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Cycle-Powered Short Radius (1.9 m) Centrifuge: Effect of Exercise Versus Passive Acceleration on Heart Rate in Humans

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Cycle-Powered Short Radius (1.9 m) Centrifuge: Effect of Exercise Versus Passive Acceleration on Heart Rate

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Summary

In addition to extensive use of lower extremity physical exercise training as a countermeasure for the work capacity component of spaceflight deconditioning, some form of additional head-to-foot (+Gz) gravitational (orthostatic) stress may be required to further attenuate or prevent the signs and symptoms (nausea, vertigo, instability, fatigue) of the general reentry syndrome (GRS) that can reduce astronaut performance during landing. Orthostatic (head-to-foot) stress can be induced by standing, by lower body negative pressure, and by +Gz acceleration. One important question is whether acceleration training alone or with concurrent leg exercise would provide sufficient additive stimulation to attenuate the GRS. Use of a new human-powered centrifuge may be the answer. Thus, the purpose for this study was to compare heart rate (HR), i.e., a stress response during human-powered acceleration, in four men (35–62 yr) and two women (30–31 yr) during exercise acceleration versus passive acceleration (by an off-board operator) at 100% (maximal acceleration = Amax), and at 25%, 50%, and 75% of Amax. Mean ($\pm$ SE) Amax was 43.7 $\pm$ 1.3 rpm (+3.9 $\pm$ 0.2 Gz). Mean HR at exercise Amax was 189 $\pm$ 13 b/min (50–70 sec run time), and 142 $\pm$ 22 b/min at passive Amax (40–70 sec run time). Regression of mean HR on the various +Gz levels indicated explained variance (correlations squared) of $r^2 = 0.88$ (exercise) and $r^2 = 0.96$ (passive): exercise HR of 107 $\pm$ 4 (25%) to 189 $\pm$ 13 (100%) b/min were 43–50 b/min higher (p < 0.05) than comparable passive HR of 64 $\pm$ 2 to 142 $\pm$ 22 b/min. Thus, exercise adds significant physiological stress during +Gz acceleration. Inflight use of this combined exercise and acceleration countermeasure may maintain work capacity as well as normalize acceleration and orthostatic tolerances which could attenuate or perhaps eliminate the GRS.

Introduction

Humans have evolved in a constant gravitational environment on Earth. Gravitational force is negated or may be eliminated on space travelers resulting in an altered adapted-deconditioned physiological state. This altered state does not appear to have significant deleterious effects on astronauts' health or well-being during short-term (<16 d) Space Shuttle or up to 15-month orbital flights, but impaired physiological responses and performance (general reentry syndrome (GRS)) have occurred during reimposition of increased +Gz (head-to-foot) gravitational force during reentry and landing on Earth in space vehicles (refs. 1 and 2).

Appropriate use of simulated gravitational countermeasures or treatments such as centrifugation, exercise, or lower body negative pressure, applied singly (refs. 3–10) or in combination (refs. 7, 8, 11–13), have been investigated. One advantage of utilizing two or more countermeasures simultaneously is economy of time, and there may also be a synergistic interaction between them: the disadvantage is a possible negative interaction. Synergistic or negative interactions can be determined by testing treatments singly and in combination.

Short-arm (radius < 2.0 m) centrifuges have been used by physicians for medical treatment early in the 19th century to increase headward (–Gz) blood flow to treat insanity (ref. 14). A dual-couch short-arm human centrifuge was designed for use in an orbital laboratory prior to 1964 (ref. 15), and there has been continuing interest in this basic gravitational countermeasure without (refs. 16–29) and with (refs. 8 and 13) concomitant physical exercise. Antonutto and colleagues (refs. 30 and 31) have proposed use of two counter-rotating bicycles that move on the inner wall of a cylindrical space module as a means of applying exercise and accelerative stimuli simultaneously.

