NASA Technical Memorandum 101045

# Exercise Countermeasures for Bed Rest Deconditioning



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National Aeronautics and Space Administration

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Edited by
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Moffett Field, California 94035

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## PREFACE

This technical memorandum is a compilation of the results of a 30-day bed rest study to investigate the effects of short-term, high-intensity isotonic and isokinetic exercise training on maintenance of working capacity (peak oxygen uptake), muscular strength and endurance, and on orthostatic tolerance, posture and gait. John Greenleaf, Ph.D., NASA Ames Research Center, Moffett Field, CA 94035, compiled the results reported by the following 14 researchers:

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## SUMMARY

The purpose for this 30-day bed rest study was to investigate the effects of short-term, high-intensity isotonic and isokinetic exercise training on maintenance of working capacity (peak oxygen uptake), muscular strength and endurance, and on orthostatic tolerance, posture and gait. Other data were collected on muscle atrophy, bone mineralization and density, endocrine analyses concerning vasoactivity and fluid-electrolyte balance, muscle intermediary metabolism, and on performance and mood of the subjects.

Nineteen men (32-42 yr) were divided into three groups: no-exercise control (peak $\dot{V}O_2$ once/wk, N = 5), isotonic exercise (electronic Quinton ergometer, supine, N = 7), and isokinetic exercise (electronic Lido ergometer, supine, N = 7). The exercise regimens were conducted near peak levels for 30 min in the morning and 30 min in the afternoon 5 days/wk. The protocol consisted of a 7-day ambulatory control period during which the subjects equilibrated on the standardized diet, 30 days of –6° head-down rest, and a final 4.5 days of ambulatory recovery.

The diet consisted of normal fresh and frozen foods; mean caloric consumption of 2,678 kcal/day (control), 2,833 kcal/day (isotonic), and 2,890 kcal/day (isokinetic) resulted in mean weight losses during bed rest of 1.01 kg, 0.85 kg, and 0.0 kg, respectively.

It was concluded that: (1) The subjects maintained a relatively stable mood, high morale, and high esprit de corps throughout the study. Performance improved in nearly all tests in almost all the subjects. Isotonic training, as opposed to isokinetic exercise training, was associated more with decreasing levels of psychological tension, concentration, and motivation; and improvement in the quality of sleep. (2) Working capacity (peak oxygen uptake) was maintained during bed rest with isotonic exercise training; it was not maintained with isokinetic or no exercise training. (3) In general, there was no significant decrease in strength or endurance of arm or leg muscles during bed rest, in spite of some reduction in muscle size (atrophy) of some leg muscles. (4) There was no effect of isotonic exercise training on orthostasis, since tilt-table tolerance was reduced similarly in all three groups following bed rest. (5) Bed rest resulted in significant decreases of postural stability and self-selected step length, stride length, and walking velocity, which were not influenced by either exercise training regimen. Most pre-bed rest responses were restored by the fourth day of recovery.

## GENERAL BACKGROUND FOR THE STUDY

J. E. Greenleaf

The physical working conditions during construction of Space Station Freedom will require that astronauts spend long hours in extravehicular activity (EVA). Sustained EVA activity can impose fatiguing work loads, and productive work performance requires that these astronauts maintain high levels of physical fitness. The major question is what types of exercise training programs should all astronauts undertake on the ground and in the space station to maintain their physical working capacity and endurance for effective EVA as well as for their normal vehicular activity. Present data indicate that arm and shoulder strength is well-maintained during flight, but it is leg strength that is reduced to a greater extent (JSC, personal communication). Thus, the training routines emphasized leg exercise, but arm exercise testing was included. A secondary, but no less important, question is the effect these exercise training regimens will have on the occasional episodes of post-flight osthostatic intolerance experienced by some crew members; and also the effect these exercise procedures will have on the astronauts' ability to regain normal 1-G gait, movement, and ambulatory patterns following flight. These two problems were investigated in this bed rest study. Additional data were collected on muscle atrophy, bone mineralization and density, hormone analyses concerning vasoactivity and fluid-electrolyte balance, muscle intermediary metabolism, and on performance and mood of the subjects.

The major objectives of the present study were to evaluate the efficacy of different modes of exercise (isotonic and isokinetic) for countering the deleterious effects of bed rest deconditioning on work capacity (peak oxygen uptake), muscular strength, orthostatic tolerance and posture, equilibrium and gait; and to collect additional data of a more fundamental nature to help understand how these deconditioning responses occur. These data will be used to assist in writing prescriptions for exercise to be utilized by astronauts for maintaining work capacity and well-being on Freedom Station, and to determine what exercise devices should be placed in the station.

## GENERAL PROCEDURE AND METHODS

Nineteen men, aged 36 ±SD 4 yr, ht 177.5 ±6.9 cm, wt 76.5 ±7.6 kg, S.A. 1.94 ±0.12 $m^2$, percent fat 15.5 ±6.5%, peak leg strength 145 ±22 Newton-m, and leg total work 6923 ±101 Newton-m, were divided into three experimental groups: no-exercise training control (N = 5), isotonic exercise (N = 7), and isokinetic exercise (N = 7). The protocol consisted of a 7-day ambulatory control period during which the subjects equilibrated on the standardized diet, 30 days of –6° head-down bed rest, and a final 4.5 days of ambulatory recovery. The study was run in two sessions (fig. 1): the first from July 1 to August 11, 1986 (12 subjects), and the second from August 19 to September 29, 1986 (7 subjects). The two exercise regimens were conducted for 30 min each in the morning and afternoon for 5 days/wk. Peak exercise testing was conducted on the remaining 2 days/wk. The diet consisted of normal fresh and frozen foods, and there were 17 different daily menus which were rotated over each 42-day session. The planned caloric intake was 2,800 kcal/day for the no-exercise control group, and 3,100 kcal/day for the two exercise groups. There was no caloric adjustment for subjects with different body weights since the average weight for the three groups was approximately the same. The actual mean caloric consumption

was 2,678 kcal/day (control), 2,833 kcal/day (isotonic), and 2,890 kcal/day (isokinetic), which resulted in mean weight (±SD) losses during bed rest of –1.01 ±1.81 kg, –0.85 ±1.56 kg, and 0.00 ±1.36 kg, respectively. The subjects were supervised 24 hr/day and were requested to remain in the –6° head-down position. All testing and excretory functions were conducted in the horizontal or –6° head-down positions. The subjects were allowed to rise on one elbow to eat. The testing schedule is given in figure 2.

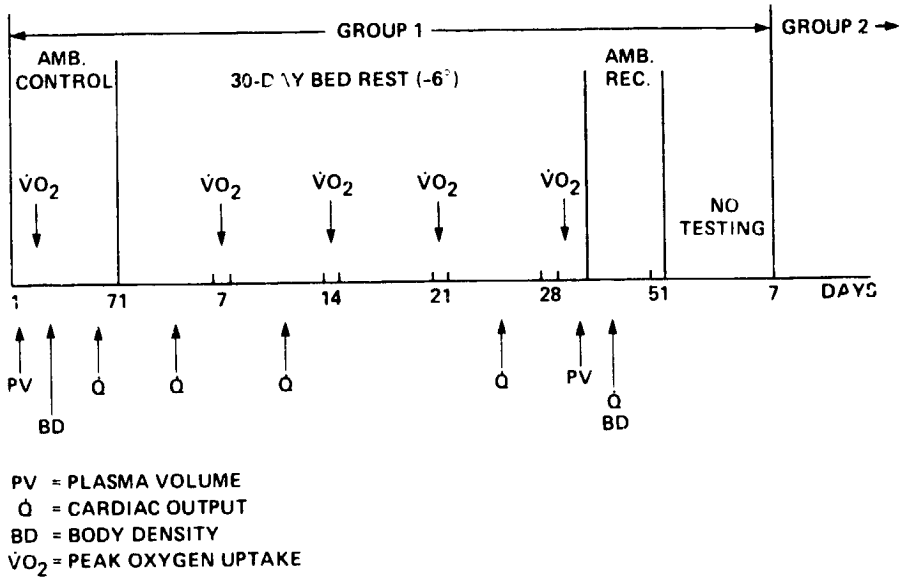
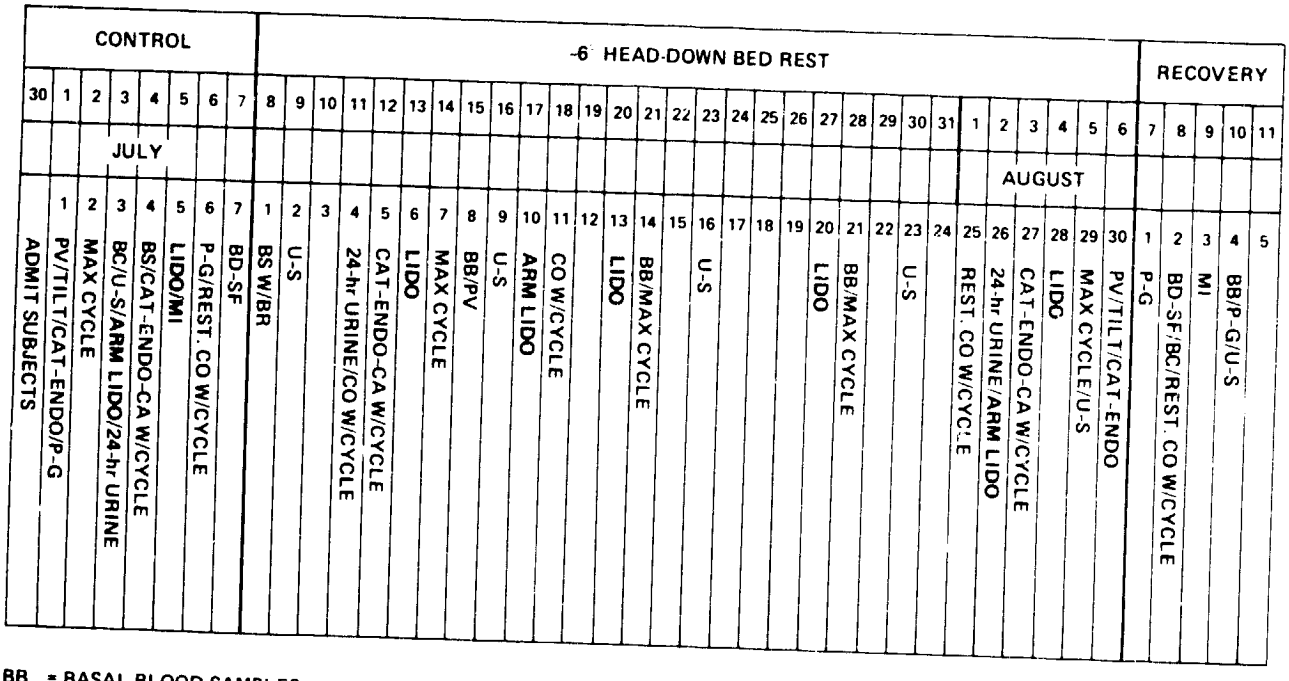



Figure 1.– Experimental protocol.




BB = BASAL BLOOD SAMPLES
P-G = POSTURE-GAIT
PV = PLASMA VOLUME
BS = BLOOD SAMPLE
BC = BODY COMPOSITION
CO = CARDIAC OUTPUT

LIDO = ISOKINETIC EXERCISE
CYCLE = ISOTONIC EXERCISE
MI-US = MAGNETIC IMAGING-ULTRASOUND
CAT-ENDO-CA = CATECHOLAMINE-ENDOCRINE-CALCIUM BLOOD SAMPLES
BD-SF = BONE DENSITOMETRY-SAN FRANCISCO
24-hr URINE = 24-hr URINE/CALCIUM

Figure 2.– Schedule of tests.

# EXERCISE TRAINING PROGRAMS

J. E. Greenleaf, E. M. Bernauer, and M. Bond

## Background

Results from a 14-day bed rest study concluded at Ames Research Center (ARC) in 1972 (ref. 1) indicated that intermittent isometric leg exercise performed during bed rest resulted in a somewhat lower (–4.8%) reduction in working capacity (peak oxygen uptake) than during isotonic ergometer exercise (–9.2%) or with no exercise (–12.3%) during bed rest. It was concluded from this and other studies that various remedial exercise procedures performed during bed rest result in less reduction in peak oxygen uptake, compared with the greater reduction in peak oxygen uptake without remedial exercise (refs. 1-3). The major question is whether isometric, isokinetic, or some type of isotonic exercise (cycle ergometer or treadmill) is better for maintaining the peak oxygen uptake during 30 days of bed rest.

## Procedure and Methods

Each training group exercised in the supine position for 30-min periods in the morning and afternoon. The daily isotonic cycle ergometer exercise (Quinton model 845, Seattle, WA) consisted of a 7-min warm-up period at a relative load of 40% of the peak oxygen uptake, followed by 2 min of exercise at 60%, 70%, 80%, 90%, and 80% loads, with each separated by 2 min at the 40% load (fig. 3). The daily testing schedule is given at the bottom of figure 3. The weekly peak cycle ergometer test protocol is presented in figure 4. Here, the same 7-min warm-up period was used, but the next load was 400 kg-m/min below the peak load for 2 min, then 200 kg-m/min below for the next 2 min, and finally the peak load for at least 2.5 min. This was followed by an appropriate cooling-down period.

The daily isokinetic exercise training was performed on the LIDO™ Isokinetic Rehabilitation System ergometer (Loredan Biomedical, Inc., Davis, CA). The daily exercise training and weekly peak exercise test protocols are given in figures 5 and 6, respectively. Five maximal leg flexions and extensions were performed in 10 sec; this exercise was repeated at the beginning of each minute for 15 min for each leg. The various measurement definitions for leg strength (peak torque, work per repetition) and endurance (endurance index, total work) are presented in figure 7. Arm peak strength and endurance utilizing abduction and adduction were measured weekly.

## Results and Discussion

**Isotonic exercise**– Mean (±SE) work capacity (peak oxygen uptake) was maintained at ambulatory control levels in the isotonic exercise training group (fig. 8). Both the isokinetic and no-exercise training groups were unable to maintain their peak oxygen uptake capacities; the isokinetic group lost about 10% of its capacity and the no-exercise group lost about 20% of its pre-bed rest capacity (fig. 9). The stimuli that facilitated the maintenance of peak oxygen uptake in the isotonic group was equivalent to that which would raise peak oxygen uptake by 20% in ambulatory subjects. Thus, intermittent, near-peak isotonic (cycle) leg exercise training for two 30-min periods/day maintained peak oxygen uptake over

30 days of –6° head-down bed rest, while peak isokinetic (flexion-extension) leg exercise training of similar intensity and duration, and no exercise training did not.

The basic hypothesis for utilizing these exercise prescriptions was that high-intensity exercise stimuli for shorter periods would be more likely to maintain peak oxygen uptake capacity at ambulatory levels during prolonged bed rest compared with the lower-intensity, longer-duration exercise prescriptions used in previous studies. This hypothesis appears to be confirmed for the maintenance of peak oxygen uptake capacity.

Isokinetic exercise– Mean (±SE) arm total work ($\overline{X}$ right + left + abduction + adduction) and total peak torque were unchanged for all three groups during bed rest without the performance of daily arm exercise training (figs. 10 and 11). There was a tendency for strength (peak torque) and endurance (total work) to increase in the isokinetic group, and for these measures to remain constant or decrease slightly in the other two groups (figs. 12 and 13).

The basic high-intensity, short-duration hypothesis appeared to function positively in the isokinetic group, but to a somewhat lesser degree than in the isotonic group. More data analysis is needed before firm conclusions can be drawn.

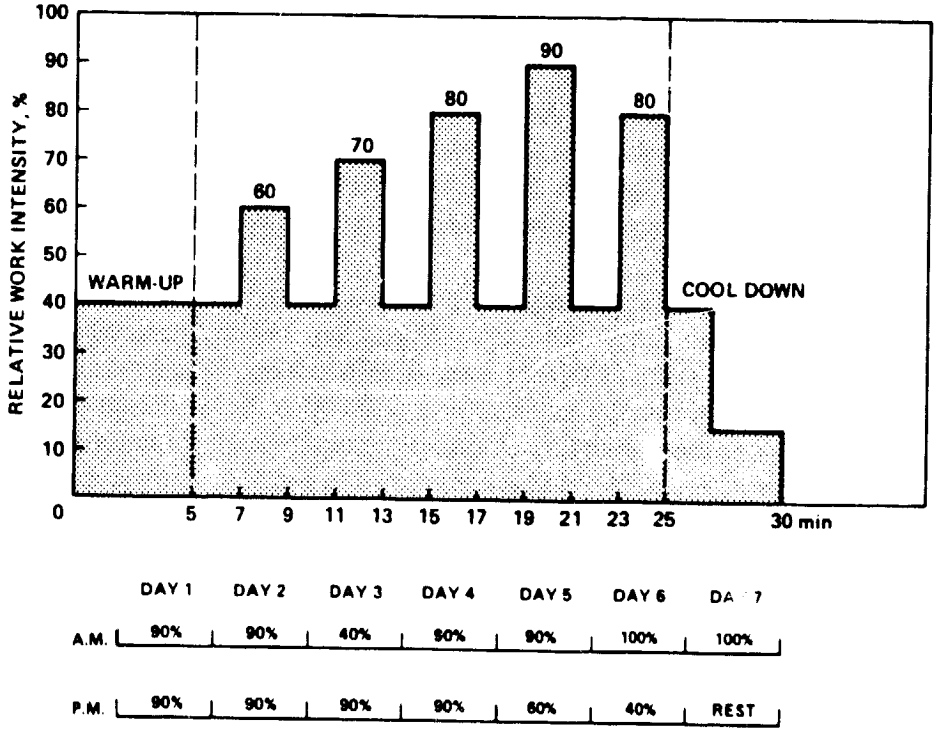



Figure 3.– Daily isotonic cycle ergometry.

