Exercise conditioning in middle-aged men after 10 days of bed rest

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ABSTRACT Of 12 healthy men with a mean age 50 ± 4 years who had been at bed rest for 10 days, six were randomly assigned to perform individually prescribed physical exercise daily for 60 days after bed rest (exercise group) and six simply resumed their customary activities (control group). Exercise group subjects were significantly more active than control subjects during this interval (p < .05). Two classic training effects observed in the 60 days after bed rest were significantly larger among exercise than among control group subjects; compared with values immediately after bed rest, heart rate at a constant submaximal workload declined by 36 ± 11 beats/min in the exercise group vs 16 ± 8 beats/min in the control group and peak oxygen consumption increased by 4.8 ± 4.2 vs 2.2 ± 5.0 ml/kg/min (both p < .05). Despite these differences in the cardiovascular response to exercise, peak oxygen consumption in both groups returned to before-bed rest levels by 30 days after bed rest, and this was accompanied by significant (p < .05) and similar increases in resting left ventricular end-diastolic and stroke volumes in both groups. Simple resumption of usual physical activities after bed rest was as effective as formal exercise conditioning in restoring functional capacity to before-bed rest levels. Circulation 68, No. 2, 245-250, 1983.

BED REST diminishes the cardiovascular tolerance for exercise in normal subjects and contributes to the physical debility of patients recovering from myocardial infarction.1, 2 The classic bed rest study of Saltin et al.1 in normal young subjects is often cited as evidence favoring in-hospital and early posthospital exercise conditioning for postinfarction patients, but that study was not designed to distinguish between the effects of exercise conditioning and those of resumption of usual activities; all five subjects underwent exercise conditioning.

In the same group of healthy middle-aged men presently under consideration, we have recently reported that orthostatic factors resulting in a diminished left ventricular end-diastolic volume contributed significantly to the diminished capacity for upright exercise after bed rest.3 In this study we sought to address the clinically relevant question of whether or not exercise conditioning is required to restore functional capacity after bed rest. This question is particularly important in postinfarction patients in whom the risk of vigorous exercise conditioning soon after the acute event may outweigh the potential benefits of hastening the restoration of functional capacity.

Methods

Twelve healthy men (mean age 50 ± 4 years, range 45 to 55) were recruited through an employment service and paid for their participation in this study. Initial exercise testing before the in-hospital phase of the study (screening evaluation) was performed by subjects in the upright position on a Monark bicycle ergometer. The initial workload was 30 W and was increased in 30 W increments until exhaustion. All workloads were performed at 60 rpm, which permitted the expression of work rate in watts. A bipolar V5 electrocardiographic lead was monitored continuously and was recorded at the end of each minute of exercise and after the first 5 min of recovery. Systolic pressure was recorded on a sphygmomanometer at the end of each third minute of exercise and at peak effort. Monitoring and recording of the electrocardiogram and blood pressure were similar for all subsequent exercise tests. Resting 12-lead electrocardiograms were normal in all 12 subjects and no chest discomfort, ischemic ST segment depression, or significant ventricular arrhythmias were noted during any of the exercise tests performed before or after bed rest.

The experimental protocol (figure 1) consisted of a 4 day in-hospital ambulatory control period followed by 10 days of in-hospital bed rest and by a 60-day ambulatory period after hospital discharge. The study group of 12 subjects was admitted to the Clinical Research Center of the Stanford University Hospital. During the 4 day ambulatory control period, subjects under-

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went baseline studies for determination of endocrine levels and metabolic function and M mode echocardiographic imaging of the heart during supine rest. Standard procedures were used for echocardiographic imaging and for calculation of cardiac volume.4

On the morning of the second day before bed rest, subjects underwent upright symptom-limited bicycle ergometric exercise testing in conjunction with the recording of radionuclide ventriculograms. A specially constructed Lucite brace was used to minimize movement of the arms and torso during exercise. In order to produce physical exhaustion within the 12 to 15 min available for imaging of left ventricular function, workload increments during testing before bed rest were larger than those used on the screening evaluation (approximately 300 vs 180 kg·m/min). As a result of these differences in protocol, the values for peak oxygen consumption (VO$_2$) were 21% lower during testing before bed rest than during the screening exercise test, despite similar peak heart rates during both tests (170 ± 3 and 173 ± 4 beats/min). Actual value for peak VO$_2$ for the entire group of 12 subjects were 25.8 ± 5.2 and 31.3 ± 5.9 ml/kg/min on before-bed rest and screening evaluations, respectively (p < .05).

