The Effect of Isometric Exercise on the Left Ventricular Volume in Normal Man

By Miltiadis A. Stefadouros, M.D., William Grossman, M.D., Mahfouz El Shahawy, M.D., and A. Calhoun Witham, M.D.

SUMMARY

The effect of isometric handgrip exercise (IHG) on left ventricular (LV) size and performance was studied noninvasively on 15 normal subjects at rest and at the end of 3 min of IHG at 50% of maximum contraction. Left ventricular internal diameter was measured at end-diastole and end-systole on LV echograms, the indirect carotid pulse was recorded, and blood pressure measured by sphygmanometry. Using the cube formula, LV end-diastolic (EDVI) and end-systolic (ESVI) volume indices were computed, and stroke (SI) and cardiac (CI) indices were derived. Mean blood pressure (BPm) and systemic vascular resistance (SVR) were calculated from these data by a technique previously described. In comparison to values at rest, IHG resulted in a significant (P < 0.01) rise in CI (3.5 ± 0.2 to 4.4 ± 0.3 L/min/m²), BPm (87 ± 2 to 120 ± 4 mm Hg) and heart rate (79 ± 3 to 97 ± 4 beats/min). The product (SI × BPm), used as an index of LV stroke work, increased substantially (52 ± 3 to 74 ± 4 gm-m/m²). No significant change was noted in EDVI (60 ± 4 to 62 ± 3 ml/m²), SI (44 ± 2 to 46 ± 2 ml/m²), and SVR (1209 ± 69 to 1275 ± 63 dynes/sec/cm²).

Thus normal hearts responded to IHG by increasing CI through tachycardia and pumping the same SI against increased afterload, without utilizing diastolic volume reserves. These data support the hypothesis that isometric muscular exercise leads to an augmentation of LV myocardial contractility in normal man.

Additional Indexing Words:
Echocardiography
Ventricular function curve

Additional Indexing Words:
End-diastolic volume
Stroke work

ISOMETRIC MUSCLE CONTRACTION represents a convenient provocative test for evaluation of the functional integrity of the human left ventricle. The interpretation customarily has been based on changes in the relationship between stroke work and end-diastolic pressure, rather than its volume, within the left ventricle because rapid sequential changes in volume are more difficult to determine by traditional angiographic or indicator dilution methods. Thus, it is not currently known to what extent, if at all, the volume reserves of the normal left ventricle are utilized to enable it to cope with the extra workload induced by isometric muscle contraction.

During the past few years several investigators have demonstrated the feasibility of noninvasively measuring one "dimension" of the left ventricular cavity by use of reflected ultrasound. It has been shown that this "dimension" can be successfully employed in the calculation of left ventricular volume and output.

The purpose of this study was to establish whether isometric muscle contraction is associated with alterations in the volume of the normal left ventricle, as determined by this technique.

Material and Methods

Fifteen healthy volunteers, whose ages and sex are listed in table 1, formed the population of this study. All were considered to be normal on the basis of history, physical examination, and electrocardiogram. A dynamometer was employed for the isometric handgrip (IHG) test. The maximal voluntary contraction was determined at least 10 min prior to the experiment, as the average of three consecutive maximal attempts. The subjects were suitably instructed in order to avoid performing the Valsalva maneuver, and carefully observed for this during IHG test. All studies were conducted in the postabsorptive state and included simultaneous measurement of arterial pressure by sphygmanometry of the noncontracting arm and recording of both indirect carotid pulse and left ventricular (LV) echocardiogram. Data were obtained just before and at the end of a 3 min period of IHG test at 50% of maximal voluntary contraction. Blood pressure was measured, and the indirect carotid pulse was recorded at the same time on
Table 1

Echocardiographic and Other Data from 15 Normal Subjects

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<th>#</th>
<th>Age</th>
<th>Sex</th>
<th>Dd (mm)</th>
<th>Ds (mm)</th>
<th>EDVI (ml/m²)</th>
<th>SI (ml/m²)</th>
<th>HR (L/min/m²)</th>
<th>Blood pressure</th>
<th>SVR (sec/cm²)</th>
<th>(SI × BPm)</th>
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Abbreviations: Dd and Ds = end-diastolic and end-systolic internal dimension of the left ventricle; EDVI = left ventricular end-diastolic volume index; HR = heart rate; CI = cardiac index; Syst = systolic; Diast = diastolic; SVR = systemic vascular resistance; BPm = mean blood pressure; F = female; M = male, R = at rest; E = exercise; SEM = standard error of the mean.

*P less than 0.01 in comparison with value at rest.

