

Hemodynamic Determinants of Exercise-Induced Abnormal Blood Pressure Response in Hypertrophic Cardiomyopathy

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OBJECTIVES	We sought to assess the hemodynamics of exercise in patients with hypertrophic cardiomyopathy (HCM), with and without an exercise-induced abnormal blood pressure (BP) response, by ambulatory radionuclide monitoring of left ventricular (LV) function with the VEST device (Capintec Inc., Ramsey, New Jersey).
BACKGROUND	Blood pressure fails to increase >20 mm Hg during exercise in about one-third of patients with HCM. This carries a high risk of sudden death.
METHODS	Forty-three patients with HCM and 14 control subjects underwent maximal symptom-limited exercise on a treadmill during VEST. The VEST data were averaged for 1 min and analyzed at baseline, 3 min and peak exercise. The LV end-diastolic, end-systolic and stroke volumes, cardiac output and systemic vascular resistance were expressed as the percentage of baseline.
RESULTS	Ejection fraction and stroke volume fell in patients with HCM, although they increased in control subjects ($p < 0.001$ and $p = 0.002$, respectively). Cardiac output increased significantly more in control subjects than in patients with HCM ($p = 0.001$). In 17 patients with HCM (39%) with an abnormal BP response, ejection fraction and stroke volume fell more ($p = 0.032$ and $p = 0.009$, respectively) and cardiac output increased less ($p = 0.001$) than they did in patients with HCM with a normal BP response. Systemic vascular resistance decreased similarly in patients with HCM, irrespective of the BP response.
CONCLUSIONS	In patients with HCM with and without an abnormal BP response, abnormal hemodynamic adaptation to exercise was qualitatively similar but quantitatively different. An abnormal BP response was associated with exercise-induced LV systolic dysfunction. This causes hemodynamic instability, associated with a high risk of sudden cardiac death. (J Am Coll Cardiol 2002;40:278–84) © 2002 by the American College of Cardiology Foundation

Sudden cardiac death is an ominous complication of hypertrophic cardiomyopathy (HCM), especially in adolescents and young adults, and it may be the first clinical manifestation in previously asymptomatic patients (1,2). An abnormal blood pressure (BP) response during exercise occurs in about one-third of patients with HCM, and it has been associated with a high risk of sudden cardiac death, as it may lead to hemodynamic instability, which may trigger life-threatening arrhythmias (1,3–6).

The mechanism responsible for exercise-induced abnormal BP in patients with HCM is still debated. Counihan et al. (3) and Frenneaux et al. (4) reported that an abnormal BP response to exercise was associated with an excessive fall in systemic vascular resistance during exercise, due to a peripheral vasodilatory mechanism. Conversely, a more recent study demonstrated that an abnormal BP response during exercise was associated with the development of subendocardial ischemia in patients with HCM (7), thus

implying a central mechanism, through a decline in left ventricular (LV) systolic function.

In patients with HCM, myocardial ischemia without significant coronary artery stenosis has been demonstrated by fixed or reversible defects (8) or cavity dilation during exercise thallium perfusion (7), myocardial lactate production during atrial pacing (9) and positron emission tomography (10). Moreover, ischemia-related LV systolic dysfunction during exercise has been observed in about one-half of patients with HCM during exercise (11,12).

The aim of our study was to assess the effects of exercise on hemodynamics in patients with HCM with and without an abnormal BP response to exercise, as well as in healthy subjects, by ambulatory radionuclide monitoring of LV function (VEST), a method that reliably allows the assessment of LV function.

METHODS

Patient group. Fifty-three consecutive patients with HCM were enrolled from our cardiomyopathy outpatient clinic at Federico II University School of Medicine. The diagnosis of HCM was based on echocardiographic evidence of hypertrophied, nondilated LVs, without an apparent cause, such as systemic hypertension and aortic stenosis (13,14). All

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Abbreviations and Acronyms

ANOVA	= analysis of variance
BP	= blood pressure
ECG	= electrocardiogram
HCM	= hypertrophic cardiomyopathy
LV	= left ventricular
VEST	= ambulatory radionuclide monitoring device

patients were in normal sinus rhythm. All cardioactive medications were withdrawn for at least five half-lives before the study in patients with HCM. Ten patients were subsequently excluded upon acknowledging the inadequacies of VEST: movement of the detector in three patients, gating problems in three patients and technical problems with the recorded data in four patients. The remaining 43 patients with HCM (age 37 ± 14 years [range 16 to 64 years]; 33 men and 10 women) were included in the final study group.

