Left ventricular function at similar heart rates during tachycardia induced by exercise and atrial pacing: an echocardiographic study

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SUMMARY   M mode echocardiography was used in 10 normal subjects to study left ventricular dimension and function variables at identical heart rates during tachycardia induced by supine bicycle exercise or atrial pacing. Echocardiographic data were analysed independently by two observers. The maximum heart rate reached during atrial pacing was lower (mean (1SD) 148 (17) beats/min) than that reached during exercise (mean (1SD) 167 (14) beats/min). The left ventricular end diastolic dimension was greater before supine exercise than before atrial pacing, probably as a result of leg raising. At each graded exercise step the end diastolic dimension remained greater than during atrial pacing and the differences became progressively greater with increasing heart rates. The left ventricular end systolic dimension was not significantly different at each step during the two stresses. During recovery the end systolic dimension was significantly smaller after exercise than at corresponding heart rates induced by atrial pacing. Left ventricular function indices—fractional shortening and peak rates of left ventricular systolic and diastolic dimensional change—were significantly higher during exercise than during atrial pacing and the differences increased with heart rate.

It is concluded that (a) the intervention used to change heart rate has an important effect on M mode echocardiographic left ventricular dimensions; (b) indices of left ventricular performance increase progressively during exercise and differ from those measured at the same heart rate during atrial pacing; (c) it is important to consider heart rate, stroke volume, and loading conditions when reference values are used and when the effects of a particular stress are to be interpreted.

Adaptation of the cardiovascular system to dynamic exercise results from integrated mechanisms including an increase in heart rate and contractility, changes in preload and afterload, and a decrease in peripheral resistance.[1] Echocardiography permits non-invasive assessment of left ventricular size and function in subjects at rest.[2][3] Exercise echocardiography has the important advantage of allowing continuous measurement of left ventricular function variables before, during, and immediately after exercise. Different types of stress and exercise have been studied echocardiographically, including isometric exercise[4]; handgrip[5]; and bicycle ergometer exercise in supine,[5][6] semisupine,[7][8] sitting,[9] and upright positions.[5][10][11]

M mode echocardiographic variables of the left ventricle are known to vary with heart rate,[12][13] but differences due to separate interventions have not been compared in the same subjects. The aim of the present study was to analyse left ventricular function at identical heart rates induced by supine bicycle exercise and atrial pacing.

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Subjects and methods

STUDY POPULATION

We studied ten healthy volunteers (medical students; mean age 24, range 20–41) with resting M mode echocardiograms of excellent quality. All had a normal history, physical examination, resting and exercise electrocardiogram, and chest x ray. We obtained informed consent from all of them. The subjects were not required to fast before the tests; hence studies were performed with normal volume loading of the left ventricle.

BICYCLE EXERCISE STRESS TEST

The 10 subjects lay supine on a table in a slightly left lateral position and were fixed at the shoulder level. They pedalled a bicycle ergometer at 60 revolutions/min. The initial workload was 50 W for three minutes; this was increased by 25 W every three minutes until the subject was exhausted. The mean (1 SD) duration of exercise was 22 (3) minutes and the maximal workload was 212 (30) W.

M mode echocardiograms were recorded before exercise with the subject's feet elevated in the cycling position, at the end of each three minute exercise period, and every minute during recovery. The heart rate was measured on the simultaneously recorded electrocardiogram. Brachial blood pressure was measured every minute with a mercury sphygmomanometer.

ATRIAL PACING STRESS TEST

The day after the exercise test, at nearly the same time, the 10 subjects were studied in the catheterisation laboratory. An electrode catheter (5F bipolar) was inserted into the right atrium via a left antecubital vein. The subjects rested until heart rate and blood pressure were stable. M mode echocardiograms were then recorded with the subject in the supine and slight left lateral decubitus position—

at rest and after a steady state period of 60 seconds of atrial pacing—at the same heart rates as those achieved at the end of each step of the exercise test. Atrial pacing was stopped in each subject when a 1:1 atrioventricular conduction failed. Arterial blood pressure was measured with a mercury sphygmomanometer during the recording of each echocardiogram.

