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Responses to LBNP in Men with Varying Profiles of Strength and Aerobic Capacity: Implications for Flight Crews

Victor A. Convertino
John F. Kennedy Space Center
Kennedy Space Center, Florida

Karen L. Mathes
Krug International
Houston, Texas

Mary L. Lasley
The Bionetics Corporation
Kennedy Space Center, Florida

Clare Marie Tomaselli
John B. Pierce Laboratory
New Haven, Connecticut

Mary Anne Bassett Frey
Lockheed Engineering and Sciences Company
Washington, D.C.

G. Wyckliffe Hoffler
John F. Kennedy Space Center
Kennedy Space Center, Florida

NASA
National Aeronautics and Space Administration
Office of Management
Scientific and Technical Information Division

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ABSTRACT

Hemodynamic, cardiac, and hormonal responses to lower-body negative pressure (LBNP) were examined in 24 healthy men to test the hypothesis that responsiveness of reflex control of blood pressure during orthostatic challenge is associated with strength and/or aerobic capacity. Subjects underwent treadmill tests to determine peak oxygen uptake (peak $\dot{V}O_2$) and isokinetic dynamometer tests to determine leg strength. Based on predetermined criteria, subjects were classified into one of four fitness profiles of six subjects each, matched for age, height, and weight: (a) low strength/average aerobic fitness; (b) low strength/high aerobic fitness; (c) high strength/average aerobic fitness; and (d) high strength/high aerobic fitness. Following 90 min of 6° head-down tilt (HDT), each subject underwent graded LBNP through -50 mmHg or presyncope, with maximal duration 15 min, while hemodynamic, cardiac, and hormonal responses were measured. All groups exhibited typical hemodynamic, hormonal, and fluid shift responses during LBNP, with no intergroup differences between high and low strength characteristics. Subjects with high aerobic capacity exhibited greater ($P < 0.05$) stroke volume and lower ($P < 0.05$) heart rate, vascular peripheral resistance, and mean arterial pressure during rest, HDT, and LBNP. Seven subjects, distributed among the four fitness profiles, became presyncopal. These subjects showed greatest reduction in mean arterial pressure during LBNP, had greater elevations in vasopressin, and lesser increases in heart rate and peripheral resistance. Neither peak $\dot{V}O_2$ nor leg strength were associated with fall in arterial pressure or with syncopal episodes. We conclude that neither aerobic nor strength fitness characteristics are associated with responses to LBNP challenge and therefore should not be used as an issue on orthostatic performance in the selection of astronauts and pilots.

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INTRODUCTION

A proposed relationship between aerobic capacity and tolerance to orthostatic challenges has stimulated controversy and confusion regarding the ideal fitness characteristics of astronauts and high-performance aircraft pilots. Results from several investigations have indicated that high aerobic capacity may be associated with low tolerance to orthostatic challenge (Luft et al. 1976, 1980; Mangseth and Bernauer 1980; Convertino 1987; Klein et al. 1977). On the other hand, data generated from other studies using cross-sectional subject populations have indicated no relationship between aerobic capacity and orthostatic tolerance (Convertino et al. 1986; Convertino 1987; Klein et al. 1969; Ludwig et al. 1987). Such discrepancy has raised issues concerning endurance exercise training by individuals in aerospace environments who are exposed to rapid changes in gravitational forces and has stimulated controversy and confusion about the ideal physical characteristics used for selection of astronauts and pilots of high performance aircraft (Klein et al. 1977; Luft et al. 1976; Tesch et al. 1983; Tipton 1983).

Fewer data have been reported on the relationships between muscle strength and responses to orthostatic challenge. Greater cardiovascular responsiveness and orthostatic tolerance have been observed in weight-trained subjects compared to aerobically-fit, endurance-trained subjects (Luft et al. 1980; Smith and Raven 1986). This may be related to less compliance in the legs of weight-trained individuals (Luft et al. 1980) since large muscle mass is associated with low limb compliance (Convertino et al. 1987), and low limb compliance is related to greater orthostatic tolerance (Luft et al. 1976, 1980). Resistive strength training, particularly of the lower extremities, may be preferable, therefore, to endurance training for protection against orthostatic challenges.

A clear relationship between fitness and orthostatic stability may be obscured by interactions of varying aerobic and strength profiles on blood pressure regulation. We are unaware of any investigations in which responses to orthostatic challenge in individuals with various combinations of aerobic and strength fitness have been examined.

We therefore chose subjects with varying combinations of high or average aerobic capacity and high or low leg muscular strength and assessed their cardiac and hemodynamic responses, vasoactive hormone levels, and fluid shifts associated with regulation of arterial blood pressure during exposure to lower-body negative pressure (LBNP) after a period of acute head-down tilt (HDT). Our purpose was to retest the hypothesis that responsiveness of reflex control of blood pressure during an orthostatic challenge is associated with strength and/or aerobic capacity by systematically evaluating the interactions between these fitness profiles. We also analyzed the responses of subjects who exhibited presyncopal limited intolerance to LBNP and compared their responses to those of subjects who did not exhibit presyncopal symptoms.

