Determination of left ventricular residual function by analysis of post-extrasystolic beat in mitral stenosis

P D PAPADOPOULOS, P C TOUTOUZAS, G SPANOS, C KOUROUKLIS,
A S PAPADOPOULOS, D G AVGOUSTASKIS

From the Department of Cardiology, University of Athens, Hippokration Hospital, Athens, Greece

SUMMARY Residual function of the left ventricle was assessed in 25 patients with mitral stenosis and a normal left ventriculogram. The post-extrasystolic beat (R_2) in sinus rhythm (nine patients) and the first beat after an early beat (R_2) in atrial fibrillation (16 patients) were analysed angiographically. Five subjects with a normal heart (controls) were also studied. The results are expressed as percentage changes in left ventricular contractility from the beat preceding the extra beat (R_1) to the beat R_2 . In the control group the mean changes from R_1 to R_2 were: end diastolic volume +68.3% (increase), end systolic volume -21.7% (decrease), ejection fraction +36.2%, mean systolic ejection rate +22.1%, and mean velocity of circumferential fibre shortening +31%. A significant increase in proportional systolic shortening of all left ventricular axes was found in R_2 compared with R_1 . In five patients with sinus rhythm and nine with atrial fibrillation the results fell within the normal range. In the remaining patients the beat R_2 indicated signs of poor left ventricular function. The mean changes from R_1 to R_2 in the patients with sinus rhythm and those with atrial fibrillation were respectively: end diastolic volume +47.8% and +36.6%, end systolic volume +20% and +27%, ejection fraction +12.5% and +6.2%, mean systolic ejection rate -23.3% and -30.2%, and mean velocity of circumferential fibre shortening -25.5% and -39.2%. The increase in the left ventricular axial systolic shortening was not significant.

Thus analysing a post-extrasystolic beat in sinus rhythm or the beat following an early beat with a long diastole in atrial fibrillation is a valuable method of determining the residual function in patients with mitral stenosis who have a normal left ventriculogram in basic rhythm.

Angiographic studies have shown that in patients with mitral stenosis the performance of the left ventricle may be either normal¹⁻³ or reduced.⁴ Dyskinesia of the posterior part and occasionally other areas of the left ventricular wall may be responsible for left ventricular dysfunction.⁵⁻⁷ In the present study we assessed the residual function of the left ventricle in those patients with mitral stenosis who had a normal left ventriculogram. For this purpose we analysed the post-extrasystolic beat in patients with mitral stenosis in sinus rhythm and also the first beat after an early beat in those with atrial fibrillation.

Patients and methods

Twenty five patients with mitral stenosis and five controls without heart disease (eight men and 22 women, age ranges 14-64, mean 44 years) were studied. Nine patients were in sinus rhythm and 16 had atrial fibrillation. A complete haemodynamic and angiographic study was carried out in all 30 cases. The controls consisted of patients undergoing tests because of a mild systolic murmur: all five were found to have healthy hearts. Table 1 shows the haemodynamic results. Left ventriculograms were obtained in the right anterior oblique position by injecting 50 ml of radiopaque material (hypaque 75%) over 2 or 3 seconds. Recordings were made simultaneously on 35 mm film with a speed of 40 frames/s. The criteria for including patients with sinus rhythm in the study were: (a) the appearance of an extra beat (R') on the

Requests for reprints to Dr Peter Papadopoulos, Department of Cardiology, Hippokration Hospital, Vassilissis Sofias Ave 114, Athens, Greece.

Table 1 Clinical and haemodynamic data in 25 patients with mitral stenosis (cases 6-30) and five controls (cases 1-5)