A concept of a self-powered human centrifuge has been patented (ref. 32), but another design, constructed at Ames research Center, is a short-arm dual-couch machine powered by a chain-linked cycle foot drive (fig. 1). Thus, the purpose for this study was to describe this centrifuge and to compare heart rate response, as an estimate of total physiological stress, in subjects undergoing active (exercise) and passive (no exercise) +Gz acceleration.
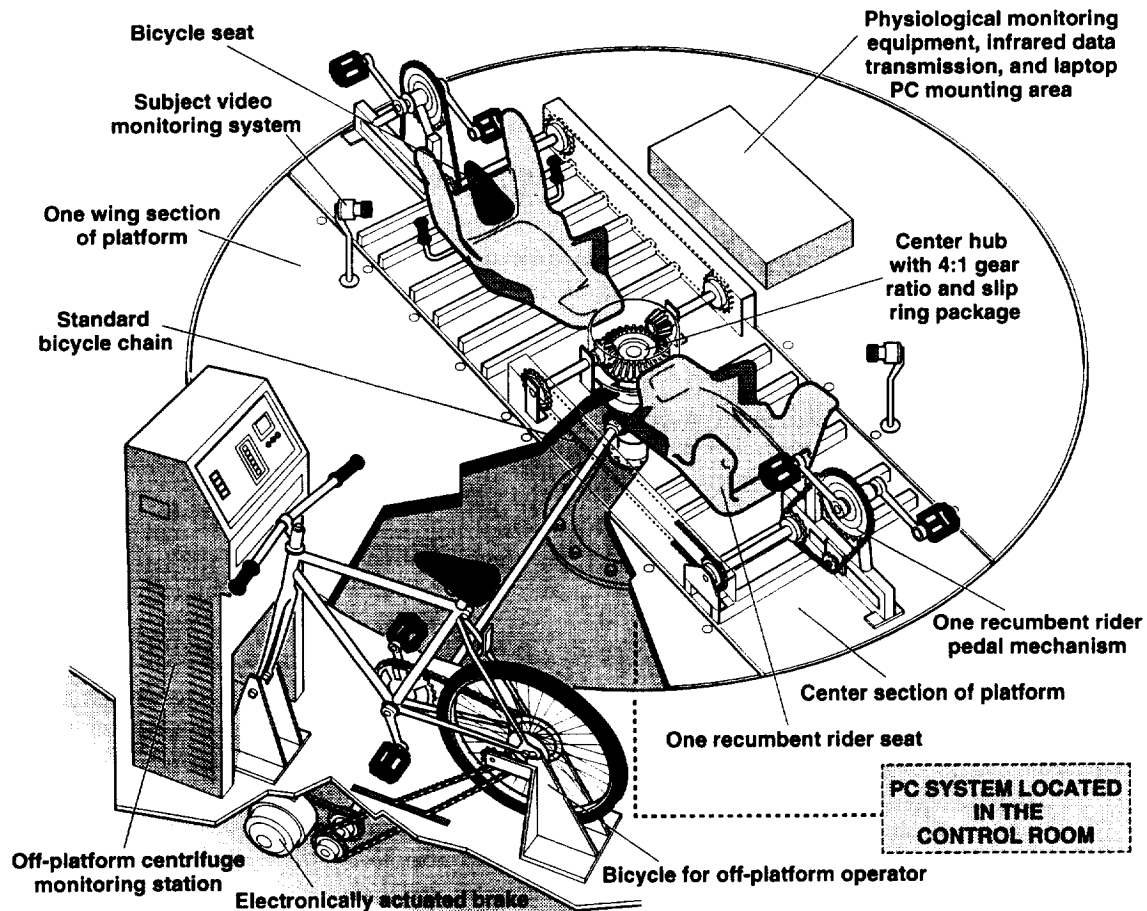
Figure 1. Human-powered short-arm centrifuge schematic.

Methods

Centrifuge Design

The platform (rotation) assembly, consisting of three sections of lightweight aluminum honeycomb and bonded aluminum pieces (weighing 295 kg), was designed and fabricated in the model and machine shops at Ames Research Center (fig. 1). The center section of the platform rides on Timken tapered roller bearings and supports the two cycle couches; Boston bevel gears (4:1 gear ratio) transform subjects' pedaling power to platform rotation. The other two platform sections (wings), bolted to the center section for ease of disassembly and maintenance, also provide an area for walking and for mounting physiological instrumentation. The supine test subjects were oriented with their heads toward the center of rotation (fig. 1). The top of the subject's head is 26 cm from the center of rotation. The three pedaling stations, two onboard at the end of each couch and the third on the operator's offboard stationary cycle, are linked by standard bicycle chains (with tubing) and sprockets to the center hub. Acceleration level was calculated at 1.9 m from the center of rotation. One 360° rotation of the centrifuge requires 1.6 pedal rotations.

The operator or the subject can actuate and control velocity (rpm), but only the operator can stop the

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centrifuge with an electronically activated spring-set disc brake (series 87,300, Stearns). Centrifuge parameters such as rpm (model P6020 Tachometer, Newport Electronics, Inc., Santa Ana, California), with an accuracy of 0.0002%, and G-level (currently set at 5 G maximal at 168 cm from the center of rotation) are displayed on the adjacent monitoring station (fig. 1). The center hub-drive differential has a slip-ring assembly (model 1067, Fabricast) that transmits physiological data from the onboard test subject(s) to the adjoining control room. Infrared or radio-frequency transmission systems are also available for conveying data.

Subjects

Six healthy volunteers (staff members), four men (35–62 yr) and two women (30–31 yr) (table 1), provided informed consent and received our standard thorough medical examination including medical history and blood and urine tests. This study, approved by the Ames Research Center Human Research Institutional Review Board, was conducted in July 1995.

