Assessment of pulmonary valve echogram in normal subjects and in patients with pulmonary arterial hypertension

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SUMMARY To assess the validity of ultrasound criteria for pulmonary arterial hypertension, we studied pulmonary valve motion in 28 patients and 20 normal subjects. In the latter group, we categorised normal movement of the posterior leaflet of the pulmonary valve in a fashion not previously described. Of the 28 patients, 19 had pulmonary arterial hypertension (pulmonary artery mean pressure more than 20 mmHg, range 22 to 72). Negative, flat, and positive e to f slopes occurred equally in normal subjects and patients. Maximal a wave excursion was less than 2 mm in 9 of 13 patients with pulmonary hypertension who had sinus rhythm, but was more than 2 mm in all normals and in the 9 patients with normal pulmonary artery pressure (69% sensitivity and 100% specificity). The b to c slope was more than 450 mm/s in 6 of 18 patients with pulmonary arterial hypertension and was less than 450 mm/s in all others (33% sensitivity and 100% specificity). The normalised right ventricular pre-ejection period was more than 0.095 (range 0.10 to 0.16) in 13 of the 19 patients with pulmonary arterial hypertension and less than 0.095 in all others (68% sensitivity and 100% specificity). A midsystolic notch occurred in patients with pulmonary arterial hypertension and in no normal subjects or patients with normal pulmonary arterial pressure. Of the 19 patients with pulmonary arterial hypertension, 18 were identified by one or more ultrasound criteria. Of the 5 patients who met only 1 criterion (increased normalised right ventricular pre-ejection period), 4 had atrial fibrillation. We conclude that measurement of the pulmonary valve e to f slope is useless for identifying pulmonary arterial hypertension. However, changes in normalised right ventricular pre-ejection period, maximal a wave excursion, b to c slope, and the presence of a midsystolic notch, while insensitive, are highly specific for pulmonary arterial hypertension.

The pulmonary valve remains the most elusive and difficult valve from which to record echocardiograms, particularly in normal subjects. There have been a number of studies of the changes in the pulmonary valve echogram in patients with pulmonary arterial hypertension (Nanda *et al.*, 1974; Weyman *et al.*, 1974b; Kaku *et al.*, 1978). The present investigation was undertaken to evaluate the previously described ultrasound criteria for pulmonary arterial hypertension, and to assess normal and abnormal variations in pulmonary valve motion on the echocardiogram.

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Subjects

Twenty-one men, aged 17 to 74 years (mean 47) and 7 women, aged 19 to 70 years (mean 51) were studied by echocardiography and cardiac catheterisation. Satisfactory pulmonary valve echograms were recorded in all 28 patients. Twenty-two of the patients were in sinus rhythm and 6 had atrial fibrillation. The diagnosis was valvar heart disease in 13 patients, congenital heart disease in 3, coronary artery disease in 2, pulmonary emboli in 2, cardiomyopathy in 2, primary pulmonary hypertension in 1, and no cardiopulmonary disease in 5 patients. The interval between echocardiogram and cardiac catheterisation was 0 to 7 days (mean 1.6) in 23 of the 28 patients. In the remaining 5 patients, the interval was 2 weeks to 3 months (mean 42 days),

but during this time there was no evident change in their clinical condition.

Of the 28 patients who had cardiac catheterisation studies, 9 had a normal pulmonary artery mean pressure of 20 mmHg or less. All 9 were in sinus rhythm. Nineteen patients, 13 of whom were in sinus rhythm, had raised pulmonary artery (PA) mean pressure (more than 20 mmHg, range 22 to 72 mmHg). Nine of these patients had a raised right ventricular end-diastolic pressure (RVEDP) more than 10 mmHg (range 11 to 16 mmHg).

Twenty normal subjects were studied by echocardiography alone. In all 20 the pulmonary valve was well visualised. This group included 15 men, aged 23 to 34 years (mean 27) and 5 women, aged 24 to 26. None of the normal subjects had any evidence of cardiopulmonary disease by history, physical examination, electrocardiogram, or echocardiography.

Methods

Echocardiograms were obtained with a commercially available device.¹ A medium internal focus 2.26 MHz transducer with a repetition rate of 1000 impulses/s was used. The technique for recording the pulmonary valve echo has been previously described (Gramiak *et al.*, 1972; Weyman *et al.*, 1974a). All 20 normal subjects were studied in the left lateral position. The optimal echocardiographic 'window' in normal subjects was in the fourth intercostal space in 19 and the third intercostal space in 1. Of the 28 patients, 24 were studied in the left lateral position; 19 had recordings in the fourth intercostal space, 4 in the third intercostal space, and 1 in the second intercostal space. The

¹Pickering Echoview System 80C coupled to an Irex Continuum Trace unit for display and recording.

remaining 4 patients were studied in the supine position in the fourth intercostal space.