Case No	Age (yr)	Sex	Diagnosis	NYHA clinical class	Pressure (mm Hg)			Cardiac index (l/min/m ²)
					LV	BA	PC	
Good left ventricular residual function								
1	14	F			100/6	100/65	10/3-6	3.8
2	21	M			110/4	135/70	10/1-5	2.70
3	45	M			105/10	100/110	13/7-10	3.8
4	54	M			125/8	125/70		3.0
5	24	F			140/5	150/70		3.3
6	39	F	MS, SR	I-II	130/8	99/55	14/6-10	2.2
7	51	M	MS, SR	II	160/5	160/100	20/4-11	2.90
8	35	F	MS, SR	II	140/9	130/90	35/15-22	2.0
9	57	F	MS, SR	II	150/6	140/85	30	3.0
10	36	F	MS, SR	II	130/7	120/70	25/4-12	2.92
11	32	M	MS, AF	II	115/8	105/65		3.35
12	53	F	MS, AF	I-II	160/6	160/80	30/10-21	2.37
13	39	F	MS, AF	II	125/10	120/75		3.7
14	36	M	MS, AF	II	110/10	115/65	25	2.19
15	52	F	MS, AF	II	120/4	115/70		2.54
16	64	F	MS, AF	II	160/10	160/100	17	2.17
17	50	F	MS, AF	II	195/12	180/115	30	2.36
18	54	F	MS, AF	I-II	155/10	150/80	20/10-13	2.29
19	37	F	MS, AF	II	100/6	100/70		3.73
Impaired left ventricular residual function								
20	22	F	MS, SR	I-II	107/9	105/70	35/17-28	2.79
21	40	F	MS, SR	II	130/11	140/89	25/12-16	2.0
22	42	F	MS, SR	II	110/8	130/90		2.3
23	50	F	MS, SR	I-II		130/70	14/4-7	3.7
24	53	F	MS, AF	II	127/7	125/68	25/12-16	2.93
25	54	M	MS, AF	II-III	130/7	130/80		2.26
26	46	F	MS, AF	II	145/5	145/80		3.44
27	50	F	MS, AF	II	150/5	160/90		2.30
28	54	M	MS, AF	II	145/2	145/90		3.6
29	61	F	MS, AF	II	110/14	110/50	20/10-17	2.05
30	64	F	MS, AF	II	175/5	170/95		3.1

MS, mitral stenosis; SR, sinus rhythm; AF, atrial fibrillation; LV, left ventricle; BA, brachial artery; PC, pulmonary capillary.

ventriculogram; and (b) the location of R' in relation to the preceding sinus beat (R₁) and the following sinus beat (R₂) being such that R₁R' < R' R₂.

In atrial fibrillation an early beat (Re) had to be identified to serve as the extra beat. In these cases the criteria were: (a) the R₁Re interval had to be at most half of the ReR₂ interval; and (b) the length of the cardiac cycle preceding the R₁ had to be about equal to the mean cycle length of the patient at the time of the study. In all the cases selected the patients' symptoms were in class II (New York Heart Association), and their left ventriculograms in basic rhythm showed the same pattern.

In each left ventriculogram we assessed the performance of the left ventricle on R₁ and R₂ beats separately by determining (a) left ventricular end diastolic and end systolic volume; (b) left ventricular contractility; and (c) wall synergy of the left ventricle.

Left ventricular end diastolic and end systolic volume

These were measured⁸⁻¹⁰ using the formula $V=0.52 LD_2$, where V was the volume in cm³, L the longitudinal axis from the middle of the aortic root to the left

ventricular apex in cm, and D the middle transverse left ventricular axis calculated from the formula

$$D = \frac{4A}{\pi L} \text{ where } A \text{ was the ellipsoid surface}$$

measured by planimetry and $\pi=3.14$. In all cases two values of end systolic volume and two values of end diastolic volume (beats R₁, R₂) were obtained. In the results the values obtained from R₂ were expressed as percentages of the corresponding volumes of the beat R₁.

Left ventricular contractility

Ejection fraction was determined using the formula¹¹⁻¹³: left ventricular ejection fraction =
$$\frac{\text{end diastolic volume} - \text{end systolic volume}}{\text{end diastolic volume}}$$

Mean systolic ejection rate in end diastolic volumes per second (EDV/s) was determined using the formula¹⁴: mean systolic ejection rate =
$$\frac{\text{end diastolic volume} - \text{end systolic volume}}{\text{ejection time} \times \text{end diastolic volume}}$$

The ejection time was determined by measuring the time interval from end diastole to end systole by means of simultaneous pressure tracings and subtracting 50 ms corresponding to the left ventricular isovolumic contraction time.

Mean velocity of circumferential fibre shortening (VCF) was determined by the formula:

$$VCF = \frac{Dd - Ds}{ET \times Dd}$$
, where Dd was the mean transverse diameter of the left ventricle in diastole; Ds the mean left ventricular transverse diameter in systole; and ET the ejection time.¹⁵ In all cases the values of the left ventricular contractility indices obtained from beat R_2 were expressed as percentage values obtained from beat R_1 .

