Comparison of Cardiovascular Responses to Static-Dynamic Effort and Dynamic Effort Alone in Patients with Chronic Ischemic Heart Disease

ROBERT DEBUSK, M.D., WILLIAM PITTS, B.S., WILLIAM HASKELL, PH.D., AND NANCY HOUSTON, R.N.

SUMMARY Thirty men, mean age 55 years, known to have treadmill-induced ischemic ST-segment depression, performed static and dynamic effort, i.e., forearm lifting and treadmill exercise, separately and combined. Static effort was sustained at 20%, 25% or 30% of maximal forearm lifting capacity. Two symptom-limited treadmill tests, one with and one without added static effort, were performed on each of two visits. Compared with dynamic effort alone, combined static-dynamic effort decreased treadmill work load and increased heart rate, systolic blood pressure and rate-pressure product at the onset of ischemic ST-segment depression or angina pectoris: 7.1 ± 0.4 vs 8.0 ± 0.5 (SEM) multiples of resting oxygen consumption (mets), estimated; 141 ± 3 vs 134 ± 3 beats/min; 170 ± 4 vs. 162 ± 4 mm Hg and 239 ± 8 vs 218 ± 9 (p < 0.001). The prevalence of angina pectoris was significantly less with combined static-dynamic effort than with dynamic effort alone. Static effort causes a resetting of the threshold at which ischemic abnormalities appear during dynamic effort.

COMBINED STATIC-DYNAMIC EFFORT is often considered hazardous to patients with chronic ischemic heart disease.1,2 This belief is primarily based on a study of normal persons in whom systolic blood pressure and, by inference, myocardial oxygen consumption increased disproportionately during combined static-dynamic effort compared with sub-maximal dynamic effort alone.3 When these two modes of effort have been directly compared in patients with chronic ischemic heart disease, the actual prevalence of ischemic abnormalities has been found to be lower with combined static-dynamic effort than with dynamic effort alone.2,4 These studies have reported the heart rate-systolic blood pressure product, or "double product," only during peak static-dynamic effort5,4 or during a fixed submaximal work load,6 but not at the onset of ischemic ST-segment depression or angina pectoris. Consequently, we do not know whether the double product at which ischemic abnormalities appear with dynamic effort is the same as that at which ischemic abnormalities appear with combined static-dynamic effort. Moreover, a systematic difference between the double product at the onset of ischemic abnormalities during these two modes of effort may imply a significant difference in mechanisms underlying myocardial ischemia.

We had three aims in this study: 1) to devise a simple, standardized and clinically useful method for


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providing combined static-dynamic effort; 2) to determine the least intensity of static effort necessary to augment the cardiovascular response compared to that observed with dynamic effort alone; and 3) to compare the hemodynamic responses and the prevalence of angina pectoris and ischemic ST-segment depression induced by static, dynamic and combined static-dynamic effort.

We thought that significant cardiovascular abnormalities manifested during combined static-dynamic effort would be more readily apparent in patients with severe coronary heart disease than in patients with relatively less severe disease. Therefore, we included only patients with exercise-induced ischemic ST-segment depression. Our conclusions must be applied cautiously to all patients with chronic ischemic heart disease.

**Methods**

Men aged 70 years or less with a history of angina pectoris or myocardial infarction who were free of clinical congestive heart failure, unstable angina pectoris and other limiting medical conditions were studied. No subject had suffered myocardial infarction within the 3 months before this study. Patients taking digitalis and those whose daily dose of propranolol exceeded 80 mg were excluded. Propranolol dosages of 80 mg were considered to be relatively low, and therefore not likely to cause exertional hypotension or significant bradycardia compared with patients not taking this medication. Propranolol and quinidine were not administered on the day of testing. Three patients, one in each of the three groups described below, were taking propranolol in daily doses of 20 mg, 40 mg and 80 mg. Four patients, two in group A, one in group B and one in group C, were taking quinidine in total daily doses of 400 mg, 600 mg, 1200 mg and 1600 mg, respectively. Within the 6 months before this study, all patients had had a treadmill exercise test demonstrating ischemic ST-segment depression of 0.1 mV or more in leads V6, V4 or V5 during exercise and a functional capacity of at least 6 multiples of estimated resting oxygen consumption (mets).