Fifteen age- and gender-matched healthy subjects with no history of cardiovascular disease, a normal clinical examination (including BP) and a normal surface electrocardiogram (ECG), volunteered to enroll in the study and served as the control group. One control subject was subsequently excluded because of VEST detector movement. The remaining 14 control subjects (age 36 ± 13 years [range 23 to 60 years]; 9 men and 5 women) were included in the final study group.

Echocardiography. Each subject underwent M-mode and two-dimensional echocardiography, followed by color flow imaging and pulsed and continuous wave Doppler ultrasonography. Echocardiography was performed using a Hewlett-Packard ultrasonic scanner (Sonos 1000, Andover, Massachusetts) equipped with 2.5-MHz and 1.9-MHz transducers. M-mode echocardiograms were obtained from the two-dimensional images under direct anatomic visualization. Left atrial, LV end-diastolic and LV end-systolic diameters were measured according to the guidelines of the American Society of Echocardiography (15) and were normalized for body surface area. Left ventricular hypertrophy was evaluated from the short-axis view by dividing the LV wall into four segments at the level of mitral valve and papillary muscle (16). An index of the extent of LV hypertrophy was calculated by adding the maximal wall thickness measured (at either the mitral valve or papillary muscle level) in each of the four ventricular segments (anterior and posterior septum and posterior and lateral walls) (17,18).

Color flow Doppler imaging was used for identifying and semiquantitatively estimating mitral regurgitation (19). The LV outflow tract gradient was recorded both at rest and during provocation by amyl nitrite inhalation, with a 1.9-MHz nonimaging transducer, using the simplified Bernoulli equation: $P = 4V^2$, where P = pressure and V = flow velocity. Care was taken to distinguish the ejection velocity from the mitral regurgitation jet (20).

Radionuclide angiography. Red blood cells were labeled in vivo with 15 to 20 mCi of technetium-99m. Each subject underwent equilibrium radionuclide angiography in the supine position, under control conditions, to determine the basal LV ejection fraction. Counts were collected by a small field-of-view Anger camera (LEM ZLC, Siemens Gamma-sonics, Des Plaines, Illinois), in a 45° left anterior oblique projection with a 15° craniocaudal tilt. Images were acquired with 2× digital zoom in frame mode by ECG gating at a framing rate of 30 frames/cardiac cycle on a dedicated computer system. Radionuclide angiograms were analyzed with standard commercial software (Starcam 300 A/M, General Electric, Milwaukee, Wisconsin). The ejection fraction was computed on the raw time-activity curve. Accuracy and reproducibility of measurements in our laboratory have been reported previously (21).

VEST. Immediately after radionuclide angiography, the VEST detector (Capintec, Inc., Ramsey, New Jersey) was placed over the subject's chest and tightened to ensure stable contact. Because VEST is a nonimaging device, it was positioned under gamma camera control over the LV (22). At the end of the protocol, a 30-s static image was obtained to confirm that the radionuclide detector had not moved during recording. At the end of monitoring, data were reviewed for technical adequacy. Briefly, the average count rate (decay-corrected) of the entire recording was displayed: if the curve had abrupt >10% deviation from a straight line, the VEST study was considered inadequate: sudden shifts in the slope of the line indicate detector movement or instrument malfunction (22,23). Radionuclide and ECG data were averaged for 60-s intervals and analyzed at baseline, 3 min and peak exercise. The ejection fraction was computed as stroke counts divided by the background-corrected end-diastolic counts. Background was determined by matching the initial rest VEST ejection fraction value to that obtained by the gamma camera. Systemic vascular resistance was calculated according to the following formula: $80 \times (\text{mean arterial pressure} / \text{LV cardiac output})$. The LV end-diastolic, end-systolic and stroke volumes, cardiac output and systemic vascular resistance were considered to be 100% at the beginning of the study and were subsequently expressed relative to this value.