Exercise tests were conducted with the use of a Schwinn electrically braked bicycle ergometer that provided standardized workload increments of 150 kg·m/min. Although the same sequence of workloads was used for exercise testing before and after bed rest, the duration of testing and the number of stages varied, tending to be greater at 60 days after than immediately after bed rest (table 1). For all subjects exercise testing consisted of three uninterrupted 3 min stages (I, II, and III of submaximal effort) that approximated 20%, 45%, and 70% of the workloads during the prehospital screening evaluation. Immediately after bed rest both groups completed 1.5 to 2 min of stage IV effort, which approximated 100% of the highest exercise workload during the prehospital screening evaluation. The absolute workload for stage IV effort was 1156 ± 194 kg/min. Sixty days after bed rest almost all of the trained subjects were capable of stage IV effort and some completed as much as 2 min of stage V effort, representing an increase of approximately 1 min over before-bed rest values (table 1). Whereas most control subjects also completed stage IV 60 days after bed rest, few reached stage V, and values for exercise duration in this group did not exceed values before bed rest.

Oxygen consumption was measured during the last 30 sec of each ergometric workload by standard techniques.5 During exercise testing subjects underwent radionuclide ventriculography with a gamma camera positioned to record left ventricular performance in the left anterior oblique position.

At the conclusion of the bed rest period all subjects were advised to resume their customary living habits and routines. On the first day of ambulation after bed rest the 12 subjects were sorted into six pairs matched on the basis of age, height, weight, body surface area, and peak VO$_2$. One member of each pair was randomly assigned to undergo exercise conditioning (exercise group) and one was randomly assigned to receive no instructions for exercise conditioning (control group).

Exercise and control subjects were evenly matched with respect to age (49 ± 3 years, range 45 to 54 vs 51 ± 4 years, range 45 to 55), height (177 ± 8 cm, range 167 to 188 vs 178 ± 7 cm, range 170 to 188), weight (82.1 ± 13.3 kg, range 65.0 to 99.3 vs 84.5 ± 9.7 kg, range 72.4 to 96.1), and peak VO$_2$ (31.6 ± 7.1 ml/kg/min, 26.0 vs 43.4 vs 31.0 ± 5.1 ml/kg/min, range 23.9 to 37.0).

Although it was not used for randomization, each subject's customary pattern of physical activity before entry into the study was obtained by questionnaire on the day before randomization into exercise and control groups. We have used this questionnaire, modified from that used by Shapiro et al.,6 to evaluate physical activity patterns in patients with myocardial infarction.

In order to assess the relative levels of physical activity in the two groups, all 12 subjects completed a daily activity log throughout the entire 60 day period after bed rest. Subjects were asked to indicate their most strenuous physical activity for each of three time periods during each day: wake up to noon, noon to

![FIGURE 1](http://circ.ahajournals.org/)

**FIGURE 1.** Experimental protocol. Echo = resting supine echocardiogram.

### TABLE 1

| VO$_2$, test duration, heart rate (HR), systolic blood pressure (SBP), and rate-pressure product (RPP) during maximal exercise before bed rest and 0, 30, and 60 days after bed rest (mean ± SD) |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
|                 | Before bed rest | After bed rest  | R = 30          | R = 60          |
|                 | Control         | Exercise        | Control         | Exercise        | Control         | Exercise        |
| Peak VO$_2$ (l/min)$^a$ | 2.15 ± 0.52     | 2.13 ± 0.36     | 2.24 ± 0.54     | 2.03 ± 0.38     | 2.10 ± 0.48     | 2.21 ± 0.32     |
| Peak VO$_2$ (ml/kg/min)$^b$ | 25.4 ± 6.2      | 26.3 ± 4.7      | 26.0 ± 6.5      | 24.5 ± 5.0      | 24.1 ± 5.3      | 26.8 ± 4.6      |
| Test duration (sec)$^b$ | 698 ± 109       | 685 ± 48        | 722 ± 113       | 741 ± 96        | 706 ± 56        | 762 ± 91        |
| HR (bpm)         | 170 ± 6         | 171 ± 16        | 173 ± 12        | 164 ± 15        | 169 ± 12        | 164 ± 17        |
| SBP (mm Hg)$^b$  | 213 ± 34        | 208 ± 19        | 217 ± 24        | 223 ± 29        | 235 ± 22        | 218 ± 27        |
| RPP × 10$^{-2}$  | 361 ± 61        | 351 ± 30        | 375 ± 48        | 366 ± 64        | 397 ± 47        | 358 ± 59        |

$^a$The difference between value after bed rest and 60 day response significantly differentiated exercise and control groups, with $p < .02$ (interaction of time and training).