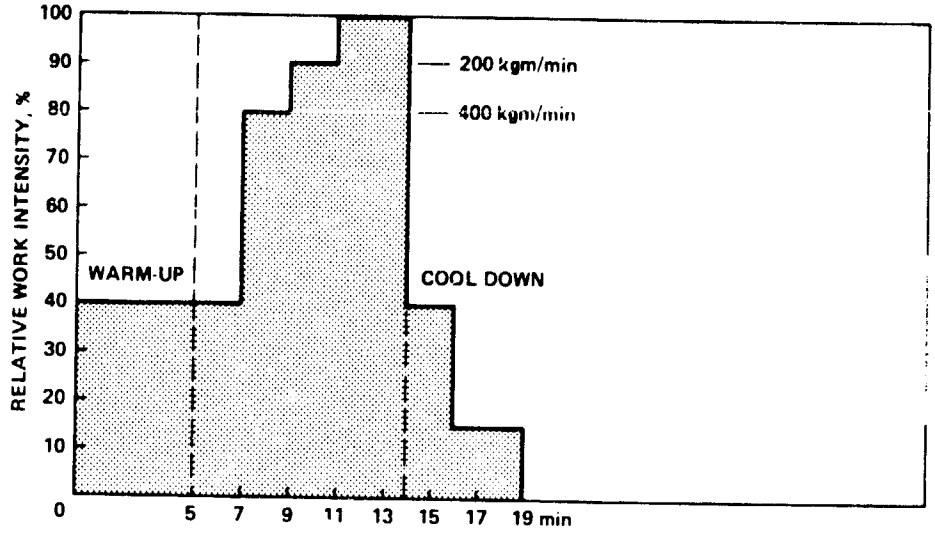



Figure 4.– Weekly maximal cycle ergometry.

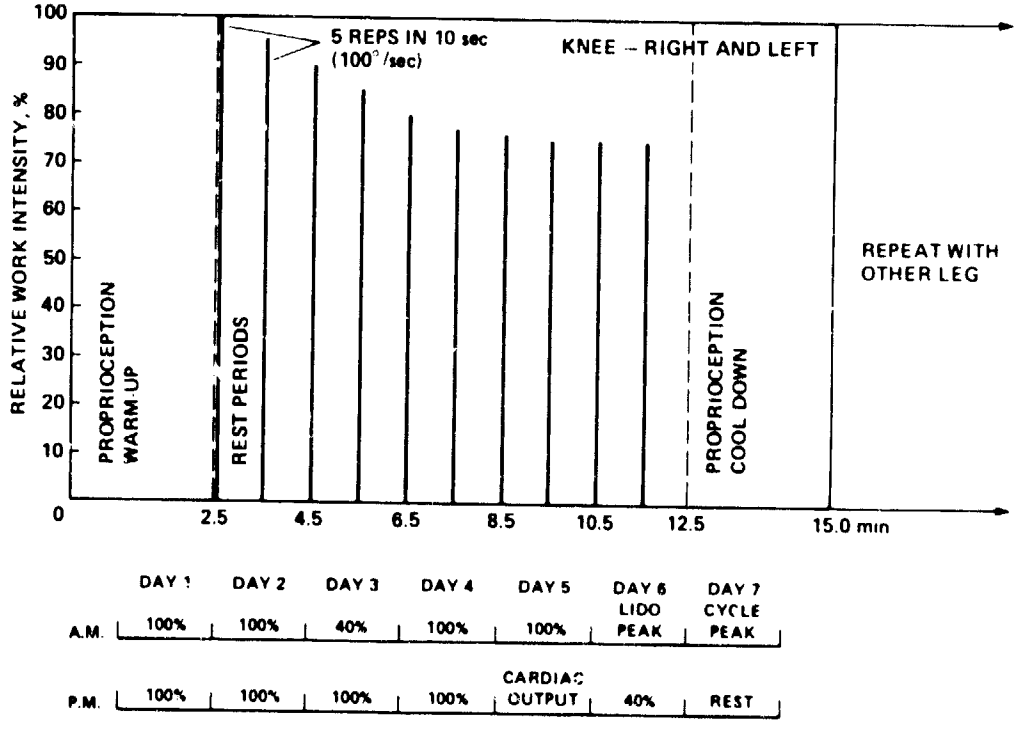



Figure 5.– Daily isokinetic (LIDO) exercise.

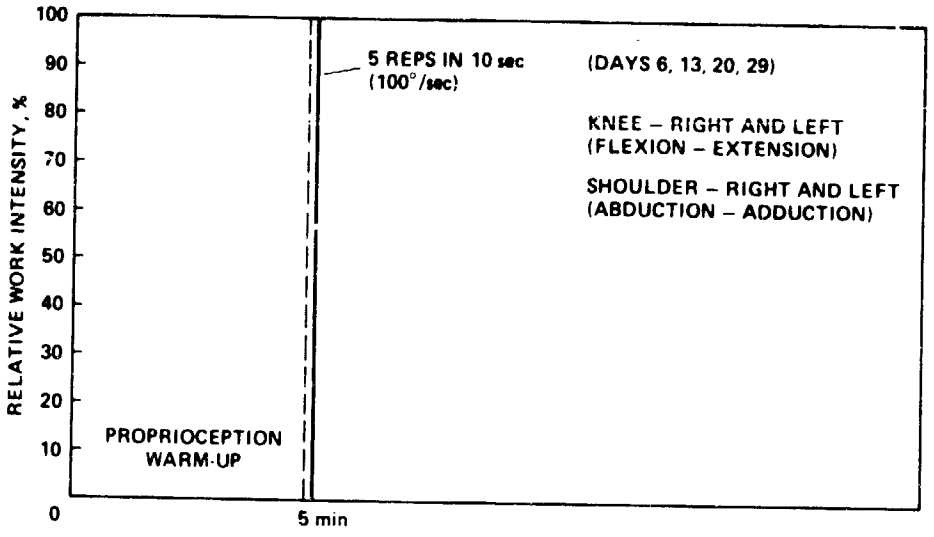



Figure 6.– Weekly maximal isokinetic (LIDO) exercise.

- PEAK TORQUE (N-m); THE MAXIMUM TORQUE PRODUCED FOR FLEXION AND FOR EXTENSION DURING AN EXERCISE BOUT AT THE SPECIFIED SPEED. IT IS MAXIMUM MUSCULAR FORCE (STRENGTH)

- TORQUE RATIO (%) = $\frac{\text{PEAK TORQUE}_{\text{FLEXION, ADD}}}{\text{PEAK TORQUE}_{\text{EXTENSION, AB}}} \times 100$

  REPRESENTS RELATIVE MUSCULAR FORCES OF OPPOSING MUSCLE GROUPS

- JOINT ANGLE AT PEAK TORQUE (DEGREES)

- WORK PER REPETITION (N-m): THE AVERAGE TIME INTEGRAL OF TORQUE X VELOCITY FOR ALL FLEXION AND EXTENSION MOVEMENTS. INDICATES THE ABILITY OF THE MUSCLE GROUP TO PRODUCE TORQUE (FORCE) THROUGHOUT THE RANGE OF MOTION

- ENDURANCE INDEX (%) = $\frac{\text{WORK PER LAST REPETITION}}{\text{WORK PER FIRST REPETITION}} \times 100$

  INDICATES THE ENDURANCE OF A MUSCLE GROUP

- TOTAL WORK (N-m) = $\Sigma$ WORK/REPETITIONS

Figure 7.– Definitions of isokinetic measurements.

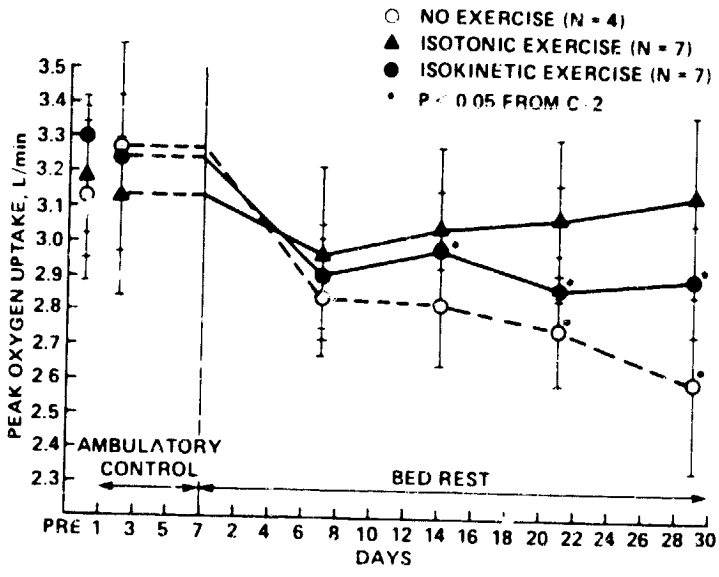



Figure 8.– Work capacity (liters/min).

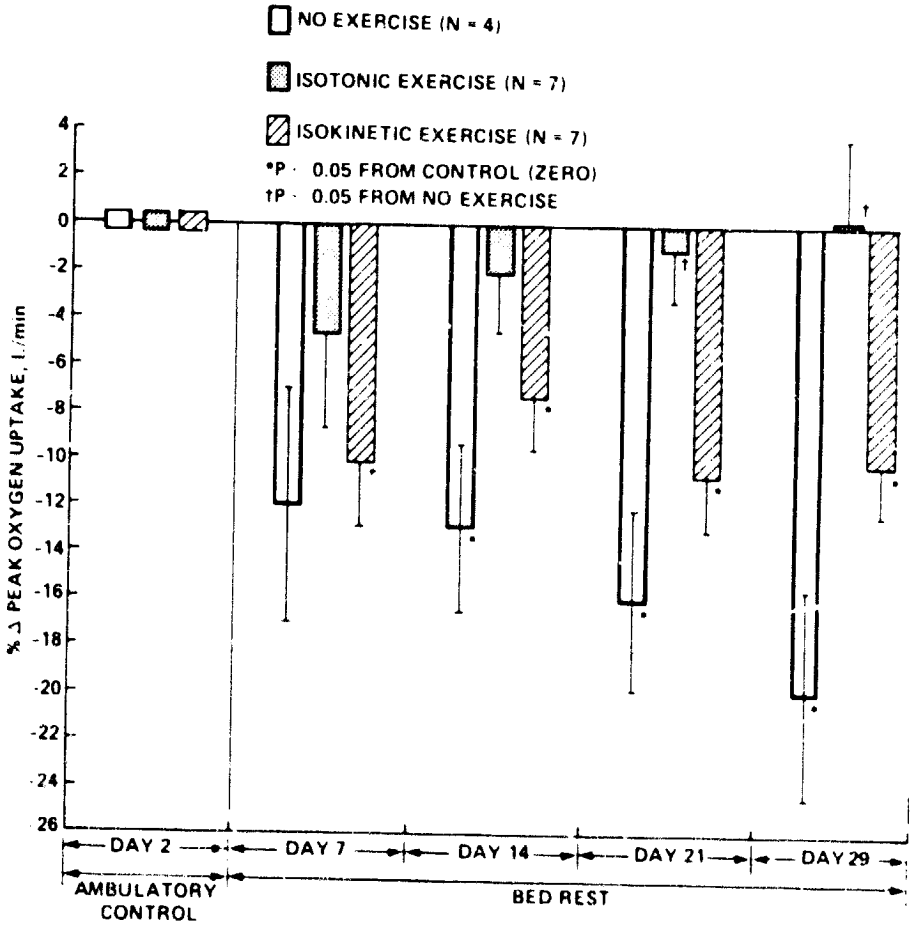



Figure 9.– Work capacity (percent change).

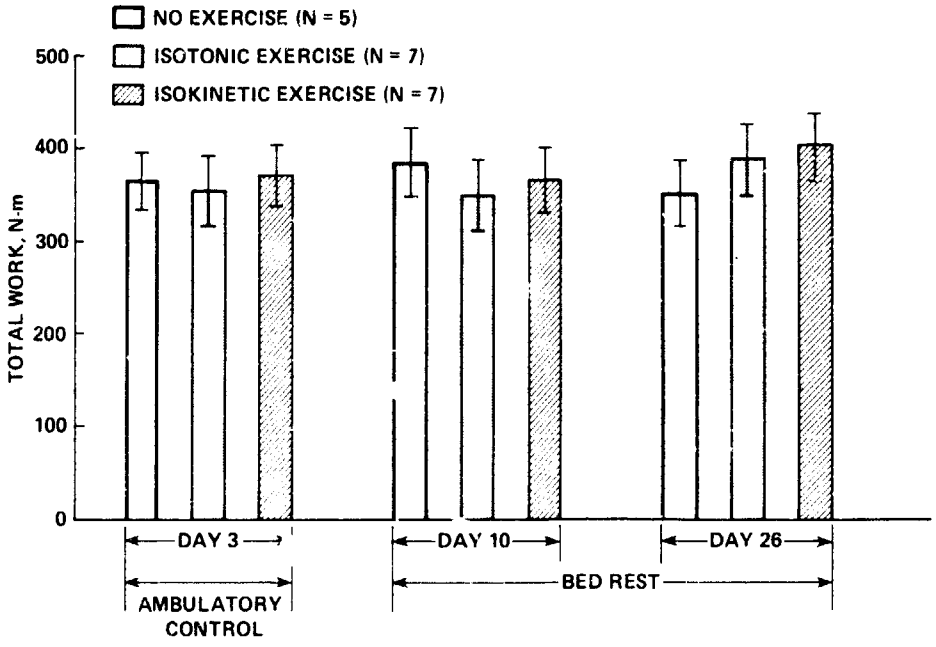



Figure 10.– Total work (arm), $\overline{X}$ right + left + abduction + adduction.

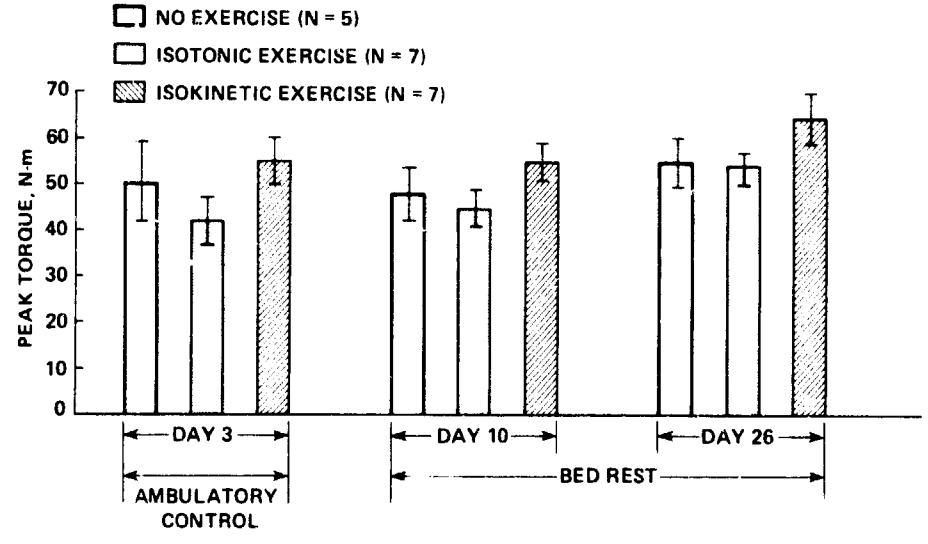



Figure 11.– Total peak torque (arm), $\overline{X}$ right + left + abduction + adduction.