At least 5 beats displaying the pulmonary valve echogram were recorded in each of the 48 subjects and patients, with a simultaneous lead II electrocardiogram. The normal motion of the pulmonary valve is shown in Fig. 1–3. The posterior leaflet of the pulmonary valve moves in a flat or posterior fashion from points e to f (Fig. 1). The rate of this motion is measured as the e–f slope (mm/s). The f point marks the beginning of the a dip and coincides with the end of the P wave on the electrocardiogram. The a wave excursion is measured as the vertical displacement between the f point and the trough of the a dip (Fig. 2). There are respiratory variations in this measurement and the maximum a wave excursion (A_{max}) is the measurement used for comparative purposes. At the end of the a excursion, the posterior leaflet of the pulmonary valve moves slightly anteriorly to the b point which corresponds to the initiation of right ventricular ejection and therefore with pulmonary valve opening. It then opens rapidly to the posteriorly located c point (Fig. 3). The most rapid motion occurs towards the end of this b–c segment and is measured as the b–c slope (mm/s). The vertical displacement between points b and c is measured as the b–c opening distance or amplitude.

The systolic movements of the posterior leaflet of the pulmonary valve are not normally recorded. When they are visible there is an anterior motion from c to the d point, followed by pulmonary valve closure from d to e. Occasionally, midsystolic notching is present giving the systolic pulmonary valve echo a 'W' configuration. Under these circumstances, the posterior leaflet moves anteriorly early in systole followed by a posterior motion and

Fig. 1 Normal variations in the pulmonary valve e–f slope. On the left, the dotted lines indicate in diagrammatic fashion the various ways of measuring the e–f slope. Actual recordings for each of the patterns are shown on the right.

Fig. 2 Measurement of A_{max} . On the left, the dotted vertical lines indicate the onset of the dip at the end of the P wave of the electrocardiogram. The horizontal lines depict the maximum depth of the a dip (A_{max}). In the panels on the right, actual recordings for each of the patterns are shown.

Fig. 3 Normal variations in the pulmonary valve b-c slope. On the left, the normal variations in the b-c slope are shown in diagrammatic fashion. In the echocardiographic tracing at the upper right, the pulmonary valve b-c slope can be seen to vary with phase of respiration (altered beam angle). The right lower panel is characteristic of pattern B-2.

Fig. 4 An echocardiographic sweep showing portions of the pulmonary and mitral valves. The pulmonary valve recording at the upper left portion of the recording shows 'notching' characteristic of pulmonary arterial hypertension.

Fig. 5 Measurement of right ventricular pre-ejection period (RPEP). The dotted line on the left identifies the onset of the QRS complex of the electrocardiogram, and the one on the right indicates the onset of pulmonary valve opening. The time between the two lines measures RPEP, which in the example shown is 80 ms.

then another anterior motion during pulmonary valve closure (Fig. 4).

Right ventricular systolic time intervals were obtained by methods previously described (Nanda *et al.*, 1974; Hirschfeld *et al.*, 1975). The right ventricular pre-ejection period (RPEP) was measured between the time of onset of electrical and mechanical right ventricular systole, that is from the onset of the QRS complex on the electrocardiogram to the b point of the pulmonary valve echo (Fig. 5). Normalised right ventricular pre-ejection

period was calculated as $RPEP/\sqrt{R-R}$, where R-R is the cycle length in seconds (Nanda *et al.*, 1974). The right ventricular ejection time (RVET) was measured between the b point and the point of pulmonary valve closure (Hirschfeld *et al.*, 1975) but could only be recorded in 9 patients with pulmonary hypertension and in none of the other patients or normal subjects. The ratio of the RPEP to the RVET was also calculated.

Results

PULMONARY VALVE ECHOGRAM IN NORMAL SUBJECTS

e-f slope

In normal individuals variations in the e-f slope, an excursion, and b-c slope occurred commonly (Fig. 1). The e-f slope occasionally moved anteriorly to an e' position followed by normal posterior motion (Fig. 1, E-2), or it moved posteriorly to e'' followed by a long flat e-f segment (Fig. 1, E-3). In both cases the terminal segment preceding the a excursion was measured as the e-f slope. In some cases only the terminal e-f segment was visualised (Fig. 1, E-4).