$^b$Test duration at both 30 and 60 days after bed rest was significantly longer for exercise than for control groups, $p < .05$ (main effect of training).

R = recovery day 30 or 60 after bed rest.
6 p.m., and 6 p.m. to bedtime. These peak activities were considered in four categories: cycle/walk/jog, lift/push/move, competitive sports, and "other." The intensity of these peak activities was evaluated with a Borg scale that rates activity levels continuously from 7 (very very light) to 19 (very very hard). This scale has been widely used in the classification of the level of exertion during physical activities in ambulatory subjects. Subjects also reported the duration of their peak activities and the highest heart rate attained during these activities. An overall rating of daily physical exertion was obtained by multiplying the duration of peak activity in minutes by the highest heart rate noted during the peak activity during each of the three recording periods each day. All 12 subjects were instructed in recording carotid pulses at rest and during stationary cycling or immediately after running or jogging. Subjects were given preaddressed envelopes in which to return daily activity logs at the end of each week of participation in the study.

The home exercise training program consisted of riding a Monark bicycle ergometer in the upright position for 30 min daily for 60 consecutive days. Each training session consisted of a 5 min warm-up at a relatively low workload followed by 20 min of continuous cycling at an intensity sufficient to elicit a heart rate of 70% to 85% of the peak heart rate measured on the screening exercise test. This corresponded to a training range of approximately 120 to 150 beats/min. Peak heart rates during screening exercise tests were used because these data were available for all subjects at the time of randomization to exercise training. A 5 min cool-down period of low-resistance cycling concluded each exercise session.

In order to ensure compliance to the home training program, the same staff member who had instructed subjects in the use of bicycle ergometers telephoned them twice a week during their exercise sessions in order to record their peak heart rate during training periods. Exercise sessions were held at a fixed prearranged time each day but subjects were not forewarned as to the days on which they would be telephoned. Subjects responded to 90 of the 96 investigator-initiated telephone calls during the 60 days of training. In all but eight of these 90 calls, subjects reported having initiated or completed their planned training sessions; intercurrent illness or injury accounted for the remainder.

Thirty and 60 days after bed rest all subjects returned to the hospital for M-mode echocardiographic imaging at rest and for symptom-limited ergometric exercise testing. The methods used were identical to those employed during previous tests. Radioisotope ventriculographic measurements were recorded before and after bed rest and are reported separately. To maintain comparability, at 30 and 60 days subjects were tested while strapped to the gamma camera in the usual manner.

Data were analyzed by analysis of variance for multiple comparisons and by paired and unpaired t tests for two-group comparisons. Differences were considered significant at p < .05.

Results

Self-reported physical activity during the 3 months before bed rest was not significantly different in subjects in the exercise and in control groups, all of which had ratings categorized as "moderately active." During the 60 days after bed rest, self-reported activity levels were significantly higher in exercise than in control group subjects; the highest heart rates during peak activity were 141 ± 12 vs 106 ± 22 beats/min and the product of highest heart rate and duration of peak activity was 4225 ± 333 vs 3152 ± 1370 beats

(bold p < .05). Mean values for peak rating of perceived exertion corresponded to "hard" and to "somewhat hard" in exercise and in control subjects, respectively.

VO₂ was measured 30 days after bed rest were significantly (p < .05) higher in both groups than those measured immediately after bed rest (table 1, figure 2). The increase in peak VO₂ that occurred between days 0 and 60 after bed rest was significantly greater in the exercise than in the control group (4.8 ± 4.2 vs 2.2 ± 5.0 ml/kg/min and 0.46 ± 0.32 vs 0.29 ± 0.34 l/min; p < .05). The pattern of change in peak VO₂ in the 60 days after bed rest was significantly different in the two groups; whereas exercise subjects demonstrated a continuing rise throughout this interval, control subjects demonstrated an increase in the first 30 days and a decline in the second 30 days after bed rest (p < .05). Peak VO₂ at 60 days significantly exceeded values before bed rest in exercise subjects but not in control subjects (p < .05; table 1, figure 2).

