Cardiovascular Responses to Dynamic and Static Effort Soon after Myocardial Infarction

Application to Occupational Work Assessment

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SUMMARY Static and dynamic work involving the arms and the legs was performed by 40 men seven weeks after myocardial infarction. Leg ergometry produced a significantly higher peak work load, systolic blood pressure (BP), heart rate (HR), and HR x BPs x 10^{-3} product (DP) than did arm ergometry: 842 ± 178 vs 546 ± 135 kg-m/min, 176 ± 24 vs 154 ± 19 mm Hg and 256 ± 54 vs 219 ± 48 (SD). Peak heart rates were 145 and 142. Endpoints were primarily muscular and generalized fatigue and dyspnea. Ischemic abnormalities and ventricular ectopy were more frequent with leg ergometry. Sustained forearm lifting elicited higher HR, BP, and DP responses than sustained handgrip contraction; 95 ± 16 vs 91 ± 16 beats/min, 162 ± 18 vs 152 ± 17 mm Hg and 154 ± 33 vs 139 ± 33 (SD). Ischemic ST segment depression and significant ventricular arrhythmias were infrequent with static effort. Dynamic leg testing is superior to dynamic or static arm testing in assessing the capacity of patients to perform physical work tasks after myocardial infarction.

FUNCTIONAL CAPACITY has been safely evaluated as early as three weeks after myocardial infarction.1 In most of these studies, however, functional capacity has been determined only during dynamic leg effort. Since upper extremity effort, required in many job tasks, is reported to produce angina and other cardiac symptoms,4,5 an occupational work assessment based solely on lower extremity dynamic effort seems inadequate.

We designed a series of dynamic and static work tasks similar to those encountered in occupational work. Our objective was to evaluate the cardiovascular responses to dynamic effort involving both the arms and legs, and to compare these responses with those noted during two forms of static effort. This evaluation was performed at a relatively early phase of recovery from myocardial infarction, when many patients are being considered for return to occupational work and before the time when functional assessment of occupational working capacity has been advocated.

Methods

Patients

The population consisted of 40 men, mean age 51 ± 7 (range 34–63 years) with myocardial infarction documented by history, cardiac enzyme elevations and characteristic evolutionary electrocardiographic changes. Infarctions were inferior in 29 patients, anterior in nine and nontransmural in two patients. Patients with a third sound gallop, clinical congestive heart failure or unstable angina pectoris at six to seven weeks after infarction were excluded from this study. As a group, our patients had less severe cardiac damage than was noted by Norris in a general population of postinfarction patients.9 The “long-term” Coronary Prognostic Index (CPI) of Norris is a method for assessing post-hospital prognosis, utilizing clinical information obtained at the time of hospital admission. Two-thirds of our patients had a CPI score of less than 3, equated with a 12% three-year mortality, compared with one-third of Norris’ unselected patients.

Baseline ST segment depression of 0.5–0.9 mm was found in only two patients, and no patient had as much as 1 mm of ischemic ST segment depression at rest. Three patients had more than 0.5 mm of resting ST segment elevation.

Five patients were taking propranolol and two were taking quinidine. No patient was taking digitalis. All medications were discontinued on the day of the test. All patients had been employed before myocardial infarction, mostly in jobs not requiring heavy physical effort. Most had been moderately active before myocardial infarction.

Test Procedure (fig. 1)

Dynamic and static exercise was performed on two occasions, usually two days (range three to eight) apart, at an average of seven weeks (range six to eight weeks) after infarction. Handgrip contraction and forearm flexion were presented in random order on each visit. On the second visit, ergometry was performed using the limbs not tested on the first visit.

1. Static Exercise

A. Handgrip contraction: Maximum handgrip strength of the dominant hand was determined by averaging three successive contractions on a Jamar dynamometer. Each trial lasted less than
3 seconds, and trials were separated by 20 seconds. While standing, patients then sustained for 1 minute a handgrip contraction equivalent to 25% of the mean maximum voluntary contraction (MVC). After heart rate and blood pressure had returned to baseline, the procedure was then performed at a level of effort equivalent to 50% of the MVC.

