P ERIPHERAL arterial occlusive disease (PAOD) is a leading cause of morbidity in elderly persons because of ambulatory limitations associated with intermittent claudication (1). It is estimated that 1 million older Americans become symptomatic each year (2), and that PAOD patients with severe intermittent claudication are physically impaired by up to 75% (3). With the population over 65 years of age projected to reach 67 million by the year 2040 (1), intermittent claudication will be a growing cause of functional disability and chronic morbidity in the United States. Consequently, interventions should focus on improving ambulatory function to maintain functional independence in PAOD patients with intermittent claudication.

In a meta-analysis, we reported that exercise rehabilitation increased distance to onset of claudication pain by 115% (178 ± 22 m to 383 ± 34 m; p < .001) and distance to maximal claudication pain by 65% (389 ± 29 m to 641 ± 34 m; p < .001). The increased distance to onset of pain was independently related to a 27% increase in calf blood flow (r = .42, p < .001) and to baseline age (r = −.26, p < .05), and the increased distance to maximal pain was predicted by a 10% increase in peak oxygen uptake (r = .41, p < .001) and a 10% improvement in walking economy (r = −.34, p < .05). Free-living daily physical activity increased 31% (337 ± 29 kcal/day to 443 ± 37 kcal/day; p < .001) and was related to the increases in treadmill distances to onset (r = .24, p < .05) and to maximal pain (r = .45, p < .001).

Conclusions. Increased claudication distances following exercise rehabilitation are mediated through improvements in peripheral circulation, walking economy, and cardiopulmonary function, with younger patients having the greatest absolute ambulatory gains. Furthermore, improved symptomatology translated into enhanced community-based ambulation.

The impact of improved claudication symptoms following exercise rehabilitation on quantitative measures of ambulatory function in the community setting is not well known. Because a low level of daily physical activity is an independent risk factor for coronary heart disease incidence and mortality (16–18), increased free-living daily physical activity in PAOD patients following exercise rehabilitation may improve their poor prognosis for long-term survival (19–21).

The purposes of this study are to identify the clinical and physiological predictors of increased treadmill claudication distances following exercise rehabilitation in PAOD pa-
tients and to determine whether improved claudication distances translate into increased free-living daily physical activity in the community setting.

Methods

Patients

Screening.—A total of 86 PAOD patients were enrolled into exercise rehabilitative research studies conducted as part of the Claude D. Pepper Older Americans Independence Center at the University of Maryland, Baltimore. The patients were recruited from the Vascular Clinic at the Baltimore location of the Maryland Veterans Affairs Health Care System and from newspaper and radio advertisements in Baltimore. All patients were classified as having Fontaine stage II PAOD (3) defined by the following inclusion criteria: (a) a positive Rose questionnaire for intermittent claudication (22), (b) intermittent claudication elicited during a screening treadmill test, and (c) an ankle/brachial index (ABI) at rest <0.97 (23). Patients were excluded from this study for the following conditions: (a) absence of PAOD; (b) asymptomatic PAOD (Fontaine stage I); (c) rest pain PAOD (Fontaine stage III); (d) exercise tolerance limited by factors other than claudication (e.g., severe coronary artery disease, dyspnea, poorly controlled blood pressure); and (e) active cancer, renal disease, or liver disease. All patients lived independently at home. The procedures used in this study were approved by the institutional review board at the University of Maryland. Written informed consent was obtained from each patient prior to investigation.

Baseline characteristics.—Of the initial 86 PAOD patients enrolled into the exercise rehabilitation program, 18 patients were noncompliant with the exercise program, and 5 patients did not finish because they developed new acute medical problems. The remaining 63 patients successfully completed the 6-month study and therefore comprise the sample studied in this investigation.

Patients had a mean age of 68 ± 1 years (mean ± standard error [SE], range = 51 to 84 years) and reported having intermittent claudication for 2.2 ± 0.2 years with a self-reported pain-free walking distance of 3.5 ± 0.5 blocks. The sample consisted of 57 (90%) men and 6 (10%) women, of whom 29 (46%) were Caucasian and 34 (54%), African American. Nineteen (30%) of the participants reported unilateral claudication, 44 (70%) reported bilateral claudication, and 8 (13%) participants had a history of lower extremity revascularization. As expected, cardiovascular risk factors were highly prevalent in this sample as 57 (90%) had a history of cigarette smoking, 49 (78%) had hypertension, 40 (64%) had hyperlipidemia, 26 (41%) had diabetes, and 20 (32%) had diagnosed coronary artery disease by medical history, hospital records, or electrocardiogram findings. Antihypertensive, antianginal, and diabetes medications were adjusted as needed to meet the safety criteria established by the American College of Sports Medicine for exercise testing and training (24).

