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

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SUMMARY Residual function of the left ventricle was assessed in 25 patients with mitral stenosis and a normal left ventriculogram. The post-extrasystolic beat (R2) in sinus rhythm (nine patients) and the first beat after an early beat (R1) 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 (R1) to the beat R2. In the control group the mean changes from R1 to R2 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 R2 compared with R1. In five patients with sinus rhythm and nine with atrial fibrillation the results fell within the normal range. In the remaining patients the beat R2 indicated signs of poor left ventricular function.

The mean changes from R1 to R2 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 (hipaque 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
Table 1  Clinical and haemodynamic data in 25 patients with mitral stenosis (cases 6–30) and five controls (cases 1–5)

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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 R₁R₂ < 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 measured using the formula V = 0.52 L/D₂, 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 = 4A/πL where A was the ellipsoid surface measured by planimetry and π = 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 R₁ were expressed as percentages of the corresponding volumes of the beat R₂.

**Left ventricular contractility**

*Ejection fraction* was determined using the formula: left ventricular ejection fraction = end diastolic volume – end systolic volume end diastolic volume

*Mean systolic ejection rate* in end diastolic volumes per second (EDV/s) was determined using the formula: mean systolic ejection rate = end diastolic volume – end systolic volume ejection time × 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. 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 systole (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 \(R_1, Re\) and \(ReR_2\) intervals were measured and the ratio \(R_1, Re:ReR_2\) was calculated.

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.

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±9.9% over the corresponding volume in beat \(R_1\) in patients with sinus rhythm and of 63.3±15.3% in patients with atrial fibrillation, whereas the end systolic volume in \(R_2\) decreased by 30.6±8.9% in sinus rhythm and 22.1±5.1% in atrial fibrillation (Table 2).

Similarly, the beat \(R_2\) compared with the beat \(R_1\)
had an increase in ejection fraction of 42.8 ± 10.8% in sinus rhythm and of 34.9 ± 12.9% in atrial fibrillation, an increase in mean systolic ejection rate of 28.6 ± 9.6% in sinus rhythm and of 20 ± 2.6% in atrial fibrillation, and an increase in mean velocity of the circumferential myocardial fibre shortening of 28.1 ± 7% in sinus rhythm and 49.6 ± 14.1% in atrial fibrillation (Table 3).

A greater systolic shortening of the left ventricular axes in beat R₂ compared with R₁ was found. Thus in patients with sinus rhythm the mean longitudinal axis, the mean transverse anterior, and the mean transverse posterior axis in R₂ were shortened by 33%, 43%, and 43% respectively, whereas the corresponding values in R₁ were 17.2%, 32%, and 23.5%. In patients with atrial fibrillation the respective values in R₂ were 27.4%, 44.6%, and 24% whereas those in R₁ 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 R₁R₂:ReR₂ 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 R₂ indicated poor left ventricular function. The R₂ showed an increase in end diastolic volume of 47.8 ± 3.7% in sinus rhythm and of 36.9 ± 6.8% in atrial fibrillation and, in contrast to the controls, an increase in end systolic volume of 20% ± 4.3% in sinus rhythm and of 27% ± 7.7% in atrial fibrillation (Fig. 2b). Also in R₂ the ejection fraction increased slightly by 12.5% ± 2.9% in sinus rhythm and by 6.1% ± 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%±6.7% in sinus rhythm and by 30.2%±6.4% in atrial fibrillation, and the mean velocity of the circumferential myocardial fibre shortening by 25.5%±8.2% in sinus rhythm and 39.28%±7.3% in atrial fibrillation (Table 3, Fig. 2b).

The increase in axial systolic shortening of the left ventricle in R2 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 R2 were shortened by 20%, 40%, and 26% respectively, whereas the corresponding values in R1 were 11%, 37%, and 22%. In patients with atrial fibrillation the respective values in R2 were 21%, 48%, and 27% and in R1 20%, 52%, and 23%.

The coupling time was 0.431±0.03 s in sinus rhythm. In patients with atrial fibrillation the ratio $R_1 Re: ReR_2$ was 0.560±0.232. Neither the coupling time in patients with sinus rhythm nor the $R_1 Re: ReR_2$ 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 rate21,22 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
an increased contraction on the following beat. The same postextrasystolic 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 Karlner 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 postextrasystolic 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 postextrasystolic 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 postextrasystolic potentia-
and studied the myocardial systolic tension of one beat (R₂) in relation to the duration of the preceding cardiac cycle (ReR₂) and the cycle before (R₁Re). ¹⁷

They found that the myocardial systolic tension was mainly related to the duration of the R₁Re interval which corresponds to the coupling interval of a ventricular extrasystole in sinus rhythm and to a lesser degree with the ReR₂ interval corresponding to the pause of the ventricular extra beat. It is maintained that the smaller the ratio RₐRe:ReR₂ the greater the contraction in R₂. 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 R₂ was clearly related to the duration of the two preceding cardiac cycles, particularly to the ratio of the R₁Re interval to the ReR₂ 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 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
Left ventricular residual function in mitral stenosis

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