Aortic regurgitation

*Detection of left ventricular dysfunction by exercise echocardiography**

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SUMMARY Left ventricular performance was assessed in 20 symptom free patients and 10 with symptoms, all with isolated aortic regurgitation, by measuring the echocardiographic peak velocity of circumferential fibre shortening (echo peak Vcf) at rest and during graded bicycle ergometer exercise in the supine position. The normal left ventricular response during such exercise was first determined in 20 healthy controls.

On the basis of their resting and exercise echo peak Vcf, the 30 patients with aortic regurgitation could be separated into three groups: Group 1 comprised 11 symptom free patients with a normal resting echo peak Vcf which increased normally with exercise; group 2 comprised nine symptom free patients with a normal resting echo peak Vcf but with a subnormal response to exercise; group 3 consisted of 10 patients with symptoms with a depressed resting echo peak Vcf which remained subnormal with exercise. Subsequent cardiac catheterisation disclosed normal ejection fractions in patients in group 1, borderline ejection fractions in those in group 2, and reduced ejection fractions in those in group 3.

Echocardiographic assessment of left ventricular performance during supine isotonic exercise may provide a simple noninvasive method for the early detection of left ventricular dysfunction in symptom free patients with aortic regurgitation.

Once heart failure has occurred in patients with chronic aortic regurgitation, valve replacement may prove ineffective in restoring normal myocardial function.¹⁻⁵ Echocardiography has been suggested as a simple means of detecting early impairment of left ventricular function in such patients^{6,7} and the finding of impaired myocardial performance in the absence of clinical heart failure should prompt additional invasive studies.⁶ On examining those reports,^{6,7} however, it is apparent that difficulties may be encountered in this assessment. Though mean values for the echocardiographic variables of

myocardial function may clearly differentiate normal from abnormal groups, there can be considerable overlap of individual values, and the diagnostic accuracy in a specific patient may be suspect. Since echocardiograms are normally done only at rest, we felt that the addition of exercise might further differentiate those patients with impaired ventricular function from those whose myocardium was normal. This approach, which has recently been used to detect wall motion abnormalities during myocardial ischaemia,⁸ could be a valuable method for the improved assessment of patients with chronic aortic regurgitation.

Methods

PATIENTS

All patients with isolated aortic regurgitation who had cardiac catheterisation at our centre over a two year

*Supported by a grant from the Ontario Heart Foundation. Drs Boughner and Paulsen are Ontario Heart Foundation Research Fellows

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period were studied. Patients with associated aortic stenosis (>10 mmHg left ventricular to aortic gradient), a malfunctioning prosthetic aortic valve, mitral valve disease, coronary artery disease ($>50\%$ stenosis), cardiomyopathy, or hypertension (diastolic blood pressure >95 mmHg), were deliberately excluded. Eight patients with isolated aortic regurgitation were also excluded because we could not obtain adequate quality echocardiograms during exercise. The final study population consisted of 30 patients, 26 men and four women, with a mean age of 46 ± 13 years (range 15 to 65 years). Twenty patients had no symptoms, i.e. no history of exercise intolerance, chest pain, lightheadedness, syncope, or heart failure. Functionally, such patients were in New York Heart Association Class I.⁹ The remaining 10 patients had one or more of these symptoms and were in New York Heart Association Classes II–IV. Left ventricular hypertrophy on electrocardiography was diagnosed by using the criteria of Romhilt and Estes (five points or more).¹⁰

At the time of the study, three of the patients with symptoms were taking digitalis. The remaining 27 patients were either on no cardiac drugs or these had been discontinued at least one week before the study.

NORMAL SUBJECTS

Twenty normal healthy subjects were also studied, found to be free of heart disease by history, physical examination, electrocardiography, and echocardiography. There were 16 men and four women with a mean age of 33 ± 9 years (range 18 to 46). None was taking any drugs.

ECHOCARDIOGRAPHY

Echocardiograms were obtained using a Unirad, Series C echocardiographic unit equipped with a Honeywell 1858 strip chart recorder. Patients and normal subjects were studied supine in the slight left lateral position with the transducer positioned in the fourth or fifth left intercostal space. The left ventricular minor axis diameter was recorded by directing the ultrasound beam just inferior to the mitral valve and recordings were made at a paper speed of 100 mm/s (Fig. 1). Echocardiograms were obtained only at end expiration and from the same interspace with the same anatomical landmarks present throughout the study in an attempt to minimise spurious changes of left ventricular dimensions.