We believed that too light a static load might not augment heart rate and systolic blood pressure significantly above the levels seen with dynamic effort alone. However, too heavy a static load would cause termination of the treadmill test due to local or generalized fatigue related to weight bearing. To assure that at least one level of static effort would accomplish the desired augmentation of the cardiovascular response to dynamic effort, we evaluated three levels of static effort: 20%, 25% and 30% of maximal forearm lifting capacity. Ten men each were assigned to perform one of these three levels of static effort, constituting groups A, B and C, respectively.

To examine the relationship between ischemic ST-segment depression and angina pectoris, group C was comprised entirely of individuals with a history of exercise-induced angina pectoris in addition to exercise-induced ischemic ST-segment depression. The choice of a 30% static load in this group was made arbitrarily. Five patients in group A and two patients in group B also had a history of treadmill-induced angina pectoris. The groups were clinically similar in age and in functional capacity. Other clinical characteristics are presented in table 1.

Two treadmill exercise tests were performed on each of two visits to the laboratory, with no more than 3 days separating visits. On each visit, patients performed one treadmill test while carrying a weight equivalent to 20%, 25% or 30% or maximum forearm lifting capacity and one treadmill test in which no weight was carried. The two tests were separated by a 15-minute recovery period. The initial test on visit 1 was randomly assigned to be static-dynamic or dynamic. The sequence of tests during the next visit was reversed. Forearm lifting capacity was determined and sustained static effort was performed on visit 1 only, before treadmill exercise.

Maximum handgrip strength was determined by averaging three successive contractions on a Jaymar hand dynamometer using the dominant hand. Maximum handgrip strength for groups A, B and C was 104 ± 6 (SEM), 101 ± 8 and 102 ± 7 lbs, respectively, without significant differences between groups. Maximum forearm lifting capacity of the dominant extremity was determined by asking patients to lift, with the hand supinated, a previously described set of wall-mounted weights. The weights were lifted through an angle of approximately 60° until the forearm was parallel to the floor. Care was taken to maintain the back straight and the elbow away from the trunk, to avoid vigorous gripping of the handle bearing the weight, and to avoid performing the Valsalva maneuver. The maximum voluntary contraction was judged to be the highest load in pounds which could be

<table>
<thead>
<tr>
<th>Table 1. Patient Characteristics</th>
<th>Group A 20% MVC</th>
<th>Group B 25% MVC</th>
<th>Group C 30% MVC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>53.6 ± 2</td>
<td>55.3 ± 2</td>
<td>54.5 ± 3</td>
</tr>
<tr>
<td>Prior treadmill capacity (mets)</td>
<td>9.1 ± 2</td>
<td>9.6 ± 1</td>
<td>9.0 ± 1</td>
</tr>
<tr>
<td>Prior myocardial infarction</td>
<td>9/10</td>
<td>10/10</td>
<td>4/10</td>
</tr>
<tr>
<td>Time since prior MI (mos)</td>
<td>35 ± 9</td>
<td>28 ± 9</td>
<td>11 ± 2</td>
</tr>
<tr>
<td>History of angina pectoris</td>
<td>5/10</td>
<td>2/10</td>
<td>8/10</td>
</tr>
</tbody>
</table>

Abbreviations: mets = multiples of resting energy expenditure (estimated); MI = myocardial infarction; NS = not significant; MVC = maximal voluntary contraction.
lifted through 60°. Loading began at 15 lbs and weights were added in 2.5- and 5.0-lb increments until the maximum load was attained. Rest intervals of 30 seconds separated increments, and peak loads were presented only once. An average of three increments was required to determine maximum forearm lifting capacity. Maximum capacity for groups A, B and C was 28 ± 3, 33 ± 3 and 31 ± 2 lbs, without significant differences between groups.

Patients performed sustained forearm effort while standing by supporting for 6 minutes a weighted metal basket equivalent to 20%, 25% or 30% of their maximum forearm lifting capacity. Electrocardiographic leads V4, V5 and V6 were continuously monitored for 1 minute at rest, for as long as 6 minutes during sustained forearm lifting and during a 2-minute recovery period. These leads were recorded during the last 10 seconds of each minute of effort. A 12-lead ECG was recorded at rest, at the end of sustained forearm lifting, and at the end of 1 and 2 minutes of recovery. Indirect arterial blood pressure was recorded by sphygmomanometry in the non-exercising arm at rest, at the end of each minute of sustained effort, and at the end of 1 and 2 minutes of recovery.