ECHOCARDIOGRAPHIC STUDY AND ANALYSIS

M mode echocardiography was performed with a 2.25 MHz transducer placed at the left parasternal border in the same position that was marked during exercise and atrial pacing stress. The recordings were made at 50 mm/second paper speed during end expiration. We took care to record left ventricular echocardiograms below the tips of mitral valve leaflets with clearly defined echoes of the interventricular septum and the posterior wall endocardium and epicardium.

The echocardiograms were obtained at rest; at the end of each step of exercise test; at 2, 3, and 5 minutes during recovery; and at identical heart rates induced by atrial pacing. The tracings were analysed on a digitising tablet connected to a PDP 11 digital computer.[14] Three consecutive beats were independently traced by two observers who used the leading edge to leading edge method. The results were averaged to obtain a single value in order to avoid beat to beat variability.

The following measurements of the left ventricle were made: left ventricular end diastolic diameter (EDD) (in mm) at the onset of the QRS complex; left ventricular end systolic diameter (ESD) (mm) defined as the minimum diameter in systole; and fractional shortening

$$\left(\text{FS} \% = \frac{(\text{EDD} - \text{ESD})}{\text{EDD}} \times 100 \right).$$

The following indices of left ventricular function

Table *Reproducibility of digitisation during exercise: interobserver variability*

	Observer		%*	R†	2 SD limit
	1	*2*			
Heart rate (beats/min)	119 (25)	119 (27)	0·6	0·87	8·9
LVEDD (mm)	57·2 (6·0)	57·3 (5·8)	0·2	0·98	1·57
LVESD (mm)	37·4 (4·8)	37·8 (4·7)	0·9	0·98	1·5
Fractional shortening (%)	34 (4)	34 (4)	1·36	0·94	2·03
VCF/EDD (s^{-1})	0·92 (0·19)	0·93 (0·18)	1·5	0·71	0·2
Max decrease dD/dt/EDD	2·75 (0·52)	2·70 (0·46)	1·8	0·88	0·36
Max increase dD/dt/EDD	4·96 (1·19)	4·86 (1·08)	2	0·84	0·91

LVEDD, left ventricular end diastolic dimension; LVESD, left ventricular end systolic dimension; VCF/EDD, velocity of circumferential fibre shortening standardised for end diastolic dimension; max decrease dD/dt/EDD, maximum rate of decrease in left ventricular dimension standardised for end diastolic dimension; max increase dD/dt/EDD, maximum rate of increase in left ventricular dimension standardised for end diastolic dimension.
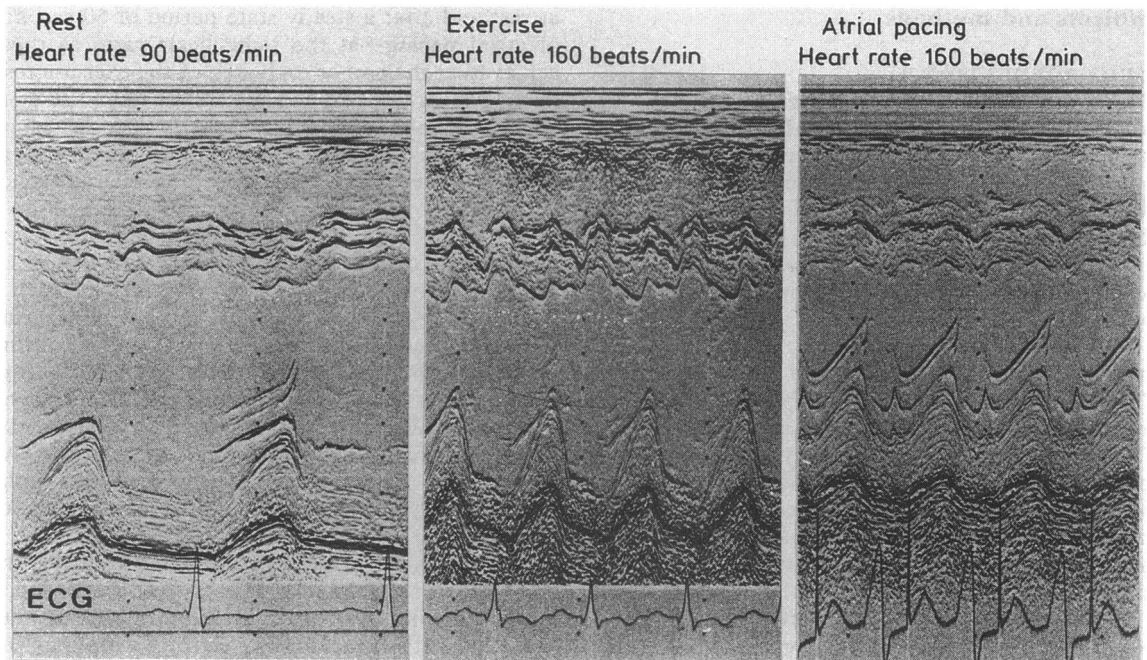*Percentage difference between means.
†Correlation coefficient.