Three electrocardiographic leads (V4–6) were continuously monitored for 1 minute before sustained handgrip, during the contraction and for a 2-minute recovery period, at which time heart rate and blood pressure had usually returned to near baseline values. A 12-lead electrocardiogram and indirect blood pressure were recorded at baseline, during the last 10 seconds of contraction and at the end of 1 and 2 minutes of recovery. Blood pressure was recorded in the non-exercising arm.

B. Forearm flexion: MVC involving flexion of the dominant forearm was determined by a set of calibrated weights and pulleys (fig. 2). 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, and to avoid vigorous gripping of the handle bearing the weights and performance of the Valsalva maneuver. The MVC was judged to be the highest load in pounds which could be lifted through an angle of 60°. Loading began at 15 pounds and weights were added in five-pound increments until the maximum load was attained. Rest intervals of 30 seconds separated increments, and the peak load was presented only once. An average of three trials was required for determination of MVC.

While standing, patients then supported for one minute a weight equivalent to 25% of their demonstrated forearm lifting capacity (MVC). After heart rate and blood pressure had returned to baseline, sustained forearm lifting was repeated at 50% of MVC. Monitoring and recording of the electrocardiogram were performed as for handgrip.

2. Dynamic exercise

Arm and leg ergometry were performed on separate days, after heart rate and blood pressure had returned to baseline following the static testing described above.

A. Leg ergometry: Leg work on a Collins bicycle ergometer began at a load of 450 kg-m/min. The work load was increased by 150 kg-m/min every 3 minutes, with intervening 1-minute rest periods. The highest work load lasting at least 2 minutes was designated as the peak work load.

Three electrocardiographic leads (V4–6) were continuously monitored for 1 minute before exercise, during exercise and during 7 minutes of recovery. A 12-lead electrocardiogram was recorded at the end of each 3-minute work load and at peak work load, at the end of each 1-minute rest period and 1, 2, 3, 5 and 7 minutes after peak work load.

Blood pressure was recorded by a sphygmomanometer immediately following

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**Table 1**

<table>
<thead>
<tr>
<th>VISIT 1</th>
<th>VISIT 2</th>
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<tr>
<td><strong>STATIC WORK</strong></td>
<td><strong>DYNAMIC WORK</strong></td>
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<tr>
<td>FA25,50</td>
<td>ARM CRANKING</td>
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<tr>
<td>HG25,50</td>
<td>OR</td>
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<tr>
<td>HG25,50</td>
<td>LEG CRANKING</td>
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**FIGURE 1.** Static-dynamic work sequence. Abbreviations: FA = forearm; HG = handgrip.

**FIGURE 2.** Device for determining maximum forearm lifting capacity.
tachycardia (three or more consecutive ventricular premature contractions). ST segment depression per se was not an endpoint.

D. Tests performed: All 40 patients performed arm and leg ergometry; 32 had duplicate isometric evaluation.

E. Data analysis: Data analysis was performed on an IBM 360/168 computer, using the Statistical Package for the Social Sciences Program.

Results

Handgrip Contraction

The mean MVC for handgrip was 105 ± 25 pounds (SD), with a range from 78–180 pounds. Maximum handgrip capacity on the two visits was highly correlated (r = 0.95). Cardiovascular responses to 25% and 50% MVC are noted in table 1. Neither level of handgrip contraction produced ST segment depression or elevation or angina pectoris. Eight patients had isolated premature ventricular contractions (PVCs) during or after sustained handgrip (fig. 4). Ventricular ectopy was equally common at each level of effort and on each visit.

