig. 1, left). When measuring right ventricular inflow velocities, the sample volume was placed just distal to the tricuspid valve ring (Fig. 1, right). In both locations the sample volume was aligned with flow to obtain the maximal velocity.

Pulmonary artery velocities alone were studied in the first 30 subjects, but in the latter 20 both pulmonary and tricuspid valve velocities were recorded. When quality of recording permitted, M-mode events in these patients were analyzed to assess diastolic motion of the pulmonary valve. In four patients motion of the pulmonary valve ring and pulmonary artery bifurcation were also assessed. In each location the structure of interest was found by two-dimensional imaging and a cursor was placed on the structure in...
question to observe motion by M-mode echocardiography. Traces from two different sites in individual subjects were compared at matched RR intervals as measured by the surface electrocardiogram.

Four additional patients—two with aortic stenosis and two with coronary artery disease (all with normal right heart pressures)—were studied to assess electromagnetically detectable blood flow velocity in the pulmonary artery. In the first two cases pulmonary artery blood flow velocity was recorded using a 6F gauge electromagnetic flow velocity catheter (Skalar Medical, Delft, The Netherlands). In the second two cases an electromagnetic transducer cuff was placed around the main pulmonary artery before coronary artery vein grafting (Cliniflow CF 580 cuff and 601D flow-meter, Carolina Medical Electronics).

Results

Obtaining pulsed Doppler recordings from the pulmonary artery and tricuspid valve did not prove difficult. However, obtaining high quality M-mode recordings of both early and late phases of diastolic pulmonary valve motion was time-consuming and relatively unrewarding; we were able to produce satisfactory M-mode traces in only 7 of the 20 patients whose right ventricular inflow velocities were recorded.

Waveform description. Anterograde diastolic pulmonary artery velocities were detected in all 50 normal patients, but appeared in varying form, depending on the heart rate. When the RR interval was greater than approximately 550 ms, two distinct diastolic velocity waveforms were detected (Fig. 1, left), the first occurring in early diastole (f wave) and the second in late diastole (a wave).

Patients with a faster heart rate (RR interval < 550 ms) did not show the two distinct phases of pulmonary diastolic velocity, but a single waveform (fa wave) that resulted from gradual merging of the early and late waves as the RR interval shortened (Fig. 2). These changes were mirrored in tricuspid flow velocities, seen in single peak pattern at a short RR interval and in a double peak pattern when the heart rate was slow. In the 50 normal patients initially studied, the peak fa wave velocities ranged from 15 to 30 cm/s (mean 21), the a wave ranged from 10 to 25 cm/s (mean 15.5) and the combined fa wave ranged from 20 to 30 cm/s (mean 25).

In the 20 patients whose right ventricular inflow velocities were also measured, comparison of these traces with those in the pulmonary artery showed the peak pulmonary a wave velocity to be temporally related to the peak right ventricular inflow a wave velocity (Fig. 3). The pulmonary a wave also coincided with the late diastolic a dip of the pulmonary valve M-mode recording (Fig. 4). The early diastolic pulmonary artery velocity peak was found to be temporally related to passive filling of the right ventricle as detected by the peak of the tricuspid e wave velocity (Fig. 3). The pulmonary artery f wave also coincided with the early diastolic posterior motion of the pulmonary valve seen on M-mode echocardiography (Fig. 4). In seven patients slight delays were apparent between the corresponding tricuspid and pulmonary diastolic flow velocity peaks. The
Figure 2. Effect of heart rate. A, Two distinct pulmonary artery diastolic waveforms at an RR interval of 690 ms. B, When the RR interval shortens to 550 ms, the diastolic waveforms fuse (fa).

Figure 3. Demonstration of the temporal coincidence of the diastolic peaks recorded in the tricuspid (TRI) and pulmonary artery (PA) areas in one RR matched cycle (dashed lines). VEL = velocity.
delays were 50 ms in one patient, 40 ms in one, 20 ms in two and 10 ms in three.

A single patient with congenital complete heart block was also studied; f waves were found in the pulmonary artery as in sinus rhythm but the a waves appeared in relation to the P waves of the electrocardiogram and followed the usual pattern of atrioventricular dissociation (Fig. 5A). In two additional subjects with atrial fibrillation in the absence of structural heart disease, the f waves again appeared as in normal sinus rhythm, but the a wave was absent in the pulmonary artery recording (Fig. 5B).

**Evaluation of cardiac structural motion.** To investigate the possibility of the pulmonary diastolic waveforms being due to pulmonary artery motion rather than blood flow, we assessed M-mode echocardiographically detectable movement of the pulmonary artery bifurcation and valve ring in four additional subjects (three normal, one postaortic valvotomy). The maximal velocity of the pulmonary artery bifurcation in diastole was compared with the diastolic Doppler velocities within the lumen (Fig. 6A). Similarly, an M-mode recording of the anterior aortic wall was taken in the short-axis plane to detect motion of the pulmonary valve ring, which is seen to move in unison with the adjacent aorta in this plane on two-dimensional echocardiography. The maximal detectable velocity of diastolic motion of the pulmonary artery bifurcation varied from nil (in the postoperative patient) to 6 cm/s, and the maximal velocity of diastolic motion of the anterior aortic wall was 5.8 cm/s (Fig. 6B). All four of these patients had diastolic pulmonary artery Doppler f wave and a wave velocities of approximately 20 and 15 cm/s, respectively. Structural velocity was therefore considerably less than the diastolic velocities found within the lumen.