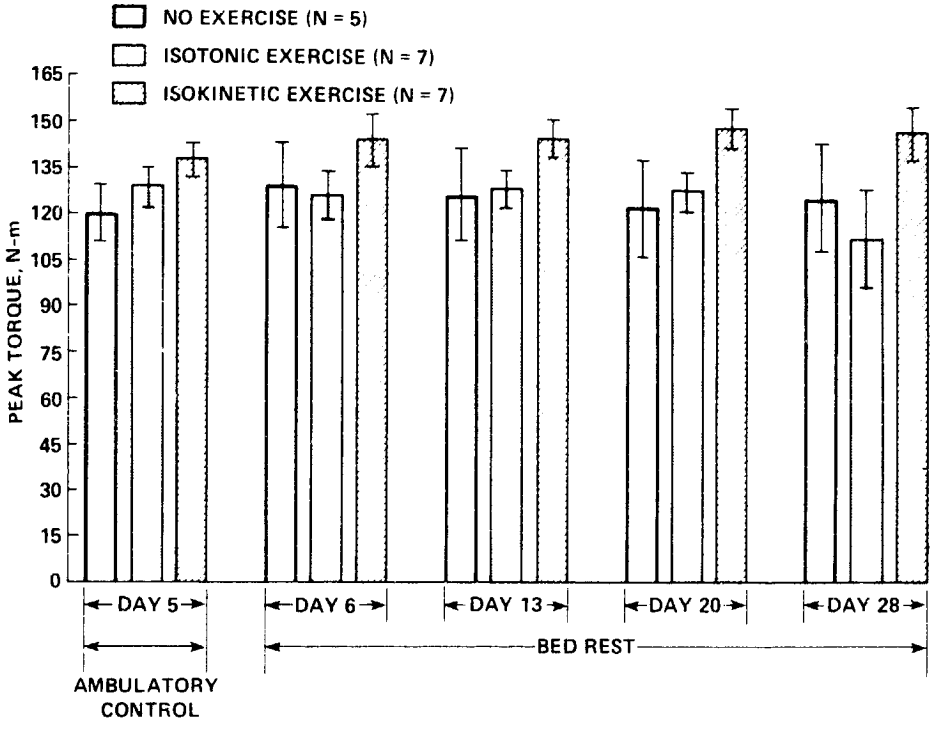



Figure 12.– Total peak torque (leg), $\overline{X}$ right + left + flexion + extension.

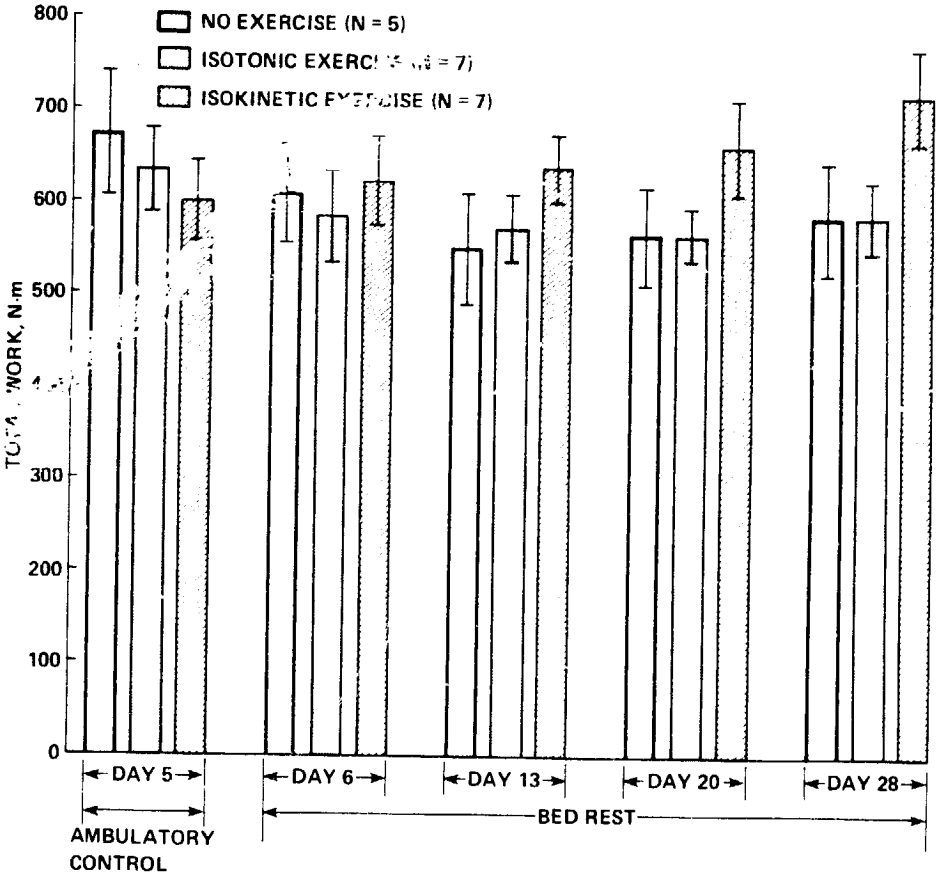



Figure 13.– Total work (leg), $\overline{X}$ right + left + flexion + extension.

# ORTHOSTATIC (TILT-TABLE TOLERANCE)

J. E. Greenleaf and C. E. Wade

## Background

There is some evidence which indicates that endurance-trained athletes have significantly lower orthostatic tolerance (greater tendency to faint in the standing position) than their untrained colleagues (refs. 4-7). There are data from a comparable number of studies which indicate either no change or an increase in orthostatic tolerance in trained athletes (see ref. 8). That trained athletes have exhibited lower orthostatic tolerance may have been a result of the exercise training per se or perhaps of a genetic predisposition. Results from studies where untrained subjects were subjected to short-term, high-intensity training (refs. 8 and 9) or to long-term (6 mo) lower intensity training (ref. 10) indicated essentially no change or, in one case, an increase in tilt-table tolerance after the training. Since moderate training does not result in decreased tilt-table tolerance, we tentatively conclude that the large reduction in tolerance in trained athletes was the result of their long-duration training over years and/or was due to a hereditary predisposition that was coupled with the ability to perform long-term endurance exercise. Thus, tilt-tolerance was measured to determine the effect of isotonic and isokinetic exercise training on orthostasis after bed rest deconditioning.

## Procedure and Methods

The subjects were given two to three practice sessions on the tilt-table. The angle was 60° head-up tilt. A footboard was used which supported about one-third of their weight, and a wide-band over the pelvic region provided additional support. The protocol was 45-min rest in the supine position (control), up to 60-min tilt unless pre-syncopal signs or symptoms occurred, and 15-30 min of recovery in a slight head-down supine position. Heart rate and indirect blood pressure were measured at 2-min intervals for the first 15 min and at 5-min intervals thereafter. Venous blood samples (20 ml each) were taken just before and again at 5 min of tilt from an indwelling Teflon catheter which was placed in the vein at the beginning of the 45-min control period.

Plasma volume was measured during the control period prior to tilting with Evans blue dye (T-1824). Eight milliliters of blood were removed, 5 ml of dye were injected, and 8 ml of blood were removed 10 min after dye injection (all through the same catheter to minimize subject anxiety).

## Results and Discussion

Seven subjects were able to tolerate 60 min of tilt before and after bed rest: 2 no-exercise, 3 isotonic exercise, and 2 isokinetic exercise (fig. 14). One subject (isotonic group) was a fainter; both of his tolerances were less than 3 min. Pre- and post-bed rest systolic and diastolic blood pressures before and during tilt were not significantly different (fig. 15). However, there was a significant increase in heart rates in all three groups at rest and during tilting following bed rest; from about 75 to 120 beats/min at the end of tilt (fig. 16). There were no significant differences in tolerances among the groups before or

after bed rest; tolerance range was 42 to 53 min before and 30 to 34 min after bed rest (fig. 17). But all the post-bed rest tolerances were significantly lower than the pre-bed rest tolerances. Also, there were no significant correlations between tilt tolerances and peak oxygen uptakes before ($r = 0.21$) or after ($r = 0.13$) bed rest.

Thus, when compared with the large reduction in peak oxygen uptake in the no-exercise group (20%), the significant training effect that maintained working capacity in the isotonic group resulted in no specific change in the normal decrease in orthostatic tolerance exhibited by all three groups. So again, a positive training response had no effect upon orthostatic tolerance. It appears that current exercise training regimens should be unrestricted for astronauts who have not previously been highly trained. These findings should be taken into consideration during selection of astronauts and in the type and intensity of the exercise training programs engaged in by those astronauts who have been or are highly trained endurance athletes.

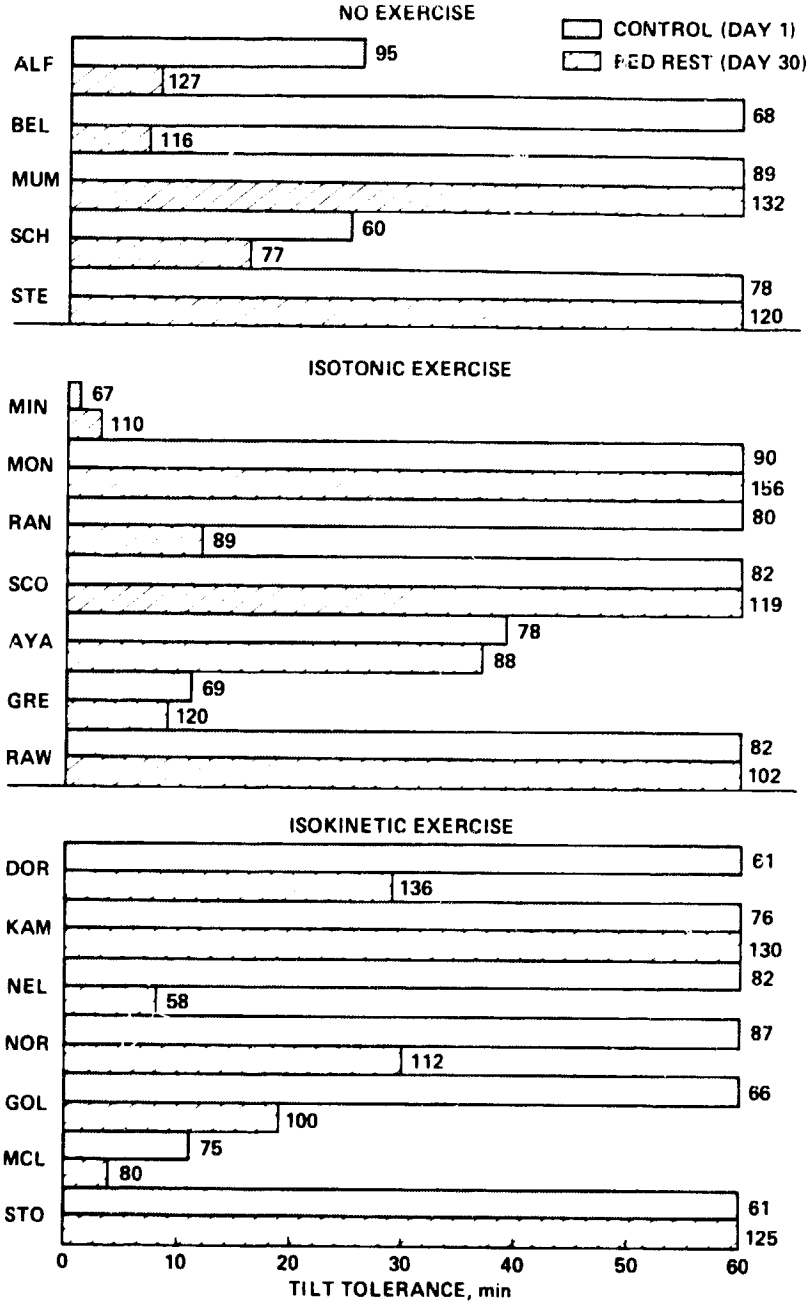



Figure 14.– Orthostatic tolerance and heart rates at tolerance.

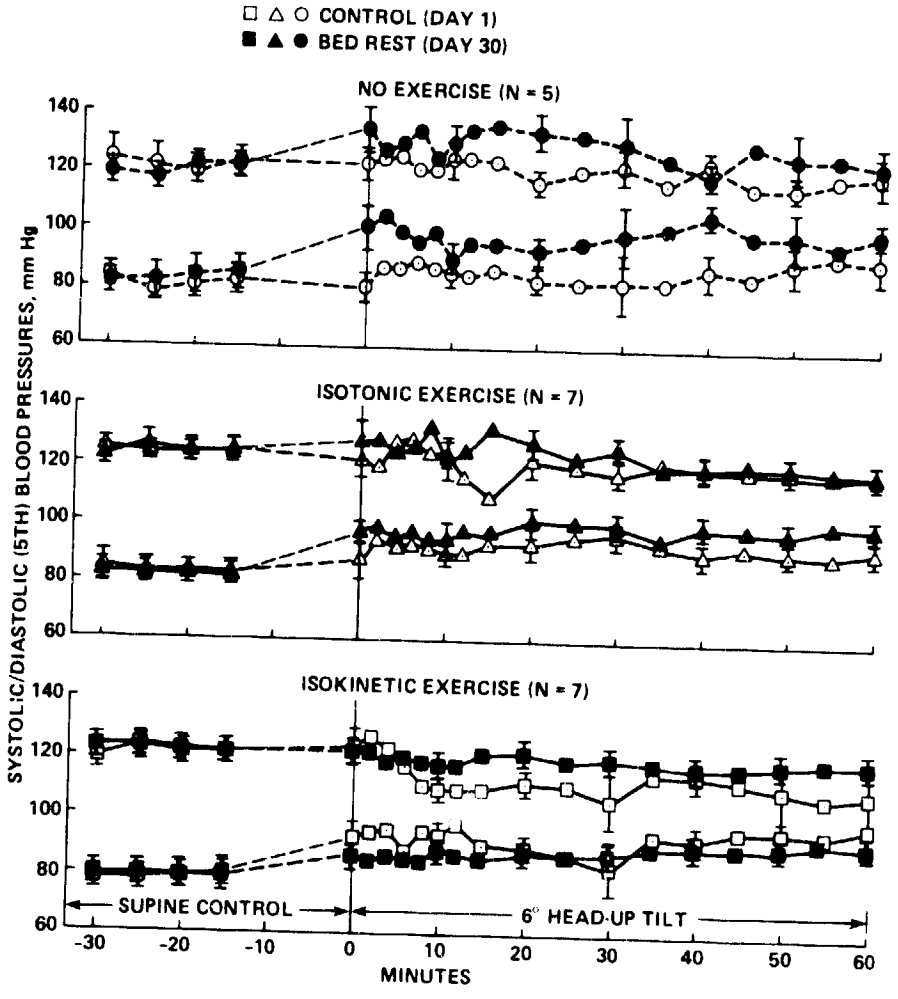



Figure 15.– Orthostasis blood pressures.

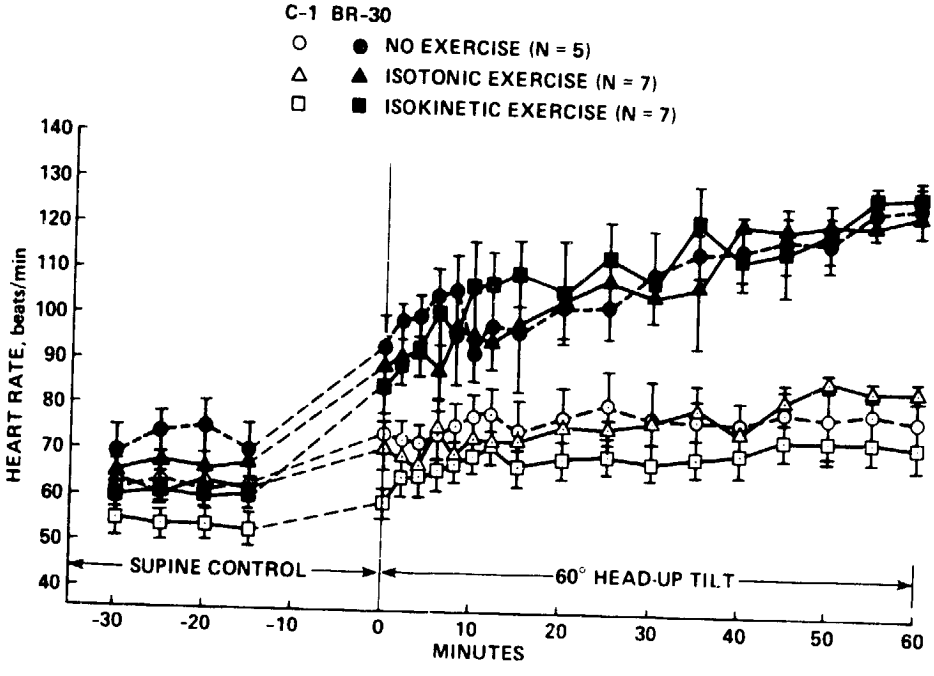



Figure 16.– Orthostasis heart rates.