Forearm Flexion

The mean MVC for forearm lifting was 36 ± 7 pounds (SD) (range 26–55). Maximum forearm lifting capacity on the two visits was highly correlated (r = 0.98). Cardiovascular responses to 25% and 50% of MVC are noted in table 1. Forearm flexion failed to elicit ST segment depression, angina pectoris or elevation. Ten patients demonstrated PVCs during sustained forearm flexion, including one patient each with couplets and bigeminy (fig. 4).

Comparison of Handgrip and Forearm Flexion

Maximum capacity for handgrip contraction and forearm flexion were significantly correlated: r = 0.72, y = 9.88 + 0.25x, SEE = 5.28. Despite the

<table>
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<th>FOREARM LIFT</th>
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<td>PRESENT</td>
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<td>7</td>
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greater cardiovascular responses to forearm contraction compared with handgrip contraction noted in table 1, the responses to 50% MVC for the two types of static effort were highly correlated for maximum heart rate \( r = 0.85, y = 11 + 0.91x \) and maximum double product \( r = 0.85, y = 66.4 + 0.63x \) and were somewhat less well correlated for maximum systolic blood pressure \( r = 0.56, y = 68.1 + 0.60x, \text{ SEE } = 13.1 \). Similar correlations were noted for 25% MVC.

The incidence of PVCs was similar for the two modes of static effort, and 28 of 32 patients (88%) demonstrated absence or presence of PVCs on both tests.

**Leg Ergometry**

The mean maximum work load was 842 ± 177 kg-m/min (range 450–1200). Ischemic ST segment depression, i.e., further depression ≥ 1 mm, compared to baseline was noted in 10 patients, only three of whom had angina pectoris during or after exercise testing. Further ST segment elevation was noted in one patient with resting ST segment elevation. Ventricular ectopy appeared during or after exercise in 15 patients, including bigeminy in three, pairs in one and ventricular tachycardia during exercise in one.

**Arm Ergometry**

The mean maximum work load was 546 ± 146 kg-m/min (SD) (range 150–900). Ischemic ST segment depression was noted in seven patients, all of whom also had ST segment depression with leg testing. No patient had further ST segment elevation with arm testing. Angina pectoris with arm testing was present in two patients, both of whom had angina with leg testing. Ventricular ectopy appeared in 20 patients during or after exercise, including bigeminy in three, couples in one and ventricular tachycardia during recovery in one.

**Comparison of Leg and Arm Ergometry (fig. 5)**

Mean peak work load with arm ergometry was 65% that of leg ergometry, with a moderately high correlation between the two modes of effort: \( r = 0.70, y = 93.4 + 0.54x, \text{ SEE } = 97 \). Cardiovascular responses are depicted in figure 5. Heart rates and double products at three work loads (450, 600 and 750 kg-m/min) were significantly higher for arm cranking than for leg cranking, but systolic blood pressure was not significantly different for two of these three work loads. Peak heart rates were not significantly different, whereas peak systolic blood pressure and double product were significantly higher for leg ergometry than for arm ergometry \( P < 0.001 \).

Ischemic ST segment responses to leg ergometry were noted in 10 patients, seven of whom also demonstrated ischemic responses to arm ergometry. No patient had ischemia with arm ergometry which was not also present with leg ergometry. Similarly, two patients had angina pectoris with leg and with arm ergometry, and one had angina with leg ergometry only. Cardiovascular parameters are compared for arm and leg ergometry in table 2. Peak work load and systolic blood pressure, but not peak heart rate or double product, were significantly higher with leg than with arm ergometry. None of these responses differed significantly from the larger group of 30 patients who did not demonstrate ischemia. At the onset of ischemic ST segment depression, only work load was significantly higher for leg than for arm exercise in the seven patients with ischemic responses to both modes of exercise.

All forms of ventricular ectopy were more frequent
with arm ergometry (20 of 40 patients) than with leg ergometry (15 of 40 patients), but this difference was not statistically significant. Ventricular ectopy was defined as complex if the peak PVC frequency was greater than three per minute or if PVCs occurred in bigeminy, couplets or triplets (ventricular tachycardia) (fig. 6). Of the 32 patients who performed all trials of static and dynamic effort, seven demonstrated isolated PVCs during at least 1 of the 10 minutes of baseline recording preceding physical effort. In all seven patients, PVC frequency and grade increased during or after each trial of physical effort.

