Effect of Sustained Isometric Handgrip Exercise on Left Ventricular Performance

By Richard H. Helfant, M.D., Maria A. DeVilla, M.D., and Steven G. Meister, M.D.

SUMMARY
Sustained isometric handgrip exercise was studied in 28 patients, 19 with and nine without catheterization evidence of heart disease. Significant increases occurred in left ventricular systolic and left ventricular end-diastolic pressures (LVEDP), heart rate, cardiac output, and cardiac index, with decreases in stroke volume and stroke index. When control and abnormal groups were compared, no differences could be demonstrated in systolic pressure or heart rate increases. However, the LVEDP increase in the abnormal subjects (9.7 ± 1.7) was significantly (P < 0.01) higher than in the controls (2.1 ± 0.7). In addition, cardiac index rose significantly (P < 0.025) in the controls (0.8 ± 0.2), but not (P < 0.1) in the abnormal subjects (0.2 ± 0.1). Conversely, there was a significant fall in stroke index in the abnormal (P < 0.005) but not in the control (P < 0.4) group.

When work or stroke-work index–LVEDP relations were compared, the controls uniformly exhibited steep curves, whereas abnormal patients demonstrated curves that were either less steep or flat. ΔWork/ΔLVEDP ratio was ≥1.0 in the controls, with one exception, and ≤0.77 in the abnormal subjects, with one exception.

The test was performed in less than 4 min and no adverse effects were observed. By virtue of its ease, simplicity, safety, and ability to distinguish normal and abnormal ventricular performance, sustained handgrip is a valuable new stress test.

Additional Indexing Words:
Systolic pressure  Left ventricular end-diastolic pressure  Cardiac output
Cardiac index  Heart rate  Stroke-work index  Left ventricular work

The effects of isotonic physical exercise on the circulation are well known and have long been used as a means of uncovering or exaggerating physiologic abnormalities of cardiac function. However, although isometric exercise is a frequent daily stress, relatively little is known about its differential effects on normal and abnormal left ventricular function. The isometric exercise of sustained handgrip causes a rapid and significant increase in systolic and diastolic blood pressure and hence imposes an acute afterload on the left ventricle. Recent preliminary reports have indicated that this stress may be useful in evaluating the hemodynamic reserve of patients with heart disease. The present investigation was undertaken to determine the effect of the isometric stress of sustained handgrip on left ventricular hemodynamics. In addition, the utility of this stress as a method of unmasking or exaggerating abnormalities of left ventricular performance was also examined.

Methods
Studies were performed in 28 patients during cardiac catheterization in the postabsorptive state and the supine position. Premedication consisted

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of 50 mg of sodium pentobarbital (Nembutal) and 50 mg of meperidine (Demerol) intramuscularly. Right-heart catheterization was performed via an antecubital vein cutdown, and left-heart catheterization was performed either via a right brachial arteriotomy or percutaneously, utilizing the right or left femoral artery.

With the patient in the resting state, pressures were measured with Statham P-23 Db strain-gauge transducers. Cardiac output was determined by the dye-dilution method with indocyanine green and a Gilford densitometer. All data were displayed and recorded on an eight-channel oscillographic, photographic recorder (Electronics for Medicine).

After control data were obtained, each patient was asked to squeeze a hand dynamometer (C. H. Stoebling Co.) to the maximum extent possible. After determination of maximum voluntary contraction, each patient was instructed to maintain contraction at one third of the predetermined maximum for 3 min. At the end of the 3-min period and prior to release of the sustained contraction, pressure and cardiac output measurements were repeated.

Left ventricular work was calculated using the formula:

\[
\frac{(LVSP - LVEDP) \times CO \times 1.36}{100}
\]

and stroke-work index was calculated using the formula:

\[
\frac{(LVSP - LVEDP) \times SI \times 1.36}{100}
\]

where \(LVSP\) = left ventricular systolic pressure, \(LVEDP\) = left ventricular end-diastolic pressure, \(CO\) = cardiac output, and \(SI\) = stroke index. Peak systolic pressure was used instead of mean pressure resulting in somewhat high absolute values, but it was felt that this would not significantly affect changes in this index with stress. Left ventricular end-diastolic pressure (LVEDP) was measured following the “a” wave and at the same Q wave–LVEDP interval in the rest and stress states.