*Model PSA Electronics for Medicine
†Smith Kline Instruments, Palo Alto, California

an Electronics for Medicine DR-6 recorder at a paper speed of 100 mm/sec, utilizing a piezoelectric transducer. The values for blood pressure were applied to the indirect carotid pulse waves, which were then planimetered to determine the mean blood pressure (BPm) which was used to calculate the systemic vascular resistance, as previously reported from this laboratory.13

Echocardiographic studies were performed using a Smith Kline Ekoline 20 echograph† equipped with a 2.25 MHz focused transducer (Model C-12). The echocardiograms were recorded on pictures taken with a Polaroid camera, during M-mode presentation at a medium sweep velocity that permits completion of a full sweep cycle within 2 sec. While most of the studies were conducted in supine position, three subjects (1, 9, and 10, table 1) had both studies conducted in the left lateral position, because this provided more stable monitoring of the LV echogram throughout the cardiac cycle. The objective of the echocardiographic study was to identify echoes from the endocardium of the left side of the interventricular septum and that of the LV posterior wall, in a plane immediately below the mitral valve, as previously described.7,18 the vertical distance between these two echoes, approximating the LV internal minor axis, was measured at the end of diastole (Dd) and end of systole (Ds) in all (2-3) available cycles on each picture and the average value, rounded to the nearest 0.5 mm, was used for calculation of LV end-diastolic and end-systolic volume from the formula:7

\[ V = 1.047 D^3 \]

End-systole was defined as the moment of the peak upward motion of the echo of the endocardium of the LV posterior
wall; end-diastole was considered to coincide with the peak of the R wave of the electrocardiogram. The effect of skeletal muscle contraction during isometric exercise on the ECG, however, was so profound that identification of the QRS complexes was impossible in seven cases, even on leads not involving the exercising arm (fig. 1). In these cases, the time relation of the peak of the R wave to the movement of the LV posterior wall endocardial echo was noted on the resting LV echocardiogram and used for the identification of the corresponding moment of end-diastole on the picture taken during exercise. Left ventricular volume, stroke volume (1.047 [Dd - Ds]) and cardiac output (CO) were indexed for body surface area. Since LV stroke work index (SWI) could not be measured noninvasively, the product of stroke volume index and mean arterial pressure (SI × BPm) was employed instead, for it was felt that despite some difference in absolute values, changes in SWI would be reflected in this product. The dimensional units of this product (ml × mm Hg/m²) were converted into the conventional gm-m/m² by multiplying by 0.0136. Systemic vascular resistance (SVR) was calculated from the ratio (BPm × 80)/CO and corrected by use of a regression formula previously developed from comparison of this ratio to the systemic vascular resistance as determined by conventional methods at catheterization. Thus,

SVR = 0.865 (BPm × 80)/CO + 216

During IHG test, specific care was taken by the examiner to avoid false changes in LV dimensions produced by involuntary alterations in either the exact point of application of the ultrasonic transducer on the chest wall or the direction of the ultrasonic beam in relation to the heart. To minimize this possibility, the following rules were adopted: 1) IHG contraction started immediately (seconds) after a satisfactory LV echocardiogram was obtained; and 2) Once the resting LV echocardiogram was obtained and throughout the 3-min period of IHG test, neither change in the position and tilt of the transducer nor modification in the setting of the several control switches of the echograph was permitted. Strict application of this policy resulted in the necessity to discard studies in several cases in whom, for unexplained reasons, unacceptable deterioration in resolution of the LV echocardiogram was observed during IHG contraction. Additional subjects were excluded because of their inability to maintain the required level of contraction for 3 min or because of performing the Valsalva maneuver during the test. Therefore, the data presented were derived from 15 subjects with high quality echocardiograms, selected from 26 persons on whom the study was attempted.

Statistical analysis of the results was done on an Olivetti (Programma 101) calculator using the t-test for paired data. The level of statistical significance was set at P < 0.01.

Results

As can be seen in table 1, during IHG contraction, Dd remained unchanged in five cases, increased in seven (by 0.5 to 4 mm) and decreased in the remaining three (by 1 to 3 mm). In the group as a whole, the observed differences in Dd (47 ± 0.9 to 47.6 ± 0.8 mm, mean ± standard error) and corresponding LV end-diastolic volume index (60 ± 4 to 62 ± 3 ml/m²) during IHG test were insignificant (fig. 2). Similarly, no significant changes were noted in DS (29.7 ± 1 to 30 ± 1 mm) and corresponding LV end-systolic volume index (16 ± 1 to 16 ± 2 ml/m²). Significant (P < 0.01) increases in heart rate (by 18 beats/min), cardiac index (by 0.9 L/min/m²) and mean arterial pressure (by 33 mm Hg) were observed during IHG test; in comparison to control values at rest, these changes were equivalent to increases of 23%, 26%, and 38%, respectively. In contrast, LV stroke volume index and systemic vascular resistance exhibited no significant changes (fig. 2). The product (SI × BPm) did increase in all cases (5% to 89%, average 44%, P < 0.01).