Left ventricular wall synergy

This was assessed by determining the extent of the percentage shortening of the left ventricular longitudinal axis and six transverse hemiaxes in systole¹⁶ (Fig. 1). The last hemiaxes were obtained by drawing three lines (basal, medial, and apical) perpendicularly to the longitudinal axis at points one quarter, one half, and three quarters of the way along its length. Thus we had three anterior and three posterior transverse hemiaxes of the left ventricle. The values of axial shortening of the left ventricular wall obtained from beat R_2 were expressed as percentage values of those obtained from R_1 .

Finally the coupling interval was measured in patients with sinus rhythm. In atrial fibrillation the R_1Re and ReR_2 intervals were measured and the ratio $R_1Re:ReR_2$ was calculated.¹⁷

Results

Tables 2 and 3 summarise the results of measurements in the five subjects with a normal heart (controls), the nine patients with mitral stenosis and sinus rhythm, and the 16 patients with mitral stenosis and atrial fibrillation.

CONTROLS

End diastolic volume, end systolic volume indices of left ventricular contractility, and assessment of wall motion of the left ventricle in longitudinal and transverse axes in all subjects fell within the normal range. The mean ($\pm$ SD) increase in end diastolic volume from R_1 to R_2 was 68.3% ($\pm$ 19.8%) of end diastolic volume of beat R_1 . The mean ($\pm$ SD) decrease in end systolic volume was 21.7% ($\pm$ 6.8%) of the corresponding volume of R_1 . The mean ($\pm$ SD) ejection fraction in beat R_1 was 58.8% ($\pm$ 2.2%) and in beat R_2 80.1% ($\pm$ 2.7%), a mean increase of 36.2%.

The mean systolic ejection rate was 2.85 ($\pm$ 0.1) EDV/s in R_1 and 3.48 ($\pm$ 0.2) EDV/s in R_2 , a mean

increase of 22.1%. The mean velocity of the circumferential myocardial fibre shortening was 1.58 ($\pm$ 0.08) circumferences/s in R_1 and 2.07 ($\pm$ 0.2) cir/s in R_2 , a mean increase of 31% (Fig. 2a).

Regarding the left ventricular axial changes, a significant increase of proportional systolic shortening of all axes was found in R_2 compared with R_1 . In R_2 the mean longitudinal axis and the mean anterior and the mean posterior transverse hemiaxes were shortened by 27%, 70%, and 27% respectively, whereas in R_1 the corresponding values were 11%, 46%, and 26%. The coupling time—that is, the interval between R_1 and the extra beat—was 0.346 ($\pm$ 0.07) s; this time correlated with the percentage changes in the left ventricular ejection fraction ($r=0.89$, $p<0.005$).

PATIENTS WITH MITRAL STENOSIS

Left ventricular residual function

In five of the nine patients with sinus rhythm and nine of the 16 with atrial fibrillation the results of left ventricular function in R_2 were similar to those in the controls (Fig. 2a).

The end diastolic volume in beat R_2 showed a mean increase of 44.8 ($\pm$ 9.9%) over the corresponding volume in beat R_1 in patients with sinus rhythm and of 63.3 ($\pm$ 15.3%) in patients with atrial fibrillation, whereas the end systolic volume in R_2 decreased by 30.6 ($\pm$ 8.9%) in sinus rhythm and 22.1 ($\pm$ 5.1%) in atrial fibrillation (Table 2).

Similarly, the beat R_2 compared with the beat R_1

Fig. 1 Measurement of left ventricular axes in systole (dotted lines) and diastole (continuous line). E, longitudinal axis; B, M, K, transverse axes and hemiaxes.

Table 2 Changes in end diastolic volume (EDV) and end systolic volume (ESV) of the left ventricle in R_2 compared with R_1 in patients with good and impaired residual left ventricular function