Isotonic exercise was performed using a Siemens-

Fig. 1 Left ventricular echogram of a symptom free patient with aortic regurgitation; paper speed 100 mm/s at rest and during supine bicycle ergometer exercise. Note the obvious increase in peak Vcf with exercise, implying normal left ventricular functional reserve. Abbreviations: IVS, interventricular septum; LVPW, left ventricular posterior wall; ECG, electrocardiogram; Vcf, velocity of circumferential fibre shortening.

Elema bicycle ergometer system mounted vertically at the foot of the bed. Recordings of left ventricular echocardiograms and measurements of heart rate and blood pressure were obtained at rest and at one minute intervals during supine bicycle ergometer exercise to a maximum of six minutes (Fig. 1). The initial ergometer workload was 25W and was increased by 25W each minute to a maximum of 150W. Though total cardiocirculatory adaptation to any magnitude of exertion does not occur for two to three minutes,¹¹ it was technically difficult to obtain adequate quality left ventricular echocardiograms throughout such a prolonged graded exercise procedure and, since the cardiovascular adjustments to a submaximal workload reach a nearly steady state within the first minute of isotonic exercise,¹²⁻¹⁴ we elected to increase the workload at one minute intervals.

MEASUREMENTS AND CALCULATIONS

Analysis of the instantaneous left ventricular dimension and calculation of its peak rate of change was performed using a Hewlett-Packard Model 1838 desk top computer, a model 986A digitiser, and x-y plotter combination by a method modified from that of Gibson *et al.*^{15 16} The recordings of the left ventricular echograms were placed on a digitising table and the endocardial echoes of the septum and posterior wall were digitised beginning at the onset of the R wave and ending with the T wave. The computer noted the position of these two echoes and automatically derived the instantaneous left ventricular diameter. This diameter measurement was divided by the initial diameter and the ratio was displayed by the x-y plotter as a "displacement ratio" versus time curve (Fig. 2).

Fig. 2 Computer plot of the displacement ratio versus time curves corresponding to the ventricular echograms shown in Fig. 1 at rest, and during bicycle ergometer exercise supine. (Displacement ratio = instantaneous left ventricular diameter divided by end-diastolic diameter.)

Peak velocity of circumferential fibre shortening (Vcf) was obtained manually by calculating the slope of the tangent to the mid portion of this curve (Fig. 3), thus giving the normalised peak Vcf in circumferences per second (circ/s). We chose to calculate peak Vcf in this fashion rather than having the computer calculate the instantaneous first derivative of the left ventricular diameter curve, as described by others,^{15 17} since we found that the normalised instantaneous diameter curve or "displacement ratio" contained sufficient noise (produced by "hand-shake" when tracing the echo) to make such a computer estimate of peak Vcf poorly reproducible. Such irregularities, however, could be easily compensated for by the manual method, resulting in a high degree of reproducibility for the peak Vcf estimate. The left ventricular end-diastolic diameter in mm was the smallest dimension measured between the endocardial surfaces of the left ventricular septum and the posterior left ventricular wall (Fig. 1). The percentage fractional shortening (% Δ D) was calculated from the formula:

$$\% \Delta D = \frac{EDD - ESD}{EDD} \times 100$$

where EDD=end-diastolic diameter and ESD=end-systolic diameter.

Measurements of four successive end expiratory beats at rest and during exercise in four randomly selected normals and four randomly selected patients with chronic aortic regurgitation were carried out by two observers independently in order to establish the beat to beat and interobserver variation in the peak Vcf calculation. One way analysis of variance indicated good reproducibility as evidenced by a coefficient of variation of less than 5%. Three of the

Fig. 3 The slope of the tangent drawn to the mid portion of each displacement ratio versus time curve provided the respective normalised peak Vcf in circ/s.

patients with chronic aortic regurgitation repeated supine bicycle exercise under similar conditions within one week of their initial studies. They each completed exercise workloads similar to those in their initial tests. The heart rate, systolic blood pressure, left ventricular end-diastolic diameter, and peak Vcf at rest and at one minute intervals during exercise were not significantly different between the two exercise tests. Thus, reproducibility did not seem to be a problem.