A_{max}

The most common a dip was classified as A-1 (Fig. 2). In this pattern there is a well-defined f point initiating the a excursion and a return anteriorly to the b point at the end of the a excursion. In pattern A-2 the end of the P wave on the electrocardiogram arbitrarily defined the f point and thus the height of the a excursion, since the e-f slope merged continuously into the trough of the a dip (Fig. 2, A-2). Where the P wave is not well defined on lead II of the electrocardiogram, a different standard lead should be employed. Nevertheless, even under the best of circumstances, this pattern may lead to inaccuracies in the measurement of A_{max}. In pattern A-3 there was a distinct

Table 1 Echocardiographic values in normal subjects

Case no.	Age/sex	Rate (bpm)	e-f slope (mm/s)	b-c slope (mm/s)	A _{max} (mm)	b-c amplitude (mm)	RPEP (s)	R-R and $\sqrt{R-R}$	RPEP $\sqrt{R-R}$
1	26/F	82	12.0	407	3.8	9.8	0.063	0.732 0.856	0.073
2	34/M	58	4.9	328	3.7	—	0.069	1.036 1.018	0.067
3	26/M	58	4.5	441	4.5	—	0.057	1.036 1.018	0.056
4	26/F	58	1.1	312	3.7	—	0.071	1.042 1.02	0.069
5	32/M	53	0.6	307	2.3	—	0.047	1.141 1.068	0.044
6	24/M	66	0.9	409	4.3	—	0.056	0.910 0.954	0.058
7	23/M	71	9.2	358	3.9	—	0.082	0.843 0.918	0.089
8	30/M	66	3.0	357	4.5	—	0.071	0.913 0.956	0.075
9	30/M	60	2.3	307	4.0	8.2	0.064	1.003 1.0	0.064
10	26/F	63	0.1	316	3.9	—	0.052	0.946 0.973	0.054
11	26/M	63	6.4	284	3.2	—	0.067	0.957 0.978	0.068
12	30/M	63	2.1	335	3.7	—	0.073	0.946 0.973	0.075
13	25/M	73	-5.0	302	3.5	—	0.073	0.823 0.907	0.080
14	23/M	55	0.2	208	4.2	8.9	0.092	1.10 1.05	0.088
15	24/M	31	2.2	—	2.7	—	0.069	1.923 1.387	0.050
16	24/M	51	1.4	314	5.1	11.7	0.071	1.179 1.086	0.065
17	25/M	53	0.4	388	7.2	11.9	0.068	1.139 1.18	0.058
18	25/M	58	2.8	288	3.2	11.9	0.068	1.04 1.02	0.067
19	26/F	57	0.8	336	6.3	11.2	0.059	1.047 1.023	0.058
20	24/F	54	0.7	263	3.4	10.2	0.078	1.109 1.053	0.074

RPEP, right ventricular pre-ejection period; R-R, interval between beats (s).

f point and a dip, but at the end of the a dip the pulmonary valve does not return to an anterior position before opening. This phenomenon has been described as a result of normal inspiration (Weyman *et al.*, 1974a), but in some of our studies the posterior leaflet of the pulmonary valve never returned to an anterior position even during expiration. In these instances the posterior leaflet opened from a partially open or domed position at the end of diastole and occasionally gave the appearance of an absent a dip. In such cases A_{max} was measured from the f point to the plateau of the a dip before the valve opened fully. In Fig. 2 the illustration for pattern A-3 shows a PR interval of 0.12 s but none of the other 3 subjects had PR intervals less than 0.14 s. In group A-4 there is a clear a dip but an incomplete e-f slope as in Fig. 2.

In this group the end of the P wave of the electrocardiogram was used to define the f point and the height of the a dip.

b-c slope

The b-c slope also varied according to the portion of the b-c segment visualised. The b-c slope was designated as B-1 when the entire opening of the pulmonary valve was seen (Fig. 1, E-4, Fig. 2, A-1). That full valve opening had occurred was confirmed by visualising the characteristic abrupt change in direction of the echo produced by rapid anterior motion at the c point (Fig. 3, B-1). The most rapid movement occurred late in the b-c segment so that visualisation only of the earlier portion gave slower b-c slope measurements (Fig. 3, B-2). Variation in b-c slope also occurs with differing cycle lengths,