Exercise test. All patients and control subjects underwent maximal symptom-limited treadmill exercise testing using the Bruce protocol (24). The test duration was measured in seconds. Blood pressure was measured using a cuff sphygmomanometer at rest, 1-min intervals during exercise and 1-min intervals for 5 min during the recovery period after exercise. A normal BP response was defined as a gradual increase of at least 20 mm Hg in systolic BP during exercise. An abnormal BP response was considered, according to most studies, as: 1) an increase or decrease in systolic BP <20 mm Hg during exercise, compared with baseline; 2) an initial increase in systolic BP with a subsequent fall of >20 mm Hg, compared with peak BP; and 3) a continuous decrease in systolic BP >20 mm Hg throughout the exercise

Table 1. Clinical, Echocardiographic and Radionuclide Baseline Characteristics of Patients With HCM and Control Subjects

	Patients With HCM (n = 43)	Control Subjects (n = 14)	p Value
Age (yrs)	37 ± 14	36 ± 13	NS
Male gender (%)	32 (74%)	9 (64%)	NS
Left atrial diameter (mm)	44 ± 6	32 ± 3	<0.001
LV end-diastolic diameter (mm)	45 ± 6	48 ± 2	NS
LV end-systolic diameter (mm)	25 ± 5	32 ± 3	<0.001
Maximal LV wall thickness (%)	21 ± 5	9 ± 1	<0.001
Ejection fraction (%)	71 ± 11	60 ± 5	<0.001
Peak filling rate (stroke counts/s)	3.2 ± 1.0	3.0 ± 0.8	NS
Exercise duration (s)	498 ± 136	558 ± 114	NS

Data are presented as the mean value ± SD or number (%) of patients or control subjects.

HCM = hypertrophic cardiomyopathy; LV = left ventricular; NS = not significant.

test, compared with baseline (3–6). These last two conditions are termed “hypotensive BP response,” whereas the first one represents a flat response. A three-lead ECG monitor allowed continuous evaluation of the heart rate, rhythm, T-wave and ST-segment.

Statistical analysis. Data are expressed as the mean value ± SD in the text and tables; however, in order for the figures to be more easily readable, standard errors of the mean value are provided there. The chi-square test with Yate’s correction for continuity was used to analyze categorical variables. Normal distribution of all continuous variables, including the percent differences from peak to baseline, was tested using the one-sample Kolmogorov-Smirnov test. All variables were normally distributed; parametric tests were therefore used. The unpaired Student *t* test was performed to determine differences between mean values for continuous variables, as appropriate. The difference in the time courses of parameters between groups was analyzed by two-way analysis of variance (ANOVA) for repeated measures. Interactions between the time course of parameters and groups were tested as: time course × groups. If any interactions were significant, a post-hoc comparison was performed using the unpaired *t* test with Bonferroni correction to detect differences between two groups at given time points. For descriptive purposes, the effect of exercise within each group was analyzed by one-way ANOVA for repeated measures.

A *p* value <0.05 was considered statistically significant. All statistical calculations were performed by SPSS for Windows, release 10.0 (SPSS Inc., Chicago, Illinois).

RESULTS

Patient characteristics. The clinical, echocardiographic and radionuclide baseline characteristics of the 43 patients with HCM and the 14 control subjects are shown in Table 1. Patients with HCM had a smaller LV end-systolic

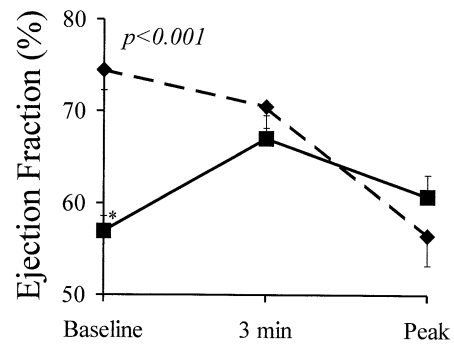


Figure 1. Mean value ± SEM of ejection fraction (%) measured at baseline, 3 min and peak exercise in the two study groups (squares = control subjects; diamonds = patients with hypertrophic cardiomyopathy [HCM]). The *p* value reflects two-way analysis of variance for repeated measures. **p* < 0.001 vs. patients with HCM, as tested by post-hoc analysis.

dimension, a larger maximal left atrial dimension and a higher baseline ejection fraction, compared with control subjects.