Exercise Rehabilitation

The exercise rehabilitation program was designed to elicit increases in claudication distances according to our published recommendations from a meta-analysis (4). This program consisted of 6 months of supervised, intermittent treadmill walking to near maximal claudication pain 3 days per week. Walking duration and intensity of the sessions were progressively increased during the program. Walking duration began at 15 minutes for the first month of the program, and increased by 5 minutes per month until a total of 40 minutes of walking was accomplished by the sixth month of rehabilitation. Walking intensity was set at grades between 50% and 80% of the maximal work load and was adjusted on an individual basis throughout the program. During each exercise session, patients walked at a speed of approximately 2 mph until their claudication pain reached a level of 3 on a 0–4 pain scale (0 = no pain, 1 = onset of pain, 2 = moderate pain, 3 = intense pain, and 4 = maximal pain) (24), after which they rested. This pattern of intermittent walking and rest continued until the prescribed number of minutes of walking was accomplished. Five minutes of cycling on a stationary bicycle ergometer served as a warm-up and cool-down during each exercise session. The program was supervised by exercise physiologists and a nurse trained in cardiopulmonary resuscitation. There were no complications that resulted from the exercise program. The following measurements were obtained before and after the exercise program.

Ambulatory Measurements

Maximal treadmill test.—Patients performed a progressive, graded treadmill protocol (2 mph, 0% grade with 2% increase every 2 minutes) until maximal claudication pain. The following three outcome measures were obtained: (a) claudication pain distances, (b) peak oxygen uptake, and (c) ABI and the ischemic window. The claudication pain scale was used to assist the patients in identifying their degree of leg pain during the exercise test, and the distances to onset and to maximal claudication pain were recorded. Peak oxygen uptake was obtained via indirect calorimetry, with gas exchange measurements obtained every 20 seconds during exercise using a metabolic measurement cart (model 2900, Sensormedics, Inc, Anaheim, CA). Peak oxygen uptake was recorded as the average of the two highest 20-second oxygen uptake values.

ABI was obtained before and at 1, 3, 5, and 7 minutes after the exercise test in the more severely diseased lower extremity by the Doppler ultrasound technique, as previously described (25,26). The reduction in ankle systolic blood pressure, following the treadmill test, from the resting value was quantified by calculating the area under the curve, referred to as the ischemic window (27). To assess the change in the ischemic window following exercise rehabilitation, patients performed an additional treadmill test at follow-up, but were stopped when they achieved the same walking distance as was measured at baseline. This permitted comparison of the change in the ischemic window for a given walking distance before and after exercise rehabilitation, as described by Carter and coworkers (28). Reliable claudica-
tion, peak oxygen uptake, and ankle systolic blood pressure measurements are obtained using this treadmill protocol (25,26).

**Submaximal treadmill test.**—This test measures the economy of walking at a constant, submaximal work rate that approximates the intensity of many daily activities performed by PAOD patients with intermittent claudication (29). Patients walked at a treadmill speed of 2 mph and a grade of 0% until maximal claudication pain, or for a maximum of 20 minutes. All patients experienced at least the onset of claudication pain by the end of the submaximal walking test, both before and after the exercise program. Oxygen uptake was measured as described for the maximal treadmill test, and walking economy was calculated by averaging the two highest 20-second oxygen uptake values during the final minute of exercise.

**Walking impairment questionnaire (WIQ).**—Self-reported ambulatory ability was assessed using a validated questionnaire for PAOD patients in which they evaluate their walking ability at various speeds and distances and their ability to climb stairs (30). A scale ranging between 0 and 100 assesses each aspect; a score of 0 represents inability and a score of 100 represents no difficulty in performing the task.