Table 2 Echocardiographic and haemodynamic data in patients

Case no.	Age/sex	Rhythm	Heart rate (bpm)	e-f slope (mm/s)	b-c slope (mm/s)	A _{max} (mm)	b-c amplitude (mm)	Midsystolic notching	RPEP (s)	R-R and $\sqrt{R-R}$
1	25/M	NSR	72	-12.1	392	4.4	—	No	0.067	0.830
2	20/M	NSR	81	2.8	171	6.9	10.4	No	0.060	0.91
3	74/M	NSR	54	—	394	6.7	—	No	0.059	0.739
4	51/M	Sinus brady-cardia	48	—	380	2.2	—	No	0.066	0.86
5	17/M	NSR	95	7.6	445	2.8	—	No	0.063	1.107
6	46/M	NSR	64	-5.3	408	3.7	—	No	0.087	1.05
7	67/M	NSR	59	0.9	243	5.0	—	No	0.069	1.242
8	57/M	NSR	58	2.4	299	2.0	12.3	No	0.080	1.11
9	45/M	NSR	76	-2.1	259	2.4	—	No	0.066	0.634
10	52/M	NSR	92	—	609	5.2	17.3	No	0.075	0.79
11	59/F	NSR	68	6.6	327	2.4	—	No	0.098	0.934
12	55/M	NSR	56	-1.0	287	2.4	—	No	0.094	0.96
13	59/M	NSR	60	13.9	413	0	6.3	Yes	0.088	1.024
14	73/M	NSR	106	2.5	392	0	10.0	No	0.103	1.01
15	44/M	NSR	92	1.0	363	0	—	No	0.088	1.033
16	25/M	NSR	72	9.5	321	1.9	—	No	0.115	1.02
17	63/M	NSR	73	7.2	346	0	—	No	0.093	0.789
18	31/M	NSR	70	1.9	270	0.9	13.7	Yes	0.060	0.89
19	28/M	NSR	39	6.2	503	2.3	14.5	Yes	0.111	0.652
20	46/M	NSR	80	7.3	394	0	19.4	Yes	0.090	0.808
21	28/F	NSR	119	0.1	478	0	8.5	Yes	0.088	0.880
22	19/F	NSR	90	27.0	495	0	12.1	No	0.059	0.938
23	70/F	Atrial fib.	71	3.3	466	0	—	No	0.102	1.081
24	66/F	Atrial fib.	95	5.6	368	0	6.3	No	0.123	1.04
25	71/M	Atrial fib.	76	12.7	399	0	12.1	No	0.141	1.0
26	46/F	Atrial fib.	107	27.0	284	0	—	No	0.116	0.566
27	68/F	Atrial fib.	91	5.5	—	0	—	No	0.098	0.752
28	38/M	Atrial fib.	102	13.5	555	0	15.1	No	0.120	0.650

NSR, normal sinus rhythm; RPEP, right ventricular pre-ejection period; R-R, interval between beats (s); RVET, right ventricular ejection time; PA, pulmonary artery; S/D, systolic/diastolic; M, mean; RV, right ventricular; ED, end-diastolic; CAD, coronary artery disease; PS, pulmonary stenosis; AR, aortic regurgitation; ASD, atrial septal defect; MS, mitral stenosis; PH, pulmonary hypertension; PE, pulmonary emboli; AS, aortic stenosis; MR, mitral regurgitation; 1°PH, 'primary' pulmonary hypertension; VSD, ventricular septal defect; MVR, mitral valve replacement.

$\frac{RPEP}{\sqrt{R-R}}$	RVET	$\frac{RPEP}{RVET}$	Diagnosis	PA pressure, mmHg S/D (m)	RV pressure, mmHg S/ED
0.073	—	—	Normal	24/5 M=10	23/2
0.063	0.338	0.179	Normal	16/7 M=11	27/5
0.056	—	—	CAD	19/8 M=12	20/7
0.059	—	—	Normal	22/10 M=14	29/6
0.080	—	—	Valvular PS	22/12 M=16	45/11
0.091	—	—	Normal	28/12 M=18	28/10
0.068	—	—	AR	33/11 M=20	32/7
0.079	—	—	Normal	23/10 M=15	25/7
0.074	—	—	CAD	23/14 M=16	25/5
0.093	—	—	ASD	27/14 M=22	32/5
0.104	—	—	MS	33/19 M=24	37/4
0.091	—	—	AR	38/18 M=24	39/14
0.088	0.373	0.22	PH, PE	46/19 M=27	49/8
0.137	0.230	0.45	AS, CAD	31/10 M=28	36/5
0.109	—	—	MR	52/22 M=31	50/5
0.126	0.274	0.42	Cardio-myopathy	60/30 M=39	60/14
0.102	—	—	MR, CAD	64/30 M=40	63/9
0.065	0.432	0.14	PH, PE	74/25 M=43	74/11
0.090	—	—	1°PH	78/40 M=55	78/15
0.105	0.371	0.244	ASD	88/40 M=55	88/4
0.124	0.207	0.426	MS	104/50 M=72	104/16
0.072	—	—	VSD	60/29 M=43	61/9
0.111	—	—	MS/CAD	43/13 M=26	46/4
0.155	0.194	0.63	MS	48/22 M=33	52/5
0.159	0.275	0.515	AS/MS/ MR/CAD	65/30 M=38	65/12
0.155	—	—	MVR	53/29 M=40	52/11
0.120	—	—	MS	83/31 M=52	83/12
0.156	0.255	0.48	Cardio-myopathy	67/45 M=58	65/14

phase of respiration, and/or transducer placement as in Fig. 3, B-3. In all cases the most rapid slope was measured.