Procedure

All subjects had active (exercise) and passive (no exercise) practice sessions before the study began. The protocol required the exercise run first (to determine the 100% maximal level) which consisted of +1 Gz for 2 min, approximately 2 min rest, +2 Gz for 2 min, rest, and then maximal acceleration (expressed as rpm) to volitional fatigue. After 10–15 min rest the submaximal exercise was performed, which consisted of 25% of maximum rpm for 2 min, rest, 50% of maximum rpm for 2 min, rest, and finally 75% of maximum rpm for 2 min (fig. 2). After heart rate (HR) had decreased to about 100 b/min during rest, the next exercise level was begun. At least two days later the offboard operator provided cycle power for the passive runs (the subject's pedals were decoupled from the drive mechanism) with the same intensity (rpm) and protocol as the active runs. The standard bicycle seat was adjusted for each subject and pedal toe-clips provided leg support for all exercise and passive runs. Hand grips were used during the maximal exercise as well as some other runs, which prevented use of the finger blood pressure data; the run termination switch was on the right grip (fig. 1). Care must be taken when two subjects ride simultaneously (either actively or passively), especially with one exercising and the other passive because

differences in their tolerance under the two conditions may lead to syncope in the passive subject. Therefore, each subject was tested individually. Most subjects elected to be blindfolded to prevent nausea or vertigo during their runs, which were conducted at least 2 hr after eating.

Instrumentation

The subjects were instrumented to provide for medical monitoring with electrocardiogram electrodes (model 14056-B, Hewlett-Packard) for wave form and heart rate (model 78203A ECG, Hewlett-Packard, Waltham, Maine), an infrared sensor (Finapres Ohmeda 2300, Englewood, Colorado) on the left middle finger for arterial blood pressure and pulse rate, and an ultrasound Doppler sensor (L&M Electronics, San Francisco, California) on the left temple for temporal arterial blood flow and pulse rate. Signals from these sensors were displayed, processed, and stored on a Gateway model 2000 personal computer (North Sioux City, South Dakota) for the medical monitor and investigators. Voice communication was maintained among the medical monitor, centrifuge operator, and test subject. Video coverage of the entire centrifuge room, including the operator and coverage from the one onboard camera focused on the subject, was available to the medical monitor and investigators in the control room.

Data analysis

Heart rate datum at a particular time was the mean of three data points; i.e., the value at that time and values recorded 10 sec before and 10 sec afterward. Heart rate data at 120 sec for the 50% and 75% levels were compared to those at 25% with Student's t-test for dependent (paired) data (program STAT 1-29A, model HP-65 calculator, Hewlett-Packard, Cupertino, California); and with linear regression (HP program STAT 1-22A). Heart rate variability was the sum of standard errors for the seven time intervals (rest + 6) for four treatments with exercise and passive acceleration.

The correlation coefficient squared (r^2) is the proportion of the Σy^2 that is associated with variation in the acceleration level; i.e., the explained variance. Therefore, $1 - r^2$ is the unexplained (independent) variance. Level of significance was $P < 0.05$ and nonsignificant differences were designed NS.

Table 1. Mean (±SD, ±SE) anthropometric and acceleration data for the four male (M) and two female (F) subjects

| | | Anthropometry | | | | Acceleration | | | | | | | | |
| | | | | | | Maximal | | | Submaximal | | | | | |
| | | | | | | 100% | | | 25% | | 50% | | 75% | |
Subject	Sex	Age, yr	Height, cm	Weight, kg	SA, m²	rpm	+Gz	sec	rpm	+Gz	rpm	+Gz	rpm	+Gz
ARA	M	35	167.5	67.5	1.79	49.4	4.9	59	12.4	0.3	24.7	1.3	37.0	2.8
GRE	M	62	175.0	95.4	2.19	43.0	3.8	66	10.8	0.2	21.5	1.0	32.2	2.1
HAR	M	51	174.5	75.9	1.93	44.5	4.1	70	11.1	0.3	22.2	1.0	33.4	2.3
LOO	F	30	158.5	60.7	1.66	40.3	3.3	77	10.1	0.2	20.2	0.8	30.2	1.9
MAR	F	31	160.0	55.2	1.58	43.0	3.8	66	10.8	0.2	21.5	1.0	32.2	2.1
MUL	M	57	179.5	82.3	2.04	42.0	3.6	74	10.5	0.2	21.0	0.9	31.5	2.0
X̄		44	169.1	72.8	1.87	43.7	3.9	69	11.0	0.2	21.8	1.0	32.8	2.2
±SD		14	8.5	14.8	0.23	3.1	0.6	6	0.8	0.1	1.5	0.2	2.3	0.3
±SE		6	3.5	6.0	0.09	1.3	0.2	3	0.3	0.0	0.6	0.1	1.0	0.1