**Physical Activity Measurements**

**Physical activity monitoring.**—Free-living daily physical activity was monitored over 2 consecutive weekdays by a Caltrac accelerometer (Muscle Dynamics, Torrance, CA) attached to the belt of each patient and worn slightly anterior over one hip. Patients wore the accelerometer during their waking hours and removed it before retiring to bed. The accelerometer assessed daily physical movements by converting vertical accelerations of the body into caloric expenditure during the 48-hour monitoring period (31,32). The accelerometer has high test-retest reliability (33) and provides valid estimates of daily physical activity assessed by the gold standard technique of doubly labeled water (34).

**Physical activity questionnaire.**—The Minnesota Leisure Time Physical Activity (LTPA) questionnaire determined the self-reported activity level of the participants (35). The Minnesota LTPA questionnaire yields an average daily energy expenditure of physical activity (kcal/day) and has been validated against the energy expenditure of physical activity measured by the doubly labeled water technique (36).

**Peripheral Circulation**

Calf blood flow was obtained under standardized resting and reactive hyperemic conditions in the more severely diseased leg using venous occlusion mercury strain-gauge plethysmography (37). Patients rested supine for 10 minutes, after which five measures were taken and averaged. Reactive hyperemia was then performed by inflating a thigh blood pressure cuff to at least 200 mm Hg to induce arterial occlusion for 3 minutes. Measurement of postocclusive reactive hyperemia (PORH) calf blood flow was obtained within the first minute following the 3-minute occlusion.

**Body Composition Measurements**

Percent body fat was determined by a total body scan with dual-energy x-ray absorptiometry (model DPX-L, LUNAR Radiation, Madison, WI) while patients rested supine after a 12-hour overnight fast. All scans were analyzed using the LUNAR Version 1.3 DPX-L extended analysis program for body composition. The coefficient of variation for the measurement of percentage of body fat is 0.9% using this technique (38). Height was recorded from a stadiometer (SECA, Germany), and body weight was measured on a balance beam scale (Health-O-Meter, Inc, Bridgeview, IL), with the patients dressed in light clothing without wearing shoes. From these measurements, body mass index was calculated as weight (kg)/height (m)².

**Statistical Analyses**

Paired t tests were used to assess the changes in functional and physiological measurements before and after exercise rehabilitation. Pearson product moment and Spearman rank correlation coefficients (r) were calculated to determine the bivariate relationships among the changes in variables and to assess the relationship between baseline characteristics and the response to exercise rehabilitation. Stepwise multiple regression was performed to identify the variables independently related to the change in claudication distances. Statistical significance was set at p < .05. Measurements are presented as means ± SE.

**RESULTS**

**Ambulatory and Physiological Responses to Exercise Rehabilitation**

The treadmill distance to onset of claudication pain increased 115% (p < .001), and the distance to maximal claudication pain increased 65% (p < .001), following the walking program (Table 1). Several patients reached a maximal grade of 18% after exercise rehabilitation. Peak oxygen uptake increased 10% (p < .01), and the economy of walking at a constant work rate improved 10% (p < .001). In addition to these improvements during a standardized treadmill test, walking distance, walking speed, and stair climbing scores from the WIQ increased 52% (p < .001), 38% (p < .01), and 23% (p = .05), respectively. Resting ABI was not changed following exercise rehabilitation, but the ischemic window improved 28% (p < .01), and calf blood flow increased under both resting (9%, p < .05) and PORH (27%, p < .001) conditions (Table 2). No changes in body weight, body mass index, or percent body fat were noted.