Comparison of Static and Dynamic Exercise

Ischemic ST segment depression was absent during static effort, whereas one-fourth of the patients demonstrated this abnormality with dynamic effort. Ventricular ectopy was significantly more common with dynamic than with static effort (fig. 7). In the three patients demonstrating ectopy with static effort only, PVCs were isolated and infrequent.

Endpoints (table 3)

General fatigue and muscle discomfort were the most common reasons for terminating both forms of ergometry. Identical endpoints were noted in 25 of 40 (65%) of patients. Strictly "cardiac" endpoints, i.e., angina pectoris and ventricular ectopy, were rare compared to muscle fatigue and cramping, or to generalized fatigue or dyspnea.

![Table 2. Cardiovascular Response to Ergometry in Patients with Ischemia](image)

<table>
<thead>
<tr>
<th></th>
<th>Peak Values (n = 10)</th>
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<th>At Ischemia (n = 7)</th>
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<tr>
<td></td>
<td>Arm</td>
<td>Leg</td>
<td>F</td>
<td>Arm</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>150 ± 11</td>
<td>146 ± 21</td>
<td>NS</td>
<td>128 ± 26</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>158 ± 12</td>
<td>175 ± 19</td>
<td>&lt;0.02</td>
<td>149 ± 18</td>
</tr>
<tr>
<td>Double product (HR × BPs/100)</td>
<td>236 ± 21</td>
<td>258 ± 57</td>
<td>NS</td>
<td>190 ± 45</td>
</tr>
<tr>
<td>Work load (kg-m/min)</td>
<td>525 ± 79</td>
<td>780 ± 155</td>
<td>&lt;0.01</td>
<td>364 ± 170</td>
</tr>
</tbody>
</table>

Abbreviations: HR = heart rate; BPs = systolic blood pressure.

Discussion

The preponderance of patients with inferior myocardial infarction (29 of 40) in this series reflects the exclusion of patients with ventricular diastolic gallops or clinical heart failure at seven weeks after infarction. Since the extent of ventricular damage is less with inferior than with anterior infarction,10 this distribution is not unexpected and has been noted in our entire group of 185 postinfarction patients. Peak enzyme elevations during hospitalization were not significantly lower, nor were peak work loads significantly higher, in the patients with inferior infarctions compared to those with anterior infarctions, suggesting that both groups of patients had an equivalent extent of ventricular damage. The higher work loads, heart rates and blood pressures with arm and with leg effort noted in our patients, compared to the less highly-selected patients reported by Schwade,11 probably reflects less ventricular dysfunction in our patients. Thus, it is the extent of myocardial damage rather than the site of infarction per se which determines the applicability of our results to other patient populations.

Exercise testing has been useful in assessing the ability of patients with coronary disease to perform occupational work.8,12 Such testing has had two shortcomings: 1) It has utilized large muscle dynamic effort to the virtual exclusion of isometric stress, a mode of effort common to many occupational tasks; and 2) it has not been applied soon after myocardial infarction, i.e., within the first 12 weeks.8 Our patients

![Figure 6. Ventricular ectopy with ergometry. Abbreviations: EX = exercise; VEA = ventricular ectopic activity; V.T. = ventricular tachycardia; N.S. = not significant.](image)
successfully completed an occupational work assessment seven weeks after infarction, at a time when many patients have not yet returned to work and while most are specifically cautioned against isometric effort.

Our patients demonstrated a close correlation between cardiovascular responses to handgrip and to forearm lifting. Although the absolute values of heart rate and blood pressure were higher for forearm contraction, the absolute differences were small and unlikely to be of clinical significance. Moreover, the cardiovascular response was highly reproducible from visit to visit for both methods at both levels of effort.