Fig 1 *M mode echocardiograms of the left ventricle obtained at rest and at the maximum comparable heart rate during exercise and atrial pacing. The 1 cm calibration is similar in the three panels. There was a reduction in left ventricular end systolic dimension during exercise and a reduction in both left ventricular end diastolic and end systolic dimensions during atrial pacing.*

(standardised for end diastolic dimension) were determined: mean velocity of circumferential fibre shortening (VCF/EDD, s^{-1}), peak rate of decrease in left ventricular diameter (maximum systolic $dD/dt/EDD$, s^{-1}), and peak rate of increase in left ventricular diameter (maximum diastolic $dD/dt/EDD$, s^{-1}).

STATISTICAL ANALYSIS
For each variable, measurements obtained at each step during the exercise test were compared with those made at identical heart rates induced by atrial pacing. Student's t test for paired data was used. Differences were regarded as significant when $p < 0.05$.

Results

REPRODUCIBILITY OF DIGITISATION OF EXERCISE ECHOCARDIOGRAMS
For each variable measured during exercise we regarded the mean value of three consecutive cardiac cycles obtained by digitisation as an individual determination. Interobserver variability was assessed by calculation of the percentage difference between the means of the two observers (table). Variance between the two observers was small for all

variables, the maximum being 2% for normalised peak rate of increase in diameter, a derived index. The correlation of the results between the two independent observers was highly significant (table).

HEART RATE
In two subjects 1:1 atrioventricular conduction failed during atrial pacing at low heart rates and thus we could not compare the results with exercise after the first step. These patients were excluded from further analysis and we compared paired variables at similar heart rates in the eight remaining subjects.

Resting heart rates were similar before exercise (73 (13) beats/minute) and before atrial pacing (73 (15) beats/minute). During exercise we obtained adequate tracings for digitisation up to maximal heart rates (167 (14) beats/minute). The mean maximal heart rate reached during atrial pacing (148 (17) beats/minute) was lower because the Wenckebach phenomenon occurred at heart rates lower than those achieved during exercise. Figure 1 shows representative echocardiograms obtained at rest and at the maximal heart rates achieved during exercise and atrial pacing.

SYSTOLIC ARTERIAL BLOOD PRESSURE
Systolic arterial blood pressure was not significantly

different before exercise (138 (5) mm Hg), and before atrial pacing (130 (11) mm Hg). It increased to 191 (14) mm Hg at maximal exercise level (p < 0·01) and decreased slightly, but not significantly, at maximal atrial pacing induced tachycardia (123 (14) mm Hg).

LEFT VENTRICULAR CAVITY DIMENSIONS

Figure 2a shows the effects of supine exercise and atrial pacing induced tachycardia on left ventricular

end diastolic dimension. At rest the end diastolic dimension was greater before exercise than before atrial pacing (p < 0·05). The end diastolic dimension remained fairly constant during exercise and decreased during atrial pacing. At each step of the two stress tests the end diastolic dimension was greater during exercise than during atrial pacing and the differences increased progressively. During recovery the differences decreased progressively and after five minutes end diastolic dimension was similar to the value obtained at the corresponding heart rate induced by atrial pacing. During recovery subjects kept their feet up on the table. Figure 2b shows the influence of exercise and atrial pacing on left ventricular end systolic dimension. End systolic dimension decreased slightly during both exercise and atrial pacing and no significant differences were seen between the two stress tests at any level. During recovery left ventricular end systolic dimension was significantly smaller after exercise than at corresponding heart rates produced by atrial pacing (p < 0·05).