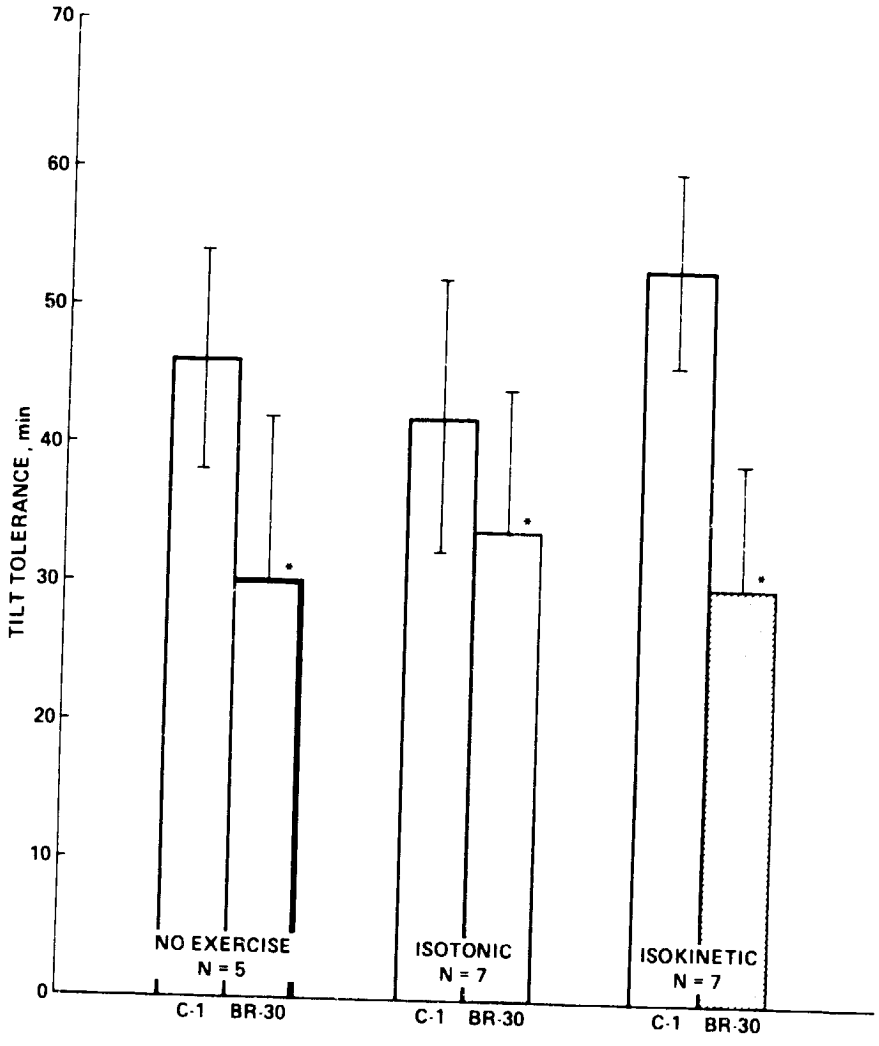



Figure 17.– Orthostatic tolerance.

# PERFORMANCE AND MOOD

C. W. DeRoshia

## Background

Results from Soviet head-down bed rest studies have consistently emphasized the increased levels of sleep impairment and asthenia, and decrements in certain performance tasks (ref. 11). The purpose of this study was (1) to evaluate the effects of head-down bed rest on a variety of carefully selected performance test abilities and mood states, and (2) to test the hypothesis that daily strenuous exercise will counteract the expected deterioration in mood, sleep, and performance tasks sensitive to the bed rest regimen.

## Procedure and Methods

A 15-min microcomputer package (NEC-8201A) consisting of 10 different performance tests (reasoning, code substitution, the Manikin test, pattern comparison, air-combat maneuvering, Sternberg short-term memory, dominant hand tapping, two-finger tapping, nondominant hand tapping, and simple reaction time), a visual analog-scale mood test with eight mood scales, and two sleep-quality scales was administered daily to each subject during the 42-day study. In addition, subject adaptability and bed rest environmental habitability were assessed by investigator-recorded observations, a performance test questionnaire, and a recorded subject interview.

## Results and Discussion

The subjects maintained a relatively stable mood, high morale, and excellent social interactions with the investigators and nursing staff throughout the study. Performance improved in nearly all tests in all groups during bed rest (fig. 18). With mood changes, the isotonic exercise (ITE) group showed significant decreases in psychological tension (TENSE) and sleep problems (SLEEP, WAKE), but they also exhibited deterioration in motivation (MOTIV) and mental concentration (CONCN) relative to responses from the isokinetic (IKE) and no-exercise (NOE) groups (fig. 19). The lower number of subjects in the second testing session (N = 7) resulted in a less crowded, quieter environment with reduced demand upon resources and staff. It was probable that these factors influenced the data, which showed that these seven subjects had more consistent improvement in mood and performance (figs. 20 and 21), higher sleep quality, and fewer expressed complaints than did the 12 subjects in the first session. The overall lack of expected deterioration in sleep quality, task performance, and mood may have been due to (1) optimal subject and nursing staff selection which facilitated excellent social interactions and cooperation, (2) the presence of four subjects who had participated in previous bed rest studies who provided a stabilizing influence, (3) the large number of experimental tests and personal activities which diminished boredom, and (4) daytime naps which may have helped to relieve sleep impairment induced by ambient noise or physical discomfort. The decreases in concentration, motivation, and cognitive performance noted in the isotonic group may have been due to an overtraining-fatigue effect.

It is concluded that the previously reported occurrences of sleep impairment, increased fatigue, irritability, and emotional lability are not normal responses to the bed rest or partial confinement mode per se.

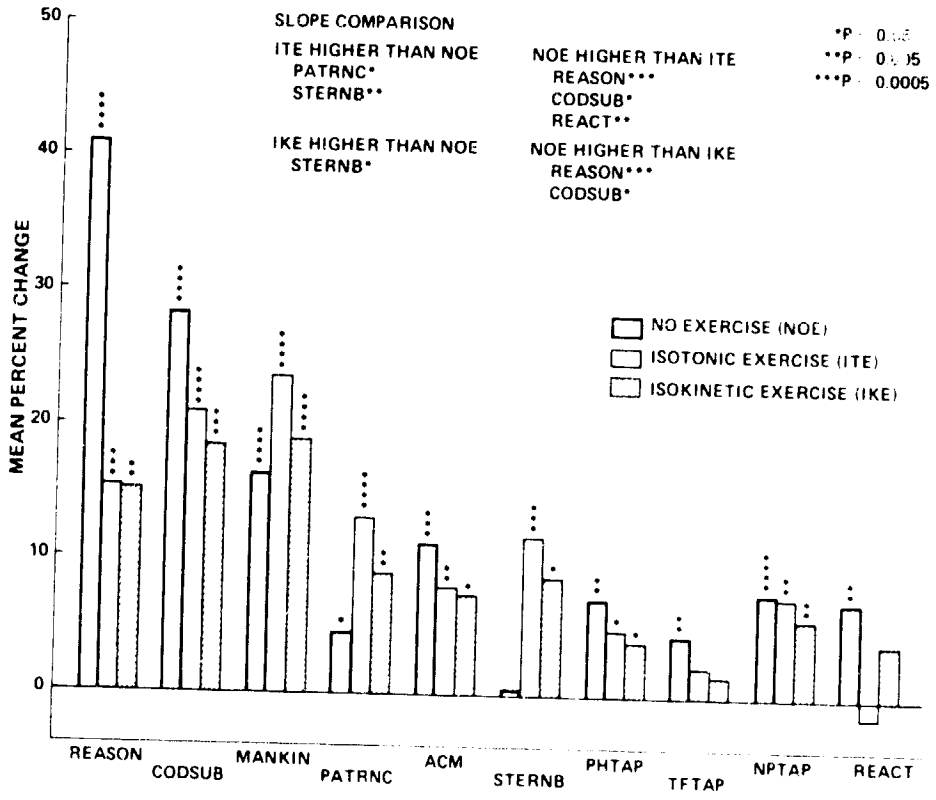
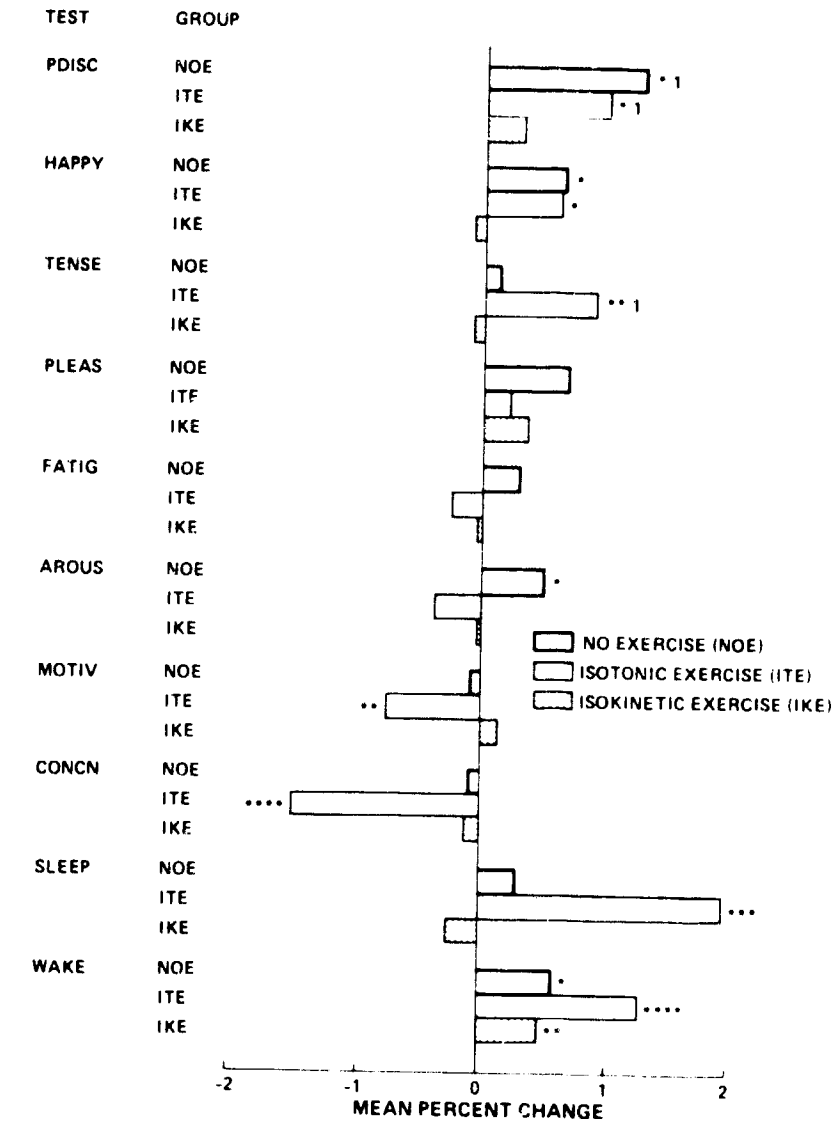



Figure 18.– Performance during bed rest (exercise groups).




*p < 0.05
**p < 0.005
***p < 0.0005
****p < 0.00005
(1) RESIDUALS FROM LINEAR REGRESSION WERE NONRANDOM

SLOPE COMPARISON

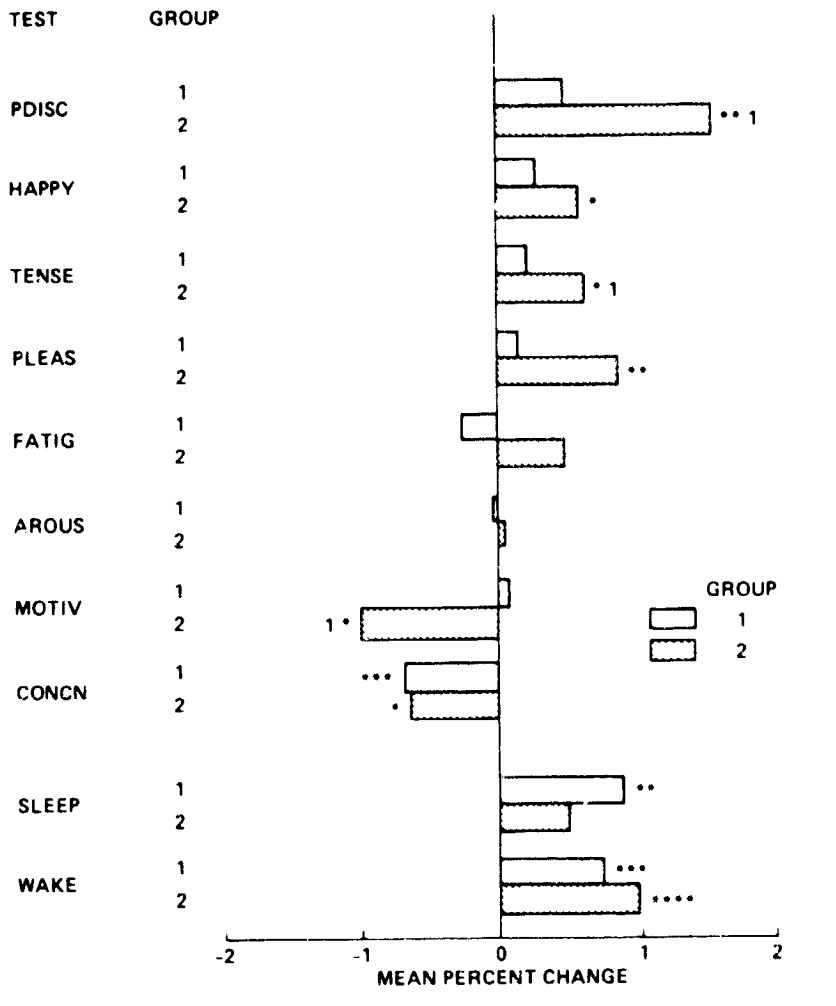
ITE HIGHER THAN NOE
TENSE**
SLEEP**
WAKE*

ITE HIGHER THAN IKE
SLEEP***
WAKE*

NOE HIGHER THAN ITE
AROUS*
CONCN****

IKE HIGHER THAN ITE
MOTIV**
CONCN****

Figure 19.– Mood change during bed rest (exercise groups).




*p < 0.05
**p < 0.005
***p < 0.0005
****p < 0.00005
(1) RESIDUALS FROM LINEAR REGRESSION WERE NON RANDOM

SLOPE COMPARISON
GROUP 2 HIGHER THAN GROUP 1
FATIG*
PDISC*
PLEAS*
GROUP 1 HIGHER THAN GROUP 2
MOTIV**

Figure 20.– Mood change during bed rest study (groups I and II).

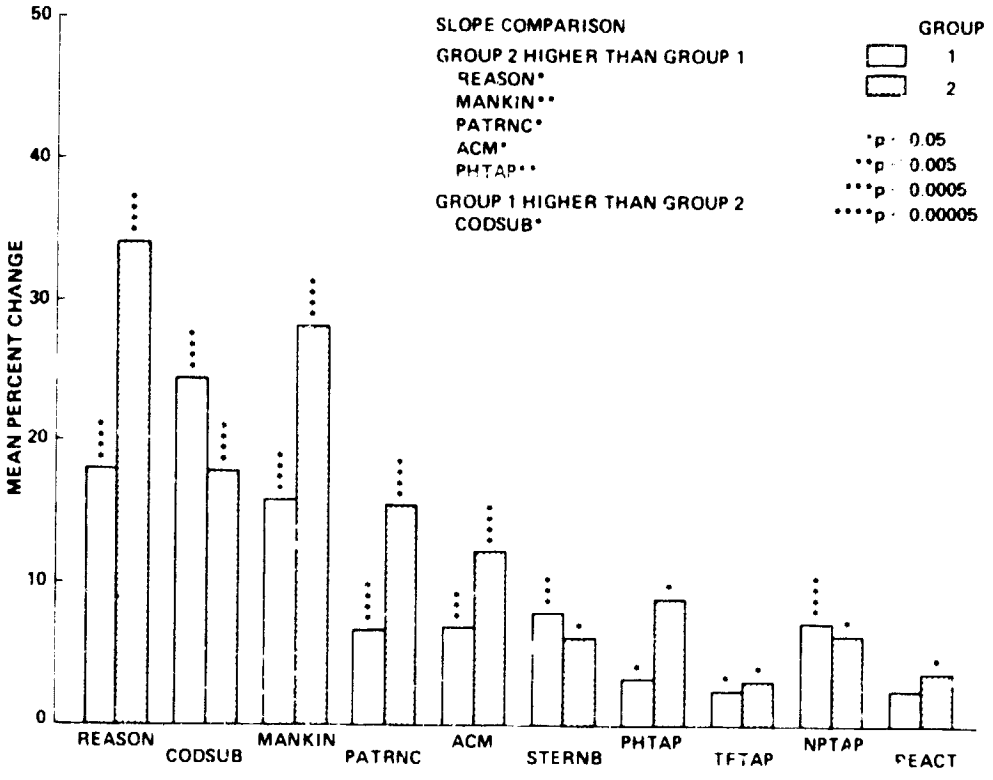



Figure 21.– Performance measures during bed rest study (groups I and II).