Hemodynamics during exercise. The achieved level of exercise was maximal in 10 control subjects (73%) and 19 patients with HCM (44%): the test was interrupted because of fatigue in 4 control subjects (27%) and 12 patients (28%) and because of shortness of breath and/or angina in 12 patients (28%). In one patient, the exercise test was stopped because syncope occurred. The mean exercise duration was similar between the two groups.

During exercise, the heart rate increased significantly (*p* < 0.001 by one-way ANOVA) and similarly in patients with HCM and control subjects (percent difference between peak exercise and baseline: 84 ± 22% and 90 ± 35%, respectively; *p* = NS by two-way ANOVA).

Systolic BP during exercise increased significantly more in control subjects than in patients with HCM (36 ± 16% vs. 22 ± 23%, respectively, over baseline; *p* = 0.032 by two-way ANOVA).

The LV end-diastolic volume did not change significantly throughout exercise in control subjects (9 ± 7% over baseline; *p* = NS by one-way ANOVA), but it rose in patients with HCM (15 ± 7% over baseline; *p* < 0.001 by one-way ANOVA). There were significant differences in end-systolic volume changes during exercise between control subjects and patients with HCM; in fact, the end-systolic volume increased less in control subjects than in patients with HCM (2 ± 20% and 41 ± 25%, respectively, over baseline; *p* < 0.001 by two-way ANOVA). Ejection fraction (Fig. 1) and stroke volume (Fig. 2) changes were significantly different between control subjects and patients with HCM. They rose in control subjects (10 ± 19% and 21 ± 27% for ejection fraction and stroke volume, respectively, over baseline), whereas they fell in patients with HCM (−20 ± 21% and −5 ± 27%, respectively, over baseline). Cardiac output increased significantly more in control subjects than in patients with HCM (132 ± 70% vs.

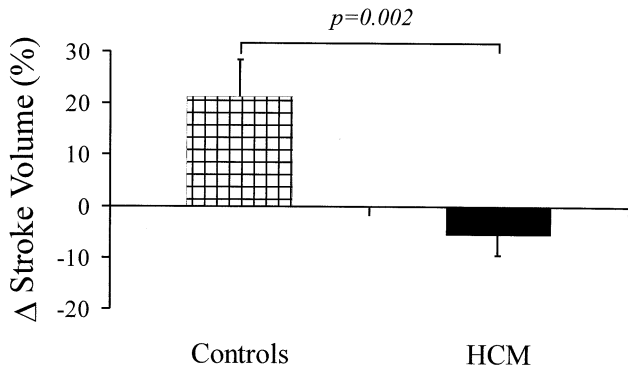


Figure 2. Mean value \pm 1 SEM of percent difference in stroke volume (%) between peak exercise and baseline in the two study groups: control subjects and patients with hypertrophic cardiomyopathy (HCM).

78 \pm 50%, respectively, over baseline; $p = 0.001$ by two-way ANOVA).

Systemic vascular resistance fell during exercise in control subjects and patients with HCM (both $p < 0.001$ by one-way ANOVA), but more so in control subjects ($-43 \pm 14\%$ vs. $-30 \pm 20\%$); however, this difference did not reach statistical significance (Fig. 3).

Hemodynamic adaptation in patients with HCM with exercise-induced abnormal systolic BP. Seventeen patients with HCM (39%) showed an abnormal BP response to exercise: in 3 patients with HCM, systolic BP fell by more than 20 mm Hg from the start of exercise (rest value); in 10 patients, there was an appropriate initial increase in systolic BP, with a subsequent fall of at least 20 mm Hg from peak BP. The remaining four patients had a flat BP response to exercise.

The baseline characteristics of patients with HCM with a normal versus abnormal BP response to exercise are shown in Table 2. The patients with HCM with exercise-induced abnormal BP were significantly younger than those with a normal BP response (age 42 ± 14 years [range 18 to 64 years] and 31 ± 12 years [range 16 to 51 years], respectively; $p = 0.011$).