Dynamic effort was performed on the treadmill using a protocol with 3-minute work increments (table 2). The initial work load, based on the results of prior treadmill exercise testing, averaged 2.7 ± 0.2 mets and was identical for all four tests in any patient.

Static effort was added to dynamic effort by asking patients to carry the same metal basket which was used to provide a static load at rest (fig. 1). To avoid terminating the treadmill test because of arm fatigue, patients were given the weight at a stage of treadmill effort at least 6 minutes before their previously-determined maximum effort, but no sooner than the end of the third minute of exercise. Patients carried the weight in their dominant hand with the forearm supinated and held parallel to the floor, and care was taken to avoid gripping of the basket handle excessively or resting the forearm against the body. Precordial leads V4, V5 and V6 were monitored for 3 minutes before the first treadmill test of each visit. A 12-lead ECG was recorded at the end of each 3-minute stage of treadmill exercise, at maximum effort, and at 1, 2, 3, 5 and 7 minutes of recovery. Precordial leads V4, V5 and V6 were recorded at 1-minute intervals throughout each test and were displayed continuously on a three-channel oscilloscope. Systolic blood pressure was recorded by sphygmomanometry in the non-dominant arm at 1- to 3-minute intervals throughout treadmill exercise.

Static, dynamic and combined static-dynamic effort was terminated in the event of limiting symptoms, that is, angina which increased with further effort, limiting dyspnea or generalized fatigue; blood pressure abnormality, defined as a fall of greater than 10 mm Hg from the peak value attained during an earlier stage of effort; or ventricular tachycardia ≥ 3 consecutive premature ventricular contractions (PVCs). ST-segment depression per se was not an end point. Flat or downsloping ST segments which, at 0.08 seconds after the J point were displaced 0.1 mV or more below a line drawn tangent to the P-Q segment, were defined as ischemic. Measurements of ischemic ST-segment depression were made to the nearest 0.05 mV.

Three primary effects and their interactions were assessed by analysis of variance: static-dynamic effort vs dynamic effort alone, first vs second tests for a given visit, and first vs second visits. Statistical analysis was performed on an interactive computer system using standard statistical test packages (Statistical Programs for the Social Sciences). Tests of significance are two-tailed.

<table>
<thead>
<tr>
<th>Minute</th>
<th>Speed (mph)</th>
<th>Gradient (%)</th>
<th>Mets (estimated)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>2.5</td>
<td>2.7</td>
</tr>
<tr>
<td>2-3</td>
<td>3</td>
<td>2.5</td>
<td>4</td>
</tr>
<tr>
<td>4-6</td>
<td>3</td>
<td>7.5</td>
<td>6</td>
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<tr>
<td>7-9</td>
<td>3</td>
<td>12.5</td>
<td>8</td>
</tr>
<tr>
<td>10-12</td>
<td>3</td>
<td>17.5</td>
<td>10</td>
</tr>
<tr>
<td>13-15</td>
<td>3</td>
<td>22.5</td>
<td>12</td>
</tr>
<tr>
<td>16-18</td>
<td>3.5</td>
<td>22.5</td>
<td>14</td>
</tr>
</tbody>
</table>

Abbreviation: Met = multiples of resting energy expenditure.

FIGURE 1. Patient carrying a weight on the treadmill.
Results

Cardiovascular responses to sustained static effort are shown in figure 2 and in table 3. Heart rate, systolic blood pressure and double product were significantly lower ($p < 0.001$) with static effort than with dynamic or static-dynamic effort. Ischemic abnormalities were absent with static effort alone. No significant difference between patient groups was noted for any cardiovascular parameter during sustained static effort. Heart rate responses were significantly elevated ($p < 0.01$) above baseline after 1 minute of sustained effort. Systolic blood pressure and double product exceeded baseline values within 2 minutes in group A and within 1 minute in groups B and C ($p < 0.02$ and $p < 0.001$, respectively). All but two patients completed the 6 minutes of sustained forearm contraction; muscular fatigue caused cessation of effort in one at 4 minutes and in the other at 5 minutes of static effort.