Compared with values immediately after bed rest, test duration increased in both groups after bed rest. The increment in test duration was significantly greater in the exercise group than in the control group, i.e., 130 vs 52 sec at 60 days after bed rest (p < .05; table 1). Test duration at 60 days significantly (p < .05) exceeded that before bed rest in exercise-trained subjects but not in control subjects (77 vs 8 sec).

Peak heart rate in both groups was lower at 30 days than immediately after bed rest and showed little decline thereafter (table 1). Peak systolic blood pressure during exercise was higher in both groups at 30 days than immediately after bed rest and rose further by 60 days in the control group but not in the exercise group (p < .05; table 1). The peak rate-pressure product showed a similar pattern, but the differences between groups were not significant.

Compared with pooled values immediately after bed rest, test duration increased in both groups after bed rest.

![Figure 2](http://circ.ahajournals.org/)

**FIGURE 2.** Peak VO₂. BR = bed rest.
rest, values of heart rate during submaximal effort at the same workload were significantly lower in exercise subjects than in control subjects 30 and 60 days after bed rest (p < .05; figure 3).

Echographically measured left ventricular end-diastolic volume at rest increased significantly (p < .05) after bed rest, mostly during the first 30 days, without significant differences between groups (table 2). Resting stroke volume after bed rest increased significantly in both groups (p < .05), without significant intergroup differences or further significant increases by 60 days. Echographically measured resting left ventricular ejection fraction did not change significantly from immediately after to 60 days after bed rest in either group.

Mean body weights measured immediately before and after bed rest were 85.2 ± 9.5 and 83.5 ± 9.3 kg in the control group and 81.9 ± 13.4 and 80.5 ± 12.7 kg in the exercise group, corresponding to a percentage decrease of approximately 2% in each group (p < .05). Thirty days after bed rest body weight had risen to 86.5 ± 10.0 and 83.5 ± 13.1 kg in control and exercise subjects, corresponding to a percentage increase over values immediately after bed rest of 3.5% and 3.7% (p < .05). No further significant increase in body weight was noted 60 days after bed rest. For the entire group of 12 subjects the increase in body weight and peak VO₂ (expressed in ml/kg/min) correlated significantly (r = .74, p < .05).

Discussion

In the same group of 12 healthy middle-aged men participating in this study we have previously reported that peak VO₂ declined 15% during upright exercise but only 6% during supine exercise after 10 days of bed rest. In the previous study echographically measured left ventricular end-diastolic volume during supine rest diminished 17% after bed rest and undoubtedly more in the upright position as a result of orthostatic stress.¹ Since orthostatic factors related to bed rest seemed to play an important role in the decline of oxygen transport capacity during upright exercise we began to consider the question of whether the reversal of these factors by simple exposure to the orthostatic stress of usual ambulatory activities might be effective in restoring functional capacity after bed rest independent of the effects of vigorous physical exercise.

The primary purpose of this study was to determine the additive effects of vigorous exercise conditioning on the cardiovascular response to exercise in subjects who had resumed their usual physical activities after bed rest. It is therefore important to establish that there was an actual difference in the “dose” of physical activity the two groups of subjects received. Subjects in both groups were rated as moderately active before the study and several in both groups were involved in personal fitness activities such as jogging. It was considered important that usual activity habits be altered as little as possible in order to enhance applicability of study results; increased physical activity in the control subjects would bias the study against demonstrating significant differences between the two groups with respect to their cardiovascular responses to exercise testing. On the other hand, training was relatively intensive and heart rates during peak activity were substantially higher in exercise subjects than in control subjects, i.e., 141 vs 106 during the first 60 days after bed rest.

It is important to note that the experimental conditions under which exercise testing was performed immediately before and after bed rest exerted a constraint on values of peak VO₂. Compared with the screening exercise test, in which subjects were allowed to grasp the handlebars and in which relatively small workload increments equivalent to 180 kg-m were used, tests performed in conjunction with the recording of radionuclide ventriculograms required restraint of the arms and torso and the use of large workload increments of 300 kg-m. These factors resulted in peak VO₂ values before bed rest that were 21% lower than on the screening evaluation. For these reasons the peak VO₂ in our subjects is slightly lower than that reported in healthy middle-aged men⁶, ¹⁰ and it seems likely that the effects of exercise training have been partially masked in our subjects.