The mean exercise duration was similar between the two HCM groups. During exercise, the heart rate rose signifi-

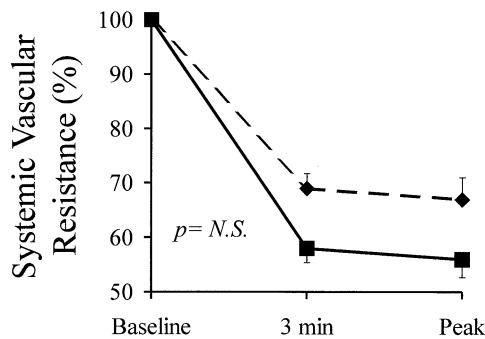


Figure 3. Mean value \pm 1 SEM of systemic vascular resistance (%) measured at baseline, 3 min and peak exercise in the two study groups (squares = control subjects; diamonds = patients with hypertrophic cardiomyopathy). The p value reflects two-way analysis of variance for repeated measures.

cantly and similarly (both $p < 0.001$ by one-way ANOVA) in patients with HCM with a normal systolic BP response and exercise-induced abnormal BP ($94 \pm 40\%$ and $85 \pm 24\%$, respectively, over baseline). The ejection fraction increased slightly in patients with HCM with a normal systolic BP response at 3 min over baseline and significantly fell at peak exercise (Fig. 4). Patients with an abnormal BP response had a similar pattern of changes; the magnitude of the peak ejection fraction decrease was greater in patients with an abnormal BP response than in those with a normal response ($-31 \pm 16\%$ and $-13 \pm 20\%$, respectively, over baseline). Likewise, changes in stroke volume during exercise were significantly different (Fig. 5) between patients with HCM with a normal versus abnormal systolic BP response to exercise. In fact, stroke volume increased in patients with HCM with a normal BP response to exercise, whereas it fell in patients with HCM with an abnormal BP response ($2 \pm 27\%$ and $-18 \pm 21\%$, respectively, over baseline). As a consequence, the increase in cardiac output was significantly different ($p = 0.001$ by two-way ANOVA) between patients with HCM with a normal BP response and those with an abnormal response to exercise ($96 \pm 47\%$ and $51 \pm 41\%$, respectively, over baseline). Systemic vascular resistance decreased similarly (both $p < 0.001$ by one-way ANOVA) (Fig. 6) in patients with HCM without and with an abnormal BP to exercise ($-33 \pm 20\%$ and $-32 \pm 26\%$, respectively, over baseline).

DISCUSSION

Hemodynamic adaptation to exercise. In this study, we demonstrated that patients with HCM show an abnormal hemodynamic adaptation to exercise, as compared with control subjects. Patients with HCM and control subjects showed a similar duration of exercise and maximal achieved heart rate, but there was a significant difference in LV systolic function. In fact, the ejection fraction (Fig. 1) and stroke volume (Fig. 2) increased significantly in control subjects, whereas they fell in patients with HCM. Furthermore, cardiac output increased significantly less in patients with HCM than in control subjects. An impaired systolic response to exercise in patients with HCM was due to the combination of a significant increase in LV end-diastolic and, more so, in end-systolic volumes. This suggests that a potential mechanism for such an abnormal response to exercise in patients with HCM may be myocardial ischemia. Myocardial ischemia has been clearly demonstrated in patients with HCM (7-12,25) and is a major risk factor for sudden cardiac death (26). It could be induced by an increased myocardial oxygen demand (27), such as occurs during exercise. Our findings were consistent with previous studies that demonstrated exercise-induced myocardial ischemia during VEST monitoring in patients with coronary artery disease (28) in whom exercise-related myocardial ischemia caused a fall in the ejection fraction and an increase in end-systolic and end-diastolic volumes. Similarly, Taki et

Table 2. Clinical, Echocardiographic and Radionuclide Baseline Characteristics of Patients With HCM With a Normal Blood Pressure Response (HCM Normal Response) and Patients With HCM With an Exercise-Induced Abnormal Blood Pressure Response (HCM abnormal response)