After collection of hemodynamic data, a left ventricular cineangiogram was obtained in the right anterior oblique projection and selective cinecoronary arteriography was performed using the Sones or Judkins technique. In patients in whom aortic regurgitation was suspected, an aortogram was also performed. Cine films were taken on a 10 by 6 inch dual-field image amplifier (Siemens) at 64 frames/second using 16-mm Kodak Shellburst film.

For purposes of analysis, subjects were divided into control and abnormal groups. Control subjects, studied for evaluation of chest pain syndromes or murmurs, exhibited normal coronary and left ventricular angiograms and rest hemodynamics. In addition, no evidence of valvular or congenital lesions was exhibited. Abnormal subjects manifested evidence of valvular disease, coronary disease (defined as >75% stenosis of left anterior descending, circumflex, and/or right coronary arteries), or cardiomyopathy (defined as diffusely hypokinetic left ventriculogram with or without abnormal rest hemodynamics and normal coronary arteriograms).

A paired-samples t-test was used to assess changes for each group, and for comparison between groups an independent-samples t-test was utilized.

Results

Table 1 comprises the data on the entire study group with mean values, standard error of the mean, and \(P\) values. Significant increases were found in systolic pressure, LVEDP, cardiac output, heart rate, and cardiac index (fig. 1). However, since heart rate increased more than cardiac output and index, stroke volume and stroke index both fell (fig. 1). (See table 1 for \(P\) values.)

Control versus Abnormal Groups

The control and abnormal groups are compared in figure 2. There was a significant rise in systolic pressure in both groups (fig. 2), but although the increase was somewhat higher in the control group, this was not significant \((P < 0.10)\). LVEDP also rose significantly in both groups, but the increase in LVEDP was significantly higher in the abnormal than in the control group \((P < 0.01)\). In addition, cardiac index rose significantly in the control but not in the abnormal group (fig. 2), and this difference in response between groups was significant \((P < 0.05)\). The relationship between cardiac index and LVEDP at rest and during sustained isometric handgrip is seen in figure 3.

Both groups increased heart rate (fig. 2) with no difference between groups. Although the decrease in stroke index was not significant in the control group, there was a statistically significant fall in stroke index in the abnormal group (fig. 2). However, this difference between groups was not statistically significant \((P < 0.5)\).


Hemodynamic Effects of Sustained Isometric Handgrip

<table>
<thead>
<tr>
<th>Patient</th>
<th>Diagnosis</th>
<th>Systolic pressure</th>
<th>Left ventricular end-diastolic pressure</th>
<th>Cardiac output</th>
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<td></td>
<td></td>
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<td>E</td>
<td>Δ</td>
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<tr>
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<td>158</td>
<td>+54</td>
</tr>
<tr>
<td>4</td>
<td>H.M.</td>
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<td>+52</td>
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<tr>
<td>5</td>
<td>E.S.</td>
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<td>147</td>
<td>+25</td>
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<td>0</td>
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<td>E.E.</td>
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<td>+40</td>
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<tr>
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<td>G.S.</td>
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<td>+25</td>
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<tr>
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<td>R.G.</td>
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</tr>
<tr>
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<td>G.F.</td>
<td>110</td>
<td>135</td>
<td>+25</td>
</tr>
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<td>140</td>
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<td>L.B.</td>
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</tr>
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<tr>
<td>28</td>
<td>A.W.</td>
<td>122</td>
<td>158</td>
<td>+36</td>
</tr>
</tbody>
</table>

Mean: 125.5, 132.3, 13.0, 20.0, 5.3, 6.0

Abbreviations: R = rest; E = exercise; Δ = change; WNL = within normal limits; CAD = coronary artery disease; CM = cardiomyopathy; ASD = atrial septal defect; MS-MR = mitral stenosis and mitral regurgitation; AS-AI = aortic stenosis and aortic incompetence; sem = ±1 standard error of the mean; P = P value.

When LV work-LVEDP and stroke-work index-LVEDP relations are examined in control and abnormal patients, clear differences are seen (figs. 4 and 5). In the work-LVEDP relation the control subjects uniformly exhibited curves that were steep, with relatively large increases in left ventricular work and small changes in LVEDP (fig. 4). On the other hand, abnormal patients demonstrated curves that were either less steep, flat, or had a negative slope (fig. 4). The stroke-work index-LVEDP relation similarly exhibited the relative steepness of the curves in the control group compared with the abnormal groups (fig. 5).