# POSTURE, EQUILIBRIUM, AND GAIT

M. M. Cohen

## Background

Upon landing after orbital missions, the ability of astronauts to locomote is frequently compromised by an impairment of gait, postural equilibrium, and balance. It has generally been assumed that these deficits result from a reinterpretation of otolith organ inputs during exposure to microgravity, although several other possibilities also exist. For example, the deficits could be due to weakened leg muscles resulting from muscle atrophy, to reduced blood flow to the brain from cardiovascular deconditioning, or to altered neuromuscular control resulting from lack of motor activity of the relevant postural muscles. Some of these alternatives were studied in bed-rested subjects where the vestibular effects of exposure to microgravity are eliminated, although effects of muscle atrophy, altered blood flow to the brain, and degraded neuromuscular control may remain. Results of the different types of exercise training were analyzed to determine their effectiveness in preventing the atrophic and other deconditioning effects of bed rest. Since independent measures of aerobic work capacity, via changes in peak oxygen uptake (indicators of cardiovascular and metabolic changes) and changes in muscle strength and size (partial indicators of muscle atrophy), were used to evaluate the efficacy of the two exercise regimens, we examined the hypothesis that postflight alterations of posture and equilibrium are due to factors other than a reinterpretation of neural inputs to the otolith organs. The purpose was (1) to examine the influence of the two types of exercise training in preventing or ameliorating changes in posture, equilibrium, and gait (PEG) after bed rest deconditioning, and (2) to test the hypothesis that factors other than a reinterpretation of inputs to the otolith organs influence post-bed rest changes in PEG since the inputs are probably not altered during bed rest as they are during exposure to microgravity.

## Procedure and Methods

Tests were conducted on pre-bed rest days 1 and 6, and recovery days 1 and 4. At the end of bed rest, the tilt-table test was performed on day 30, one day prior to PEG testing. In four separate 20-min sessions, each subject was tested for his ability to maintain posture and equilibrium, and to locomote a specified course. For the posture and body equilibrium tests, each subject was required to stand with his arms folded over his chest and his feet were placed on a force platform stabilometer where measures of body sway were obtained. An ABCDE/EDCBA design was used. Conditions were as follows:

| | A | B | C | D | E |
|---|---|---|---|---|---|
| Eyes: | open | closed | open | closed | open |
| Target: | fixed | fixed | fixed | fixed | moved |
| Platform: | fixed | fixed | moved | moved | fixed |

Immediately following the initial tests on the stabilometer, each subject was required to walk along a standardized, prescribed course while measures of locomotive ability and gait were obtained. Electromyogram (EMG) signals from the anterior tibialis and gastrocnemius-soleus muscles and signals from

foot switches indicated contact with the ground, swing/stance phases of the gait, and timing for each step. Ink pads placed on the soles were used to record the path of motion, including step width, step length, and walking velocity (fig. 22).

## Results and Discussion

Data were analyzed for amplitude of body sway as a function of the exercise training conditions. The dispersion index, representing the standard deviation of 400 samples of the body's center of gravity (a measure of postural instability) increased by approximately 20% following the 30-day bed rest period, and returned to pre-bed rest values by the fourth day of recovery. The increase of postural instability due to bed rest was not a function of either exercise training condition. On the initial walking attempts following the 30-day bed rest period, there were significant decreases in step length, stride length, and walking velocity (figs. 23-25). These changes were not a function of the exercise training regimens. By the fourth day of recovery, pre-bed rest values were restored. No other significant changes were observed. Thus, while the bed rest deconditioning resulted in deterioration of body stability and gait parameters, neither intensive isotonic nor isokinetic exercise training during bed rest had a significant influence on posture, equilibrium, or walking gait measurements.

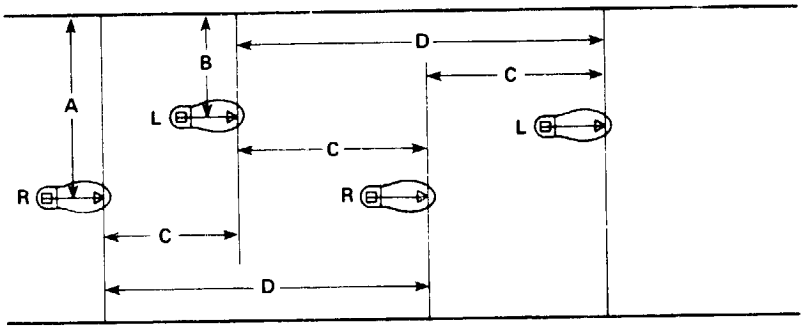



Figure 22.– Gait measures.

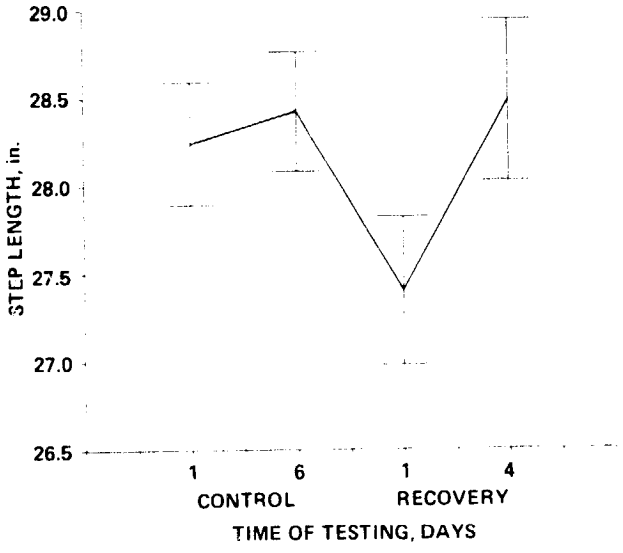



Figure 23.– Step length.

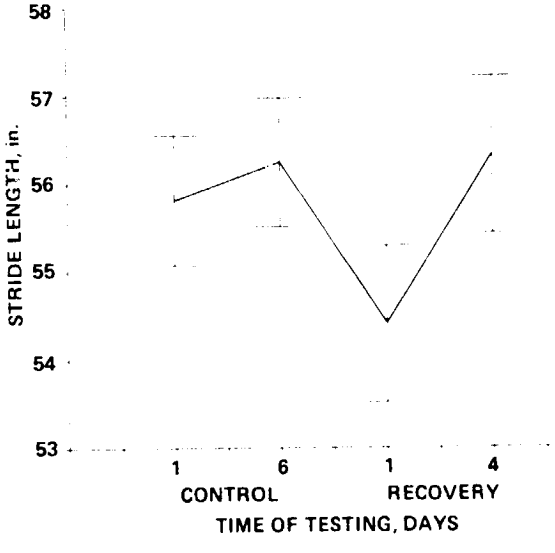



Figure 24.– Stride length.

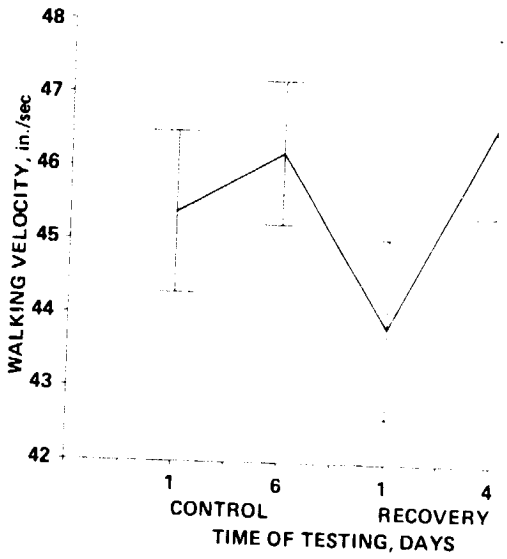



Figure 25.– Walking velocity.

# BONE, CALCIUM, AND MUSCLE METABOLISM

S. B. Arnaud, M. Powell, R. Marcus, P. Berry, B. Silver, and B. Harris

## Background

There is histologic and biochemical evidence that bed rest deconditioning produces deficits in bone mass because of reduced synthesis of bone associated with either normal (ref. 12) or increased resorption (ref. 13). A new biochemical marker for these bone changes is a noncollagenous bone-specific protein which is incorporated into the hydroxyapatite crystal of bone and synthesized by the osteoblast (ref. 14). Blood concentration of this bone-specific protein reflects the combined resorptive and formative activities of bone metabolism. Bone turnover in patients with post-menopausal osteoporosis was detected as early as 2 wk by radioimmunoassay of this protein (ref. 15). Plasma concentrations of bone-specific protein are low in hypoparathyroid patients and high in hyperparathyroid patients, consistent with the known effects of parathyroid hormone on bone turnover. Measurements of parathyroid hormone in blood of bed rest patients have not followed a consistent pattern and have raised questions concerning the specificity of the analysis (ref. 16). A nonspecific product of bone cell activity, which is synthesized by the osteoblast in response to mechanical stimuli, is prostaglandin $E_2$ (ref. 17). Increased concentrations of prostaglandin $E_2$ occur in the bones of patients with senile osteoporosis (ref. 18) and stimulate bone formation in infants to accelerate closure of the patent ductus arteriosus (ref. 19). These various analyses for estimating changes in bone turnover were performed in this study.

The early biochemical measurements of possible changes in skeletal metabolism mentioned above that occur during bed rest are followed by reduced mineral content in the calcaneous after 6 wk of rest (ref. 20), and in the lumbar spine after 4 wk of rest (ref. 21), as measured by photon absorptiometry. The relatively high proportion (75%) of trabecular bone in the vertebrae probably accounts for the greater sensitivity of vertebrae for early bone loss (ref. 22). Trabecular bone structure has a higher surface-to-volume ratio than cortical bone and may more likely be affected by metabolic perturbations associated with diet and exercise. Whether habitual patterns of either exercise or dietary mineral intake are important influences on the rate of demineralization of skeletal mass during bed rest is an unresolved issue (ref. 23).

The purpose was to evaluate the effect of isotonic and isokinetic exercise training during bed rest on bone turnover, cell calcium content, and muscle intermediary metabolism, and to test the hypothesis that the bed rest-induced decrease in bone turnover will be prevented by the daily exercise training.

## Procedure and Methods

Bone mineral content (density) was measured in the second to fourth lumbar vertebrae by dual photon and in the radius by single photon absorptiometry on pre-bed rest day 7 and on post-bed rest day 2. Twenty-four-hour urine collections were done on pre-bed rest day 3, and bed rest days 4 and 26. These were followed by renal clearance tests on pre-bed rest day 4, and bed rest days 5 and 27. Bone resorption was measured by 24-hr urine calcium and urine hydroxyproline concentrations and serum

parathyroid hormone concentrations. The rate of bone formation was estimated from the serum osteocalcin concentration.

Sublingual epithelial cell calcium content was analyzed by first visualizing the cells with a scanning electron microprobe and then the mineral content was determined by energy dispersive X-ray analysis. The intracellular ion concentration was measured from the X-ray fluorescent pattern generated by the electron excitation of the calcium ions and expressed in EXA units:

$$\text{EXA units} = \frac{\text{X-ray intensity (peak/background)}}{\text{unit cell vol } (800u^3)}$$

Serum-ionized calcium was measured with the Radiometer Ionized Calcium Analyzer (Model ICA-1).

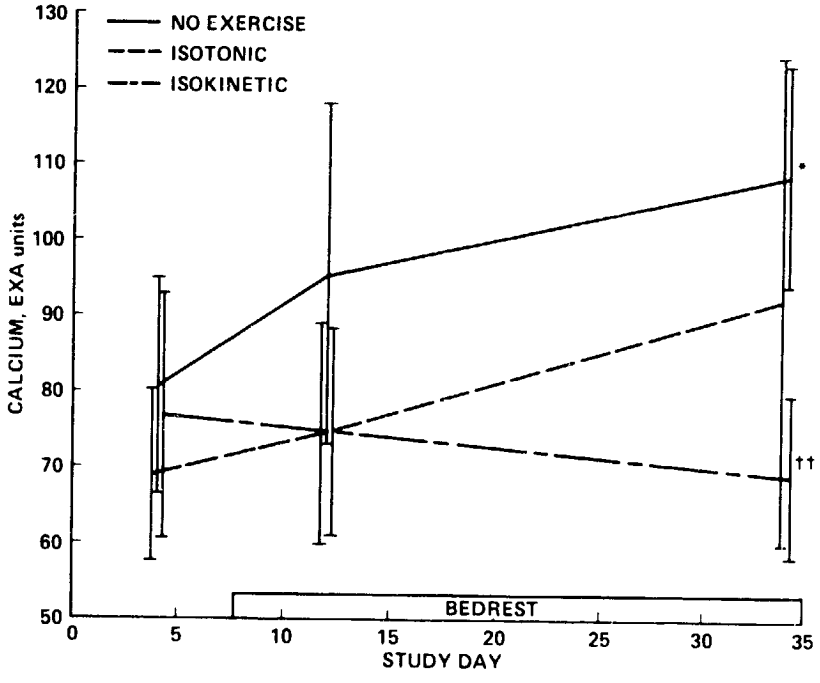
Intermediary metabolism of forearm and calf muscles at rest and during isometric contractions before and during bed rest was estimated by P-31 magnetic resonance spectroscopy. P-31 spectra were obtained at 2 Tesla (34.6 MHz) from a 25-cm bore superconducting magnet (General Electric CS1) located at the Magnetic Resonance Spectroscopy Laboratory, Department of Radiology, University of California at San Francisco. To allow for forearm and calf muscular contraction during spectra measurement, a wooden bench was constructed to fit inside the spectrometer. A pedal, under tension, was used to provide resistance for foot plantar flexion and wrist flexion. The nondominant forearm and the contralateral leg were measured.

## Results and Discussion

There were no significant changes in vertebrae or radial bone densities in any group after 30 days of bed rest. The indices of calcium and skeletal metabolism showed essentially no significant changes. One subject had a 10% increase and another a 7.1% decrease in lumbar spine density. Following bed rest there was a modest increase in serum-ionized calcium concentration (fig. 26) which may have been related to a slight decrease in venous blood pH. There was no change in serum osteocalcin concentration.

Total sublingular intracellular calcium concentration increased significantly in the no-exercise group, and was unchanged in the two exercise groups (fig. 27). Changes in intracellular calcium concentration paralleled the increases in intracellular phosphorus in all subjects. The changes in intracellular calcium were related to changes in intracellular phosphorus, sodium, potassium, and total calcium, but not to serum-ionized calcium. The parallel increases in both the intracellular Ca and P concentrations suggest the accumulation of calcium-phosphorus complexes within the cell. The close association between the change in intracellular Ca and P concentrations suggest the accumulation of calcium-phosphorus complexes within the cell. The close association between the change in intracellular Ca and the other ions indicated that fluid shifts were probably the cause for the increased intracellular calcium concentration. This method for measuring intracellular calcium in sublingual cells is a practical and sufficiently sensitive method for monitoring early metabolic changes that may precede actual bone demineralization during bed rest and possibly during spaceflight.

The P-31 magnetic resonance spectroscopy procedure detected differences in phosphorus, phosphocreatine, and pH, but not ATP, during five 3-min isometric exercise bouts performed at half maximum force; bouts were separated by 2-min rest periods. The isotonic and no-exercise training groups exhibited higher phosphorus/phosphocreatine ratios with leg exercise when compared with the isokinetic group. There was no significant change in any parameter, including pH and ATP, in any group during bed rest.