MEASUREMENTS IN NORMAL SUBJECTS AND IN PATIENTS WITH PULMONARY ARTERIAL HYPERTENSION (Tables 1, 2)

e-f slope

The pulmonary valve e-f slope was compared with the PA mean pressure (Fig. 6). In 20 normal

subjects the e-f slope had a mean value of 2.4 ± 3.7 mm/s (SD) with a range of -5.0 to 12.0 mm/s. The mean value for the patients with pulmonary arterial hypertension was 8.3 ± 8.1 mm/s which was higher than for the normal subjects ($P < 0.006$), but the overlap in values was too great to allow any separation between groups.

A_{max}

A_{max} was measured in all 48 subjects. The relation between *A_{max}* and PA mean pressure is shown in Fig. 7. There is a general inverse relation showing that higher PA mean pressures are associated with lower values of *A_{max}*. The 20 normal subjects had a mean *A_{max}* of 4.1 ± 1.1 mm with a range of 2.3 to 7.2 mm. The 9 patients with normal PA mean pressures had *A_{max}* values ranging from 2.0 to 6.9 mm with mean *A_{max}* nearly identical to that for the normal subjects (Fig. 7). Thus, *A_{max}* was 2.0 mm or greater in all 29 normal subjects and patients with a normal PA mean pressure (100%).

In the 13 patients with raised PA mean pressure and sinus rhythm, the mean *A_{max}* was 1.2 ± 1.6 mm (range of 0 to 5.2 mm). The mean was lower than in the normal subjects ($P < 0.001$). *A_{max}* was zero in 7 of the 13 patients (54%), between 0 and 2 mm in 2 (both also had an elevated RVEDP), and exceeded 2 mm in 4 (2 of these also had an elevated RVEDP). Thus, there were 9/13 (69%) who had *A_{max}* values of less than 2 mm whereas in all the normal subjects *A_{max}* exceeded 2 mm. Therefore, an *A_{max}* of less than 2 mm had 100 per cent specificity and 69 per cent sensitivity as a criterion for pulmonary arterial hypertension. Of the 4 false negatives (those with pulmonary hypertension who had an *A_{max}* value of 2 mm or greater), 3 had only slightly raised PA mean pressures (22 to 24 mmHg). Thus, of the 10 patients with PA mean pressures of 25 mmHg or greater, 9 (90%) had *A_{max}* values of less than 2 mm.

As might be expected, all 6 patients with pulmonary hypertension and atrial fibrillation had no a dip.

b-c slope

The b-c slope was measured in 96 per cent (46/48) of subjects, and is compared with the PA mean pressure in Fig. 8. In 19 normal subjects, the mean b-c slope was 328 ± 55 mm/s (range 208 to 441 mm/s). Mean b-c slope in the 9 patients with normal PA mean pressure was nearly identical to that in the normal subjects. The 18 patients with pulmonary arterial hypertension in whom the b-c slope was measured had higher values (mean 403 ± 96 mm/s, range 270 to 609 mm/s) ($P < 0.007$). A rapid b-c slope of 450 mm/s or more was found

Fig. 6 Individual e-f slope data. In the upper panel mean value ± 1 standard deviation is shown for each group. In the lower panel it can be seen that there is a rough correlation between the e-f slope and PA mean pressure, but the scatter is too great to be of any predictive value. There is also no relation between any of the e-f slope patterns (E1-4) and PA pressure. PAP, pulmonary artery mean pressure; NSR, normal sinus rhythm; A. fib., atrial fibrillation.

only in patients with raised PA mean pressure and was thus 100 per cent specific for pulmonary arterial hypertension. The sensitivity was 33 per cent since only 6 of the 18 patients with pulmonary hypertension had such rapid b-c slopes.

b-c opening amplitude

The b-c opening amplitude was measured in 44 per cent (21/48) of all subjects, and is plotted against PA mean pressure in Fig. 9. The 8 normal subjects had values from 8.2 to 11.9 mm (mean 10.5 ± 1.4 mm). There was no significant difference between normal subjects and the 11 patients with raised PA mean pressure who had a mean value of 12.3 ± 4.3 mm (Fig. 9). However, none of the normal subjects had b-c opening amplitudes greater than 13 mm whereas 5 of 11 (45%) with

pulmonary hypertension met this criterion. When an abnormally large b-c opening amplitude greater than 13 mm was used as a criterion for pulmonary arterial hypertension, there was 100 per cent specificity and 45 per cent sensitivity.