Two patients in the heart disease group were somewhat at variance with the abnormal group as a whole. Patient C.M. had predominant mitral stenosis (mitral valve gradient, 25 mm Hg) and minimal mitral regurgitation. With stress, left ventricular end-diastolic pressure and work changed little (fig. 3, no. 13), while the mitral valve gradient rose to 33. Patient R.G. (fig. 4, no. 16) had single-vessel coronary artery disease (90% left anterior descending stenosis) and a normal ventriculogram exhibiting no evidence of

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Table 2 depicts the ratio of change in work to change in LVEDP with handgrip. In the control group, with the exception of J. W. (a patient with isolated left bundle-branch block), the ratio of $\Delta$work/$\Delta$ LVEDP exceeded 1.0 in all cases (table 2). In contrast, the abnormal patients, with the exception of R. G. (the same patient discussed in the preceding paragraph), all exhibited ratios that were 0.77 or less (table 2).

Patients J. G. and E. E. in the abnormal group had ratios of 0.77 and 0.75, respectively. J. G., a 42-year-old Negro male, was admitted to the hospital with atypical chest pain and a history of heavy alcohol intake. He had been in congestive heart failure at another hospital prior to admission and had been taking a digitalis preparation. No evidence of congestive heart failure was apparent at the time of cardiac catheterization. Rest hemodynamics and coronary arteriograms were normal (table 1, patient 24), and left ventriculography exhibited mild diffuse hypokinesis consistent with a mild cardiomyopathy.

Patient E. E., a 50-year-old Negro male, entered the hospital with sudden precordial chest pain. No evidence of myocardial infarction was found and the electrocardiogram revealed stable nonspecific changes. Coronary arteriograms were normal. At rest LVEDP was abnormal, with normal cardiac index.
Hemodynamic values at rest and during sustained isometric handgrip (EXERCISE). Significant increases in systolic pressure, left ventricular end-diastolic pressure (LV EDP), cardiac output, heart rate, and cardiac index and significant decreases in stroke volume and stroke index occurred. I s.e.m. = ±1 standard error of the mean; BPM = beats per minute; B = beat.

Figure 1

Table 1, patient 14). Left ventriculography revealed left ventricular hypertrophy with a thickened wall and papillary muscle hypertrophy in the absence of hypertension (table 1). Ventricular gradients were not found at rest or with nitroglycerin. The diagnosis was mild cardiomyopathy of the hypertrophic type.

Figure 6 depicts the relationship between systolic pressure and LVEDP. If all patients are considered, a correlation exists (P < 0.01) between the increase in systolic pressure and LVEDP (fig. 6). However, when the control and abnormal groups are compared, differences become apparent. In the control group the mean increase in systolic pressure of 37.2 ± 5.2 was accompanied by a rise in LVEDP of only 2.1 ± 0.72 (fig. 2). In contrast, the mean systolic pressure rise of 22.9 ± 4.4 in the abnormal group was accompanied by an increase in LVEDP of 9.7 ± 1.7 (fig. 2). Although there was a correlation between the increase in systolic pressure and LVEDP in the abnormal group (P < 0.01), no such correlation between these parameters could be demonstrated in the control subjects (P < 0.2).

Figure 7 demonstrates the relationship between the change in systolic pressure and cardiac index before and during sustained isometric handgrip. No relationship could be demonstrated between these parameters.

Discussion

Sustained handgrip has been found to increase blood pressure markedly and rapidly as well as increase heart rate and cardiac index.
Changes in hemodynamics induced during sustained isometric handgrip in control (normal) and abnormal groups. P values refer to significance for each group. There was no significant difference between groups in systolic pressure, heart rate, or stroke index. Significant changes (Δ) between groups were seen in left ventricular end-diastolic pressure (LV EDP) and cardiac index. See Results. Abbreviations as in figure 1.

output in healthy young men. After sustained contraction ends, blood pressure and heart rate quickly return to control values. It has been postulated that the pressure response is reflex in origin, serving to increase perfusion pressure to the active muscles, in which blood flow is impeded by the sustained muscular contraction. Indirect evidence for this is the finding that forearm blood flow decreases during handgrip. The precise nature of this reflex remains to be defined.