Nearly two-thirds of static-dynamic and dynamic tests were terminated because of generalized fatigue and dyspnea. Arm fatigue stopped three static-dynamic tests in two patients of group B and six static-dynamic tests in six patients of group C. In six of these nine tests, the peak heart rate and systolic blood pressure responses to static-dynamic effort equalled or

![Figure 2](image1.png)

**Figure 2.** Cardiovascular responses to sustained static effort. MVC = maximum voluntary contraction; HR = heart rate; BPs = systolic blood pressure. Bars represent $\pm$ SEM.

![Figure 3](image2.png)

**Figure 3.** Cardiovascular responses to dynamic and static-dynamic effort. ST = ST-segment depression; HR = heart rate; BPs = systolic blood pressure. Dotted lines connect points at which measurements were available for all patients.
Table 3. Cardiovascular Responses to Dynamic and Static-Dynamic Testing

<table>
<thead>
<tr>
<th>At onset of ischemic ST-segment depression (100 tests)</th>
<th>At peak effort (120 tests)</th>
</tr>
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<tbody>
<tr>
<td>Work load (mets)</td>
<td>Heart rate (beats/min)</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Static tests (n = 60)</td>
<td>---</td>
</tr>
<tr>
<td>Dynamic tests (n = 60)</td>
<td>8.0 ± 0.5</td>
</tr>
<tr>
<td>Static-dynamic tests (n = 60)</td>
<td>7.1 ± 0.4§</td>
</tr>
<tr>
<td>Within visit comparison</td>
<td>Average 1st test response</td>
</tr>
<tr>
<td></td>
<td>Average 2nd test response</td>
</tr>
</tbody>
</table>

*§p < 0.001 vs baseline and vs dynamic. 
†p < 0.05 vs dynamic. 
‡p < 0.01 vs dynamic. 
§p < 0.001 vs dynamic. 
¶p < 0.001 vs first tests.

Abbreviations: mets = multiples of resting energy expenditure; HR = heart rate; BP = systolic blood pressure.

The responses to dynamic effort noted on the same visit.

Hypotensive responses stopped tests in three patients of group B and were nearly equally divided among static–dynamic and dynamic tests. No test was terminated because of ventricular tachycardia, although we noted self-limited ventricular tachycardia during recovery in one patient performing a static–dynamic test.

Cardiovascular parameters at the onset of treadmill-induced ischemic ST-segment depression and at peak effort are shown in table 3. Work loads were lower and heart rates and blood pressures were significantly higher for static–dynamic effort than for dynamic effort alone. Submaximal and maximal values for heart rate, systolic blood pressure and double product are plotted in figure 3. When the two exercise tests within a given visit were compared, the heart rate at the onset of ischemic ST-segment depression and at peak effort was higher on the second test than on the first (table 3). These changes from test 1 to test 2 were of similar magnitude whether static–dynamic or dynamic effort alone was performed first during each visit. Cardiovascular parameters measured during the first static–dynamic test were not significantly different from those measured during the second static–dynamic test. We also found this constancy of cardiovascular response from visit to visit for dynamic testing alone.

Of the 100 ischemic ST-segment responses to treadmill exercise, three occurred during recovery only, 54 occurred during exercise only, and 43 occurred during and after exercise. The pattern of these responses was similar for static–dynamic and for dynamic tests. The peak magnitude of ischemic ST-segment depression was 0.20 ± 0.01 mV for static–dynamic and for dynamic tests. Ischemic ST-segment depression persisted for 2.2 ± 0.2 minutes after static–dynamic and dynamic tests. The 20 tests not demonstrating ischemic ST-segment depression were divided equally between type of test (10 static–dynamic, 10 dynamic) and between patient groups (three in A, three in B, four in C). Angina pectoris occurred in all but six of these 20 tests (table 4). Five of these six tests were static–dynamic and one was dynamic.