Midsystolic notching

Midsystolic notching was not seen in any of the 20 normal subjects, in the 9 patients with normal PA mean pressure, or in the 9 patients with raised PA mean pressure and atrial fibrillation. Midsystolic notching (Fig. 4) was present in 5 of the 13 (38%) patients with raised PA mean pressure who were in sinus rhythm. Thus, for this last group there was 100 per cent specificity, with 38 per cent sensitivity. Five of these 13 patients had PA mean pressures ranging from 43 to 72 mmHg and midsystolic

Fig. 7 Individual data for maximal a wave excursion (A_{max}). Most of the patients with sinus rhythm and an increased mean pulmonary artery pressure had an A_{max} value of less than 2 mm (upper panel). There is, however, no relation between any of the a wave patterns (A1-4) and PA pressure. PAP, pulmonary artery mean pressure; NSR, normal sinus rhythm; A. fib., atrial fibrillation.

notching was present in all but one of these (PA mean pressure 43 mmHg). Midsystolic notching was also present in the 4 patients with the highest PA systolic pressures (74 to 100 mmHg).

Right ventricular pre-ejection period

The right ventricular pre-ejection period (RPEP) was measured in all 48 subjects, and is compared with the PA mean pressure in Fig. 10, which shows a prolongation in RPEP at higher PA mean pressures. The 20 normal subjects had a mean RPEP of 0.068 ± 0.010 s (range of 0.047 to 0.092 s). The RPEP values of the 9 patients with normal PA mean pressure had a similar range and mean to the normal subjects. Fig. 10 shows that the 19 patients with raised PA mean pressures had a mean RPEP of 0.098 ± 0.021 s (range 0.059 to 0.141 s). RPEP was significantly longer than in normals ($P < 0.001$). The 4 longest RPEP values were in the patients with atrial fibrillation. None of the normal subjects had RPEP values of 0.095 s or greater whereas 10 of 19 (53%) patients with pulmonary hypertension had such values. Thus, a RPEP of greater than

0.095 s had a specificity of 100 per cent for pulmonary hypertension and a sensitivity of 53 per cent. The relation between RPEP and PA end-diastolic pressure was also examined but did not improve the separation between normal subjects and those with pulmonary hypertension.

Normalised RPEP

None of the normal subjects or patients with normal PA mean pressure had normalised RPEP values of 0.095 or greater, while 13 of 19 (68%) of those with pulmonary hypertension did (Fig. 11). Thus, the specificity remained at 100 per cent and the sensitivity improved to 68 per cent. Mean normalised RPEP was also significantly greater in patients with pulmonary hypertension than in normal subjects ($P < 0.001$).

Right ventricular ejection time

Right ventricular ejection time (RVET) could be measured in none of the 20 normal subjects and in only 1 of the 9 patients with normal PA mean pressure. RVET was measured in 9 out of 19 (47%)

Fig. 8 Individual *b-c* slope data. All those with a *b-c* slope more than 450 mm/s had pulmonary hypertension (upper panel). There is a rough correlation between the *b-c* slope and the level of PA mean pressure, but the scatter is wide. PAP, pulmonary artery mean pressure; NSR, normal sinus rhythm; A. fib., atrial fibrillation.

with pulmonary arterial hypertension. Values ranged from 0.207 to 0.432 s (mean 0.315 s) in those with sinus rhythm and from 0.480 to 0.630 s (mean 0.542 s) in those with atrial fibrillation. The ratio RPEP/RVET ranged from 0.14 to 0.45 (mean 0.32) in those in sinus rhythm and exceeded 0.3 in 3 of 5 patients; this ratio ranged from 0.48 to 0.63 (mean 0.54) in 3 patients with atrial fibrillation in whom it could be measured.

Discussion

Variations in pulmonary valve motion in normal subjects indicated to us that a classification of the

variations seen in pulmonary valve echo motion in normal subjects was a prerequisite for a definition of the changes found in patients with pulmonary arterial hypertension. To our knowledge there have been no previously published standard methods of measurement for the various portions of the pulmonary valve echogram.

e-f SLOPE

Of the 4 types of e-f slope (Fig. 1), pattern E-2 includes the previously described anterior e-e' motion followed by the normal posterior e'-f segment and is believed to reflect transmitted aortic pulsation (Weyman *et al.*, 1974a, b). Recent

evidence also indicates that left atrial size and pulsation also may play a role in determining the shape of the e-f slope (Pocoski *et al.*, 1978).

Weyman and colleagues (1974b) also have described a pattern in which the normal posterior e-f motion terminates early and is followed by a short flat segment. They reported that the length of this flat segment was directly related to cycle length and for measurement used the initial rapid e-f slope. In such instances we also measured the predominant rapid slope. However, we found a large variation in the length of the terminal flat segment as compared with the initial rapid posterior motion. In many normal subjects the initial motion was short, rapid, and posterior followed by a long flat slope. Fig. 1 shows an example in which the e-f slope is divided equally between posterior motion

in the first half followed by flat motion in the second half (pattern E-3). This common variation occurred in 30 per cent (6/20) of our normal subjects. When the flat motion predominated in this group, it was used for measurement of the e-f slope.