In the 20 controls, peak Vcf was calculated at one minute intervals throughout the six minutes of exercise in order to determine the normal ventricular response to graded exercise to a maximum load of 150W. In the 30 patients with chronic aortic regurgitation, the peak Vcf was calculated at rest and during the final stage of exercise achieved by the patient. The echocardiographic measurements were made during expiration and represented the average of three cardiac cycles.

CARDIAC CATHETERISATION

All 30 patients with chronic aortic regurgitation underwent resting left and right heart cardiac catheterisation within three days of the echocardiographic evaluation. Left ventricular ejection fractions were calculated from the right anterior oblique cineangiograms using the single plane method of Sandler and Dodge.¹⁸ Left ventricular cardiac output was calculated by multiplying angiographically determined stroke volume by heart rate. The Fick cardiac output was determined before angiography, and the difference between the ventricular output

and the Fick output provided the percentage of aortic regurgitation.¹⁹ Coronary arteriography was performed in all patients over 40 years old (23 patients). The controls did not undergo cardiac catheterisation.

Statistical analysis was performed using Student's *t* test and analysis of variance.

Results

NORMAL SUBJECTS

In the normal subjects, the peak Vcf at rest ranged from 1.4 to 2.2 circ/s with a mean of 1.78 ± 0.21 (standard deviation) with the 95% confidence limits lying between 1.68 and 1.88 circ/s. We used these values to define the normal range of peak Vcf at rest. With six minutes of exercise, peak Vcf increased in a stepwise fashion to 2.89 ± 0.22 circ/s (95% confidence limits 2.79 to 2.99 circ/s), and when the heart rate exceeded 100 beats/min, it invariably increased at least 0.30 circ/s above the value obtained at rest. Since all patients in this study attained heart rates above 100 beats/min during exercise an increase of peak Vcf of 0.30 circ/s or more was accepted as normal in the patients with aortic regurgitation.

PATIENTS WITH AORTIC REGURGITATION

The clinical data for the 30 patients with chronic aortic regurgitation are presented in the Table. On the basis of their resting and exercise echocardiographic peak Vcf, they could be divided into three groups (Fig. 4).

Group 1 consisted of 11 symptom-free patients who

Fig. 4 Changes in echocardiographic peak Vcf from rest to exercise in normal subjects and patients with aortic regurgitation. Groups 1 and 2 represent symptom free patients and group 3 those with symptoms (open circles with vertical bars denote mean $\pm$ 1 SD).