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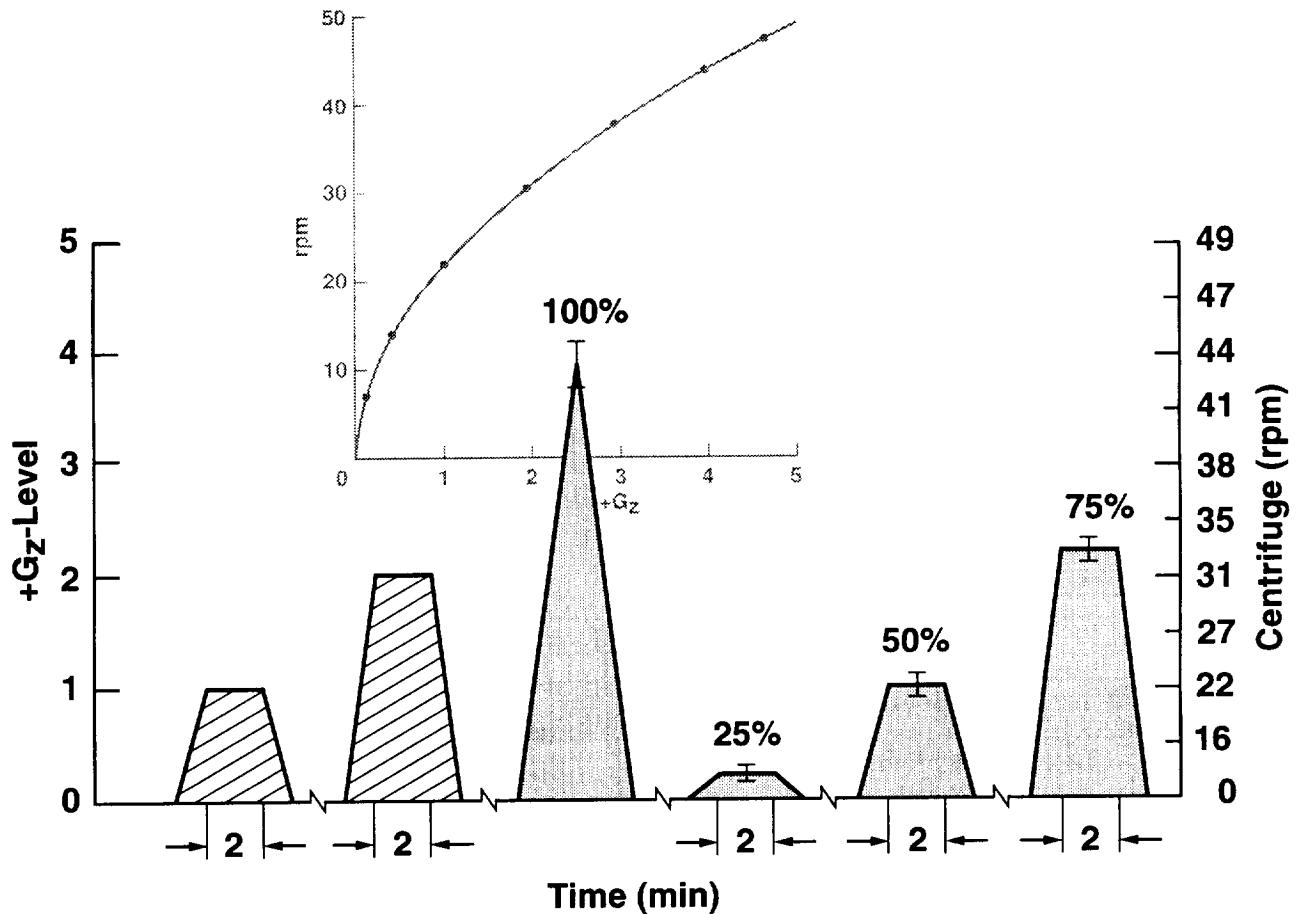
Figure 2. Experimental protocol with +Gz-level and comparable rpm on Y-axes. Maximal acceleration is 100%. Background graph is calibration curve: X-axis is +Gz-level and Y-axis is rpm. The rpm are not related linearly to +Gz-level.

Results

Maximal (Amax) and Submaximal Acceleration

Mean (±SE) Amax was +3.9 ± 0.2 Gz (43.7 ± 1.3 rpm). Submaximal acceleration levels were +0.2 ± 0.0 Gz (11.0 ± 0.2 rpm) for 25%, +1.0 ± 0.1 Gz (21.8 ± 0.6 rpm) for 50%, and +2.2 ± 0.1 Gz (32.8 ± 1.0 rpm) for 75% of Amax (table 1).

Warm-Up

Equilibrium HR with 3 min of exercise + acceleration was 114 ± 15 b/min (Δ from time 0 = 31 ± 13 b/min) at +1 Gz, and 150 ± 14 b/min (Δ from time 0 = 64 ± 13 b/min) at +2 Gz (table 2). Comparable HR with passive acceleration was 74 ± 5 b/min (Δ from time 0 = 1 ± 4 b/min) at +1 Gz, and 112 ± 17 b/min (Δ from time 0 = 38 ± 18 b/min) at +2 Gz.