Recent preliminary reports have indicated that the stress of handgrip is useful in quantitating left ventricular performance. These findings are substantiated by the present study. Ventricular performance in
subjects with and without catheterization evidence of heart disease was markedly different (figs. 4 and 5). In the control group the response to handgrip was characterized by an increase in work (fig. 4) and stroke-work index (fig. 5) with relatively little change in LVEDP. Conversely, abnormal patients exhibited a smaller rise or a fall in work or stroke-work index and a relatively marked increase in LVEDP. Thus the slopes were much steeper in the control group compared with those in the abnormal group (figs. 4 and 5).

When the relationship between change in work and LVEDP is expressed as a ratio, the control group, with one exception, had ratios greater than 1.0. The abnormal group, also with a single exception, had ratios less than 0.77 (table 2). These data indicate that this ratio may be useful as an index of left ventricular performance with sustained handgrip. An index greater than 1.0 is consistent with normality, whereas an index less than 0.75 would suggest an abnormal left ventricle. The range of 0.75 to 1.0 is intermediate, since one control and two mildly abnormal patients were in this group (see Results).

In assessing the value of sustained handgrip stress, several disadvantages and advantages should be considered. One disadvantage of
SUSTAINED ISOMETRIC HANDGRIp

Figure 4

Relationship between left ventricular (LV) work in kg-m/min and left ventricular end-diastolic pressure (LV EDP) in mm Hg at rest and during sustained isometric handgrip in control (normal) and abnormal subjects. See Results. Numbers are those assigned to patients for identification.

this test may be that occasional poor patient cooperation can result in a relatively submaximal contraction and therefore minimal stress to the circulation. This can be offset by taking the rise in systolic pressure as a measure of the adequacy of the stress (fig. 6) in much the same way as heart rate increase is commonly taken as a measure of isotonic exercise. It should be pointed out parenthetically that poor patient cooperation is also one of the limitations of isotonic muscular exercise testing.

Another possible disadvantage of sustained handgrip is the recently reported arrhythmogenic effect of this stress. Although in the present study isolated ventricular premature complexes occurred during handgrip, this has not been a significant problem in our experience. Ventricular premature complexes were never more than occasional, did not occur in runs, and never necessitated stopping the procedure.

A third theoretic disadvantage is the possibility that some patients perform a Valsalva maneuver during the study, which would affect the hemodynamics. This is easily avoided by simple patient instruction, the use of a sustained 3-min contraction, and careful observation by the physician during study.

Sustained handgrip has several distinct advantages. The test has the prime virtues of ease, simplicity, and safety. In contrast to isotonic exercise it can be performed within 4 min, and our experience confirms that systolic pressure and heart rate rapidly return to baseline on cessation of stress. Except for the occasional ventricular premature complexes mentioned, no adverse effects were
observed, in contrast with occasional hypotension reported following cessation of angiotensin infusion\textsuperscript{13} and angina with isotonic exercise in subjects with coronary disease.\textsuperscript{14} Pressure recordings are clear and undistorted (particularly end-diastolic pressure determinations), and the procedure is much less cumbersome than that required to quantitate isotonic exercise, particularly when femoral vessels are utilized for catheter insertion.

**Acknowledgment**

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*Figure 5*

Relationship between stroke work index and left ventricular end-diastolic pressure (LV EDP) before and during sustained isometric handgrip in control (normal) and abnormal subjects. See Results. Numbers are those assigned to patients for identification.
Table 2

Ratio of Change in Work and Left Ventricular End-Diastolic Pressure with Sustained Isometric Handgrip

<table>
<thead>
<tr>
<th>Patient</th>
<th>Control</th>
<th>ΔWork</th>
<th>ΔLVEDP</th>
<th>ΔWork/ΔLVEDP</th>
<th>Abnormal</th>
<th>ΔWork</th>
<th>ΔLVEDP</th>
<th>ΔWork/ΔLVEDP</th>
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<tr>
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<td>7.98</td>
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<td>7.98</td>
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<td></td>
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<tr>
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<tr>
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<td>6.20</td>
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<tr>
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</table>

Abbreviations: Δ = change; LVEDP = left ventricular end-diastolic pressure.

Figure 6

Relationship between systolic pressure and left ventricular end-diastolic pressure (LV EDP) before and during isometric handgrip in control (normal) and abnormal subjects. See Results. Numbers are those assigned to patients for identification.
Figure 7

Relationship between systolic pressure and cardiac index before and during isometric hand-grip in control (normal) and abnormal subjects. See Results. Numbers are those assigned to patients for identification.


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