Table

Case No.	Age/ Sex	NYHA Class	ECG LVH	Echocardiography												Catheter study	
				HR (beats/min)		BP (mmHg)		EDD (mm)		ESD (mm)		p Vcf (circ/s)		% ΔD			
				Rest	Ex	Rest	Ex	Rest	Ex	Rest	Ex	Rest	Ex	Rest	Ex	EF(%)	RF(%)
Group 1: Symptom free																	
1	49/M	I	+	70	112	160	205	58	57	36	29	1.9	2.6	38	49	62	38
2	19/M	I	−	69	148	145	175	60	61	36	30	1.8	2.8	40	51	78	48
3	24/M	I	−	66	108	120	145	54	53	32	28	1.7	2.9	41	47	72	32
4	48/F	I	+	78	105	155	185	63	63	44	40	1.4	2.5	30	37	61	49
5	55/M	I	−	85	133	165	210	66	65	40	34	1.9	2.9	39	48	59	54
6	18/F	I	−	64	108	134	172	64	63	41	39	1.6	2.8	36	40	76	58
7	42/M	I	−	72	104	140	170	70	68	50	44	1.6	2.5	29	35	60	56
8	47/M	I	+	80	120	172	208	72	70	45	43	1.8	2.6	38	39	68	60
9	46/F	I	−	71	125	140	190	62	63	41	36	1.6	2.8	34	43	64	50
10	15/M	I	−	68	130	125	150	64	64	42	39	1.8	2.9	35	41	70	51
11	48/M	I	−	65	118	160	182	60	59	41	34	1.7	3.2	32	40	62	48
Mean 37				72	119	147	181	63	62	41	36	1.71	2.77	36	43	67	49
SD 14				6	13	16	21	5	5	5	6	0.15	0.22	4	5	6	8
Group 2: Symptom free																	
12	36/F	I	−	69	112	155	200	59	60	35	32	1.9	1.8	41	46	48	46
13	56/M	I	−	78	139	155	180	65	65	40	38	1.7	1.6	39	42	52	57
14	42/M	I	+	60	110	120	140	66	63	43	42	1.4	1.4	35	33	54	50
15	35/M	I	−	70	124	145	180	65	64	44	40	1.8	1.9	32	38	50	48
16	29/M	I	+	78	107	130	178	70	69	45	43	1.5	1.6	36	40	50	60
17	49/M	I	−	76	130	145	190	64	65	42	40	1.6	1.5	34	38	56	48
18	53/M	I	−	74	138	160	205	67	67	40	37	1.8	1.6	40	45	49	52
19	46/M	I	−	82	140	156	176	62	63	39	36	1.4	1.3	37	43	57	44
20	53/M	I	−	73	106	150	174	71	72	50	47	1.8	1.6	30	36	55	61
Mean 44				73	123	146	180	65	65	42	39	1.66	1.59	36	40	52	52
SD 9				6	14	13	18	4	3	4	5	0.15	0.17	4	4	3	6
Group 3: With symptoms																	
21	59/M	II	−	80	130	165	210	68	69	48	47	1.3	1.2	29	32	43	53
22	45/M	II	+	70	120	150	200	67	68	46	47	1.2	1.0	31	31	52	48
23	60/M	II	+	79	120	140	205	70	69	52	50	1.1	0.9	26	29	46	54
24	53/M	III	+	69	105	150	172	74	73	52	54	1.2	1.0	30	27	38	58
25	58/F	II	+	78	106	130	200	72	71	55	56	1.2	0.8	24	21	40	55
26	65/M	III	+	74	118	140	195	76	74	53	55	0.9	0.7	30	26	35	59
27	61/M	III	+	82	116	154	186	78	77	60	62	0.8	0.6	23	19	29	56
28	59/F	IV	+	88	126	160	215	82	83	59	62	1.0	0.7	28	25	30	62
29	62/M	III	+	68	135	162	190	80	80	61	61	1.2	1.0	24	24	39	61
30	62/M	IV	+	84	144	125	155	79	77	58	60	1.0	0.7	27	22	36	59
Mean 58				77	122	148	193	75	74	54	55	1.09	0.86	27	26	39	56
SD 5				6	12	13	17	5	5	4	6	0.15	0.18	3	4	7	4

Abbreviations: NYHA, New York Heart Association; ECG, electrocardiogram; LVH, left ventricular hypertrophy; HR, heart rate; EDD, left ventricular end-diastolic diameter; ESD, left ventricular end-systolic diameter; Ex, exercise; pVcf, peak velocity of circumferential fibre shortening; EF, ejection fraction; RF, regurgitant fraction; % ΔD , % fractional shortening.

had a normal resting peak Vcf (≥ 1.4 circ/s) which increased normally with exercise. Their resting peak Vcf of 1.7 ± 0.15 circ/s (range 1.4 to 1.9 circ/s) was not significantly different from that of normal subjects at rest (1.78 ± 0.21 circ/s). With exercise, peak Vcf increased significantly ($p < 0.001$) to 2.77 ± 0.20 circ/s,

a value not significantly different from that of normal subjects during exercise (2.89 ± 0.21 circ/s). In addition, each patient had increased peak Vcf by at least 0.30 circ/s above the resting value. At subsequent cardiac catheterisation, all 11 patients in this group had normal ejection fractions, the mean

being 67 ± 6 with a range of 59 to 78 (normal $\geq 55\%$).

Group 2 consisted of nine symptom-free patients who had a normal resting peak Vcf which failed to increase normally with exercise. Thus, though their resting peak Vcf of 1.66 ± 0.17 circ/s (range 1.4 to 1.9 circ/s) was not significantly different from that of normal subjects at rest (1.78 ± 0.21 circ/s), their peak Vcf during exercise decreased slightly but insignificantly from 1.65 ± 0.17 to 1.59 ± 0.17 circ/s. Moreover, no patient in this group increased peak Vcf by greater than 0.3 circ/s with exercise. At subsequent cardiac catheterisation, patients in this group were found to have slightly reduced ejection fractions, the mean being $52 \pm 3\%$ (range 48 to 57%).