Figure 1

Representative left ventricular echocardiogram at rest and during isometric handgrip exercise. LV = left ventricular cavity; LVPW = left ventricular posterior wall echo, RV = right ventricular cavity; Dd and Ds = end-diastolic and end-systolic left ventricular internal dimension.

Figure 2

Hemodynamic alterations observed during isometric exercise. HR = heart rate; CI = cardiac index; BP = blood pressure; S = systolic; M = mean; D = diastolic; SVR = systemic vascular resistance; Dd and Ds = end-diastolic and end-systolic internal dimension of the left ventricle; EDVI and ESVI = end-diastolic and end-systolic volume index; SI = stroke index; BPm = mean blood pressure.
Discussion

The hemodynamic alterations induced by sustained isometric muscle contraction in both normal subjects and patients with impaired myocardial function have been adequately established. However, no information is currently available concerning the effects of this intervention on left ventricular volumes, either because of the technical difficulties, or the potential risks associated with determining LV volume by conventional angiographic or indicator dilution techniques simultaneously with isometric exercise stress. Using purely noninvasive techniques, we have studied the effects of isometric handgrip (IHG) exercise on LV chamber volumes and other hemodynamic changes in normal man. The validity of using these methods for measuring LV dimension, volume, and output as well as for calculating mean arterial pressure and systemic vascular resistance has been previously documented by use of cardiac catheterization as reference. Our findings, apart from those pertinent to LV volume, were qualitatively and quantitatively similar to those obtained from normal subjects by catheterization, both at rest and during similar levels of isometric exercise. Thus, tachycardia was primarily responsible for the observed increase in cardiac output during exercise, with only minimal increase in stroke volume. Increasing cardiac output, in turn, was the chief mechanism that led to the observed elevation in arterial pressure in the presence of a practically unaltered systemic vascular resistance. The magnitude of these hemodynamic changes confirmed the adequacy of the stress imposed on the heart by IHC test.

Catheterization studies have shown that despite considerable increase in LV stroke work during IHG exercise, normal hearts exhibit minimal increase, no change, or even a small decrease in left ventricular end-diastolic (LVED) pressure. These observations by others are consistent with our observations and support the hypothesis that isometric muscular exercise leads to an augmentation of left ventricular myocardial contractility in normal man. Although in our group as a whole, LVED volume remained unaffected by IHG exercise, small changes did appear in most cases. These data should not be construed as suggesting that the observed changes in LVED volume reflected proportional changes in LVED pressure, for the LV pressure-volume relation is not linear. Even directional changes in LVED pressure should not be inferred from alterations in LVED volume, unless it is assumed that the LV pressure-volume relationship is not modified by isometric exercise. This assumption, however, is not necessary in evaluating the physiologic importance of the observed response to isometric exercise, in the context of the preload-stroke work relation. This is because preload can be expressed more appropriately in terms of initial fiber length than LVED pressure. Thus a shift to the higher ventricular function curve appears to be the only possible explanation for the response exhibited by the eight subjects in whom an increase in the product (SI \times BPm) was accompanied by either a decrease (subjects 2, 5, and 8, table 1) or no change in LVED volume (subjects 6, 11, 12, 13, and 14, table 1). Similar conclusions are probably justified for subjects 3, 4, and 9 (table 1) in whom the observed increase in LV internal dimension was too small (\(\leq 2 \text{ mm}\)) to be construed as representing a true increase in LVED volume, in view of the accuracy of measurements of echocardiographic LV internal dimensions, as documented by Pombo et al. The response observed in the remaining three subjects (1, 7, and 15, table 1) is more difficult to analyze. These subjects exhibited an average increase of 24% in LVED volume accompanied by a 48% increase in the product (SI \times BPm); although a movement along the steep ascending limb of a normal ventricular function curve seems very likely, the contribution of a change in the inotropic state of the heart can neither be established nor excluded.

The vigorous arterial pressure response to IHG exercise per se is not adequate evidence of a similar rise in afterload (wall tension), for the latter depends also on the geometry, volume, and wall thickness of the LV. If one assumes no change in LV geometry during IHG exercise, however, an increase in afterload must have occurred according to Laplace's law, since LV volume (and consequently wall thickness) remained practically unchanged in this group.

In summary, this study has demonstrated that the normal left ventricle is able to meet the extra work load requirements imposed on it by isometric exercise with either minimal drawing on its diastolic volume reserve, or none at all, assisted (in the latter case at least) by an augmented contractility of varying degree.

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