**Predictors of Increased Claudication Distances**

In bivariate analyses, the change in distance to onset of claudication pain following rehabilitation correlated with age, laterality of intermittent claudication, baseline distance to onset of pain, baseline WIQ speed score, baseline ABI, and improvements in peak oxygen uptake, walking economy, resting calf blood flow, and PORH calf blood flow (Tables 3 and 4). Using stepwise multiple regression, the change in PORH calf blood flow and age were the only in-
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Table 1. Ambulatory Function of 63 Peripheral Arterial Occlusive Disease Patients With Intermittent Claudication Before and After Exercise Rehabilitation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before Exercise</th>
<th>After Exercise</th>
<th>Delta</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distance to onset of claudication pain (m)</td>
<td>178 ± 22</td>
<td>383 ± 34</td>
<td>205 ± 31***</td>
</tr>
<tr>
<td>Distance to maximal claudication pain (m)</td>
<td>389 ± 29</td>
<td>641 ± 34</td>
<td>252 ± 31***</td>
</tr>
<tr>
<td>Peak oxygen uptake (mL/kg/min)</td>
<td>13.2 ± 0.4</td>
<td>14.5 ± 0.4</td>
<td>1.3 ± 0.4**</td>
</tr>
<tr>
<td>Walking economy (mL/kg/min)</td>
<td>11.6 ± 0.3</td>
<td>10.4 ± 0.3</td>
<td>−1.2 ± 0.3***</td>
</tr>
<tr>
<td>WIQ distance (%)</td>
<td>31 ± 4</td>
<td>47 ± 5</td>
<td>16 ± 5***</td>
</tr>
<tr>
<td>WIQ speed (%)</td>
<td>32 ± 3</td>
<td>44 ± 4</td>
<td>12 ± 4.5***</td>
</tr>
<tr>
<td>WIQ stairs (%)</td>
<td>47 ± 4</td>
<td>58 ± 4</td>
<td>11 ± 5*</td>
</tr>
</tbody>
</table>

Notes: Values are mean ± standard error. WIQ = walking impairment questionnaire. *p < .05; **p < .01; ***p < .001.

dependent predictors of the change in distance to onset of claudication pain; the change in PORH calf blood flow entered the model first explaining 18% of the variance, and age entered the model on the second and final step explaining an additional 7% of the variance. The regression equation predicting the change in distance to onset of claudication pain is: Change in distance (m) = 779 + (35 × change in PORH calf blood flow [mL/100 mL/min]) − (10 × age [years]), $R = .50$, $R^2 = .25$, standard error estimate (SEE) = 247 m, $p < .001$.

The change in distance to maximal claudication pain following rehabilitation correlated with laterality of intermittent claudication, baseline distances to onset and to maximal pain, and improvements in peak oxygen uptake and walking economy (Tables 3 and 4). Using stepwise multiple regression, the change in peak oxygen uptake and the change in walking economy were the only independent predictors of the change in distance to maximal claudication pain; the change in peak oxygen uptake entered the model first, explaining 26% of the variance, and the change in walking economy entered the model on the second and final step, explaining an additional 15% of the variance. The regression equation predicting the change in distance to maximal claudication pain is: Change in distance (m) = 139 + (45 × change in peak oxygen uptake [mL/kg/min]) − (64 × change in walking economy [mL/kg/min]), $R = .64$, $R^2 = .41$, SEE = 219 m, $p < .001$.

Physical Activity Response to Exercise Rehabilitation

Physical activity level of the patients also increased following exercise rehabilitation, as free-living daily physical activity from the accelerometer increased 31% ($p < .001$) (Figure 1), and self-reported activity from the Minnesota LTPA questionnaire increased 62% (93 ± 8 kcal/day to 151 ± 9 kcal/day; $p < .05$). The changes in treadmill claudication distances were related to the change in free-living daily physical activity (Figures 2 and 3), but not to the change in self-reported activity. The regression equation predicting the change in physical activity from the change in distance to onset of claudication pain is: Change in physical activity (kcal/day) = 51 + (0.18 × change in distance to onset of pain [m]), $R = .27$, $R^2 = .07$, SEE = 234, $p < .05$.

The regression equation predicting the change in physical activity from the change in distance to maximal claudication pain is: Change in physical activity (kcal/day) = −10 + (0.35 × change in distance to maximal pain [m]), $R = .45$, $R^2 = .20$, SEE = 216, $p < .001$.

DISCUSSION

The primary findings of this study were that 6 months of exercise rehabilitation increased the distance to onset of claudication pain by 115% and distance to maximal claudication pain by 65%. These improvements in ambulatory function translated into a 31% increase in free-living daily physical activity and a 62% increase in self-reported physical activity. Furthermore, exercise rehabilitation improved the self-reported quality of life as assessed by the disease-specific WIQ instrument. This study demonstrates that exercise rehabilitation improves ambulatory function, physical activity, calf blood flow, and disease-specific quality of life in middle-aged and older PAOD patients with intermittent claudication, and supports current recommendations for exercise rehabilitation in this patient population (39).