Angina pectoris occurred in 47 of 120 tests and in 28 of 60 visits (table 5) and was significantly more frequent in dynamic than in static–dynamic tests — 27 vs 20 (p < 0.05 by McNemar’s test). Angina as an endpoint was also significantly more frequent in dynamic than in static–dynamic tests — 19 vs 10 tests (p < 0.05). Cardiovascular parameters at the onset of angina pectoris were analyzed only for group C patients, all of whom had angina on at least one test. Treadmill work loads were lower and heart rates, systolic blood pressures and double products were significantly higher at the onset of angina for static–dynamic compared with dynamic tests: 7.0 ± 0.5 vs 8.0 ± 0.9 mets (p < 0.01), 140 ± 4 vs 134 ± 5 beats/min (p < 0.01); 166 ± 7 vs 160 ± 7 mm Hg (p < 0.05) and 234 ± 14 vs 214 ± 17 (p < 0.05), respectively. Heart rate and double product at the onset of angina pectoris were significantly higher on

Table 4. Angina Pectoris and Ischemic ST-Segment Depression

<table>
<thead>
<tr>
<th>Angina</th>
<th>Present</th>
<th>Absent</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST§</td>
<td>33</td>
<td>67</td>
<td>100</td>
</tr>
<tr>
<td>Absent</td>
<td>14</td>
<td>6</td>
<td>20</td>
</tr>
</tbody>
</table>

| ST§    | 47      | 73     | 120 tests |
The second of two tests performed on each visit: 142 ± 4 vs 132 ± 5 beats/min (p < 0.01) and 235 ± 13 vs 215 + 16 beats/min (p < 0.02), respectively. No difference between visits was noted for work load, heart rate or systolic blood pressure at the onset of angina. Angina pectoris and ischemic ST-segment depression occurred almost simultaneously in static-dynamic and in dynamic tests.

PVCs were noted during sustained static effort in six patients, one in group A, three in group B and two in group C. PVCs occurred in 21 static-dynamic and in 16 dynamic tests. The two modes of effort did not differ with respect to the grade of PVCs: ventricular bigeminy or couplets were noted in nearly half of those with PVCs. The prevalence and grade of PVCs was similar for all three patient groups. Five of the six patients taking propranolol or quinidine had PVCs. The frequency and grade of these PVCs was not significantly different from that of patients with PVCs who were not taking antiarrhythmic medications.

Discussion

Forearm lifting as a method for providing a static stress is particularly well-suited to a study of combined static-dynamic effort: 1) The weight required to elicit a significant cardiovascular response is substantially less with forearm flexion than when a weight is carried with the forearm extended, as in carrying a suitcase.4 While sustained forearm lifting of as little as 6 lbs significantly augmented heart rate and blood pressure in our patients, a suitcase providing a proportional static stress would have weighed 20 lbs.6 We have found that weight carrying of this magnitude makes treadmill walking awkward and is more likely than forearm flexion to interfere with recording the systolic blood pressure and the ECG. 2) It is easier to maintain static effort within predetermined limits with forearm flexion than with hand dynamometry, another method used to provide a static stress.5,6 3) Finally, weight carrying with the forearm flexed better simulates many occupational work tasks than carrying with the forearm extended or gripping a dynamometer.

Static effort sustained to the point of muscular fatigue did not elicit ischemic ST-segment depression or angina pectoris in our patients, though all had previously demonstrated treadmill-induced ischemic ST-segment depression and some had experienced treadmill-induced angina pectoris. Since the heart rate and double product at the onset of treadmill-induced ischemic ST-segment depression or angina pectoris in our patients substantially exceeded the peak values noted with static effort, it is not surprising that dynamic effort elicited ischemic responses when static effort did not. We have previously reported a lack of ischemic responses to static effort in patients with ischemic responses to dynamic effort.5 Ferguson et al. noted a similar disparity between the two modes of effort.7 On the other hand, Haissly et al. noted ischemic ST-segment depression in nearly one-third of men (17 of 60) with chronic ischemic heart disease who performed sustained handgrip.8 The maximal heart rate responses to static effort in Haissly’s patients were higher and peak heart rate responses to maximal leg ergometry were lower than in our patients. Sustained static effort thus appears to elicit myocardial ischemia only in patients with a severely restricted coronary circulation.