Group 3 consisted of 10 patients with symptoms who all had a depressed resting peak Vcf (< 1.4 circ/s). With exercise, the peak Vcf either remained unchanged or was further reduced. Thus, their resting peak Vcf of 1.09 ± 0.15 circ/s (range 0.8 to 1.3 circ/s) was significantly lower than that of normal subjects at rest (1.78 ± 0.21 circ/s) ($p < 0.001$), and with exercise it decreased to 0.86 ± 0.18 circ/s ($p < 0.05$). In this group, ejection fractions at cardiac catheterisation were all conspicuously reduced, with a mean of $39 \pm 7\%$ (range 29 to 46%).

The heart rate during exercise did not differ significantly between the three groups; in group 1 119 ± 13 beats/min (range 104 to 148 beats/min), in group 2 123 ± 14 beats/min (range 107 to 140 beats/min), and in group 3 122 ± 12 beats/min (range 105 to 144 beats/min) (Table).

PER CENT FRACTIONAL SHORTENING

At rest, the $\% \Delta D$ of the 10 patients with symptoms (group 3) was significantly lower than that of the 20 symptom free patients (groups 1 and 2); 27 ± 3 versus $36 \pm 4\%$ ($p < 0.001$) (Table). It was 31% or less in all 10 patients with symptoms and greater than 31% in 17 of the 20 without them. There was no significant difference between the resting $\% \Delta D$ in group 1 ($36 \pm 4\%$) and in group 2 ($36 \pm 4\%$).

With exercise, the $\% \Delta D$ of patients in group 1 increased from 36 ± 4 to $43 \pm 5\%$ (NS) and that of patients in group 2 increased from 36 ± 4 to $40 \pm 4\%$ (NS). Thus, echocardiographic $\% \Delta D$, unlike echocardiographic peak Vcf, was not able to distinguish symptom free patients with catheter proven normal left ventricular function (group 1) from those with borderline left ventricular function (group 2). Patients in group 3 decreased their $\% \Delta D$ insignificantly from 27 ± 3 to $26 \pm 4\%$ with exercise.

END-DIASTOLIC DIMENSIONS

The resting left ventricular end-diastolic diameter of the 10 patients with symptoms was significantly larger than that of the 20 who were symptom free, 75.5 ± 5

versus 63.5 ± 5 mm ($p < 0.001$). Thus, all patients with symptoms had an end-diastolic diameter greater than 66 mm and, in addition, they all had a depressed peak Vcf at rest. Of the 20 symptom free patients, however, all of whom had a normal resting peak Vcf, there were five with end-diastolic diameters greater than 66 mm (Table). Two of these five showed a normal increase in peak Vcf with exercise while in the remaining three, exercise peak Vcf was subnormal. It was therefore necessary to determine whether the exercise peak Vcf was affected by the degree of left ventricular dilatation in the 20 symptom free patients. We found that there was wide variation in exercise peak Vcf at any level of resting end-diastolic diameter ($r = 0.35$, not significant) so that end-diastolic diameter at rest in symptom free patients could not be used to predict their exercise peak Vcf.

The end-diastolic diameter did not change significantly between rest and exercise for any of the subgroups (Table). The 20 normal subjects also showed no significant change in end-diastolic diameter during exercise supine (50.2 ± 5 mm at rest versus 50.8 ± 6 mm during exercise).

END-SYSTOLIC DIMENSIONS

At rest the end-systolic diameter of the 10 patients with symptoms (group 3) was significantly larger than that of the 20 symptom free subjects (groups 1 and 2); 54 ± 4 versus 41 ± 5 mm ($p < 0.001$) (Table). The end-systolic diameter was greater than 46 mm in all of the former and less than 46 mm in 18 of the 20 of the latter. Of the two symptom free patients with resting end-systolic diameters greater than 46 mm, one showed a normal increase of peak Vcf with exercise while in the other exercise peak Vcf was subnormal. There was no correlation ($r = 0.22$, not significant) between resting end-systolic diameter and exercise peak Vcf for the 20 symptom free subjects.