Case No	EDV (cm ³)			ESV (cm ³)		
	R_1	R_2	% Change	R_1	R_2	% Change
<i>Good left ventricular residual function</i>						
Controls						
1	88	107	+ 21.6	37	22	-40.5
2	140	205	+ 46.4	66	61	- 7.6
3	140	198	+ 41.4	52	40	-23
4	120	242	+101.7	54	34.7	-35.7
5	118	268	+127	42	39	- 7.1
Mitral stenosis (SR)						
6	108	156	+ 44.4	51	45	-11.8
7	102	182	+ 78.4	38	25	-34.2
8	70	88	+ 25.7	39	15	-61.5
9	93	114	+ 22.6	47	39	-17
10	76	110	+ 44.7	27	16	-40.7
Mitral stenosis (AF)						
11	69	118	+ 71	25	22	-12
12	60	115	+ 91.6	23	22	- 4.3
13	55.7	115	+106	20	19	- 5
14	120	180	+ 50	64	50	-22
15	105	205	+ 95	60	45	-25
16	134	165	+ 23	65	40	-38.4
17	160.5	186	+ 16	89	61	-31
18	140.5	190	+ 35	72	70	- 2.8
19	113	292	+158	46	32.2	-30
<i>Impaired left ventricular residual function</i>						
Mitral stenosis (SR)						
20	92	120	+ 30	46	48	+ 4
21	68	92	+ 35	32	36	+12
22	119	176	+ 47.8	50	85	+70
23	188	302	+ 60.6	62.4	68	+ 9
Mitral stenosis (AF)						
24	124	180	+ 45	62	86	+38.7
25	135	170	+ 26	58	70	+20.6
26	223	348	+ 56	105	150	+43
27	210	275	+ 31	120	150	+25
28	91.6	163	+ 78	38	62	+63
29	180	220	+ 22.2	95	100	+ 5.2
30	133	145	+ 9	54	58	+ 7.4

SR, sinus rhythm; AF, atrial fibrillation.

had an increase in ejection fraction of $42.8 \pm 10.8\%$ in sinus rhythm and of $34.9 \pm 12.9\%$ in atrial fibrillation, an increase in mean systolic ejection rate of $28.6 \pm 9.6\%$ in sinus rhythm and of $20 \pm 2.6\%$ in atrial fibrillation, and an increase in mean velocity of the circumferential myocardial fibre shortening of $28.1 \pm 7\%$ in sinus rhythm and $49.6 \pm 14.1\%$ in atrial fibrillation (Table 3).

A greater systolic shortening of the left ventricular axes in beat R_2 compared with R_1 was found. Thus in patients with sinus rhythm the mean longitudinal axis, the mean transverse anterior, and the mean transverse posterior axis in R_2 were shortened by 33%, 43%, and 43% respectively, whereas the corresponding values in R_1 were 17.2%, 32%, and 23.5%. In patients with atrial fibrillation the respective values in R_2 were 27.4%, 44.6%, and 24% whereas those in R_1 were 17.6%, 39%, and 17%.

The coupling time was 0.440 ± 0.2 s in sinus

rhythm; this correlated poorly with left ventricular ejection fraction, possibly because of the prolonged coupling time in these patients. In patients with atrial fibrillation the ratio $R_1\text{Re:Re}R_2$ interval was 0.548 ± 0.193 . An excellent correlation was found between this ratio and percentage changes of left ventricular ejection fraction ($r=0.93$, $p<0.001$).

Impaired left ventricular residual function

In the remaining four patients with sinus rhythm and seven with atrial fibrillation the beat R_2 indicated poor left ventricular function. The R_2 showed an increase in end diastolic volume of $47.8 \pm 3.7\%$ in sinus rhythm and of $36.9 \pm 6.8\%$ in atrial fibrillation and, in contrast to the controls, an increase in end systolic volume of $20\% \pm 4.3\%$ in sinus rhythm and of $27\% \pm 7.7\%$ in atrial fibrillation (Fig. 2b). Also in R_2 the ejection fraction increased slightly by $12.5\% \pm 2.9\%$ in sinus rhythm and by $6.1\% \pm 4.3\%$ in atrial fibrillation, while

Fig. 2 Percentage changes in indices of left ventricular function in the post-extrasystolic beat in controls and patients with mitral stenosis in sinus rhythm (SR) and the corresponding changes in beat following an early beat with a long diastole in patients with mitral stenosis and atrial fibrillation (AF): (a) good left ventricular residual function; (b) reduced left ventricular residual function. EDV, end diastolic volume; ESV, end systolic volume; EF, ejection fraction; MSER, mean systolic ejection rate; VCF, mean velocity of circumferential fibre shortening.

the mean systolic ejection rate decreased by $23.3\% \pm 6.7\%$ in sinus rhythm and by $30.2\% \pm 6.4\%$ in atrial fibrillation, and the mean velocity of the circumferential myocardial fibre shortening by $25.5\% \pm 8.2\%$ in sinus rhythm and $39.28\% \pm 7.3\%$ in atrial fibrillation (Table 3, Fig. 2b).