In pattern E-4 an incomplete e-f slope was recorded. This variation could easily represent the terminal portion of any of the other patterns. Because many pulmonary valve echo recordings are incomplete, we advocate routine measurement of the terminal motion of all e-f slopes because this would represent the most consistently reproducible slope. For example, if the initial posterior motion is measured as in pattern E-3, subsequent echo recordings may only include the terminal portion, invalidating any comparisons.

Weyman and colleagues (1974b) reported that

Fig. 9 Individual data for b-c opening distance. This measurement could not be made in many patients and normal subjects. PAP, pulmonary artery mean pressure; NSR, normal sinus rhythm; A. fib., atrial fibrillation.

Fig. 10 Data for right ventricular pre-ejection period (RPEP). All patients with RPEP more than 0.095 have pulmonary hypertension (upper panel). Nevertheless, many patients with increased mean pulmonary artery pressure have normal values (upper and lower panels). $\overline{PAP}$, pulmonary artery mean pressure; NSR, normal sinus rhythm; A. fib., atrial fibrillation.

patients with pulmonary hypertension tended to have flat e-f slopes but the overlap with normal subjects limited the usefulness of this measurement. However, they reported that negative e-f slopes, present in 6 of 32 patients but none of their normal subjects, were specific for pulmonary hypertension. Our data suggest that the overlap between e-f slopes in normal subjects and in patients with pulmonary hypertension is too great to be of any value. Moreover, negative slopes were found in 4 of 27 (15%) normal subjects and patients with normal PA mean pressure but in only 1 of 18 (6%) with pulmonary arterial hypertension.

a DIP

In normal subjects deep inspiratory a dips, in which the pulmonary valve opens from a partially open or domed position, have been described (Weyman *et al.*, 1974a). This phenomenon occurs especially in the presence of a short PR interval, but the normal anterior motion to the b point is seen again during expiration. However, in 4 of our 20 normal subjects the posterior leaflet of the

pulmonary valve never moved anteriorly following the trough of the a dip. In these subjects, the pulmonary valve opening began from a position at approximately the same level as the trough of the a dip. This occurred throughout the tracing, even during expiration and with a normal PR interval. In this group (pattern A-3) measurement of the a wave excursion was from f point to plateau of the a dip.

Several investigators have reported that an A_{max} of 2 mm or less is useful in separating patients with normal from those with raised PA mean pressure (Nanda *et al.*, 1974; Weyman *et al.*, 1974a, b; Kaku *et al.*, 1978). A greater frequency of absent a dips has been reported at higher PA mean pressures (Nanda *et al.*, 1974). Our data are in agreement with the hypothesis that A_{max} values are very useful in separating patients with pulmonary arterial hypertension from those with normal PA pressures. We found that when A_{max} of less than 2 mm was used as a criterion of pulmonary hypertension (PA mean pressure more than 20 mmHg), specificity was 100 per cent and sensitivity 69 per cent. The

sensitivity was improved to 90 per cent when only patients with PA mean pressure exceeding 25 mmHg were considered.

It is believed that the a dip represents opening or doming of the pulmonary valve in response to the transmitted pressure rise from right atrial contraction. In pulmonary arterial hypertension there is a raised pressure distal to the pulmonary valve that prevents or reduces the opening motion after atrial contraction. However, some patients with pulmonary hypertension may have misleadingly high $A_{\max}$ values, greater than 2 mm, in the presence of right ventricular failure. One explanation for this phenomenon is that the raised right ventricular end-diastolic pressure reduces the effective diastolic gradient across the pulmonary valve, thus allowing movement (often more than 2 mm) despite the raised PA mean pressure. Such an inverse relation was found in our data. Patients with pulmonary arterial-right ventricular end-diastolic pressure gradients of 10 mmHg or more tended to have smaller $A_{\max}$ values than patients with an end-diastolic gradient across the pulmonary valve of

less than 10 mmHg. Of the 6 patients with pulmonary arterial hypertension who had an identifiable a dip, 4 had a raised right ventricular end-diastolic pressure with reduced pulmonary arterial-right ventricular end-diastolic pressure gradient.

b-c SLOPE

Nanda and associates (1974) reported that the b-c slope was less steep in normal subjects than in patients with pulmonary hypertension. By contrast, Kerber and Maximov (1976) showed in dogs that the b-c slope failed to increase with an acute increase in mean pulmonary arterial pressure. Kaku *et al.* (1978) found that the b-c slope did not help to identify patients with pulmonary hypertension. Our data suggest that patients with pulmonary hypertension do have steeper b-c slopes than normals ($P=0.006$) but there is great overlap. When used as a criterion for pulmonary hypertension, a very rapid slope of 450 mm/s or more was found to be 100 per cent specific but sensitivity was only 33 per cent. The more rapid b-c slope in some of these patients may be explained by prolongation of the