Table 2. Peripheral Hemodynamic and Body Composition Measurements of 63 Peripheral Arterial Occlusive Disease Patients With Intermittent Claudication Before and After Exercise Rehabilitation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before Exercise</th>
<th>After Exercise</th>
<th>Delta</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle/Brachial Index</td>
<td>0.63 ± 0.02</td>
<td>0.63 ± 0.03</td>
<td>0.00 ± 0.01</td>
</tr>
<tr>
<td>Ischemic window (AUC)</td>
<td>209 ± 20</td>
<td>150 ± 16</td>
<td>−59 ± 21**</td>
</tr>
<tr>
<td>Calf blood flow: rest (mL/100 mL/min)</td>
<td>3.19 ± 0.14</td>
<td>3.47 ± 0.15</td>
<td>0.28 ± 0.12*</td>
</tr>
<tr>
<td>Calf blood flow: PORH (mL/100 mL/min)</td>
<td>8.36 ± 0.43</td>
<td>10.63 ± 0.49</td>
<td>2.27 ± 0.40***</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>79.9 ± 1.7</td>
<td>79.8 ± 1.6</td>
<td>0.1 ± 0.4</td>
</tr>
<tr>
<td>Body mass index</td>
<td>27.5 ± 0.6</td>
<td>27.6 ± 0.6</td>
<td>0.1 ± 0.2</td>
</tr>
<tr>
<td>Percent fat</td>
<td>29.2 ± 1.2</td>
<td>29.1 ± 1.3</td>
<td>0.1 ± 0.5</td>
</tr>
</tbody>
</table>

Notes: Values are mean ± standard error. AUC = area under curve; PORH = post occlusive reactive hyperemia. *p < .05; **p < .01; ***p < .001.
Different mechanisms appear to explain the exercise-mediated increase in the two treadmill claudication distances. The increase in distance to onset of claudication pain was primarily due to an increase in hyperemic calf blood flow. This finding suggests that exercise rehabilitation improves the perfusion of the calf musculature in response to an ischemic challenge, thereby delaying the onset of claudication during exercise. Exercise-mediated improvements in peripheral circulation in PAOD patients with intermittent claudication is not a consistent finding, as some studies report an increase (5,8,9,12,27), although others report no change in perfusion (6,10,11,13,15,28). A closer examination of the literature (4) reveals that nine studies that measured maximal calf blood flow using venous occlusion plethysmography reported an average increase of 19% following exercise rehabilitation, closely approximating the 27% increase found in the present investigation. Because the sample size of these studies ranged from 7 to 21 patients, it is likely that inadequate statistical power partially accounts for the inconsistent findings of significant exercise-mediated increases in the peripheral circulation. When our findings are considered along with that of other studies (4), calf perfusion increases approximately 20% in PAOD patients with intermittent claudication following a program of exercise rehabilitation.

The increase in distance to onset of claudication pain also was independently related to the baseline age of the patients. The indirect relationship between age and improved symptomatology suggests that older patients respond less favorably to exercise training than their younger counterparts on an absolute basis. This probably occurred because all patients were trained at the same relative intensity; there-

### Table 3. Correlation Coefficients Between Baseline Characteristics of 63 Peripheral Arterial Occlusive Disease Patients and the Change in Claudication Pain Distances Following Exercise Rehabilitation

<table>
<thead>
<tr>
<th>Baseline Measures</th>
<th>Change in Distance to Onset of Claudication Pain</th>
<th>Change in Distance to Maximal Claudication Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>−0.26*</td>
<td>−0.11</td>
</tr>
<tr>
<td>Laterality of IC</td>
<td>−0.26*</td>
<td>−0.25*</td>
</tr>
<tr>
<td>Distance to onset of claudication pain</td>
<td>−0.25*</td>
<td>−0.39*</td>
</tr>
<tr>
<td>Distance to maximal claudication pain</td>
<td>−0.10</td>
<td>−0.35*</td>
</tr>
<tr>
<td>Peak oxygen uptake</td>
<td>−0.12</td>
<td>−0.19</td>
</tr>
<tr>
<td>Walking economy</td>
<td>0.05</td>
<td>0.21</td>
</tr>
<tr>
<td>WIQ distance</td>
<td>0.18</td>
<td>0.05</td>
</tr>
<tr>
<td>WIQ speed</td>
<td>0.31*</td>
<td>0.00</td>
</tr>
<tr>
<td>WIQ stairs</td>
<td>0.12</td>
<td>−0.02</td>
</tr>
<tr>
<td>Ankle/Brachial Index</td>
<td>0.25*</td>
<td>0.16</td>
</tr>
<tr>
<td>Ischemic window</td>
<td>0.01</td>
<td>−0.05</td>
</tr>
<tr>
<td>Calf blood flow: rest</td>
<td>0.00</td>
<td>−0.05</td>
</tr>
<tr>
<td>Calf blood flow: PORH</td>
<td>0.02</td>
<td>0.14</td>
</tr>
</tbody>
</table>