INDICES OF LEFT VENTRICULAR FUNCTION

Figure 3a shows the effects of exercise and atrial pacing on fractional shortening. Fractional shortening values were similar before exercise and atrial pacing. This variable remained constant during atrial pacing and increased progressively during exercise. This difference was significant at all levels of stress. After five minutes of recovery from exercise, fractional shortening values remained higher than at corresponding heart rates produced by atrial pacing.

Figures 3b and 3c show the effects of exercise and atrial pacing on the rate of left ventricular dimensional decrease (maximum systolic dD/dt/EDD) and increase (maximum diastolic dD/dt/EDD). Both indices showed a tendency to increase slightly during atrial pacing (NS) and a progressive and greater significant increase during exercise (p < 0·0001 at all steps of exercise). The difference between these values during the two stresses was statistically significant.

Discussion

Previous studies have shown that M mode echocardiography is an accurate and reproducible method for measuring left ventricular cavity dimensions at rest.[15-17] Interest in exercise echocardiography is increasing but the technique is difficult to perform, especially with the subject in the upright position.[10] In this study we obtained good quality M mode echocardiograms during maximal
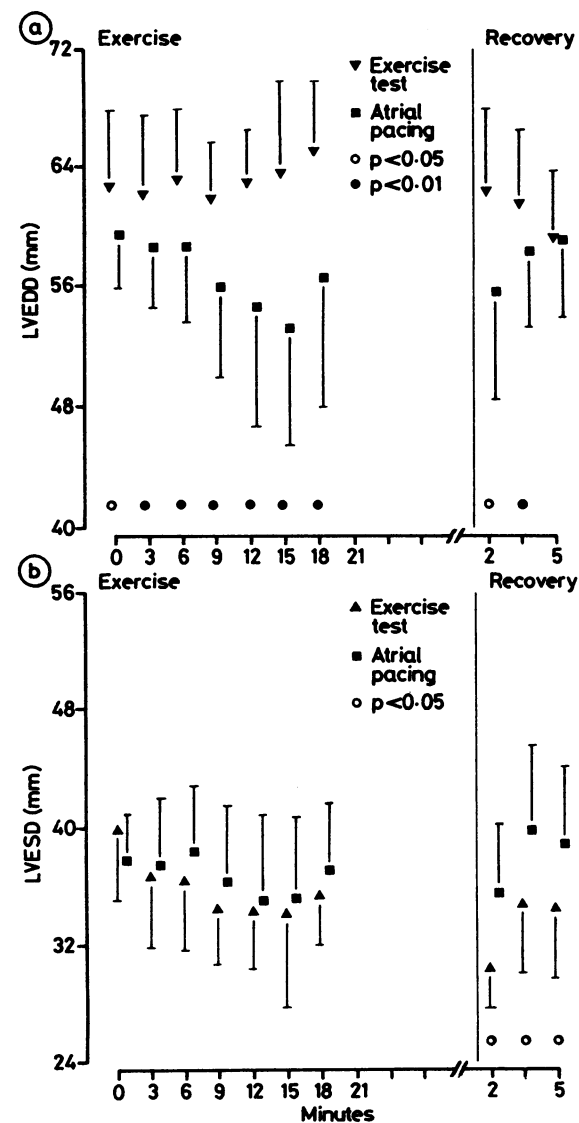
Fig 2 *Measurements of (a) left ventricular end diastolic dimension (LVEDD) and (b) left ventricular end systolic dimension (LVESD) at each step of exercise and recovery and at identical heart rates induced by atrial pacing. Mean (1SD) values of all subjects are indicated.*
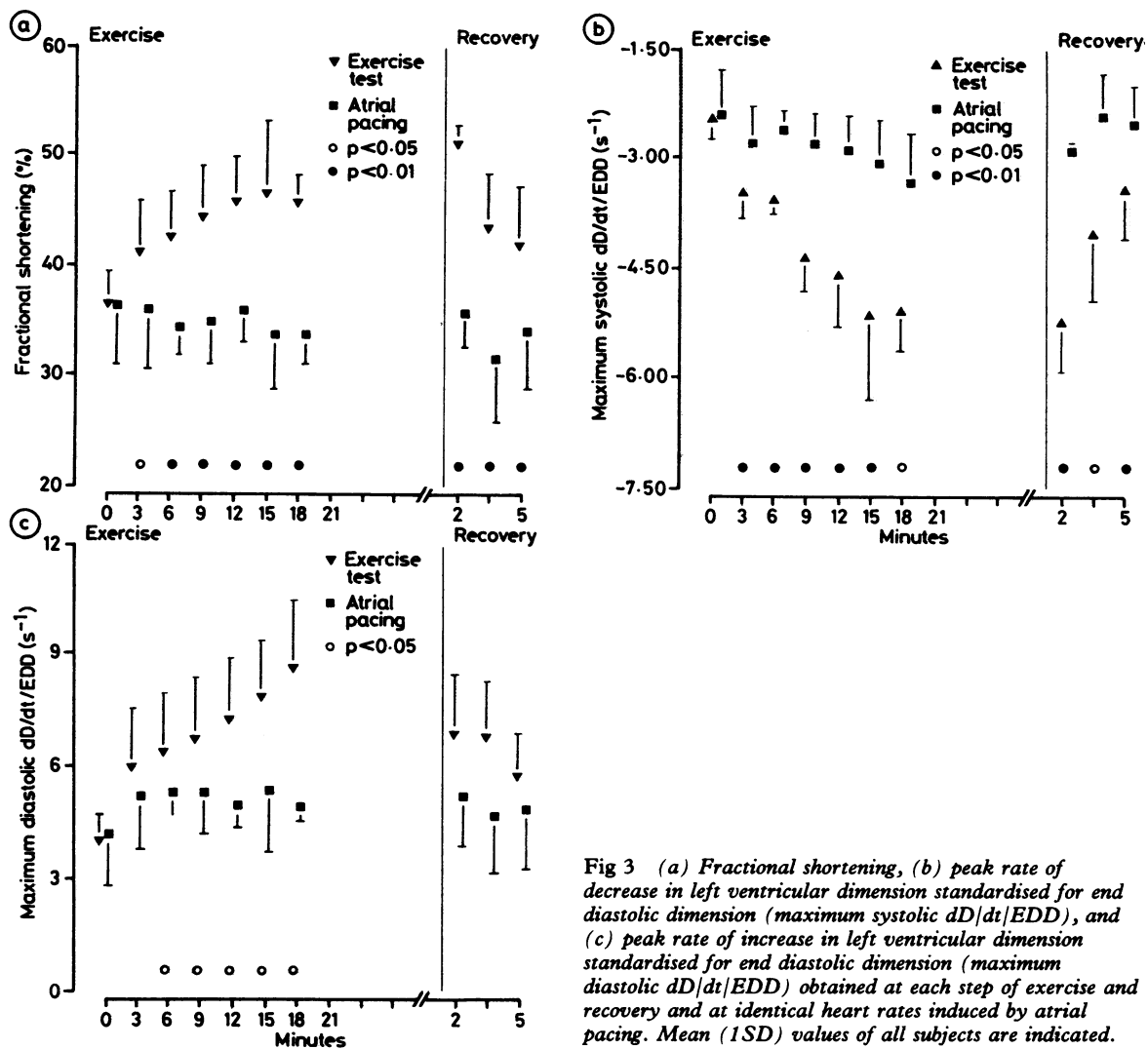
Fig 3 (a) Fractional shortening, (b) peak rate of decrease in left ventricular dimension standardised for end diastolic dimension (maximum systolic $dD/dt/EDD$), and (c) peak rate of increase in left ventricular dimension standardised for end diastolic dimension (maximum diastolic $dD/dt/EDD$) obtained at each step of exercise and recovery and at identical heart rates induced by atrial pacing. Mean (1 SD) values of all subjects are indicated.

supine exercise tests in young selected healthy subjects.