Fig. 11 Right ventricular pre-ejection period corrected for heart rate ($RPEP/\sqrt{R-R}$). This is little better than RPEP alone for identifying patients with pulmonary hypertension, but is somewhat more sensitive. PAP, pulmonary artery mean pressure; NSR, normal sinus rhythm; A. fib., atrial fibrillation.

right ventricular pre-ejection period (see below). Under these conditions pulmonary valve opening occurs later, at a time when right ventricular dP/dt is higher, thereby leading to the more rapid opening of the pulmonary valve.

b-c OPENING AMPLITUDE

The pulmonary valve b-c opening amplitude in pulmonary hypertension was examined by Weyman *et al.* (1974b), who found no significant difference from normal, and by Nanda *et al.* (1974) who found that normal subjects had significantly smaller amplitudes (Nanda *et al.*, 1974). The b-c opening amplitude was measurable in fewer than half of all our normal subjects and patients. The overlap was considerable with no significant difference in mean opening amplitude between the 2 groups. However, none of the normal subjects had b-c opening amplitudes of greater than 13 mm as compared with 5 of 11 (45%) patients with raised PA mean pressures. Thus, a b-c opening amplitude more than 13 mm was by itself a useful criterion with 100 per cent specificity and 45 per cent sensitivity. Though a rapid b-c slope might be expected to occur in pulmonary stenosis, such an abnormality has not been described (Weyman *et al.*, 1974a). Further, in the presence of sinus rhythm, the amplitude of the a dip would distinguish pulmonary hypertension from pulmonary stenosis. The b-c opening amplitude is probably related to the anatomical size of the pulmonary artery and an increased value should therefore be related to radiographic evidence of an enlarged pulmonary artery.

MIDSYSTOLIC NOTCHING

Midsystolic notching of the pulmonary valve produces a 'W' configuration during systole and has been reported only in patients with pulmonary hypertension (Sakamoto *et al.*, 1974; Weyman *et al.*, 1974b). We also found this pattern to be very specific (100%) for pulmonary hypertension.

The sensitivity of this sign in patients with sinus rhythm was 38 per cent (5/13). It occurred in all 4 of the patients with the highest PA mean pressure and in the 4 patients with the highest PA systolic pressures (74 to 104 mmHg). The genesis of this abnormality is unknown. It has been shown by Sakamoto *et al.* (1974) in patients with pulmonary hypertension and pulmonary systolic murmurs that the end of the murmur coincides with the peak of the midsystolic notch (the tip of the centre of the 'W'). They also observed a systolic click occasionally occurring at the same time, believed to result from the sudden checking of pulmonary cusp motion.

RIGHT VENTRICULAR SYSTOLIC TIME INTERVALS

Nanda *et al.* (1974) reported that the right ventricular pre-ejection period (corrected for heart rate) was prolonged in the presence of pulmonary hypertension. Hirschfeld *et al.* (1975) found that both the right ventricular pre-ejection period and the right ventricular ejection time shortened with increasing heart rates and with decreasing age in their paediatric population. They reported that the ratio RPEP/RVET was independent of heart rate and age and correlated well with the PA diastolic and mean pressures and with the pulmonary vascular resistance. As the ratio increased so did the percentage of patients with PA diastolic pressures greater than 25 mmHg. Mills *et al.* (1975) measured the time between tricuspid valve closure and pulmonary valve opening by echocardiography (right ventricular isovolumic contraction time) and reported prolongation of this interval in patients with raised PA end-diastolic pressure.

In our study right ventricular pre-ejection period was useful in identifying patients with pulmonary hypertension. This measurement was especially helpful because it could be recorded in all 48 of the echocardiograms analysed. A right ventricular pre-ejection period of more than 0.095 s was found in none of the normals but occurred in 10 of 19 (53%) with pulmonary hypertension. As an ultrasound criterion for pulmonary hypertension, RPEP had 100 per cent specificity and 53 per cent sensitivity. Right ventricular pre-ejection period normalised for heart rate also had a 100 per cent specificity and improved sensitivity (68%).

Right ventricular ejection time could not be measured in any of the normal subjects or patients with normal PA mean pressure. It could be measured in only 47 per cent (9/19) of those with pulmonary hypertension. Though values were obtained for the ratio of right ventricular pre-ejection period to right ventricular ejection time, the inability to measure the latter in all normal subjects and in many patients reduced its usefulness in the identification of pulmonary hypertension.

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