*Notes: IC = intermittent claudication; PORH = post occlusive reactive hyperemia; WIQ = walking impairment questionnaire. *p < .05.

### Table 4. Correlation Coefficients Between the Change in Physiological Measures and the Change in Claudication Pain Distances Following Exercise Rehabilitation in 63 Peripheral Arterial Occlusive Disease Patients

<table>
<thead>
<tr>
<th>Change in Physiological Measures</th>
<th>Change in Distance to Onset of Claudication Pain</th>
<th>Change in Distance to Maximal Claudication Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak oxygen uptake</td>
<td>0.27*</td>
<td>0.41**</td>
</tr>
<tr>
<td>Walking economy</td>
<td>−0.25*</td>
<td>−0.34*</td>
</tr>
<tr>
<td>WIQ distance</td>
<td>0.11</td>
<td>0.02</td>
</tr>
<tr>
<td>WIQ speed</td>
<td>−0.13</td>
<td>0.10</td>
</tr>
<tr>
<td>WIQ stairs</td>
<td>0.04</td>
<td>−0.01</td>
</tr>
<tr>
<td>Ischemic window</td>
<td>−0.12</td>
<td>−0.15</td>
</tr>
<tr>
<td>Calf blood flow: rest</td>
<td>0.30*</td>
<td>0.17</td>
</tr>
<tr>
<td>Calf blood flow: PORH</td>
<td>0.42**</td>
<td>0.19</td>
</tr>
</tbody>
</table>

*Notes: PORH = post occlusive reactive hyperemia; WIQ = walking impairment questionnaire. *p < .05; **p < .01.

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**Figure 1.** Free-living daily physical activity of 63 peripheral arterial occlusive disease patients with intermittent claudication before and after exercise rehabilitation. Values are mean ± standard error.

**Figure 2.** The relationship between the change in free-living daily physical activity and the change in distance to onset of claudication pain in 63 patients with peripheral arterial occlusive disease following exercise rehabilitation.
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Figure 3. The relationship between the change in free-living daily physical activity and the change in distance to maximal claudication pain in 63 patients with peripheral arterial occlusive disease following exercise rehabilitation.

fore, older patients exercised at a lower absolute intensity due to their lower baseline exercise capacity (40). The training response of older patients is closer to the response of their younger counterparts when expressed on a relative basis.

The exercise-mediated increase in distance to maximal claudication pain was independently related to improved cardiopulmonary function. The 10% increase in peak oxygen uptake following exercise rehabilitation is consistent with the 19% to 29% improvement found in previous studies (41,42). These results suggest that the distance to maximal pain is not solely dependent upon local perfusion of the active musculature, but that central factors responsible for oxygen delivery enable PAOD patients with intermittent claudication to walk a greater distance before maximal leg pain is attained.

In addition to peak oxygen uptake, walking economy is an important factor in explaining the increase in distance to maximal claudication pain following exercise rehabilitation. The 10% improvement in walking economy supports the 9% to 17% improvement found in previous studies (41–43) and suggests that exercise rehabilitation improves the biomechanical efficiency of ambulation, thereby lowering the metabolic demand of walking on the active calf musculature. An important clinical implication of the present investigation is that the combined 10% improvement in peak oxygen uptake and 10% improvement in walking economy following exercise rehabilitation may enable patients to ambulate at a 20% lower relative exercise intensity in the community setting.