During exercise, the end-systolic diameter of the 10 patients with symptoms (group 3) was significantly larger than the 20 without them (groups 1 and 2) 55 ± 6 versus 38 ± 5 mm ($p < 0.001$). The end-systolic diameter was 47 mm or greater in all 10 of the former and less than 47 mm in 19 of the latter. There was no correlation between exercise end-systolic dimension and exercise peak Vcf ($r = 0.42$, not significant) in the 20 symptom free subjects. Thus, echocardiographic end-systolic dimension, unlike echocardiographic peak Vcf, was not able to distinguish between the symptom free patients with normal left ventricular function (group 1) and those with borderline left ventricular function (group 2).

SYSTOLIC ARTERIAL PRESSURE

The maximum systolic arterial pressure during exercise did not differ significantly between the three

groups. The maximum systolic arterial pressure during exercise in patients in group 1 was 181 ± 21 mmHg (range 145 to 210 mmHg), for those in group 2 180 ± 18 mmHg (range 140 to 205 mmHg) and for those in group 3 193 ± 17 mmHg (range 155 to 215 mmHg) (Table). These values were not significantly different from those found in normal subjects during exercise (183 ± 17 mmHg, range 135 to 200 mmHg).

There was no correlation between exercise peak Vcf and maximum systolic pressure during exercise either for the entire group of patients with aortic regurgitation ($r=0.20$, not significant), or the subgroups (groups 2 and 3) with a subnormal peak Vcf response during exercise ($r=0.16$, not significant). Thus, the maximum systolic pressure attained during exercise could not be used to predict peak Vcf during exercise.

Discussion

In patients with chronic aortic regurgitation, the detection of early impairment of left ventricular performance remains an important clinical problem. Once heart failure has occurred valve replacement may prove ineffective in restoring normal myocardial function.¹⁻⁵ Though contrast angiographic ejection phase indices of myocardial performance have been recommended as the most reliable means of assessing left ventricular function,²⁰⁻²⁴ their clinical application, for periodic assessments aimed at detecting early impairment of myocardial function, are limited by the need for repeated cardiac catheterisation. Echocardiography is a simple non-invasive technique which provides similar information about left ventricular performance but has the advantage of safety, repeatability, and relatively low cost. Several studies have shown that resting echocardiographic indices of left ventricular performance provide a satisfactory means of separating groups of patients with normal left ventricular function from groups of patients with depressed left ventricular function.^{6, 7, 25-28} The presence of significant overlap between such groups, however, can lead to difficulty in distinguishing normal function from impaired function in an individual patient.

Our results indicate that in symptom free patients with chronic aortic regurgitation, echocardiography performed during exercise is considerably more sensitive in detecting mild left ventricular dysfunction than echocardiography performed at rest. All 20 symptom free patients had a normal echocardiographic peak Vcf at rest. Only 11 (group 1), however, had a normal increase in peak Vcf with exercise (Fig. 4). These 11 patients all had normal left ventricular function at subsequent cardiac catheterisation. The nine remaining symptom free patients (group 2) with

normal resting echocardiographic peak Vcf failed to increase their peak Vcf normally during exercise and all had slightly depressed ejection fractions at cardiac catheterisation, suggesting the presence of mild left ventricular dysfunction. Moreover, in these nine patients, the abnormal peak Vcf response during exercise could not be predicted from the presence of electrocardiographic left ventricular hypertrophy, the left ventricular diastolic or systolic dimensions, the maximum systolic pressure attained during exercise, or a combination of these factors.

In contrast, all 10 patients with symptoms (group 3) had echocardiographically detectable left ventricular dysfunction at rest, and the addition of exercise merely confirmed this fact and added no further useful information. Subsequent cardiac catheterisation in this group further confirmed the presence of severely depressed left ventricular function.