The increase in axial systolic shortening of the left ventricle in R_2 was not significant in these patients compared with good left ventricular residual function. Thus in patients with sinus rhythm the longitudinal axis, the average transverse anterior and posterior left ventricular axes in R_2 were shortened by 20%, 40%, and 26% respectively, whereas the corresponding values in R_1 were 11%, 37%, and 22%. In patients with atrial fibrillation the respective values in R_2 were 21%, 48%, and 27% and in R_1 20%, 52%, and 23%.

The coupling time was 0.431 ± 0.03 s in sinus rhythm. In patients with atrial fibrillation the ratio

$R_1Re:ReR_2$ was 0.560 ± 0.232 . Neither the coupling time in patients with sinus rhythm nor the $R_1Re:ReR_2$ ratio in those with atrial fibrillation correlated well with the percentage changes in left ventricular ejection fraction.

Discussion

Normally the post-extrasystolic beat is characterised by a pronounced increase in the force of left ventricular contraction.¹⁸⁻²⁰ This is attributed mainly to the increased influx of calcium ions into the myocardial cell caused by the temporary change in heart rate^{21 22} and also to the increased left ventricular preload.²³

It has been shown that with electrical paired pacing, when the second myocardial depolarisations are so close that they have no mechanical effect, there is

Table 3 Left ventricular ejection fraction, mean systolic ejection rate, and mean velocity of circumferential fibre shortening in R_2 compared with R_1 in patients with good and impaired left ventricular residual function

Case No	Ejection fraction			Mean systolic ejection rate (EDV/s)			Mean velocity of circumferential fibre shortening (cir/s)		
	R_1	R_2	% Change	R_1	R_2	% Change	R_1	R_2	% Change
<i>Good left ventricular residual function</i>									
Controls									
1	58	80	+38	2.9	3.53	+21.7	1.52	1.70	+ 11.8
2	53	70	+32	3.09	3.52	+14	1.56	1.92	+ 23
3	63	80	+27	2.28	2.46	+ 7.8	1.3	1.59	+ 22.3
4	55	85.6	+55.6	3.15	3.89	+23.4	1.80	2.25	+ 25
5	65	85	+30.7	2.87	4.0	+39.3	1.75	2.9	+ 65.7
Mitral stenosis (SR)									
6	53	71	+34	2.01	2.03	+ 1	0.98	1.13	+ 15.3
7	63	86	+36.5	2.5	3.45	+38	1.87	2.30	+ 23
8	44	83	+88.6	2.53	3.68	+45.4	1.81	2.24	+ 23.7
9	49	65	+32.6	2.19	3.29	+50	0.96	1.5	+ 56.2
10	64	85	+32.8	3.9	4.4	+13	2.06	2.65	+ 28.6
Mitral stenosis (AF)									
11	63	81	+28	2.40	3.10	+29	1.4	2.3	+ 64
12	61.6	80	+29.8	2.65	3.28	+23	1.2	3.08	+156
13	64	83.4	+30	2.32	2.75	+18	1.20	1.75	+ 46
14	46	72	+56	2.10	2.45	+16.6	1.35	1.84	+ 36
15	57	71	+24.5	3.42	3.53	+ 3.2	1.48	1.86	+ 25.6
16	51	75	+47	2.25	2.90	+29	1.75	1.90	+ 8.5
17	45	67	+50	1.78	2.25	+26.4	0.81	0.87	+ 7.4
18	48	63	+31	1.95	2.40	+23	1.10	1.52	+ 38
19	71	84	+18	3.17	3.76	+19	1.76	2.87	+ 63
<i>Impaired left ventricular residual function</i>									
Mitral stenosis (SR)									
20	50	60	+20	3.33	2	-40	1.56	0.88	- 43
21	53	61	+15	2.94	2.06	-29	1.45	0.90	- 38
22	58	51	+12	4.38	4.03	- 8	2.29	2.16	- 6
23	66.8	77	+15.2	3.44	2.76	-20	1.92	1.42	- 26
Mitral stenosis (AF)									
24	50	52	+ 4	2.46	1.32	-46	1.29	0.44	- 65
25	57	58.8	+ 3.21	3.2	2.7	-15.6	1.2	0.98	- 18.3
26	53	57	+ 7.5	4.24	2.85	-32	2.97	1.34	- 54.8
27	43	45.4	+ 4.6	3.1	2.75	-11.2	1.8	1.2	- 33
28	58	62	+ 6.8	5.15	2.26	-56	2.46	1.12	- 54
29	47	54.5	+15.8	2.55	2.05	-19	2.4	1.78	- 25.8
30	59	60	+ 1.7	3.82	3.17	-17	2.20	1.82	- 17

