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R. M. Peters, T. Silberstein

In patients with resting left ventricular diastolic abnormalities, it is not known if their transmitral diastolic flow velocity patterns in response to exercise are different from the response seen in normal subjects.

Treadmill stress echocardiography was performed on 31 normotensives (Group 1), 16 hypertensives without LVH (Group 2), and 14 hypertensives with mild LVH on resting echo (Group 3). All tests were negative for ischaemia, and all subjects reached greater than 85 % of predicted maximum heart rate for age. Transmitral flow was measured by pulsed Doppler with the sample volume at the mitral anulus in the 4 chamber view at rest, immediately post-exercise at peak heart rate, and 12 minutes post exercise.

Lower E/A mean ratios were found at rest in Group 2 (0.88) and Group 3 (0.87) compared to Group 1 (0.95) indicating a resting diastolic abnormality in the hypertensive groups ($p < 0.05$). With exercise, all 3 groups showed similar significant ($p < 0.05$) increases in both the mean E velocity (+15.7 %, +11.2 %, +16.5 %) and the mean A velocity (+18.8 %, +11.2 %, +16.9 %) compared to rest values. All 3 groups showed no significant change in the E/A ratio or the deceleration time with exercise. At 12 minutes, the mean E and mean A velocities had returned to mean resting values in all 3 groups.

The response of left ventricular filling dynamics to treadmill exercise in hypertensives is similar to that seen in normal subjects even in the presence of resting diastolic abnormalities and when mild LVH is present. *J Clin Basic Cardiol 1999; 2: 89–91.*

Key words: diastolic dysfunction, exercise, hypertension

Pulsed wave Doppler transmitral flow velocities are the most extensively studied noninvasive methods for examining diastolic resting filling abnormalities of the left ventricle in a variety of cardiac diseases [1]. In patients with arterial hypertension and left ventricular hypertrophy an "impaired relaxation" pattern is often seen [2]. This consists of reduced peak early LV filling (E velocity) and increased atrial filling (A velocity) with consequent reduction in the E/A ratio. It is thought that these resting abnormalities are at least partly responsible for the dypsnea on exertion and diminished exercise capacity commonly found in hypertensive patients [3], but ventricular diastolic filling patterns in response to dynamic exercise have not been well studied in hypertensives.

In the normal left ventricle, increased sympathetic stimulation associated with exercise accelerates the speed of relaxation resulting in increased elastic recoil [4]. This, coupled with the increased preload associated with dynamic exercise, leads to an increase in both the E and A velocities which is the pattern of response of the normal ventricle [5–8]. It is not known if hypertensives with resting diastolic abnormalities have the same ventricular filling pattern in response to exercise. In this study we examined left ventricular diastolic filling patterns in response to treadmill exercise using Doppler echocardiography in normotensive subjects and in hypertensive patients with and without left ventricular hypertrophy.

Methods

Subjects

Sixty-one randomly selected patients were divided into 3 groups. Group 1 consisted of 31 normotensive subjects (16 males, 15 females, mean age 61, range 45 to 77 years). Group 2 consisted of 16 hypertensive patients without LVH (8 males, 8 females, mean age 62, range 36 to 73 years). Group 3 consisted of 14 hypertensive patients with LVH (9 males, 5 females, mean age 66, range 38 to 80 years). No patient in any group had any clinical history of coronary artery disease, valvular heart disease, IHSS, diabetes mellitus, pericardial disease, peripheral vascular disease or lung disease. Only 18 % of these 61 patients were referred for stress echocardiography for evaluation of atypical chest pains, the rest were asymptomatic.

Arterial hypertension was defined as blood pressure readings of greater than 140/90 on at least two separate office visits in the past three years. Left ventricular hypertrophy (LVH) was defined as greater than 1.2 cm thickness of either the interventricular septum or the posterior wall of the left ventricle, or both, recorded by resting M-mode echocardiography obtained at the time of the study.

Study protocol

Treadmill stress echocardiography was performed on all subjects using the Bruce exercise protocol. Tests were done off all medications for at least 24 hours.