The belief that patients with chronic ischemic heart disease should avoid static and combined static-dynamic effort is primarily theoretical. Because added static effort substantially augments the heart rate and especially systolic blood pressure response to low-level dynamic effort in normal persons,1,2 it has been postulated that patients with chronic ischemic heart disease might experience angina pectoris with combined static-dynamic effort but not with dynamic effort alone.1 When static-dynamic and dynamic effort have been directly compared in patients, we have not noted a worsening of the ischemic response. Kerber et al. noted fewer ischemic ST-segment responses when suitcase carrying was added to near-maximal treadmill walking than with treadmill walking alone.4 Similarly, Haissly noted a higher intra-arterial double product with sustained handgrip combined with submaximal bicycle ergometry than with maximal leg ergometry. Yet, despite their higher double products during combined static-dynamic effort, these patients had fewer anginal responses than during maximal dynamic effort alone.4 Like Kerber and Haissly, we found evidence that the myocardial oxygen supply-demand relationship during dynamic effort was altered by the addition of a static load: The heart rate and systolic blood pressure response at ischemia was uniformly and significantly increased.

The addition of a static effort caused the heart rate and systolic blood pressure at the onset of ischemic abnormalities to rise above the level noted during dynamic effort alone. This upward shift in the ischemic “threshold” tended to delay the onset of angina pectoris and to maintain treadmill capacity (fig. 3). Without this shift, angina pectoris would have occurred 1 minute earlier than it did. An upward shift in the ischemic threshold cannot be considered clinically beneficial if it is accompanied by a significant decrease in effort tolerance. In fact, the treadmill work load at the onset of angina pectoris was only slightly less with combined static-dynamic effort than with dynamic effort alone: 7.1 ± 0.4 vs 7.9 ± 0.5 mets, respectively. Although statistically significant, this difference has little clinical importance.
with a functional capacity of 7 mets at the onset of
angina pectoris probably will not suffer significant
restriction of physical activities. On the other hand,
coronary patients with a diminished functional reserve
often develop significant myocardial ischemia and left
ventricular dysfunction in response to static effort. While coronary patients with good myocardial reserve
show a nearly normal rise in stroke work and little or
no change in ventricular end-diastolic pressure with
static effort, those with a poor myocardial reserve
show a fall in stroke work and a substantial rise in left
ventricular end-diastolic pressure in response to static
effort. Similarly, patients in New York Heart Association (NYHA) class I or II show a normal
response to static effort measured by systolic time in-
tervals, while patients in NYHA classes III or IV
show abnormalities of ventricular function in response
to static effort. Hence, the effect of static effort com-
bined with dynamic effort may depend greatly on
the status of ventricular function and of functional
capacity in a subject. Our data indicate that in patients
with good functional capacity, angina pectoris is un-
likely to be precipitated by combined static-dynamic
effort, but not by maximal or near-maximal dynamic
effort alone. In fact, the prevalence of angina pectoris
in our patients was significantly less with static-
dynamic effort than with dynamic effort alone.

The lower prevalence of ischemic abnormalities
during static-dynamic effort than during dynamic
effort alone may reflect the higher diastolic blood
pressure noted with the former. Haissly et al. found
that angina was less frequent with combined static-
dynamic effort than with dynamic effort alone, despite
the higher peak double product associated with com-
bined effort. This may be explained by the higher
intra-arterial diastolic blood pressure noted with com-
bined effort compared with dynamic effort alone, i.e.,
115 ± 5 vs 87 ± 4 mm Hg. Our finding of a higher
double product at the onset of ischemic abnormalities
with combined static-dynamic effort than with
dynamic effort may reflect a resetting of the balance
between oxygen supply and demand which is due to
the increase in diastolic blood pressure which predict-
ably accompanies sustained static effort.

The double product measured during dynamic
effort is highly correlated with myocardial oxygen
consumption and with coronary blood flow in patients
with chronic ischemic heart disease and in normal indi-
viduals. Similarly, the double product during
static-dynamic effort is highly correlated with myocar-
dial oxygen consumption and coronary blood flow in
normal persons. This readily measured noninvasive
parameter thus reflects myocardial oxygen consump-
tion during vocational and avocational activities. The
double product at the onset of ischemic ST-segment
depression or angina pectoris induced by dynamic
effort is highly reproducible.