Exercise Acceleration

Mean HR at maximal exercise + acceleration was 189 ± 13 b/min at 50 sec of exercise (N = 6), and 184 ± 12 b/min at 60 sec where N = 5 (fig. 3, upper half). Only two subjects exercised maximally for over 70 sec. Compared with mean HR at 120 sec at 25% (110 ± 5 b/min), HR was elevated significantly at 50% (141 ± 15 b/min, t = 3.10, p < 0.05) and at 75% (184 ± 11 b/min, t = 9.19, p < 0.001). There was no significant difference at 40 sec between HR at 75% (176 ± 15 b/min) and 100% (164 ± 12 b/min, t = 1.03, p < 0.3 NS) maximum loads. The rates of rise of HR (ΔHR/Δtime) for the first 20 min were not different among the three higher loads (fig. 3, upper half).

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Table 2. Mean (±SE) heart rate at rest (0 min) and at 60 sec intervals during the sequential 1G and 2G warm-up runs

	1 G (22 rpm)				2 G (31 rpm)			
	0 sec	60 sec	120 sec	Δ (120–0) sec	0 sec	60 sec	120 sec	Δ (120–0) sec
Exercise acceleration								
$\overline{X}$	83	113	114	31	86	151	150	64
±SE	6	9	15	13	7	15	14	13
Passive acceleration								
$\overline{X}$	73	79	74	1	74	96	112	38
±SE	3	7	5	4	3	11	17	18

Passive Acceleration

Compared with exercise acceleration, there were different patterns of ΔHR/Δtime and equilibrium levels of HR with comparable Gz loads during passive acceleration (fig. 3, lower half). Heart rate at 100% load was 136 ± 22 b/min at 40 sec (N = 6), and 115 ± 24 b/min at 50 sec (N = 4). Only one subject endured 70 sec and his HR was just 85 b/min. Compared with mean HR at 120 sec at 25% (63 ± 2 b/min), HR was not elevated significantly at 50% (70 ± 3 b/min, t = 2.32, p > 0.05, NS), but was elevated significantly at 75% (110 ± 16 b/min, t = 3.07, p < 0.05). There was no significant difference at 40 sec between HR at 75% (111 ± 20 b/min) and 100% (136 ± 22 b/min, t = 1.65, p > 0.01, NS) (fig. 3, lower half).

Heart Rate Variability

This summated variability at 25%, 50%, 75%, and 100% of Amax was 29, 78, 91, and 65 b/min, respectively (Σ = 263 b/min), with exercise acceleration; and 18, 29, 114, and 90 b/min, respectively (Σ = 251 b/min t = 0.17, NS, df = 3), with passive acceleration.

Regression of Heart Rate on Acceleration Level

The correlation coefficient (r) for regression of HR on acceleration level was 0.80 (r^2 = 0.64, p < 0.01, df = 22) for exercise acceleration, and 0.62 (r^2 = 0.39, p < 0.10, NS, df = 22) for passive acceleration (fig. 4, upper half). The comparable linear regressions for mean data at each acceleration level indicated r = 0.94 (r^2 = 0.88, p > 0.10, NS, df = 2) for exercise acceleration, and r = 0.98

(r^2 = 0.96, p < 0.05, df = 2) for passive acceleration (fig. 4, lower half).

Discussion

While the physiological effects of physical exercise and passive (nonexercise) +Gz acceleration have been studied extensively, it was not until 1967 that Rosenhamer (refs. 6 and 12) and Bjurstedt et al. (ref. 33) combined the two stresses; i.e., exercise alone and exercise while accelerating to counteract the acceleration. The Karolinska centrifuge was powered independently of the exercise. They studied the effect of passive acceleration and exercise + acceleration (leg exercise at 300 and 600 kpm/min) on men sitting in a centrifuge while undergoing +3 Gz acceleration. Compared with responses during passive acceleration, exercise + acceleration greatly attenuated circulatory collapse and improved circulatory stability as evidenced by enhanced cardiac output and reduced heart rate variability.