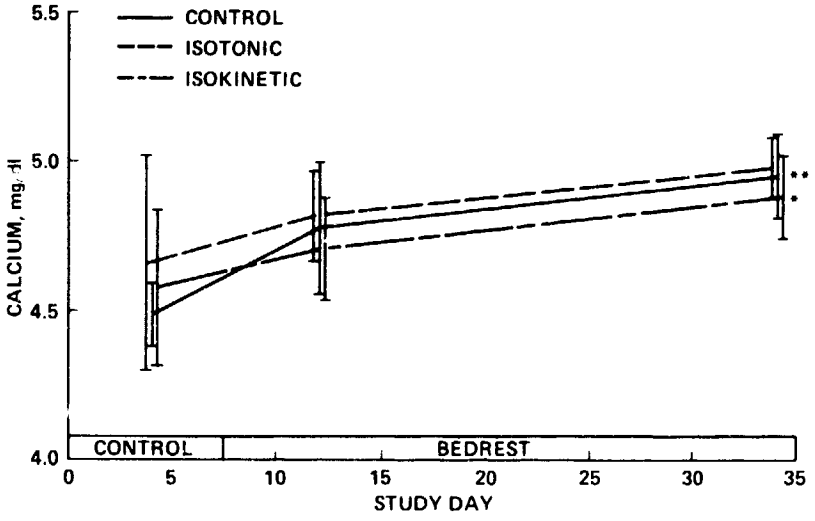

INTRACELLULAR CALCIUM
(EXA UNITS)

| | | STUDY DAY | | |
|---|---|---|---|---|
| GROUP | N | DAY 4 | DAY 12 | DAY 34 |
| NO EXERCISE | 5 | 80.8 ± 14.2 | 95.6 ± 22.4 | 108.6 ± 14.6* |
| ISOTONIC | 7 | 69.0 ± 11.3 | 74.6 ± 14.6 | 92.1 ± 32.2 |
| ISOKINETIC | 7 | 76.9 ± 16.1 | 74.9 ± 13.7 | 68.9 ± 10.7†† |

COMPARED TO PRE-BED REST
*$p < 0.05$
**$p < 0.01$

COMPARED TO NO EXERCISE
†$p < 0.05$
††$p < 0.01$

Figure 26.– Effects of exercise and no exercise on intracellular calcium.




SERUM IONIZED CALCIUM
(mg/dL)

STUDY DAY

| GROUP | N | DAY 4 | DAY 12 | DAY 34 |
|---|---|---|---|---|
| NO EXERCISE | 5 | 4.49 ± 0.11 | 4.78 ± 0.22 | 4.95 ± 0.14** |
| ISOTONIC | 7 | 4.66 ± 0.36 | 4.82 ± 0.15 | 4.98 ± 0.10 |
| ISOKINETIC | 7 | 4.58 ± 0.26 | 4.71 ± 0.17 | 4.88 ± 0.14* |

COMPARED TO CONTROL
*$p < 0.05$
**$p < 0.01$

COMPARED TO NO EXERCISE
$^{\dagger}p < 0.05$
$^{\dagger\dagger}p < 0.01$

Figure 27.– Serum ionized calcium during control and bed rest periods.

# MUSCLE MAGNETIC RESONANCE IMAGING

S. Ellis, P. Lee, and R. Selzer

## Background

Skeletal muscles which have been load-free for extended periods undergo reductions in muscle mass and strength roughly in proportion to the duration of unloading. This effect has been described in individuals whose limbs have been immobilized by casting. Removal of the cast after several weeks of immobilization reveals a striking reduction in limb girth and strength, particularly in lean individuals. The atrophic changes are ameliorated after several weeks of normal activity. Sargeant et al. (ref. 24) found that leg casting for 131 days produced a 12% reduction in total leg volume relative to the non-casted leg. However, the reduction in muscle cross-sectional area was much greater as indicated by the decreases in the areas of type I (slow twitch) and type II (fast twitch) muscle fibers, which were reduced by 46% and 37%, respectively. Thus, limb girth measurements are not always a reliable indicator of the actual changes in muscle size. It is anticipated that isokinetic leg exercise would be the most effective for maintaining muscle mass. This hypothesis is supported by results from studies which indicated that the largest relative muscle areas were found in heavy weight lifters (203 and 270 $cm^2$ total muscle area at mid-thigh), whereas those people who engaged in endurance training had muscle areas of the same size as untrained and previously sedentary individuals (128, 150, and 151 $cm^2$) (ref. 25). Haggmark and Eriksson (ref. 26) measured cross-sectional areas of muscles after 6 wk of casting following Achilles tendon rupture; calf muscle area was reduced by 11%, but the area of the soleus and gastrocnemius was reduced by 23%, and mean fiber area was decreased by 25% in the soleus. It is our supposition that changes in cross-sectional area and volume of the total muscle as well as of individual muscles can be most accurately measured by quantitative magnetic resonance (MR) imaging. Moreover, this technique should be highly useful for evaluating the extent of muscle atrophy sustained during bed rest and after extended weightlessness, as well as for monitoring the efficacy of exercise training regimens. Although MR imaging is being used extensively for diagnosing anatomical malformations in organs, the quantitative measurement of changes in muscle dimensions has yet to be developed into a reliable technique. The measurement of cross-sectional areas should present no major difficulty since good results were obtained using computerized X-ray tomography (ref. 27). This bed rest study offered the necessary subject population for the development of this technique and its application to the problem of weightlessness-induced muscle atrophy.

The purpose of this study was to employ quantitative magnetic resonance imaging to measure the cross-sectional areas and the volumes of selected leg muscles of human subjects who have undergone 30 days of bed rest with and without exercise training.

## Procedure and Methods

Magnetic resonance images were taken at the University of California-San Francisco Radiology Imaging Laboratory. The subjects were scanned on pre-bed rest day 5 and recovery day 3. Limousines were used to transport the subjects between Ames Research Center and the UCSF-RIL.

The 19 subjects were imaged in a Diasonics MT/S imager operating at a field strength of 0.35 Tesla. All imaging was done in a quadrature detection head coil which gave an inplane resolution of 0.95 × 0.95 mm. The imager did not have sufficient field of view to allow the entire calf to be imaged at once, so it was necessary to image the leg in three 20-cm-long sections with the leg translated 18 cm each time. The standard protocol was a spin echo image with TR = 1.0 sec and TE = 30 ms. In addition, TR = 0.5 sec images were taken at the middle position to provide for T1 information. The same protocol was used for the pre- and post-bed rest measurements.

A lucite leg rest was made to facilitate the 18-cm translation of the subjects. The foot of the subject was taped to the leg rest and three fiducial marks on the leg rest provided for proper positioning for the pre- and post-bed rest measurements. To monitor changes in the MR imager, four lucite tubes filled with oil were used as phantoms.

The images were recorded on magnetic tapes which were sent to the Jet Propulsion Laboratory. The tapes were then transformed to the MINI-VICAR format and cataloged. The 2660 cross-sectional images were each enlarged and filtered to give the basic images for computer analysis (fig. 28).

As a first step in the computer analysis, an edge-detection program was used to outline the total muscle. The resulting area included everything except the subcutaneous fat. As a first-order correction, the areas of the tibia and the fibula were delineated and subtracted from the total area. The image quality was such that the boundaries for the soleus and the gastrocnemius were ambiguous in the middle sections of the leg. So the group of muscles posterior to the tibia, the fibula, and their connecting interosseous membrane were outlined. The posterior compartment group consisted of the following muscles: soleus, lateral and medial gastrocnemius, flexor digitorum longus, tibialis posterior, and the flexor hallucis longus. The computer edge-detection program proceeded in a semiautomatic fashion with operator intervention at marginal areas.

## Results and Discussion

The soleus and gastrocnemius muscles for subject 409 were traced manually. The muscles separate at the two ends. However, the middle sections were sometimes ambiguous and difficult to delineate. Typical examples from each of the three positions are shown in figure 28–upper. After a sequence of outlines was found, the outlines were combined into a three-dimensional (3-D) shaded surface display where the locations of the light source and the observer could be varied at will. The volume of a particular muscle or muscle group was calculated after appropriate smoothing. Examples of 3-D displays for the soleus and the gastrocnemius muscles are shown in figure 28–lower.

As a monitor of the imaging conditions at the pre- and post-bed rest studies, the separations between the centers of the oil-filled tubes were measured (fig. 28–upper). The separations for tubes 1 and 4 in pixel units are:

| | Pre-bed rest | Post-bed rest |
|---|---|---|
| Position 1 | 209.8 ±2.3 | 209.4 ±1.2 |
| Position 2 | 207.1 ±1.3 | 207.9 ±1.5 |

All these values agree to within 1%.

Table 1 presents the ratio of the total area of the posterior compartment muscle groups measured pre- and post-bed rest for the three exercise groups. The post-bed rest volumes were: no exercise 93.7%, isotonic exercise 95.7%, and isokinetic exercise 92.3%. The mean percentage change was 93.9%. No difference was statistically significant, but only subject MIN had an apparent increase in muscle area.

Because of the enormous quantity of data involved, the image processing is being done in increasing levels of sophistication. Further refinement will be done to smooth out gaps and rough edges. Only the TR = 1.0 sec images have been used. The TR = 0.5 sec images can be analyzed to give T1 values.

Thus, the mean change in cross-sectional area of the posterior leg and thigh muscles was –6%. There was no influence of either isotonic or isokinetic leg exercise training on this decrease in muscle area.

With improvements in resolution, magnetic resonance imaging and the computer analysis will be a viable method to estimate volumes of individual muscles quantitatively.

TABLE 1.– TOTAL AREA OF THE POSTERIOR LEG AND THIGH MUSCLES PRE- AND POST-BED REST (PIXEL UNITS)

| Subject | Pre | Post | Post/pre ×100, % |
|---|---|---|---|
| No exercise | | | |
| ALF | 457,400 | 441,972 | 96.6 |
| BEL | 625,509 | 574,886 | 91.9 |
| MUM | 500,720 | 462,181 | 92.3 |
| SCH | 458,663 | 429,955 | 93.7 |
| STE | 640,100 | 601,956 | 94.0 |
| $\bar{X}$ | 536,478 | 502,190 | 93.7 |
| ±SD | 89,792 | 80,129 | 1.8 |
| ±SE | 40,156 | 35,834 | 0.8 |
| Isotonic exercise | | | |
| MIN | 699,199 | 717,271 | 102.6 |
| MON | 512,305 | 466,321 | 91.0 |
| RAN | 586,837 | 548,913 | 93.5 |
| SCO | 391,390 | 389,348 | 99.5 |
| AYA | 470,062 | 429,770 | 91.4 |
| GRE | 583,301 | 550,687 | 94.4 |
| RAW | 526,694 | 514,507 | 97.7 |
| $\bar{X}$ | 538,541 | 516,688 | 95.7 |
| ±SD | 97,740 | 107,036 | 4.3 |
| ±SE | 36,942 | 40,456 | 1.6 |
| Isokinetic exercise | | | |
| DOR | 632,374 | 586,170 | 92.7 |
| KAM | 514,331 | 503,787 | 97.9 |
| NEL | 542,297 | 461,375 | 85.1 |
| NOR | 583,010 | 516,086 | 88.5 |
| GOL | 560,310 | 520,152 | 92.8 |
| MCC | 614,370 | 591,050 | 96.2 |
| STO | 577,311 | 537,626 | 93.1 |
| $\bar{X}$ | 574,858 | 530,892 | 92.3 |
| ±SD | 40,573 | 45,865 | 4.4 |
| ±SE | 15,335 | 17,336 | 1.6 |

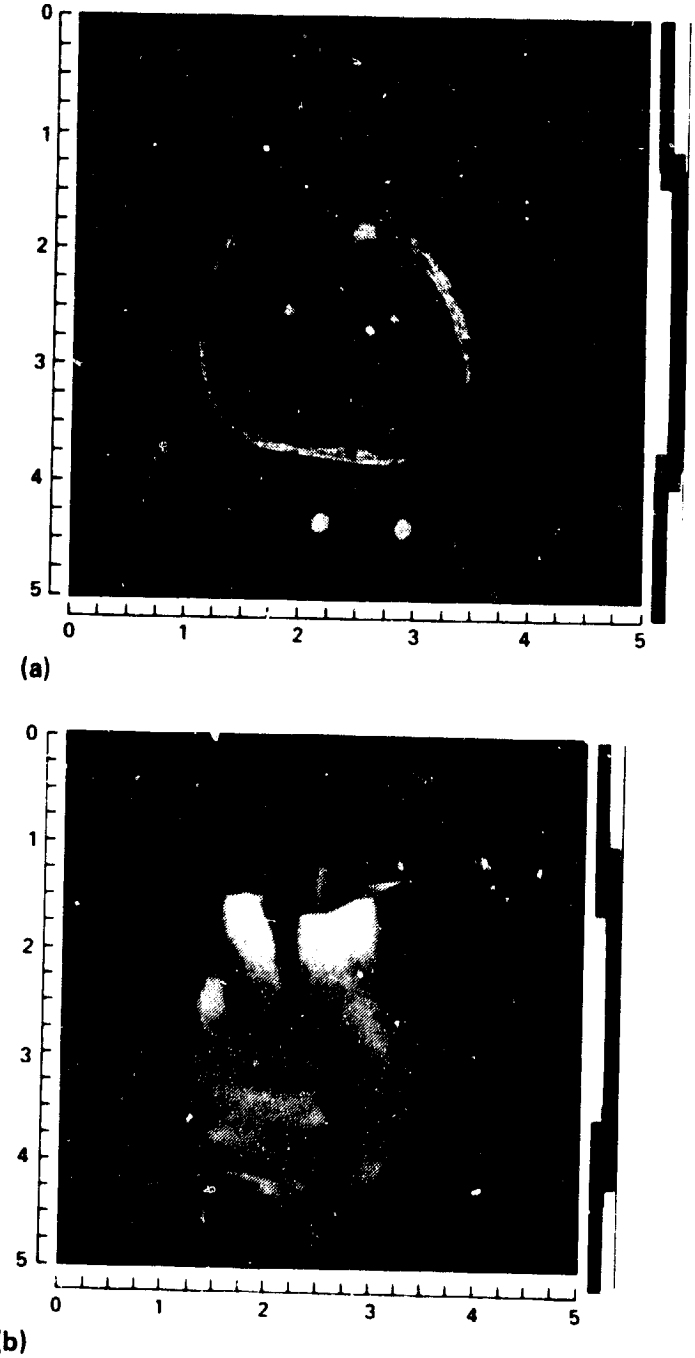



Figure 28.– MR images of calf and middle leg muscles. (a) Cross section of the calf taken about 20 cm above the ankle. The soleus and the two parts of the gastrocnemius are outlined. (b) Three-dimensional shaded surface reconstruction of the middle leg. The soleus muscle is hidden in this view.

# MUSCLE ULTRASOUND

S. Ellis and L. C. Kirby

## Background

Leg muscle wasting is a well-known effect of disuse, but the rate at which it occurs and the effectiveness of various types of exercise in forestalling the atrophy has never been measured quantitatively. Computerized axial tomography (CT), magnetic resonance imaging (MR), and ultrasonic imaging are some techniques for measuring skeletal muscle cross-sectional area or thickness. The first and second methods are limited by radiation hazard and size and weight of the equipment, respectively, as well as cost. Ultrasound, on the other hand, is an innocuous technique which can yield estimates of muscle thickness with relatively simple and portable equipment which can be readily adapted for Shuttle or space station use.

There have been no longitudinal studies where the decrease in thigh and calf muscle thickness over a period of disuse of 30 days has been measured. That changes in muscle thickness can be detected by ultrasound has been demonstrated by several investigators (refs. 28-30), but these studies were semi-quantitative in nature. Recently a quantitative method for measuring leg muscle thickness and cross-sectional areas with a high degree of accuracy and repeatability has been described (ref. 31).

The purpose was to measure the rate at which the thickness of the thigh and calf muscles decrease (atrophy) during bed rest, and to determine the effectiveness of isotonic and isokinetic exercise training during bed rest to attenuate the decrease in muscle thickness.

## Procedure and Methods

Ultrasonic measurements of muscle thickness were taken (B-MODE, 7.5 MHz) on pre-bed rest day 3, at weekly intervals during bed rest (days 2, 9, 16, 23, and 29), and on recovery day 4. Measurement locations were marked on the skin. Pressure of the ultrasonic probe on the skin is critical for reproducible measurements. The subjects were measured in standardized prone and supine positions. Muscles measured were the anterior thigh (quadriceps), posterior thigh (hamstrings), and posterior leg (gastrocnemius, soleus, and flexor hallucis longus).

## Results and Discussion

There was a significant decrease in thickness in the soleus-flexor hallucis longus (SOL + FHL) muscle group after bed rest that was not attenuated by exercise training (fig. 29). The thickness of the rectus femoris (RF) was unchanged in all groups during bed rest (figs. 29 and 30). The vastus intermedius (VI) showed substantial atrophy in the isokinetic and no-exercise groups, but was unchanged in the isotonic group (fig. 31).

It is concluded that ultrasound can be used successfully to detect changes in muscle thickness in leg muscles during bed rest.