None of the baseline characteristics, except for age, were independent predictors of the change in treadmill claudication distances in response to an exercise rehabilitation program. Consequently, PAOD patients with intermittent claudication who are not excluded because of severe coronary artery disease, dyspnea, and poorly controlled blood pressure are capable of increasing their treadmill distances to onset and to maximal claudication pain following exercise, regardless of gender, race, laterality of intermittent claudication, duration of claudication symptoms, and cardiovascular disease risk factors. Furthermore, baseline measures of claudication distances, perceived ambulatory function, ABI and calf perfusion, and body composition did not predict response. This suggests that all PAOD patients with intermittent claudication, who can safely exercise from a clinical, cardiopulmonary, and orthopedic standpoint, should be considered as candidates for a treadmill walking program to improve their symptomatology. Patients with severe cardiopulmonary disease should be targeted for other therapies to treat their claudication.

The 65% to 115% increase in treadmill claudication distances following exercise rehabilitation translated into a 31% higher free-living daily physical activity in the community setting. This supports a recent study reporting a significant increase in the daily percentage of time spent in physical activity from 4.1% to 6.9% in a small cohort of PAOD patients who successfully completed a program of exercise (44). We believe that the increase in free-living daily physical activity of 106 kcal in the present study following rehabilitation is clinically important for several reasons. First, it suggests that improved symptomatology enables PAOD patients to perform an additional 20 to 30 minutes of community-based ambulation each day. Second, the higher level of physical activity may have a protective effect on subsequent cardiovascular morbidity and mortality (16,18), thereby improving the poor survival prognosis typical in this population (19–21).

The primary limitation of this study was the lack of a nonexercise control group. We believe that the lack of a control group had a minor impact on the interpretation of the change in treadmill claudication distances, because these parameters would be expected to gradually decline over time in this chronically ill patient population. In addition, our findings are consistent with numerous other studies (4). However, the improvements in peripheral circulation, peak oxygen uptake, walking economy, and free-living daily physical activity following exercise rehabilitation have not been well established in PAOD patients, and future randomized controlled trials are needed to replicate these findings. Of these measures, the increase in physical activity level is most prone to bias, as community-based activity may be influenced by many factors other than improved claudication distances. For example, it is possible that when PAOD patients volunteer to participate in an exercise research study, they concomitantly adopt other behaviors that promote better health in addition to the exercise intervention. Consequently, a randomized controlled trial is required to better isolate the effect of exercise rehabilitation on free-living daily physical activity from other extraneous factors.

In summary, the primary findings from this study on the efficacy of a treadmill walking program in PAOD patients were: (a) the exercise-mediated increase in treadmill distance to onset of claudication pain was independently explained by improved peripheral circulation and baseline age, (b) the exercise-mediated increase in distance to maximal claudication pain was independently explained by improved cardiopulmonary function and walking economy,
and (c) the increased claudication distances were related to greater free-living daily physical activity in the community setting. In conclusion, increased treadmill claudication distances following exercise rehabilitation are mediated through improvements in peripheral circulation, walking economy, and cardiopulmonary function, with younger patients having the greatest absolute ambulatory gains. Furthermore, improved symptomatology translated into enhanced community-based ambulation.

Acknowledgments

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**Johns Hopkins University School of Medicine**

**Division of Geriatric Medicine and Gerontology**

Applications are invited for a faculty position in the Division of Geriatric Medicine and Gerontology at the Johns Hopkins University School of Medicine.

Candidates should possess a Ph.D. degree in a discipline related to the health care of the elderly and have demonstrated ability in research. A track record of externally funded research is desirable. The candidate eventually will be expected to obtain grant-based funding for her/his research investigations and ultimately to contribute to existing, nascent research projects, and to advise and interact with fellows and junior faculty in a geriatric medicine clinical/research fellowship program, which now has 20 faculty including 5 Ph.D.s and 10 fellows. The potential for collaboration with colleagues in other Divisions Departments and Schools in the University is great.

Applications will be received until October 31, 2000 or until an outstanding candidate is identified. The search will give full consideration to applications that include a curriculum vitae (including grant funding history, 2-3 representative publications, and the names of three references (including addresses, telephone/fax numbers, e-mail address).

Application materials should be sent to Dr. John R. Burton, Director, Division of Geriatric Medicine and Gerontology, Johns Hopkins Geriatrics Center, 5505 Hopkins Bayview Circle, Baltimore, MD 21224.

The Johns Hopkins Bayview Physicians, the employer of the Divisional faculty is an equal opportunity employer.