In the present study mean heart rate variability (± SE) was not different with exercise + acceleration when compared with that during passive acceleration with the ultimate purpose of having the +Gz and/or exercise counteract the spaceflight deconditioning. The high correlation coefficients with the two treatments between heart rate and acceleration level were probably due to the use of relative exercise + acceleration loads, where the submaximal loads were percentages (relative levels) of the maximal level. This procedure reduces variability of many physiological variables among test subjects who have different maximal work levels (ref. 34).
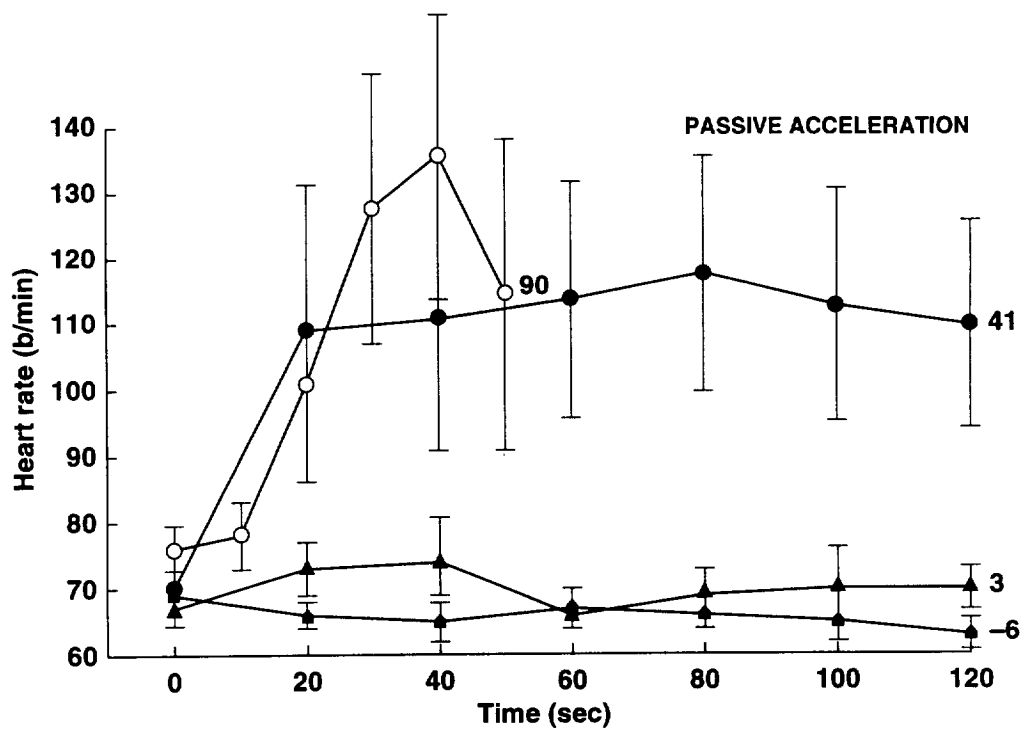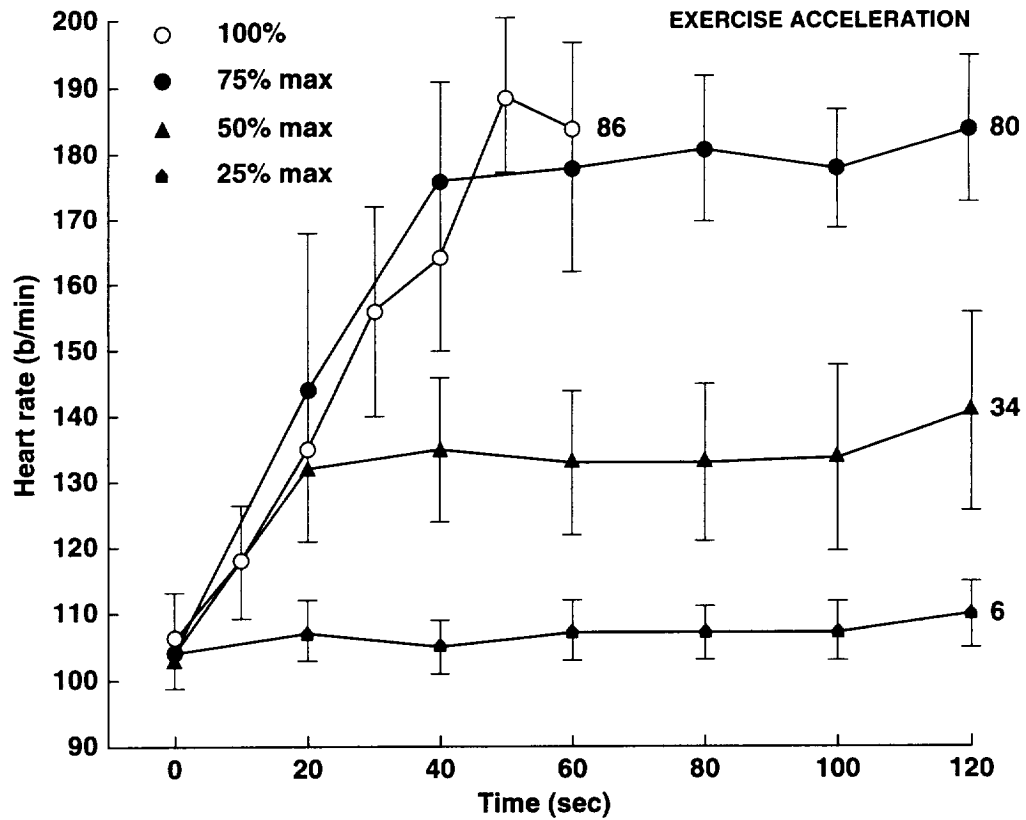
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Figure 3. Mean (±SE) heart rate for the 120 sec submaximal (25%, 50%, 75%) and maximal (100%) exercise acceleration (upper panel) and passive acceleration (lower panel) runs. Numbers at the end of each run are change in heart rate at end of run minus heart rate at 0 sec.
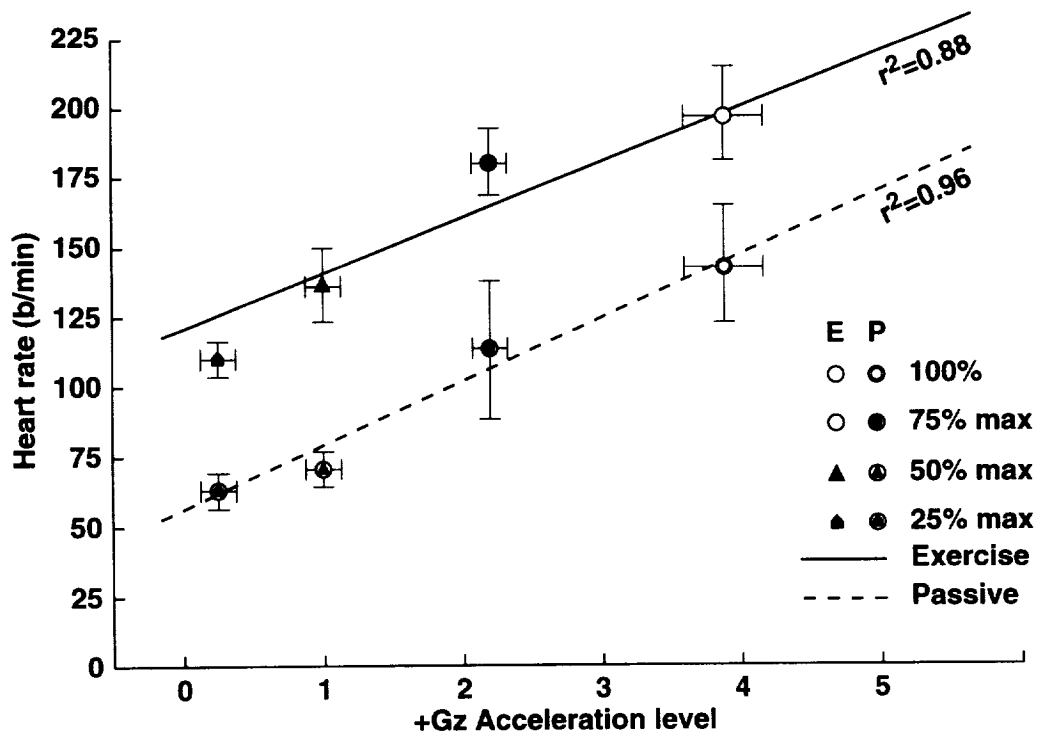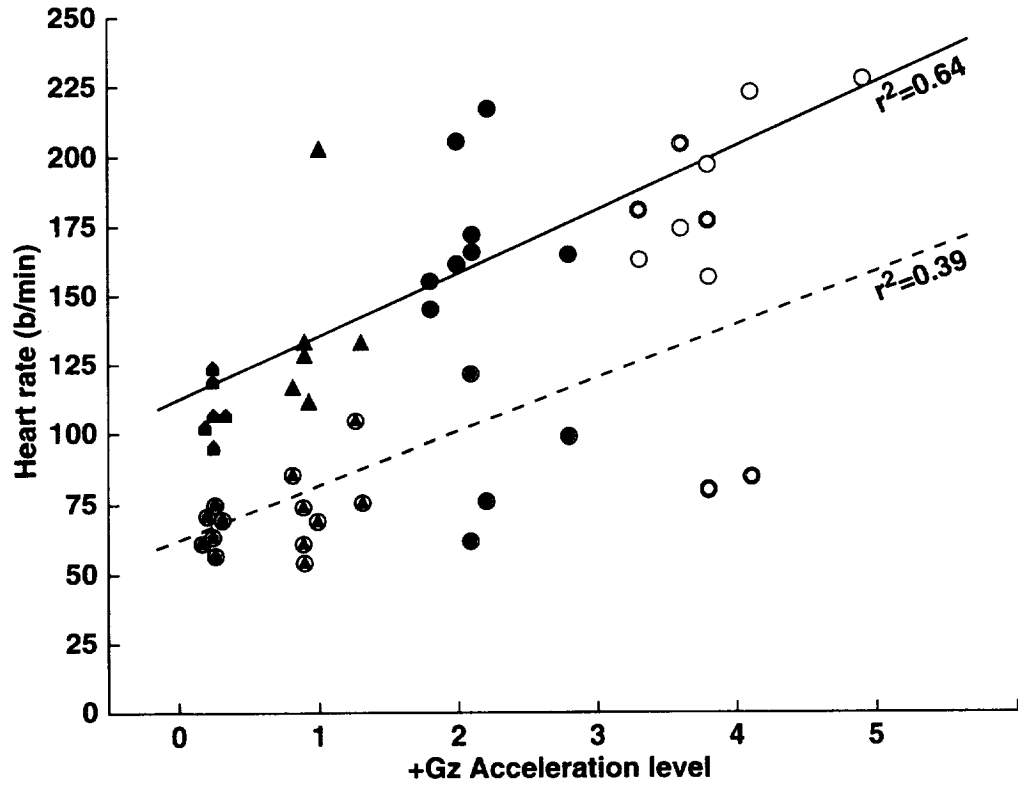
Figure 4. Regression of heart rates on acceleration level for the 120 sec submaximal (25%, 50%, 75%) and maximal (100%) exercise (E) acceleration (solid line) and passive (P) acceleration (dashed line) runs. Individual (upper panel) and mean (lower panel) heart rates are given.