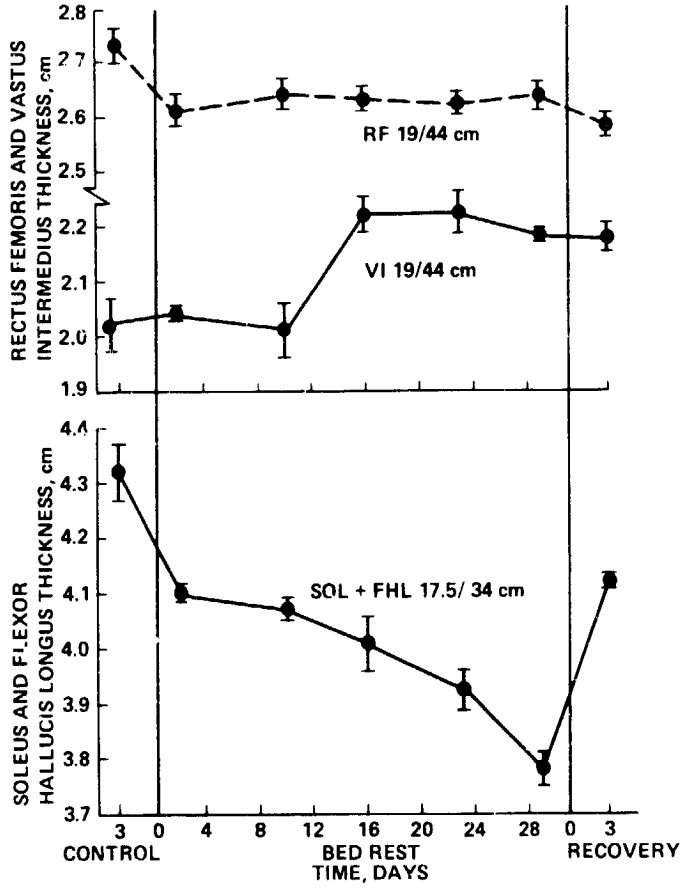



Figure 29.– Muscle thicknesses during control, bed rest, and recovery periods in one subject in the isotonic group.

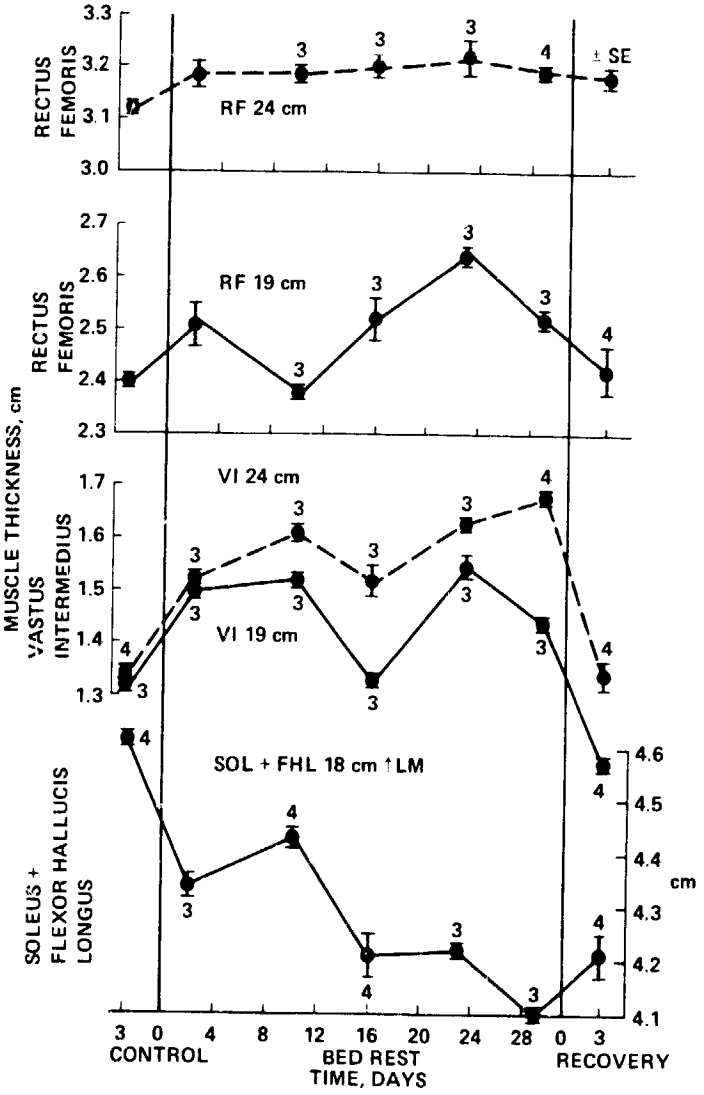



Figure 30.– Muscle thicknesses during control, bed rest, and recovery periods in one subject in the no-exercise group.

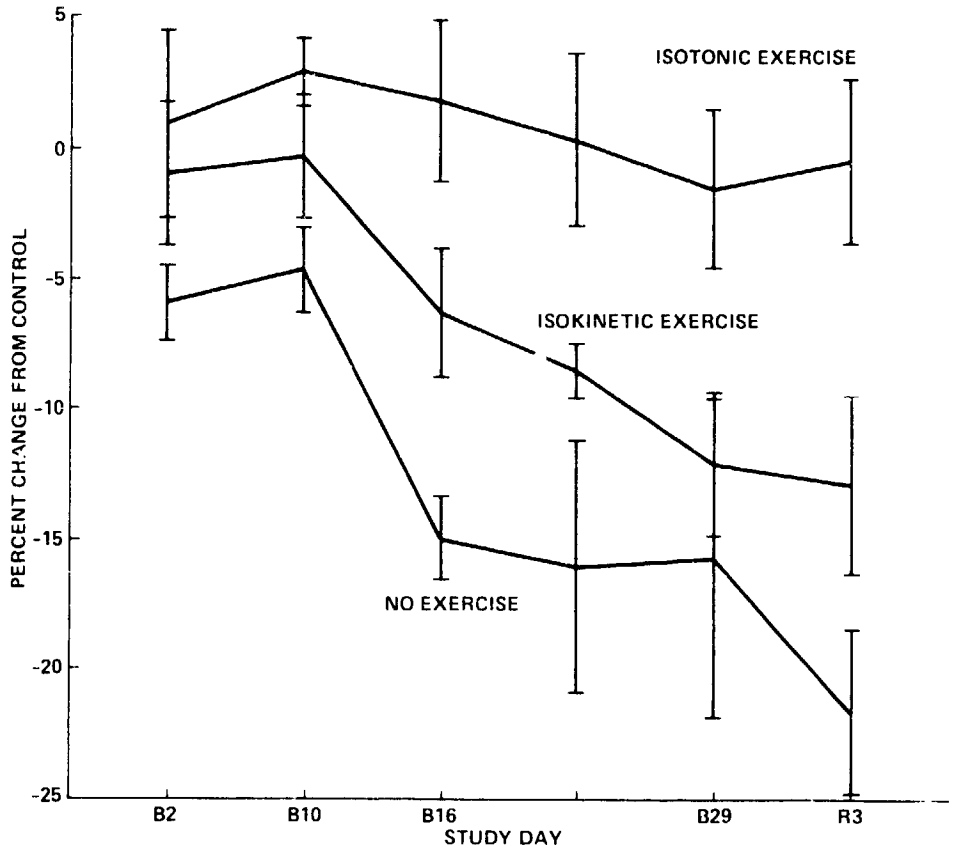



Figure 31.– Mean (±SE) percent change in vastus intermedius muscle thickness in the three groups during bed rest and recovery periods.

# HORMONAL RESPONSES TO EXERCISE AND ORTHOSTASIS

C. E. Wade, L. C. Keil, and J. Vernikos

## Background

A physiological deconditioning occurs when humans are exposed to a weightless environment. This deconditioning is associated with decreases in work capacity and muscle strength, and increased orthostatic intolerance. The decreases in work capacity and orthostatic tolerance may, in part, result from a functional change of the responses of vasoactive hormones important in maintaining cardiovascular integrity. In response to standing, there is normally a release of vasoactive hormones but, following weightlessness, the responsiveness of these hormones may be muted for days. Exercise also produces an increase in vasoactive hormones, but these responses during weightlessness are not known. However, following 7 days of bed rest, the responsiveness of some of these hormonal systems to primary stimuli is depressed. Thus, the decreases in responsiveness of vasoactive hormones may, in part, contribute to a variety of detrimental changes during weightlessness. It is possible that daily exercise will counteract the decreased responsiveness of vasoactive hormonal systems.

The effects of head-down (–6°) bed rest beyond 10 days on the regulation of fluids and electrolytes have not been determined under controlled conditions. Information from 7 days of head-down bed rest studies in both male and female middle-aged subjects has indicated an early diuresis, natriuresis, and kaliuresis. It is not known whether these continue, get progressively worse, or recover. Furthermore, the counteractive effects of various exercise regimes can be assessed rapidly by determining changes in fluid and electrolyte balance on a daily basis. The hormones responsible for the regulation of these effects (which also happen to be primary vasoactive systems underlying the cardiovascular aspects of bed rest deconditioning) show a characteristic uncoupling between the regulating hormone and the target system (e.g., PRA and aldosterone). Whether this dissociation is greatest in individuals prone to orthostatic intolerance is not known, but it is clear that such individuals are less able, in response to orthostatic stress, to sustain the increased responses in these hormones that seem essential to avoid syncope. It is possible that some form of exercise training regime may prevent or delay these changes.

The purpose was to determine whether isotonic or isokinetic exercise training during bed rest would alter the sensitivity of some vasoactive hormones to exercise and orthostatic stresses.

## Procedure and Methods

There were three parts to this experiment that were carried out on all 19 subjects: basal fluid-electrolyte hormonal responses, orthostatic hormonal responses, and exercise hormonal responses.

1. Basal responses. Intake of sodium, potassium, and fluids was recorded daily. Twenty-four-hour urine volumes were collected and a 50-ml aliquot was retained for determination of $Na^+$, $K^+$, osmolality, cortisol, and aldosterone. Basal blood samples were drawn on pre-bed rest day 4 and on bed rest days 1, 8, 14, and 21. Samples were analyzed for plasma renin activity, atrial natriuretic factor, vasopressin, adrenocorticotrophic hormone, cortisol, aldosterone, norepinephrine, epinephrine, dopamine, sodium,

and potassium. Plasma volume (T-1824) was measured on pre-bed rest day 1 and on bed rest days 8 and 30.

2. Orthostatic responses. Blood was drawn before and during tilting on pre-bed rest day 5 and on bed rest day 28. Venous blood samples (20-ml) were drawn from a Teflon catheter after 30 min of the pre-tilt control period, and at 5 min into the tilt. Blood pressure and heart rate were measured each 5 min prior to tilt, each second minute during tilt, and every 5 min during recovery. Plasma was analyzed for hematocrit, hemoglobin, lactate, $Na^+/K^+$, osmolality, creatinine, norepinephrine, epinephrine, angiotensin II, plasma renin activity, vasopressin, ACTH, cortisol, and aldosterone; urine was analyzed for creatinine, $Na^+/K^+$, and osmolality. This experiment assessed the hormonal, cardiovascular, and renal responses to head-up tilt.

3. Exercise responses. The subjects were post-absorptive at least 8 hr prior to this experiment. Sixty minutes prior to exercise the subject voluntarily voided his bladder to begin a timed urine collection. Before the start of the prescribed daily exercise, a urine and blood sample (20 ml) were obtained. Upon completion of the exercise training session, urine and blood samples were collected. Urine collection continued for an additional 2 hr for the post-exercise sample. This exercise test was performed before bed rest on day 5 and on bed rest days 5 and 28 by only the isotonic and isokinetic groups. This experiment assessed the hormonal, cardiovascular, and renal responses to daily exercise training.

All of the above hormones were measured by radioimmunoassay (RIA). Vasopressin and atrial natriuretic factors were assayed on extracts of plasma that had been reconstituted with assay buffer prior to RIA. Plasma renin activity determinations were made on plasma samples treated with angiotensianase inhibitors and incubated at 37°C for 1 hr. Measurement of angiotensin 1 before and after the incubation period was used as an index of renin activity. RIA of aldosterone was made on aliquots of plasma using an antibody-coated-tube technique. Plasma and urine electrolytes ($Na^+/K^+$) were determined by specific ion electrodes, and osmolality by freezing-point depression.

## Results and Discussion

1. Basal responses. Mean body weight of all 19 subjects decreased significantly by the second day of bed rest and remained at that level throughout bed rest. It increased significantly during recovery (fig. 32). The negative fluid balance on the first day of bed rest and the positive fluid balance on the first day of recovery, coupled with the relatively constant caloric intake, indicate clearly that these weight changes were due mainly to fluid exchange. Sodium balance for all three exercise regimens followed water balance (fig. 33). Urinary aldosterone concentration in all three groups was relatively constant in the pre-bed rest period, but was increased on the first recovery day (fig. 34). Plasma potassium concentration was reduced progressively during bed rest in all groups (fig. 35). Fluid and sodium homeostases were maintained during bed rest. Plasma volume was maintained in the isotonic group, but was reduced significantly in the two other groups during bed rest; i.e., by 17-18% (fig. 36). So part of the total body water loss was derived from the extracellular fluid compartment. During bed rest the basal plasma aldosterone concentrations were unchanged, while cortisol levels were elevated slightly but significantly.

2. Orthostatic responses. There were no significant changes in heart rate, plasma volume, or systolic and diastolic blood pressures (fig. 37) from pre-tilt to 5 min of tilt in any group in the pre-bed rest control period (day 3). After bed rest (day 28) all but systolic blood pressure were changed significantly. Similar results were observed with the vasoactive hormone responses; all were unchanged on day 3 and all were significantly elevated on day 28 (fig. 38).

3. Exercise responses. The daily isotonic exercise load was maintained throughout bed rest as evidenced by the maintenance of the peak oxygen uptake. The daily isokinetic exercise work outputs for leg flexion and extension were also maintained during bed rest (fig. 39). For each of the three groups, the change (from rest to steady-state exercise) in heart rate, plasma lactate, epinephrine, norepinephrine, renin activity, and vasopressin were not significantly different from pre-bed rest during the exercise tests on days 5 and 28 of bed rest. The responses of the two exercise groups were, for all of the tests, higher than those of the no-exercise group that did not perform the exercise test.

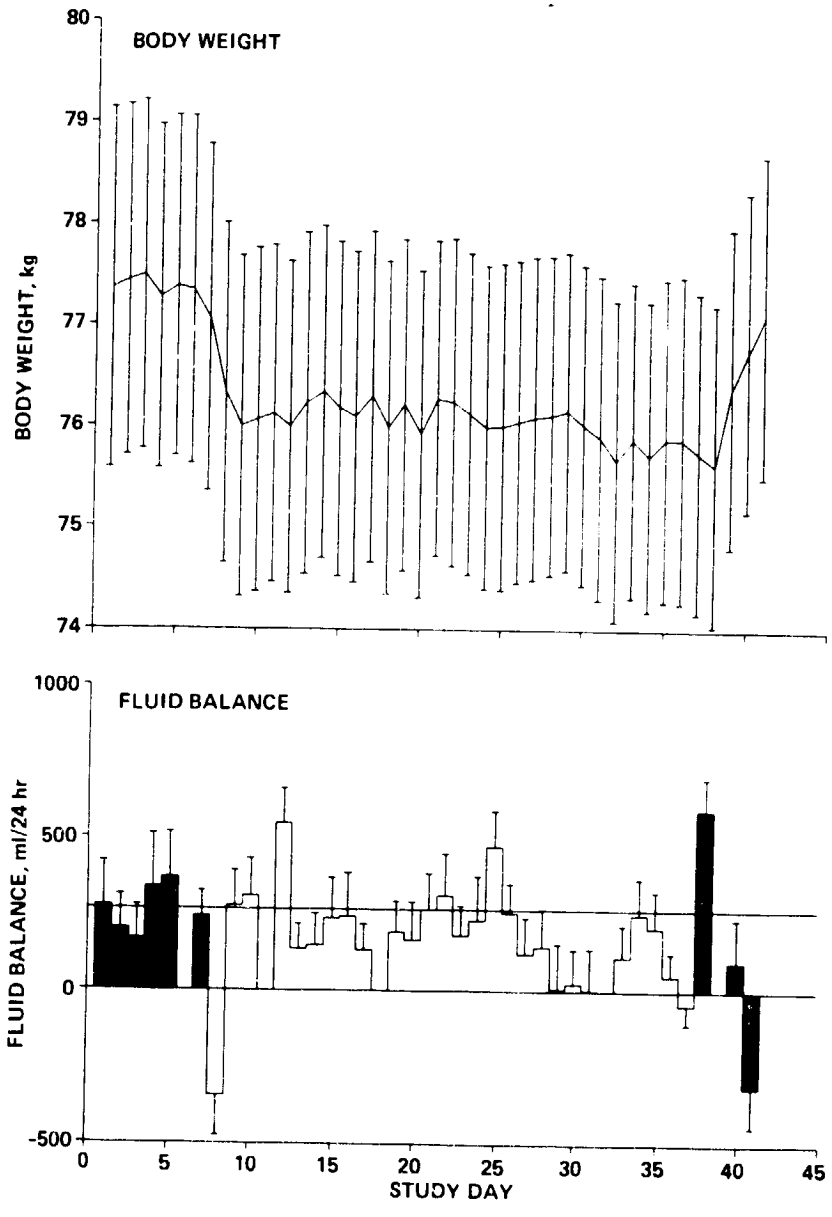



Figure 32.– Mean body weight and fluid balance.