In general, a failure of peak Vcf to increase during exercise does not necessarily imply that myocardial contractility is abnormal. Vcf is not only sensitive to inotropic stimulation, but is also inversely related to acute changes in afterload.²⁹ Since afterload increases during exercise it could preclude an increase in the Vcf if it acted alone. Exercise, however, is associated with a distinct increase in contractility³⁰ which tends to increase Vcf. Apparently, the latter effect predominates resulting in a net increase in Vcf during exercise in subjects with normal left ventricular functional reserve.¹⁶ In the present study, the increase in afterload during exercise was not measured. The systolic blood pressure, however, which is an indirect reflection of afterload,³¹ was measured both at rest and during exercise and did not differ significantly between the three groups of patients (Table). Thus, the influence of afterload upon exercise peak Vcf was probably the same for each group and can be excluded from consideration for purposes of comparison. Finally, the presence of myocardial dysfunction in patients who failed to increase their echocardiographic Vcf significantly with exercise is supported by the finding of reduced ejection fractions in these patients at subsequent cardiac catheterisation. Though the resting angiographic ejection fraction has limitations similar to those associated with the Vcf, it has proved to be a valuable estimate of left ventricular function clinically.²⁰⁻²⁴

Our finding, that the noninvasive assessment of left ventricular performance during exercise is a valuable means of detecting early ventricular dysfunction in patients with aortic regurgitation, is in keeping with recent observations by Borer *et al.*³² Using non-invasive radionuclide cineangiography, these workers showed that though the resting ejection fraction was normal in 21 symptom free patients with isolated aortic regurgitation, in only 13 was the exercise

ejection fraction normal. The eight remaining patients had subnormal exercise ejection fractions, suggesting the presence of impaired myocardial performance not apparent at rest. Compared with exercise radionuclide cineangiography, exercise echocardiography examines instantaneous events in each cardiac cycle rather than combining several cycles, imposes no radiation exposure, and is relatively inexpensive. The greatest limitation of exercise echocardiography is the difficulty of obtaining high quality left ventricular echograms during exercise in all patients examined. Our success rate of obtaining such echograms during exercise in patients with aortic regurgitation was approximately 75 per cent. This compares favourably with the experience of Sugishita and Koseki who obtained satisfactory exercise left ventricular echocardiograms in 83% of their patients.³³

In the present study, we found echocardiographic peak Vcf to be a more reliable predictor of contrast angiographic ejection fraction than either the echocardiographic $\% \Delta D$ or the end-systolic dimensions. This is consistent with previous observations where peak Vcf was found to be a more sensitive measure of left ventricular function than other echocardiographic indices currently in use.²⁸

CLINICAL IMPLICATIONS

This study shows that in symptom free patients with chronic aortic regurgitation, exercise echocardiography appears to be a simple noninvasive method of detecting early left ventricular dysfunction not apparent at rest. Evidence of impaired myocardial function in such patients may signal the need for additional invasive studies and consideration for operation.

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Subacute bacterial endocarditis

A survey is currently being carried out by the British Cardiac Society and the Medical Services Study Group of the Royal College of Physicians. Though improvement of dental prophylaxis is one objective, the survey is already yielding other valuable information. It is hoped that proformas will be received in respect of a high proportion of patients with subacute bacterial endocarditis in the British Isles seen during 1981 and 1982 and readers are asked to arrange for them to be submitted in respect of any cases that come to their notice. Proformas can be obtained from Sir Cyril Clarke, Medical Services Study Group, King's Fund Centre, 126 Albert Street, London NW1 7NF (tel. 01-267 6111, ext. 263) to whom they should be returned.

Future meetings

- (1) Cardiovascular System Dynamics Society Vth International Conference, Oxford, 28 September to 1 October 1982. For further details write to Dr G d J Lee, Cardiac Department, John Radcliffe Hospital, Oxford.
- (2) International Symposium on Coronary Arteries in Infants and Children, Tel Aviv, Israel, 17 to 22 October 1982. For further details write to D A Schneeweiss, Secretariat: PO Box 29784, Tel Aviv 61297, Israel.
- (3) VIIth World Symposium on Cardiac Pacing, 1 to 5 May 1983, Vienna, Austria. For information write to A 1107, PO Box 80, Vienna, Austria.
- (4) The Autumn Meeting of the British Cardiac Society will take place at Wembley on 6 and 7 December 1982 and the closing date for receipt of abstracts is 11 August 1982.

Erratum

Aortic regurgitation: detection of left ventricular dysfunction by exercise echocardiography (1981; 46: 380-8). On page 383, Fig. 4 should have displayed the p values as follows:

p<0.001 for normal, p<0.001 for group 1, NS for group 2, and p<0.05 for group 3. We regret the error.