The expected proportional increase in exercise + acceleration heart rate with increasing relative loads occurred at the 25%, 50%, and 75% levels, but the maximal heart rate was not significantly higher than the 75% rate. Conversely, while the maximal heart rate with passive acceleration was not significantly higher than the 75% rate, the 25% and 50% heart rates were not different and did not increase from resting levels. The difference between the exercise and passive HR/acceleration regressions (ΔHR of 65–55 b/min) reflects the additional "exercise" effect at the higher acceleration levels on heart rate during acceleration. Note that the 0 min HR began at about 100 b/min for exercise acceleration, and at about 70 b/min for passive acceleration because the 25%, 50% and 75% runs were conducted consecutively before HR could return to true resting levels. But this higher beginning level would not have appreciably influenced the end exercise level. Nevertheless, it seems the exercise + acceleration may result in some nonlinear cardiovascular responses involving cardiac output via stroke volume and heart rate. The earlier termination of some maximal passive acceleration runs was due to excessively elevated heart rates in some subjects; only one subject reported transient dizziness after the passive run. Because of similar heart rate variability between the two treatments, we cannot confirm Rosenhamer's conclusion that, in spite of significantly greater absolute heart rates with exercise + acceleration, there is enhanced cardiovascular stability with exercise acceleration versus passive acceleration. Thus, our data indicate that greater metabolic stress does not necessarily result in lesser +Gz acceleration stress.