Most studies of isometric effort have utilized handgrip, a convenient method for providing a standardized quantitative isometric stress. Since most tasks involving isometric effort are accomplished by lifting rather than by gripping, the handgrip method fails to answer a clinically important question: How much weight can the patient safely lift and carry? By first measuring the capacity for forearm flexion, the percentage of maximum forearm contraction represented by a specific weight can be readily determined. The cardiovascular response to isometric effort is a function of the relative intensity of contraction, and is independent of the mass of contracting muscle. Therefore, contraction of one forearm at 50% of its maximum capacity would be expected to elicit a response similar to that of simultaneous contraction of both forearms at 50% of their MVC. If no significant cardiac abnormalities are noted during handgrip or forearm contraction at 50% of MVC, sustained to the point of muscular fatigue, our data provide reasonable assurance that the limiting factor in isometric effort is muscular rather than cardiac.

A further practical advantage of forearm lifting is that it facilitates assessment of the cardiovascular response to combined static-dynamic effort, a circumstance commonly encountered in occupational work.

Elicitation of maximum cardiovascular responses in the laboratory provides evidence that a greater response is unlikely to be encountered during similar tasks performed on the job. Regarding the duration of effort, it is known that above 15% of maximum voluntary contraction, the cardiovascular response to isometric effort increases almost linearly with time until the onset of muscular fatigue. Our patients required considerable urging to sustain a handgrip or forearm contraction of 50% MVC for a full minute. It is unlikely that a more prolonged stress would have been possible, owing to muscle fatigue. Lind et al. also noted a maximum duration of 1 minute in normal volunteers performing handgrip at 50% of MVC. Kerber et al. also noted difficulty in their patients sustaining 50% handgrip for 2 minutes. Thus, a 1-minute stress at 50% of MVC represented a near maximum physical stress for our patients, and probably elicited a near maximum cardiovascular response. Since the duration of each trial of maximum handgrip or forearm lifting used in establishing MVC was less than 3 seconds, and since each trial was followed by 20 seconds of rest, it seems unlikely that muscular fatigue significantly altered the response to sustained static effort which followed.

Regarding the intensity of isometric effort, we noted a significantly higher cardiovascular response to 50% than to 25% effort, a finding similar to that of Lind in normal individuals, but differing from the findings of Haissley et al., who noted no significant difference in the heart rate response to 30%, 50% and 70% of maximum handgrip contraction.

It is not surprising that static effort did not produce ST segment abnormalities in our patients, since their maximum double products with isometric effort were far below those at which ischemic ST segment depression appeared during dynamic effort. In normal individuals performing dynamic, static and combined dynamic-static effort, the heart rate-blood pressure product correlates well with myocardial oxygen consumption and with coronary blood flow, the heart rate alone showing nearly as close a relationship as that of the heart rate-blood pressure product. Patients with angina pectoris who perform dynamic exercise also demonstrate the same close relationship between myocardial oxygen consumption and the heart rate-blood pressure product. It seems likely, therefore, that an ischemic "threshold" of heart rate and blood pressure exists in coronary patients, and that the relative incidence of ischemic ST segment depression and angina pectoris with isometric or with dynamic

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**TABLE 3. Endpoints in Arm and Leg Ergometry**

<table>
<thead>
<tr>
<th>Endpoint</th>
<th>Leg Ergometry</th>
<th>Arm Ergometry</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>No. of Patients</td>
<td>Percent</td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>3</td>
<td>7.5</td>
</tr>
<tr>
<td>Fatigue, dyspnea</td>
<td>15</td>
<td>37.5</td>
</tr>
<tr>
<td>Muscle fatigue, cramps</td>
<td>21</td>
<td>52.5</td>
</tr>
<tr>
<td>Ventricular ectopic activity</td>
<td>1</td>
<td>2.5</td>
</tr>
</tbody>
</table>

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*p < 0.05

**Figure 7. Ventricular ectopy with exercise. Static vs dynamic effort.**
effort relates more to the likelihood of exceeding this double product than to the nature of the work load per se. Littler et al. noted that the threshold of heart rate and blood pressure at which angina pectoris occurred was similar for a variety of conditions, including physical effort, smoking and eating.