Transmitral flow was measured by pulsed Doppler with the sample volume at the mitral valve anulus level in the four chamber apical view at rest, immediately (within 15 seconds) post-exercise, and at 12 minutes post-exercise in all subjects. All measurements were taken in the supine position. Care was taken to obtain the immediate post-exercise measurements at the same heart rate as peak treadmill heart rate. Peak values for E velocity (early passive filling) and A velocity (late atrial filling) were recorded and E/A ratios calculated. Deceleration time was measured as the time from the peak of the E filling velocity to the intersection of the declining slope with the zero baseline. If rapid heart rate resulted in onset of the A filling velocity before the conclusion of the E filling velocity, the declining slope of the E velocity profile was extrapolated to the baseline from the point of onset of the A velocity. The values reported are averages of measurements derived from 3 consecutive representative cardiac cycles.

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Statistical analysis

Differences among multiple groups were examined with analysis of variance (Scheffe F test). A twotailed unpaired Student's t-test was used for comparisons between two groups. P value of < 0.05 was considered significant.

Results

All subjects exercised to greater than 85 % of predicted maximum heart rate for age. No subject in any group developed angina, ST-segment depression or elevation, or regional ventricular wall motion abnormalities with exercise. Resting systolic left ventricular function was normal in all subjects. No subject in Group 3 had a thickness of greater than 1.5 cm in either the interventricular septum or left ventricular posterior wall.

For Group 1, Group 2, and Group 3 there were no significant differences in the resting mean heart rates (76, 74, 74 beats per minute, respectively), immediate post-exercise mean heart rates (149, 145, 150 beats per minute), the 12 minute post-exercise mean heart rates (84, 83, 81 beats per minute), and total treadmill exercise times (7:30, 7:48, 7:18 minutes). At rest, both Groups 2 and 3 had evidence of diastolic left ventricular filling abnormalities. Lower E/A mean ratios were found in Group 2 (0.88) and Group 3 (0.87) compared to the Group 1 (0.95) normals ($p < 0.05$). This was due to the higher mean A wave velocities seen in Group 2 (88.4 m/s) and Group 3 (86.5 m/s) compared to Group 1 (79.7 m/s) ($p < 0.05$). There were no significant differences in the resting E wave velocities between the three groups.

With exercise, all 3 groups showed similar significant ($p < 0.05$) increases in both the mean E velocity (+15.7 %, +11.2 %, +16.5 %) and the mean A velocity (+18.8 %, +11.2 %, +16.9 %) as shown in Figure 1. All 3 groups showed no significant change in the E/A ratio or the deceleration time with exercise. At 12 minutes post-exercise, the mean E and A velocities had returned to mean resting values in all 3 groups. The results are summarized in Table 1.

Thus, even in the presence of resting diastolic abnormalities, the response of left ventricular filling dynamics to treadmill exercise in Groups 2 and 3 was similar to that seen in the Group 1 normal subjects.

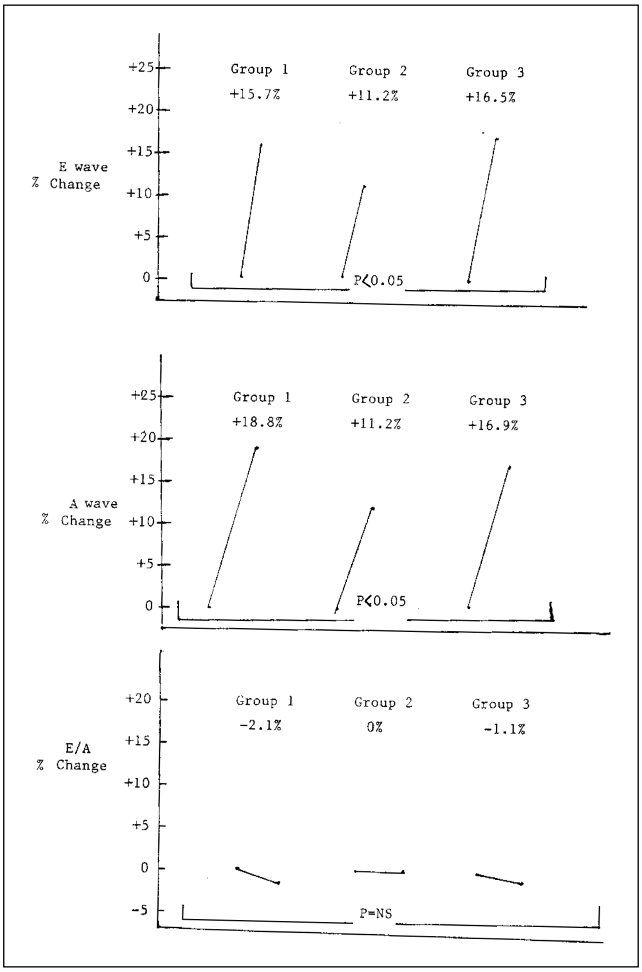

Figure 1. Changes in Doppler left ventricular filling indexes at peak rates immediately post exercise, compared to rest. E % change, percentage change of the mean peak velocity of early filling; A % change, percentage change of the mean peak velocity of late filling; E/A % change, percentage change of mean early to late filling ratio