Diastolic velocity waveforms detected by both electromagnetic methods were similar in both appearance and magnitude to those detected by Doppler echocardiography and confirmed the presence of two distinct diastolic phases of flow (Fig. 7).

**Discussion**

We have shown that forward diastolic blood flow in the pulmonary artery may be readily demonstrated in young patients with a normal heart. The waveforms usually occur in two phases, one related to atrial contraction and one to passive filling of the right heart. The presence of one or two waveforms depends on the heart rate and rhythm. Our findings in atrial fibrillation demonstrate that atrial contraction is not essential to produce diastolic flow in the pulmonary artery. The appearance of a single diastolic waveform in the Doppler trace when the RR interval shortens may be considered analogous to the change of the well-known M-mode double peak pattern of atrioventricular valve motion to a single peak pattern at fast heart rates.

The delay found between tricuspid and pulmonary artery diastolic waveforms in seven patients is of uncertain significance. The method of matching timing of the two traces was relatively crude, making accuracy difficult to attain. Delays, therefore, may have been due to observer error. At present the technology for obtaining Doppler velocity information from two different cardiac sites simultaneously does not exist and the comparison of similar but not identical cardiac cycles was the best technique available.

**Other possible causes of diastolic waveforms.** The diastolic velocities demonstrated in the pulmonary artery are far in excess of the velocity of motion of the pulmonary artery bifurcation measured by M-mode echocardiography and of the velocity of motion of the pulmonary valve ring measured indirectly by M-mode echocardiography. Our structural motion velocities are of the same magnitude as those reported by Strunk et al. (6). The positioning of an electromagnetic flow cuff around the main pulmonary artery excludes any effect of motion transmitted from adjacent structures and is conclusive evidence that the diastolic waveforms described are due to blood flow. The possibility of "rebound" flow causing the early diastolic f wave is ex-
Figure 5. A, Pulmonary artery velocity in complete heart block. The a wave is coincident with the electrocardiographic P wave irrespective of the location of the P wave within the cardiac cycle. B, In atrial fibrillation, only an f wave is found.

Figure 6. Two methods of evaluating cardiac motion. A, Recording from a postoperative patient with the M-mode and Doppler cursor set as shown (insert, left panel). The pulmonary (PUL) artery velocity tracing (left panel) shows the a and f waves as indicated. A, right panel, An M-mode recording obtained with the cursor line positioned at the pulmonary artery bifurcation (PAB), shown with a dotted line. No motion of the bifurcation was found, but the a and f velocity waves are clearly demonstrated. ao = aortic. B, left panel, The M-mode cursor (M) placed through the anterior aortic (AO) wall. B, right panel, The recording taken from it. PV = pulmonary valve. The maximal diastolic slope of the anterior aortic wall is 5.8 cm/s, but the f wave and the a wave velocities in this patient were 20 and 15 cm/s, respectively. MAX = maximal.
cluded by the presence of identical detectable waveforms in the right ventricular outflow tract immediately proximal to the pulmonary valve.

Some degree of forward blood flow in the pulmonary arteries might be expected after left atrial filling, owing to the low impedance of the pulmonary vascular bed in normal subjects. However, the pulmonary bed has variable capacitance throughout the cardiac cycle, and for this reason it is unlikely that left atrial filling alone would cause forward blood flow as far retrograde as the main pulmonary artery. If, indeed, left atrial filling did produce some forward flow at this site, consistent delay between atrioventricular valve opening and the diastolic flow velocities in the main pulmonary artery might be expected. Such delay was detected in only seven subjects and ranged from 10 to 50 ms.

It has previously been suggested that diastolic motion of the pulmonary valve seen on M-mode echocardiography is due, at least in part, to movement of the heart itself or to transmitted pulsation from left atrial filling and contraction (4,5,7). Strunk et al. (6) provided convincing evidence that these factors are responsible for the diastolic patterns of aortic valve motion. Although we cannot exclude transmitted structural motion as a contributory factor, our findings demonstrate that the diastolic velocities we have described in the pulmonary artery and the previously described movements of the pulmonary valve are principally due to blood flow caused by the two phases of right ventricular filling.

Whether right ventricular filling produces blood flow in the pulmonary artery by causing partial opening of the pulmonary valve or by a piston-like effect without actual leaflet separation remains a matter for conjecture. However, it seems unlikely that flow velocities of the magnitude described would occur without some degree of cusp separation. Pulmonary valve opening due to passive right ventricular filling has previously been reported in the context of constrictive pericarditis, when right heart filling pressures are very high (8). Our data suggest that normal venous pressure produces a similar effect in the absence of cardiac or pulmonary vascular abnormalities.

Figure 7. Pulmonary artery (PA) blood velocity. a, As recorded with an electromagnetic flow velocity catheter positioned in the main pulmonary artery. b, As recorded with an electromagnetic flow cuff placed around the main pulmonary artery. The diastolic f and a waves are clearly demonstrated in both cases. ECG = electrocardiogram.
We conclude that in the normal heart passive right ventricular filling and atrial contraction both produce blood flow in the pulmonary artery.

References