Whether the additional stress of acceleration, when combined with exercise, will enhance the exercise + acceleration-training process to attenuate the GRS remains to be studied. It is also probable that short arm acceleration training can counteract the reduced "orthostatic" tolerance and disorientation that usually occur after deconditioning (refs. 8 and 13). Head-to-foot acceleration redistributes and retains blood in the venous system of the lower extremities similar to that resulting from quiet standing or application of lower body negative pressure. On the other hand, Bjurstedt and Eiken (ref. 35) have proposed use of lower body *positive* pressure as a method for controlling lower limb blood flow, which may have some positive effects for induction of an enhanced exercise-trained state. Human-powered acceleration provides a method to examine the interaction between exercise and gravitational stress, and may also provide an effective countermeasure for physiologic deconditioning during spaceflight.

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13. ABSTRACT (Maximum 200 words)

In addition to extensive use of lower extremity physical exercise training as a countermeasure for the work capacity component of spaceflight deconditioning, some form of additional head-to-foot (+Gz) gravitational (orthostatic) stress may be required to further attenuate or prevent the signs and symptoms (nausea, vertigo, instability, fatigue) of the general reentry syndrome (GRS) that can reduce astronaut performance during landing. Orthostatic (head-to-foot) stress can be induced by standing, by lower body negative pressure, and by +Gz acceleration. One important question is whether acceleration training alone or with concurrent leg exercise would provide sufficient additive stimulation to attenuate the GRS. Use of a new human-powered centrifuge may be the answer. Thus, the purpose for this study was to compare heart rate (HR), i.e., a stress response during human-powered acceleration, in four men (35–62 yr) and two women (30–31 yr) during exercise acceleration versus passive acceleration (by an off-board operator) at 100% (maximal acceleration = Amax), and at 25%, 50%, and 75% of Amax. Mean ($\pm$ SE) Amax was 43.7 $\pm$ 1.3 rpm (+3.9 $\pm$ 0.2Gz). Mean HR at exercise Amax was 189 $\pm$ 13 b/min (50–70 sec run time), and 142 $\pm$ 22 b/min at passive Amax (40–70 sec run time). Regression of mean HR on the various +Gz levels indicated explained variance (correlations squared) of $r^2 = 0.88$ (exercise) and $r^2 = 0.96$ (passive): exercise HR of 107 $\pm$ 4 (25%) to 189 $\pm$ 13 (100%) b/min were 43–50 b/min higher ($p < 0.05$) than comparable passive HR of 64 $\pm$ 2 to 142 $\pm$ 22 b/min. Thus, exercise adds significant physiological stress during +Gz acceleration. Inflight use of this combined exercise and acceleration countermeasure may maintain work capacity as well as normalize acceleration and orthostatic tolerances which could attenuate or perhaps eliminate the GRS.

14. SUBJECT TERMS

Gravitational force, Heart rate, Maximal exercise, Human-powered centrifuge

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