EDV, end diastolic volume; cir, circumferences; SR, sinus rhythm; AF, atrial fibrillation.

an increased contraction on the following beat. The same post-extrasystolic potentiation phenomenon may occur in atrial fibrillation with a high ventricular rate, when a very early ventricular depolarisation may produce no mechanical reaction, while the following beat has a forceful ventricular contraction.²⁴⁻²⁵ These findings were corroborated by Shabetai *et al* in a study of healthy men and patients with different heart rates induced by electrical pacing.²⁶ They concluded that the increase in myocardial contractility in sinus rhythm or atrial fibrillation is due to changes in the heart rate. A different explanation was proposed by Karliner *et al*, who studied patients with mitral stenosis and atrial fibrillation.²³ These investigators considered that the variations in left ventricular performance were mainly related to the changes of left ventricular end diastolic volume and, to a lesser degree, to temporary changes in heart rate.

Studies of coronary heart disease have shown that a premature ventricular contraction often leads to an increase in left ventricular contraction of the following beat and may activate some viable areas in the asynergic myocardium.²⁷⁻²⁹ This feature is regarded as prognostic of the functional revascularisation of the myocardium.³⁰⁻³² In chronic rheumatic valvular disease several workers³³⁻³⁵ observed that the ventricular pressures of the post-extrasystolic beat did not change in patients without signs of heart failure, whereas in most patients with heart failure the ventricular systolic pressure increased in that beat.

The results of the present study indicate that post-extrasystolic potentiation can be used to determine left ventricular residual function. Thus in patients with mitral stenosis with the same clinical symptoms, haemodynamic data, and the same pattern of left ventricular cineangiogram the post-extrasystolic potentia-

Fig. 3 Correlation of the $R_1Re:ReR_2$ ratio with the percentage change in ejection fraction ($r = 0.961$, $p < 0.0001$) in nine patients with good left ventricular function ($\blacktriangle$) ($r = -0.961$, $y = -64.022 \times +69.999$, $SEE\ 3.783$, $t = -9.257$, $p < 0.0001$) and in seven patients with reduced left ventricular function ($\bullet$) ($r = 0.445$, $y = 7.732 \times +1.704$, $SEE\ 4.255$, $t = 1.111$, NS). The difference in the slopes of the intercept between the two groups was highly significant ($t = 7.337$, $p < 0.00006$). R_1Re , cycle length between the early (Re) and the preceding (R_1) beat; ReR_2 , cycle length between the Re and the following (R_2) beat.

tion is different. Those patients with mitral stenosis and with no heart disease, who had an increase in the left ventricular ejection indices and a decrease in the end systolic volume in the post-extrasystolic beat, were characterised as having good left ventricular residual function (Fig. 2a). In contrast the patients with mitral stenosis and reduced left ventricular residual function showed a decrease in the mean systolic ejection rate, mean velocity of the circumferential myocardial fibre shortening, and an increase in the end systolic volume in beat R_2 (Fig. 2b). In these patients the left ventricular ejection fraction increased by a small percentage compared with that in patients with good left ventricular residual function.