	HCM Normal Response (n = 26)	HCM Abnormal Response (n = 17)	p Value
Age (yrs)	42 ± 14	31 ± 12	0.011
Male gender (%)	22 (85%)	11 (65%)	NS
Family history of sudden cardiac death (%)	3 (12%)	7 (41%)	NS
NYHA functional class >2 (%)	7 (28%)	8 (47%)	NS
History of angina (%)	3 (12%)	6 (35%)	NS
History of syncope (%)	7 (28%)	3 (18%)	NS
Left atrial diameter (mm)	44 ± 7	44 ± 5	NS
Left atrial fractional shortening (%)	20 ± 8	21 ± 7	NS
LV end-diastolic diameter (mm)	46 ± 6	44 ± 4	NS
Incidence of LV outflow tract obstruction >30 mm Hg at rest or >50 mm Hg during stimulation by amyl nitrate inhalation (%)	11 (44%)	8 (47%)	NS
Maximal LV wall thickness (mm)	22 ± 5	21 ± 4	NS
Degree of LV hypertrophy (mm)	66 ± 14	61 ± 11	NS
Ejection fraction (%)	72 ± 12	72 ± 5	NS
Peak filling rate (stroke counts/s)	3.1 ± 1.0	3.2 ± 1.2	NS
Exercise duration (s)	514 ± 130	474 ± 147	NS

Data are presented as the mean value ± SD or number (%) of patients or control subjects. NYHA = New York Heart Association; other abbreviations as in Table 1.

al. (11) demonstrated that LV dysfunction during exercise, as assessed by VEST, is a common phenomenon in patients with HCM, and they postulated that it was due to myocardial ischemia. In a recent report, Okeie et al. (12) showed that myocardial ischemia was the primary reason for the decrease in ejection fraction in patients with HCM during exercise, as assessed by VEST. This was associated with the development of dobutamine-induced new wall motion abnormalities. Besides, there was a significant correlation between changes in ejection fraction and the number of dysfunctional LV segments (12).

Hemodynamics of exercise-induced abnormal BP. An abnormal BP response to exercise has been regarded as evidence for hemodynamic instability and has been proposed as a marker of an increased risk of sudden cardiac death in patients with HCM (1,3,4), especially in young

patients (5,6). In our study, 13 patients with HCM (30%) showed a hypotensive systolic BP response to exercise and 4 (9%) showed a flat response.

Patients with HCM with an abnormal BP response to exercise were younger than those with a normal systolic BP response, according to previous studies (5,6). Furthermore, they exhibited a more abnormal hemodynamic adaptation to exercise and impairment in systolic function, as shown by a more pronounced fall in ejection fraction (Fig. 4), stroke volume (Fig. 5) and cardiac output. Hence, the mechanism for exercise-induced abnormal BP could be a cascade from marked LV systolic dysfunction, to a decrease in cardiac output and finally to hypotension. Thus, we hypothesize a “central” mechanism responsible for abnormal BP during

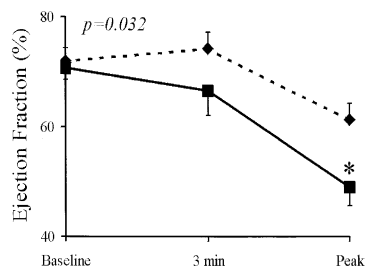


Figure 4. Mean value ± SEM of ejection fraction (%) measured at baseline, 3 min and peak exercise in the two study groups (squares = patients with hypertrophic cardiomyopathy [HCM] and a normal systolic blood pressure [BP] response to exercise; diamonds = patients with HCM and an abnormal systolic BP response to exercise). The p value reflects two-way analysis of variance for repeated measures. *p = 0.003 vs. HCM abnormal response, as tested by post-hoc analysis.

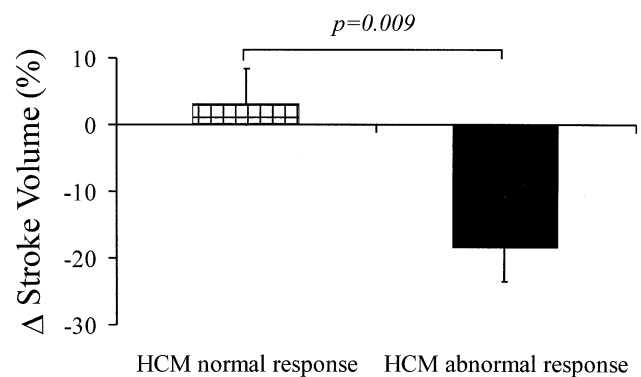


Figure 5. Mean value ± SEM of percent difference in stroke volume (%) between peak exercise and baseline in the two study groups: patients with hypertrophic cardiomyopathy (HCM) and a normal systolic blood pressure (BP) response to exercise and patients with HCM and an abnormal systolic BP response to exercise.