LEFT VENTRICULAR DIMENSIONS

Most studies in exercising dogs have shown an increasing left ventricular end diastolic dimension.[18-20] Several techniques have been used to measure left ventricular dimension change during exercise in man: M mode echocardiography,[5-10] myocardial markers,[21-24] left ventricular angiography,[25 26] and radionuclide angiography.[11 27-30] Radionuclide studies performed in upright subjects have demonstrated an increase in left ventricular end diastolic dimension[11 28 29] whereas Crawford et al

using M mode echocardiography did not observe any change during moderate upright exercise.[5] Data on left ventricular dimension changes during exercise in the supine position are even more conflicting. An increase in left ventricular end diastolic volume was demonstrated by Poliner et al using radionuclide techniques[29] and this was confirmed by Sharma et al using cineangiography.[26] Left ventricular end diastolic volume remained unchanged in Gorlin et al's angiographic study[25] and in that of Slutsky et al who used radionuclide angiography.[28] Weiss et al found an increase in echocardiographic end diastolic dimension during maximal semisupine exercise.[7] In most echocardiographic studies, however, no

significant changes were found in end diastolic dimension during supine exercise.[5 6 8] Studies with myocardial markers showed that the end diastolic dimension decreased[21 31] or remained unchanged.[23]

In the denervated heart of cardiac transplant recipients an early increase during exercise has been demonstrated.[24] The findings of most of these studies, and particularly those using echocardiography and radionuclide techniques, have been interpreted on the basis of the presence or absence of a Frank-Starling mechanism without considering the influence of heart rate. Our findings suggest that leg elevation just before supine exercise significantly increases left ventricular end diastolic dimension. Because the feet were raised at rest just before exercise, end diastolic dimension was greater than before atrial pacing when the feet were horizontal. The mean difference of 3·2 mm (5·2%) was outside the 95% confidence interval of temporal variability in serial echocardiograms and must be regarded as significant.[32] This increase in preload was not accompanied by an inotropic effect since all the indices of left ventricular function—fractional shortening, mean velocity of circumferential fibre shortening, and peak rate of decrease in diameter—were not significantly different before the two tests were started. We observed that at any given heart rate during stress, left ventricular end diastolic dimensions were greater during exercise than during atrial pacing induced tachycardia. These observations accord with those of Sonnenblick *et al* who studied patients by means of myocardial markers after cardiac operations.[22] An increase in heart rate tends to decrease end diastolic dimension whereas increased venous return has the opposite effect. Thus the maintenance of end diastolic dimension during supine exercise suggests that the Frank-Starling mechanism is active in the cardiac response to exercise.

Though most of the previous studies have shown a decrease in end systolic dimension during both supine and upright exercise,[5 6 8 20 23 25 26] indicating increased emptying of the left ventricle, others have not confirmed these findings.[7 19] Poliner *et al* found no change in end systolic volume in the supine position, but they did report a significant decrease during upright exercise.[29] Our results show that there is a trend towards a decrease in end systolic dimension during exercise, but the measurements are not significantly different from those made at corresponding heart rates induced by atrial pacing. During recovery after exercise the end systolic dimension was smaller than at similar heart rates during atrial pacing. These findings are in accord with increased left ventricular emptying during recovery caused by persistently high catecholamine

concentration and a decreased afterload due to peripheral vasodilatation.

LEFT VENTRICULAR FUNCTION

The positive inotropic effect of increasing the frequency of contraction has been described by many investigators.[22 33-38] In our study fractional shortening remained constant during atrial pacing and there was a tendency to a slight not statistically significant increase in mean normalised velocity of circumferential fibre shortening and peak rate of decrease in left ventricular diameter. These results accord with those reported by DeMaria *et al*.[12]

During exercise, however, there was a progressive increase in fractional shortening, normalised velocity of circumferential fibre shortening, and peak rate of decrease in left ventricular diameter. Thus it appears that the tachycardia per se is not the major factor in the increase of contractility during exercise.

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