In atrial fibrillation the changes in the RR intervals may be regarded as similar to those induced by ventricular extrasystoles in sinus rhythm; an early atrial beat with the following long diastole resembles the coupling interval and the following pause of the ventricular extrasystole in sinus rhythm. Rogel and Mahler provoked atrial fibrillation experimentally

and studied the myocardial systolic tension of one beat (R_2) in relation to the duration of the preceding cardiac cycle (ReR_2) and the cycle before (R_1Re).¹⁷ They found that the myocardial systolic tension was mainly related to the duration of the R_1Re interval which corresponds to the coupling interval of a ventricular extrasystole in sinus rhythm and to a lesser degree with the ReR_2 interval corresponding to the pause of the ventricular extra beat. It is maintained that the smaller the ratio $R_1Re:ReR_2$ the greater the contraction in R_2 . The experimental studies of Edmands *et al* led to similar results.²⁵

Our findings in patients with mitral stenosis and atrial fibrillation agree with the experimental studies of Rogel and are consistent with the explanation that changes in left ventricular contraction are due to temporary changes in heart rate. We found that the increase in left ventricular contraction in R_2 was clearly related to the duration of the two preceding cardiac cycles, particularly to the ratio of the R_1Re interval to the ReR_2 interval: the lower the ratio the greater the left ventricular contraction. The correlation between this ratio and the percentage change in left ventricular ejection fraction was excellent ($r = -0.961$, $p < 0.0001$) in the nine patients with good left ventricular function whereas it was poor ($r = 0.445$, NS) in the seven patients with impaired left ventricular function (Fig. 3).

In conclusion, an analysis of a post-extrasystolic beat in sinus rhythm or the beat following an early beat with a long diastole in atrial fibrillation, is a valuable method of determining the residual left ventricular function in patients with mitral stenosis who have a normal ventriculogram in basic rhythm. Although in this study we carried out detailed measurements, it was usually apparent from the left ventriculogram alone. Observation of this beat can provide accurate information for assessing left ventricular function in patients with mitral stenosis which may be particularly useful when surgery is being considered.

References

- 1 Feigenbaum H, Campbell RW, Wunsch CM, Steinmetz EF. Evaluation of the left ventricle in patients with mitral stenosis. *Circulation* 1966; **34**: 462-72.
- 2 Horwitz LD, Mullins CB, Payne RM, Curry GC. Left ventricular function in mitral stenosis. *Chest* 1973; **64**: 609-14.
- 3 Curry GC, Elliott LP, Ramsey HW. Quantitative left ventricular angiocardiographic findings in mitral stenosis. *Am J Cardiol* 1972; **29**: 621-7.
- 4 Hildner FJ, Javier RP, Cohen LS, *et al*. Myocardial dysfunction associated with valvular heart disease. *Am J Cardiol* 1972; **30**: 319-26.
- 5 Ryan TH. Mitral valve disease. In: Levine HJ, ed.