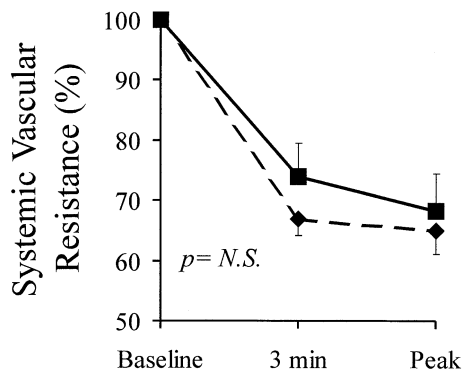


Figure 6. Mean value \pm SEM of systemic vascular resistance (%) measured at baseline, 3 min and peak exercise in the two study groups (squares = patients with hypertrophic cardiomyopathy [HCM] and a normal systolic blood pressure [BP] response to exercise; diamonds = patients with HCM and an abnormal systolic BP response to exercise). The p value reflects two-way analysis of variance for repeated measures.

exercise. In patients with coronary artery disease, exercise-induced hypotension is strictly associated with severe coronary artery disease and is related to global and regional LV systolic dysfunction (29,30).

Our results complement those of Yoshida et al. (7), who demonstrated that in patients with HCM, an exercise-induced hypotensive response was related to subendocardial ischemia, as assessed by thallium scintigraphy, with a higher prevalence of LV cavity dilation than in those with a normal BP response. Our findings shed light on the mechanism by which subendocardial ischemia is related to an abnormal BP response.

An exercise-induced BP fall was abolished by alcohol ablation of the septum in patients with HCM with LV outflow tract obstruction (31). Because LV outflow tract obstruction is a major determinant of oxygen consumption in patients with HCM, its decrease should reduce or abolish exercise-induced ischemia. This suggests a central mechanism, rather than a peripheral mechanism, for an abnormal BP response to exercise in patients with HCM.

Previous studies (3,4) have suggested that in patients with HCM, exercise-induced hypotension was due to an exaggerated decrease in systemic vascular resistance. These investigators assessed systemic vascular resistance from the forearm blood flow, and they supposed that activation of ventricular mechanoreceptors may relate to exercise-induced hypotension or increased sensitivity of arterial baroreflex, hence a “peripheral” mechanism. In our study, we assessed global systemic vascular resistance indirectly, as the ratio of mean BP to cardiac output, and we showed a similar decrease in systemic vascular resistance during exercise in patients with HCM with exercise-induced hypotension and in patients with a normal BP response to exercise. The difference in systemic vascular resistance responses during exercise between these previous studies and ours may be explained by the difference in methods; in fact, systemic vascular resistance changes differently in different regions. Global systemic vascular resistance is the sum of different

adaptation processes to exercise: the local effect of exercise metabolites, cortical influences, reflex activation of metabolic receptors in skeletal muscle and increased ventricular baroreceptor activity (32). Therefore, it is reasonable that local changes in resistance do not reflect modifications in global systemic vascular resistance.

An alternative explanation is that an abnormal decrease in systemic vascular resistance may cause or aggravate ongoing myocardial ischemia during exercise. Nevertheless, this hypothesis is unlikely, because in our study systemic vascular resistance showed a similar decrease during exercise in patients with HCM with and without exercise induced-hypotension.

Study limitations. The VEST monitoring is a functional tool that assesses relative LV volume curves. We hypothesize that the mechanism responsible for exercise-induced abnormal BP be myocardial ischemia; nevertheless, we only have evidence of exercise-induced systolic dysfunction, with a fall in stroke volume, ejection fraction and, hence, cardiac output.

Conclusions. Patients with HCM showed an abnormal hemodynamic adaptation to exercise, with LV systolic dysfunction likely due to exercise-related myocardial ischemia, as compared with control subjects.

One-third of patients with HCM had an abnormal systolic BP response to exercise. The abnormal hemodynamic adaptation to exercise in patients with HCM with and without an abnormal BP response to exercise was qualitatively similar but quantitatively different and may have a common denominator in exercise-related myocardial ischemia. Exercise-induced abnormal BP was associated with exercise-induced LV systolic dysfunction, and this causes hemodynamic instability, which may be responsible for the high risk of sudden cardiac death.

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