Discussion

Dynamic exercise involves a complex interaction of tachycardia, increased sympathetic activation, altered loading conditions, and reduced diastolic LV filling time [4]. The normal ventricle responds with increased elastic recoil secondary to accelerated relaxation [4]. The increase in both E and A wave velocities in the normal subjects in this study is consistent with these alterations, and studies on normal subjects and experimental animals have yielded very similar results [5–8].

Although isometric handgrip exercise has been found to unveil ventricular filling abnormalities in hypertensive patients [9], it is not known, especially in the presence of resting diastolic abnormalities, if hypertensives respond to dynamic exercise in the same way. Several mechanisms could cause

Table 1. Mean Values of Left Ventricular Filling Parameters

	Group 1	Group-2	Group 3
E-Wave (m/s)			
Rest	75.6 ± 11.9	77.5 ± 13.4	75.1 ± 13.9
Peak	87.5 ± 12.0	86.2 ± 21.7	87.5 + 20.5
12 min.	74.4 ± 10.3	72.2 ± 13.5	74.8 ± 13.7
A-Wave (m/s)			
Rest	79.7 ± 15.1	88.4 ± 19.1	86.5 ± 20.0
Peak	94.7 ± 16.1	98.3 ± 17.5	101.2 ± 16.6
12 min.	80.4 ± 17.8	89.0 ± 15.7	89.9 ± 15.5
E/A Ratio			
Rest	0.95 ± 0.21	0.88 ± 0.22	0.87 ± 0.24
Peak	0.93 ± 0.24	0.88 ± 0.22	0.86 ± 0.22
12 min.	0.93 ± 0.26	0.83 ± 0.23	0.83 ± 0.22
Deceleration (msec)			
Rest	255 ± 57	248 ± 84	275 ± 81
Peak	236 ± 53	258 ± 83	258 ± 85
12 min.	245 ± 65	260 ± 70	254 ± 77
Heart Rates			
Rest	76	74	74
Peak	149	145	150
12 min.	84	83	81

For all 3 groups, the differences between rest and peak mean E and mean A wave velocities are statistically significant ($p < 0.05$). There are no differences between mean rest and peak E/A ratios and deceleration times ($p = ns$). Values are reported as ± 1 standard deviation. Mean heart rates are reported in beats per minute.

diastolic filling patterns in exercising hypertensives to be different from normal exercise filling patterns. A hypertensive myocardium showing impaired diastolic relaxation at rest may not be able to accelerate relaxation under the haemodynamic stress of exercise, leading to further abnormalities in ventricular filling. Even in the absence of coronary artery disease, alterations in subendocardial perfusion associated with decreased coronary flow reserve in the hypertrophied ventricle could be severe enough during exercise to cause further diastolic dysfunction [4].

Despite these considerations, a study by Kapuku et al. showed that borderline hypertensives responded to bicycle exercise with increases in E and A velocities similar to those seen in normotensive subjects [8]. In this study we likewise found that hypertensives, with or without mild LVH, responded to treadmill exercise with a similar pattern to that seen in the normal subjects: increased E and A wave velocities, no change in the E/A ratios or deceleration times. Comparisons between gender specific responses were not done because of the small number of females in Group 3.

The findings of this study apparently contradict the hypothesis that dynamic exercise enhances diastolic left ventricular dysfunction in hypertensive patients. It may be that the level of haemodynamic stress in this exercise study was not large enough to cause a change from the normal pattern in hypertensives. It is possible that greater degrees of LV hypertrophy may be necessary before the exercise LV filling pattern deviates from the normal response. It is possible that imaging the patients in the supine position while exercising them upright may have had some effect on our results, especially as Doppler filling velocities are sensitive to changes in preload [10]. However, as we took great care to obtain the images within seconds of stopping exercise while still at or very close to the true peak heart rate, we think that the patterns seen are primarily the effect of exercise.

Future larger studies may help to further clarify these issues.

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