- Clinical cardiovascular physiology*. New York: Grune-Stratton, 1976: 523-61.
- 6 Selzer A, Cohn K. The "myocardial factor" in valvular heart diseases. *Cardiovasc Clin* 1973; 5: 177-89.
 - 7 Heller SJ, Carleton RA. Abnormal left ventricular contraction in patients with mitral stenosis. *Circulation* 1970; 42: 1099-110.
 - 8 Kennedy JW, Baxley WA, Figley MM, Dodge HT, Blackmon JR. Quantitative angiocardiology. 1. The normal left ventricle in man. *Circulation* 1966; 34: 272-8.
 - 9 Greene DG, Carlisle R, Grant C, Bunnell IL. Estimation of left ventricular volume by one-plane cine-angiography. *Circulation* 1967; 35: 61-9.
 - 10 Sandler H, Dodge HT. The use of single plane angiocardigrams for the calculation of left ventricular volume in man. *Am Heart J* 1968; 75: 325-34.
 - 11 Krayenbühl HP, Bussmann WD, Turina M, Lüthy E. Is the ejection fraction an index of myocardial contractility? *Cardiologia* 1968; 53: 1-10.
 - 12 Cohn PF, Gorlin R, Cohn LH, Collins JJ Jr. Left ventricular ejection fraction as a prognostic guide in surgical treatment of coronary and valvular heart disease. *Am J Cardiol* 1974; 34: 136-41.
 - 13 Tyers GF, Williams DR, Babb JD, Levenson L, Zelis RF, Waldhausen JA. The changing status of ejection fraction as a predictor of early mortality following surgery for acquired heart disease. *Chest* 1977; 71: 371-5.
 - 14 Peterson KL, Skloven D, Ludbrook P, Uther JB, Ross J Jr. Comparison of isovolumic and ejection phase indices of myocardial performance in man. *Circulation* 1974; 49: 1088-101.
 - 15 Karlner JS, Gault JH, Eckberg D, Mullins CB, Ross J Jr. Mean velocity of fiber shortening. A simplified measure of left ventricular myocardial contractility. *Circulation* 1971; 44: 323-33.
 - 16 Herman MV, Heinle RA, Klein MD, Gorlin R. Localized disorder in myocardial contraction. Asynergy and its role in congestive heart failure. *N Engl J Med* 1967; 277: 222-32.
 - 17 Rogel S, Mahler Y. Consumption and cumulation of the inotropic effect of depolarization. *Isr J Med Sci* 1970; 6: 90-102.
 - 18 Hoffman BF, Bindler E, Suckling NE. Postextrasystolic potentiation of contraction in cardiac muscle. *Am J Physiol* 1956; 185: 95-106.
 - 19 Fisher VJ, Lee RJ, Marlon AM, Kavalier F. Paired electrical stimulation and the maximal contractile response of the ventricle. *Circ Res* 1967; 20: 520-33.
 - 20 Braunwald E, Ross J, Frommer PL, Williams JF Jr, Sonnenblick EH, Gault JH. Clinical observations on paired electrical stimulation of the heart. Effects on ventricular performance and heart rate. *Am J Med* 1964; 37: 700-11.
 - 21 Anderson PAW, Manring A, Johnson EA. Force-frequency relationship. A basis for a new index of cardiac contractility? *Circ Res* 1973; 33: 665-71.
 - 22 Arentzen CE, Rankin JS, Anderson PAW, Feezor MD, Anderson RAW. Force-frequency characteristics of the left ventricle in the conscious dog. *Circ Res* 1978; 42: 64-71.
 - 23 Karlner JS, Gault JH, Bouchard RJ, Holzer J. Factors influencing the ejection fraction and the mean rate of circumferential fibre shortening during atrial fibrillation in man. *Cardiovasc Res* 1974; 8: 18-25.
 - 24 Siebens AA, Hoffman BF, Craneffeld PF, Brooks MC. Regulation of contractive force during ventricular arrhythmias. *Am J Physiol* 1959; 197: 971-7.
 - 25 Edmands RE, Greenspan K, Bailey JC. Role of the premature action potential in contractive potentiation: a study of paired stimulation. *Cardiovasc Res* 1972; 6: 368-74.
 - 26 Shabetai R, Uther JB, Peterson KL, Ross J Jr. Potentiation of contraction by premature activation in patients with normal and abnormal left ventricular function [Abstract]. *Am J Cardiol* 1972; 29: 291.
 - 27 Cohn PF, Gorlin R, Herman MV, et al. Relation between contractive reserve and prognosis in patients with coronary artery disease and a depressed ejection fraction. *Circulation* 1975; 51: 414-27.
 - 28 Hamby RI, Aintablian A, Wisoff BG, Hartstein ML. Response of the left ventricle in coronary artery disease to postextrasystolic potentiation. *Circulation* 1975; 51: 428-35.
 - 29 Dyke SH, Cohn PF, Gorlin R, Sonnenblick EH. Detection of residual myocardial function in coronary artery disease using postextrasystolic potentiation. *Circulation* 1974; 50: 694-9.
 - 30 Popio KA, Gorlin R, Bechtel B, Levine JA. Postextrasystolic potentiation as a predictor of potential myocardial viability: preoperative analysis compared with studies after coronary by-pass surgery. *Am J Cardiol* 1977; 39: 944-53.
 - 31 Boden WE, Liang C-S, Hood WB Jr. Postextrasystolic potentiation of regional mechanical performance during prolonged myocardial ischemia in the dog. *Circulation* 1980; 61: 1063-70.
 - 32 Cohn PF. Evaluation of inotropic contractile reserve in ischemic heart disease using postextrasystolic potentiation [Editorial]. *Circulation* 1980; 61: 1071-5.
 - 33 Beck MM, Chesler E, Schrire V. Postextrasystolic ventricular pressure responses. *Circulation* 1971; 44: 523-33.
 - 34 Kvasnička J, Liander B, Broman H, Varnauskas E. Quantitative evaluation of postectopic beats in the normal and failing human heart using indices derived from catheter-tip manometer readings. *Cardiovasc Res* 1975; 9: 336-41.
 - 35 Merillon JP, Motte G, Aumont MC, Masquet C, LeCarpentier Y, Gourgon R. Postextrasystolic left ventricular peak pressure with and without left ventricular failure. *Cardiovasc Res* 1979; 13: 338-44.