Ischemic ST segment depression was absent with static effort in all of our patients who demonstrated ischemia with dynamic effort, consistent with the results of Haissly and Helfant, who noted a much higher incidence of ischemia with bicycle ergometry than with handgrip contraction.

Ventricular ectopy was much less frequent with static than with dynamic effort in our patients, contrary to the findings of Atkins et al. Both forms of physical effort substantially increased the frequency and grade of PVCs noted at rest.

Some patients give a history of angina pectoris with arm work but not with leg work. Therefore, it was interesting to compare the hemodynamic response and the relative incidence of myocardial ischemia for dynamic arm and leg effort. Ischemic responses were more frequent with leg than with arm ergometry in our patients, though this difference was not statistically significant. No patient demonstrated ischemic ST segment abnormalities or angina pectoris with arm effort but without leg effort. The double product at the onset of ischemia was not significantly different for the two modes of effort in our patients, similar to the results of Schwade regarding ischemic ST segment depression and to those of Wahren regarding angina pectoris. Clausen et al. noted a higher systolic blood pressure and heart rate at the onset of angina in response to arm exercise compared to leg exercise.

In our patients, arm ergometry provided no independent yield of ischemic ST segment abnormalities or angina pectoris compared to leg ergometry. Even among Schwade's seven patients with a history of angina pectoris precipitated by arm work but not by walking, five reported angina pectoris with leg ergometry, including one who failed to experience angina pectoris with arm ergometry. Our results support Schwade's suggestion that the sensitivity to angina pectoris with arm effort simply reflects the tendency of some patients to use their arms more than their legs in daily activities, rather than a basic pathophysiological difference between arm and leg effort.

If the elicitation of myocardial ischemia with ergometry depends on the ability to reach the ischemic threshold of heart rate and systolic blood pressure in a given patient, leg ergometry appears to be the better test, for in our patients maximum double product was higher with leg ergometry than with arm ergometry. Leg ergometry has the advantage of greater familiarity and the use of standardized equipment. Moreover, the capacity of leg ergometry to elicit a maximum cardiovascular response is comparable to that of treadmill exercise. Where leg ergometry or treadmill exercise is impossible due to effort claudication of the legs or to orthopedic abnormalities, arm ergometry has demonstrated hemodynamic responses similar to those elicited by large muscle dynamic effort.

Symptom-limited dynamic exercise with the legs or with the arms obviates isometric testing in most circumstances, since the magnitude of the cardiovascular response and the incidence of cardiac abnormalities is so much greater with dynamic than with static effort.

While none of these test methods simulates the non-exertional factors which may influence the cardiovascular response to occupational work such as extremes of temperature, air pollutants or psychological stress, they provide an index of maximum cardiovascular response to the major types of physical effort likely to be encountered under a wide variety of circumstances. In assessing the capacity to perform physical work tasks, the maximum cardiovascular response appears to be a more useful focus than the nature of the physical tasks. Dynamic leg exercise elicits a greater cardiovascular response than a variety of tasks involving arm work which may simulate vocational and avocational conditions, but which provide a less significant cardiovascular challenge. Measurements of cardiovascular parameters during vocational and avocational activities are needed to validate the laboratory assessment.

Conclusions

1) A combination of isometric and dynamic tests of function, performed as soon as seven weeks following infarction, is safe and may provide useful information regarding the ability of patients to return to work.

2) The cardiovascular tolerance for tasks involving lifting can be inferred from the cardiovascular response to a load representing 50% of the maximum forearm lifting capacity.

3) Leg ergometry is more effective than arm ergometry in eliciting latent cardiac abnormalities, and both forms of dynamic exercise are more effective in eliciting cardiac abnormalities than is static effort.

References

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