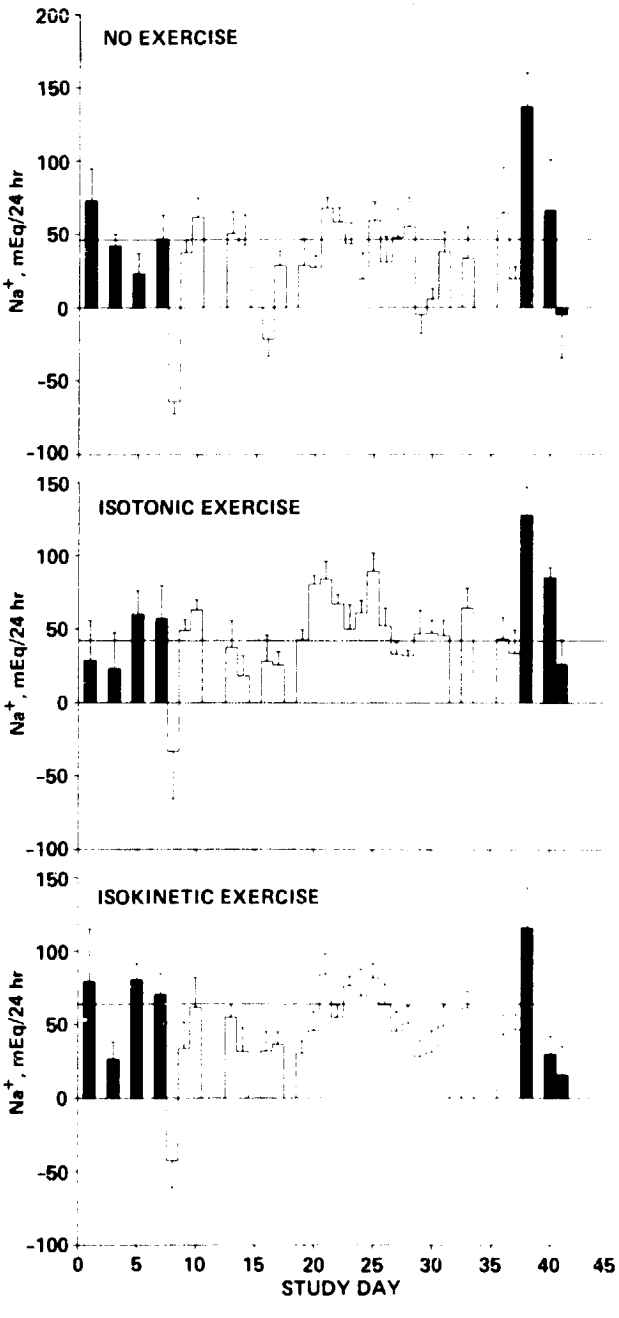



Figure 33.-- Sodium balance.

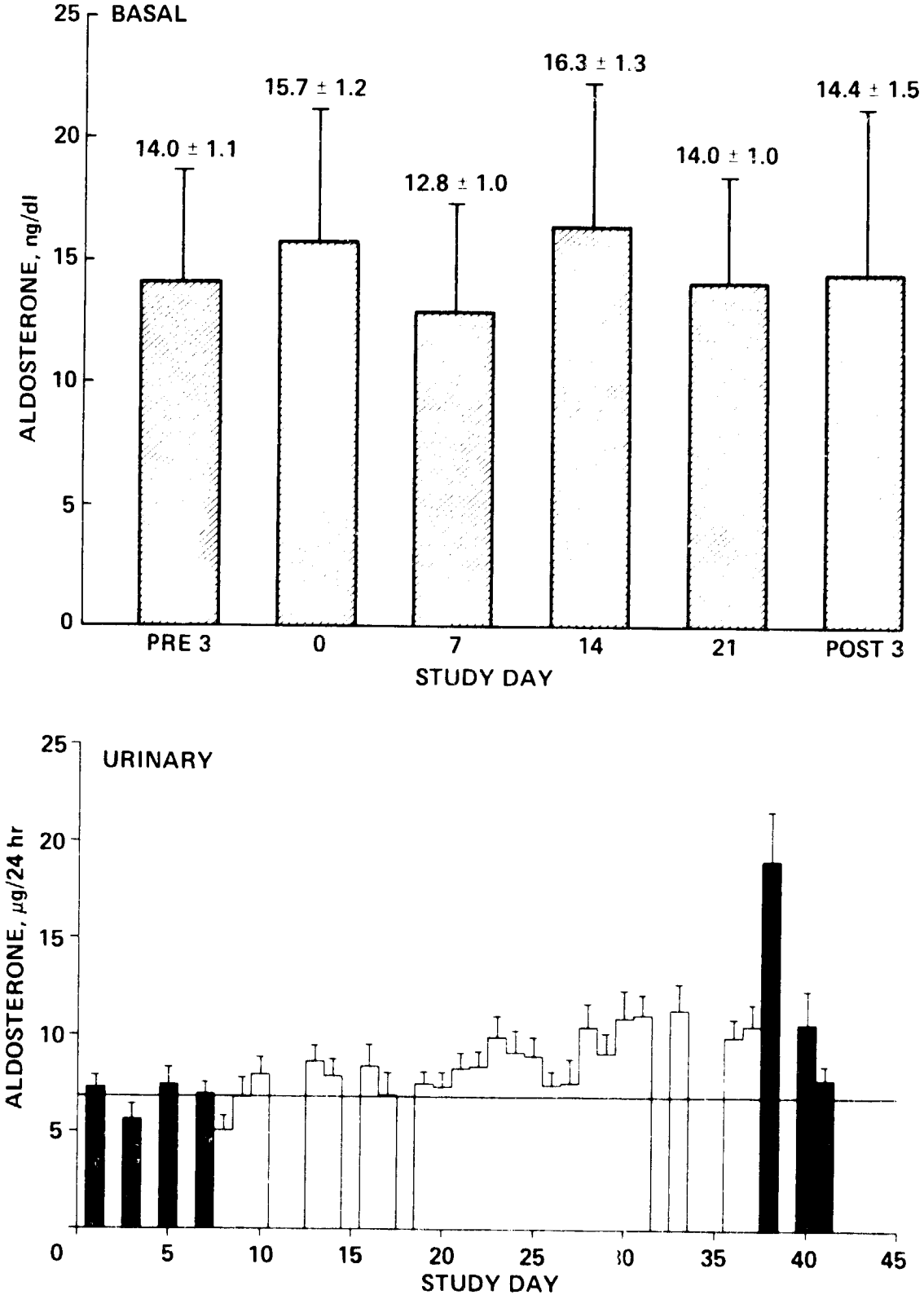



Figure 34.– Urinary aldosterone.

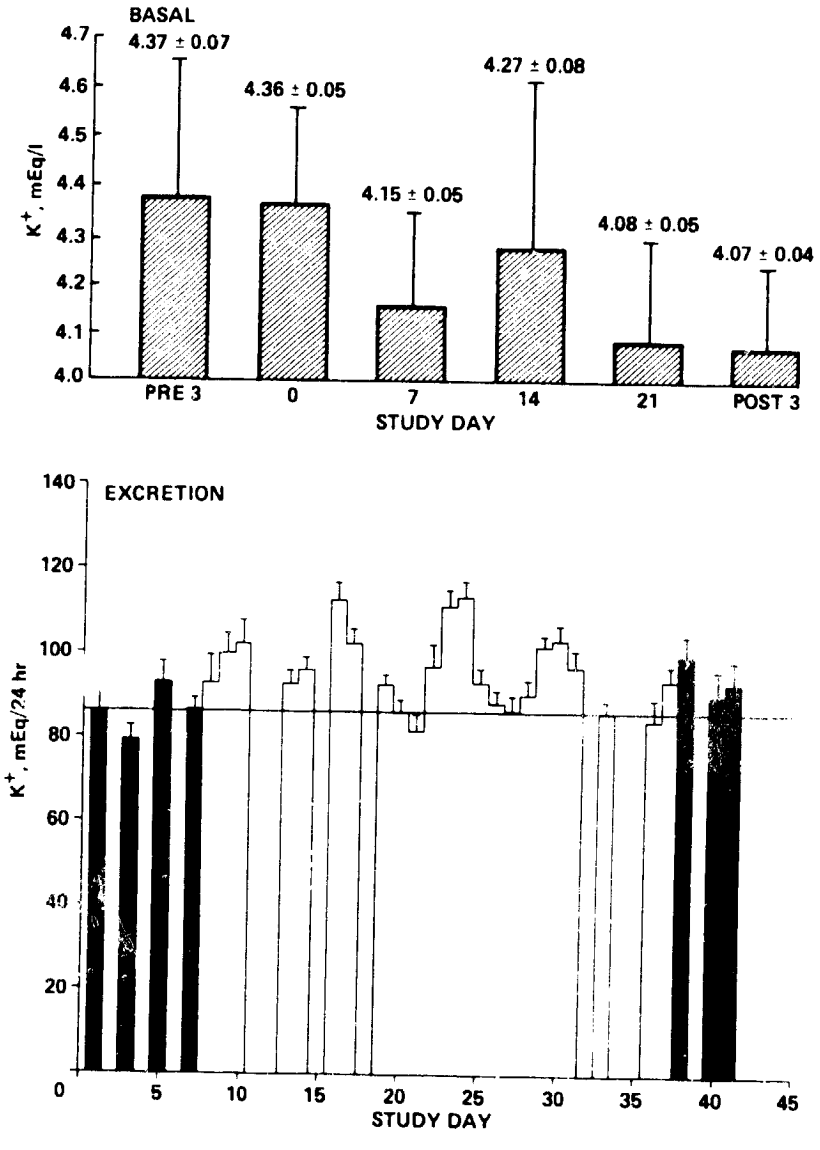



Figure 35.– Plasma potassium and urinary excretion.

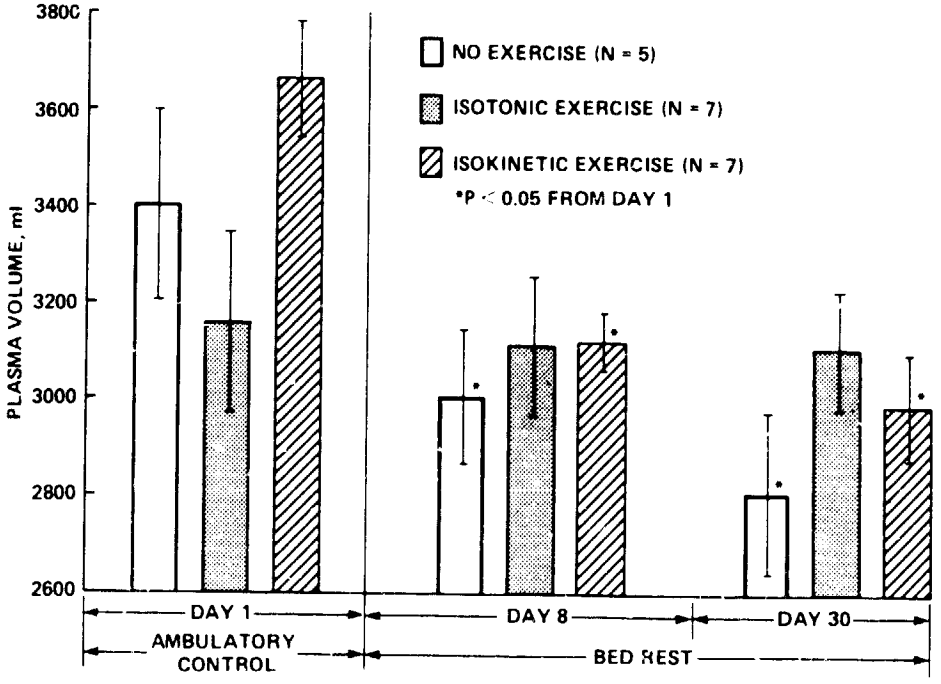



Figure 36.– Plasma volume.

| | DAY -3 | DAY 28 |
|---|---|---|
| HEART RATE, beats/min | 12 ±1 | 30* ±4 |
| PLASMA VOLUME, % | -0.8 ±0.6 | -3.8* ±0.7 |
| SYSTOLIC BLOOD PRESSURE, mm Hg | 0 ±2 | 4 ±2 |
| DIASTOLIC BLOOD PRESSURE, mm Hg | 7 ±1 | 14* ±2 |

Figure 37.– Mean (±SE) changes in cardiovascular parameters with tilting in 19 subjects before and after bed rest.

| | DAY -3 | DAY 28 |
|---|---|---|
| NOREPINEPHRINE, pg/ml | 164 ±20 | 249* ±24 |
| EPINEPHRINE, pg/ml | 16 ±5 | 50* ±13 |
| VASOPRESSIN, pg/ml | 0.1 ±0.3 | 3.8* ±1.8 |
| PLASMA RENIN ACTIVITY, ngAI/ml/hr | 0.03 ±0.03 | 0.51* ±0.17 |
| ACTH, pg/ml | 5 ±3 | 23* ±9 |

Figure 38.– Mean (±SE) changes in vasoactive plasma hormone concentrations with tilting in 19 subjects before and after bed rest.

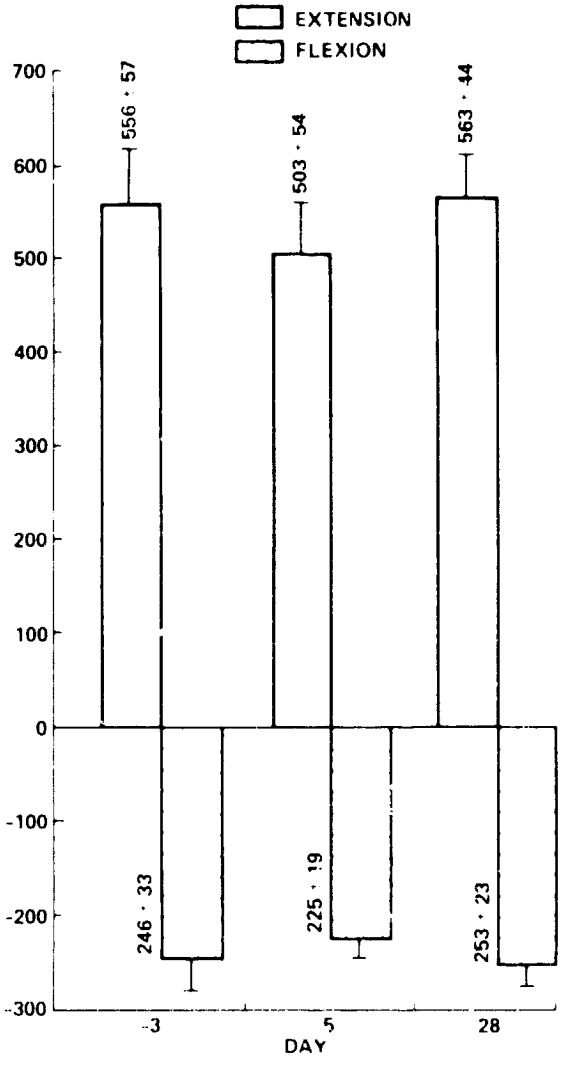



Figure 39.– Isokinetic exercise workload.

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16. Abstract

The purpose for this 30-day bed rest study was to investigate the effects of short-term, high-intensity isotonic and isokinetic exercise training on maintenance of working capacity (peak oxygen uptake), muscular strength and endurance, and on orthostatic tolerance, posture and gait. Other data were collected on muscle atrophy, bone mineralization and density, endocrine analyses concerning vasoactivity and fluid-electrolyte balance, muscle intermediary metabolism, and on performance and mood of the subjects.

It was concluded that: (1) The subjects maintained a relatively stable mood, high morale, and high esprit de corps throughout the study. Performance improved in nearly all tests in almost all the subjects. Isotonic training, as opposed to isokinetic exercise training, was associated more with decreasing levels of psychological tension, concentration, and motivation; and improvement in the quality of sleep. (2) Working capacity (peak oxygen uptake) was maintained during bed rest with isotonic exercise training; it was not maintained with isokinetic or no exercise training. (3) In general, there was no significant decrease in strength or endurance of arm or leg muscles during bed rest, in spite of some reduction in muscle size (atrophy) of some leg muscles. (4) There was no effect of isotonic exercise training on orthostasis, since tilt-table tolerance was reduced similarly in all three groups following bed rest. (5) Bed rest resulted in significant decreases of postural stability and self-selected step length, stride length, and walking velocity, which were not influenced by either exercise training regimen. Most pre-bed rest responses were restored by the fourth day of recovery.

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