Our study extends these observations in demon-
strating the following: 1) ischemic ST-segment depres-
sion and angina pectoris occur at nearly the same dou-
ble product during symptom-limited static-dynamic
effort; 2) the double product at the onset of ischemic
ST-segment depression or angina pectoris is relatively
constant during combined static-dynamic effort; 3) the
double product at the onset of ischemic ST-segment
depression or angina pectoris is statistically higher
during static-dynamic effort than during dynamic
effort alone, but the range of these differences is small
and may be of little clinical importance.

Determination of the double product at which
ischemic abnormalities appear during dynamic testing
is a useful baseline against which to judge the car-
diovascular effects of other forms of physical effort.
For example, our patients demonstrated ischemic ST-
segment depression during combined static-dynamic
effort at a double product similar to that observed
with dynamic effort alone, while static effort, which
produced a substantially smaller cardiovascular
response, was not associated with ischemic abnor-
malities. Accordingly, there appears to be no advan-
tage to combined static-dynamic testing compared
with the simpler and more familiar method of
symptom-limited dynamic testing for detecting
myocardial ischemia. This is probably true even for
patients whose jobs involve combined static-dynamic
effort. Our findings provide good assurance that if
symptom-limited dynamic effort does not elicit
ischemic ST-segment depression or angina pectoris,
combined static-dynamic effort is unlikely to do so.
If symptom-limited treadmill exercise does elicit
ischemic abnormalities, they are likely to appear at
an even higher heart rate during combined static-
dynamic effort.

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Impaired Maximal Rate of Left Ventricular Relaxation in Patients with Coronary Artery Disease and Left Ventricular Dysfunction

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SUMMARY

It has been suggested that the rate of left ventricular (LV) relaxation is related to the inotropic state, end-systolic fiber length and peak LV pressure, but little information is available regarding the rate of LV relaxation in patients with coronary artery disease (CAD) and LV dysfunction. To assess the rate of LV relaxation, we obtained high-fidelity LV pressure measurements with manometer-tip catheters in 39 patients. The signal was analyzed by a digital computer to yield the maximal rate of pressure rise (pos dP/dt) and the maximal rate of pressure fall (neg dP/dt). Selective coronary arteriography and biplane LV angiography with determination of LV volumes, ejection fraction (EF) and percent abnormally contracting segments (ACS), when present, were performed in all patients. In 10 patients with normal LV function (EF > 0.50, no asynergy) mean neg dP/dt (2074 ± 121 mm Hg/sec) was significantly (p < 0.01) greater than in 29 patients with CAD and LV dysfunction (1695 ± 66 mm Hg/sec). In nine patients with LV dysfunction and EF < 0.35, mean neg dP/dt was reduced to 1405 ± 107 mm Hg/sec, significantly (p < 0.01) lower than in patients with normal LV function. Neg dP/dt correlated well with pos dP/dt (r = 0.75), with EF (r = 0.74), and with ACS (r = -0.74), and less well with LV end-systolic volume (r = -0.67). There was very poor correlation between neg dP/dt and peak LV pressure (r = 0.30).

These data suggest that the rate of LV relaxation, as assessed by neg dP/dt, is impaired in patients with CAD and LV dysfunction, and the extent of impairment is related to the severity of the dysfunction as determined hemodynamically by pos dP/dt, and angiographically by EF and ACS. In these patients the maximal rate of LV relaxation is inversely related to LV end-systolic volume, and is not related to peak LV pressure.

RELAXATION OF CARDIAC MUSCLE is an energy-dependent process that can be altered independently of contraction and can be modified by pharmacologic agents, and disease states, and changes in the inotropic state of the myocardium. and animal studies have shown that changes during isovolumic left ventricular (LV) relaxation can be a sensitive and early indicator of myocardial dysfunction, and the study of the relaxation phase may be important in the hemodynamic evaluation of LV performance. In patients with coronary artery disease (CAD), LV function can be significantly impaired with marked alterations in the isovolumic and ejection phases of contraction, but the changes in isovolumic relaxation in these patients have not been well defined.

In this study we assess LV relaxation in patients with LV dysfunction secondary to CAD, and determine relationships between the maximal rate of isovolumic relaxation and hemodynamic and angiographic parameters of LV performance.
Comparison of cardiovascular responses to static-dynamic effort and dynamic effort alone in patients with chronic ischemic heart disease.
R DeBusk, W Pitts, W Haskell and N Houston

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