By 60 days after bed rest peak VO₂ increased in both groups, but to a greater extent in exercise than in con-

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**FIGURE 3.** Heart rate (HR) after bed rest. Dotted lines indicate that not all subjects contributed to stage IV data.
control subjects. Heart rate at fixed submaximal workload diminished significantly in both the exercise and control groups 30 and 60 days after bed rest, with a significantly greater decrement in the exercise group at 30 and at 60 days. Despite these differences in the cardiovascular response to exercise, in both groups of subjects peak VO₂ returned to before-bed rest levels by 30 days. Apart from its association with the upright posture, formal exercise training appeared to contribute relatively little to the restoration of oxygen transport capacity 30 days after bed rest.

These data should not be interpreted as disparaging the value of exercise training in general, especially in cardiac patients in whom declines in submaximal heart rate and rate-pressure product may significantly reduce myocardial oxygen demand and thus delay or prevent the onset of angina pectoris.11 Also, exercise training is associated with an increase in peak aerobic capacity in patients with coronary heart disease.12 Finally, in patients with chronic ischemic heart disease even very low-level physical activity such as walking has been shown to lower the submaximal heart rate–pressure response to exercise, even in the absence of an increase in peak VO₂.13

The magnitude of the decrease in body weight that occurred in our subjects after bed rest is similar to that measured in other studies,14–16 in which the loss of body weight after bed rest was closely related to the loss of plasma volume. Mean values of echographically measured left ventricular end-diastolic and stroke volume showed a consistent increase of nearly 30% in both groups of subjects within 30 days after bed rest, with little further increase by 60 days. As in the study of Saltin et al.,1 the increase in peak VO₂ that occurred in our subjects 0 to 30 days after bed rest was associated with an increase in body weight and in cardiac volume that occurred during this interval. However, we demonstrated that this can occur as a result of the assumption of the normal upright position and a return to customary activities without the necessity for formal exercise training. Although restoration of intravascular volume is important in reversing the cardiovascular effects of bed rest, volume repletion alone is not sufficient to accomplish this purpose. In normal subjects Hyatt et al.17 found that oral saline did not restore intravascular fluid volume or tolerance for the gravitational stress of lower body negative pressure after 7 days of bed rest. In contrast, oral saline given in conjunction with exposure to lower body negative pressure, which simulates gravitational stress, helped to sustain fluid volume and gravitational tolerance. These data underscore the importance of exposure to orthostatic stress in preventing and reversing the cardiovascular effects of bed rest.

The design of this study did not permit the detection of any differences in oxygen transport capacity that may have existed between the two groups in the interval 0 to 30 days after bed rest. In the three habitually sedentary subjects studied by Saltin et al. before–bed rest levels of peak VO₂ were restored within 10 to 14 days.1 These subjects had lower peak VO₂s that were similar to those noted in our older normal subjects. In contrast, the two habitually active subjects of Saltin et al. required 30 to 40 days of physical activity to restore peak VO₂ to before–bed rest levels. It is reasonable to consider that the time course of recovery in our subjects may also have been dependent on their peak VO₂s before bed rest. The major clinical implication of this study is that resumption of usual physical activities is as effective as exercise conditioning in restoring the functional capacity of normal individuals by 30 days after bed rest. Although these data cannot be directly extrapolated to patients recovering from myocardial infarction, it seems likely that the mechanisms responsible for improvement in their functional capacity are also more closely related to orthostatic factors such as a restoration of intravascular and left ventricular volume than to

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<td>Resting echocardiographic determinations of heart rate, end-diastolic volume, stroke volume, and ejection fraction (mean ± SD)</td>
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A<sub>p < .05</sub> vs after bed rest.
B<sub>p < .05</sub> vs before bed rest.
the intensity of physical activity to which they are exposed after bed rest.

Results of a recent randomized trial of in-hospital exercise conditioning in postinfarction patients did not support the role of exercise conditioning in improving treadmill performance 10 days after the acute event. These authors suggested two reasons for the failure to demonstrate a superior treadmill performance in patients who had received in-hospital exercise conditioning: (1) a duration of bed rest that was too short (2.7 to 4.1 days) to result in cardiovascular deconditioning in either patient group and (2) an inadequate differential in the intensity of physical activity in the two groups. To these reasons we would add a third, that restoration of functional capacity after bed rest is more closely related to exposure to the orthostatic stress of usual physical activities than to exposure to higher levels of exercise. Our data provide physiologic support for a policy of early ambulation in mitigating the deleterious effects of bed rest on cardiovascular performance after myocardial infarction but do not support the need for formal exercise training to accomplish this purpose.

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