METHODS

Subjects

Twenty-four healthy men, 25 to 39 years, gave written consent to participate as volunteers for this study after all procedures and possible risks were explained. All subjects underwent a screening evaluation that consisted of a detailed medical history and physical examination, which included blood tests, urinalysis, a resting 12-lead electrocardiogram, and pulmonary function tests.

Experimental Protocol

The experimental protocol consisted of four testing sessions. During the first session, screening evaluations were performed and

leg strength was determined. Peak oxygen uptake (peak $\dot{V}O_2$) was determined during the second testing session. During the third test session, body density was measured and subjects were familiarized with LBNP, which was performed on the fourth testing day. Subjects were asked to refrain from strenuous exercise 24 hours prior to all testing.

Anthropometric and Fitness Measurements

Body density, lean weight, and relative fat were determined by hydrostatic weighing (Wilmore and Behnke 1969), using the oxygen dilution technique to assess residual lung volume. Percent body fat was calculated from body density, and lean mass was derived by subtracting calculated total body fat from total body weight. The strength test consisted of measuring peak torques produced from four extensions of each knee performed as rapidly and forcefully as possible at a speed of 60°/sec with a isokinetic dynamometer (Cybex, Lumex, Inc.), which was calibrated daily. The greatest torques produced from the four extensions for each leg were designated as peak torques. To standardize strength for body size, our index of leg strength was peak torque divided by body weight. Peak $\dot{V}O_2$ was measured during the final 30 sec of a standard graded-intensity treadmill protocol (Bruce et al. 1973). Subjects breathed through a two-way respiratory valve (Hans Rudolph Model 2700) while the volume of expired gases and fractional percentages of expired oxygen and carbon dioxide were measured with a metabolic measurement cart (Beckman). Oxygen uptake was calculated from ventilation volume and expired gas measurements using appropriate corrections from the Haldane transformation.

Fitness Profile Classification

Subjects were classified into fitness profiles by peak $\dot{V}O_2$ and leg strength data (Table 1). Peak knee-extension torque greater than

103 percent of body weight for both legs was the criterion for high strength level; 91 percent or less of body weight for both legs was low strength level. Peak $\dot{V}O_2$ greater than 50 ml/(kg•min) was the criterion for high aerobic capacity; less than 45 ml/(kg•min) was average aerobic capacity. Subjects with leg strength between 91 and 103 percent of body weight or with peak $\dot{V}O_2$ between 45 and 50 ml/(kg•min) were excluded from this study. Classification limits for average strength and aerobic capacity profiles were similar to average (±SE) values for leg strength (91 ± 2 percent body weight; N = 74) and peak $\dot{V}O_2$ (45.8 ± 0.9 ml/(kg•min); N = 121) from a population of men studied at the Kennedy Space Center. This combined reference population was similar in age (31 ± 1 yr), height (179 ± 1 cm), and weight (80.6 ± 1.2 kg) to our subjects in the present study.

Based on these dual classifications, six subjects were assigned to each of the following fitness profiles: a) low strength/average aerobic fitness, b) low strength/high aerobic fitness, c) high strength/average aerobic fitness, and d) high strength/high aerobic fitness. The subjects within each fitness profile were matched for age, height, and total body weight (Table 1).

Lower Body Negative Pressure Test

Subjects fasted for at least six hours and abstained from strenuous exercise for at least 24 hours before their LBNP test, which was performed in the morning. Electrocardiogram recording (Frank lead configuration) was used for heart rate determination and medical monitoring. Three electromyogram (EMG) electrodes were placed on the right leg for monitoring muscle activity of the subjects' leg. The electrodes were attached to a signal conditioner for amplification, and the EMG signal was recorded continuously on a vertical strip-chart recorder (Brush). Four silver tape electrodes, two around the neck and two around the thorax, were attached to a impedance cardiograph (Minnesota Model

304B) for impedance rheographic determination of stroke volume according to the technique of Kubicek et al. (1970). This technique has been shown to be accurate when compared with invasive techniques (Judy et al. 1969). In addition, thoracic impedance was measured as an indicator of thoracic fluid volume. Following instrumentation, the subject was assisted into the LBNP chamber. A foam-padded saddle was adjusted for subject stabilization, and an airtight seal was secured between the chamber and waist of the subject at the level of the iliac crests. The left calf was measured at the point of maximal circumference after the subject had been lying supine at least five minutes. A mercury-in-silastic strain gauge was placed at the point of maximal circumference to measure changes in calf circumference during LBNP.

A 21-gauge polyethylene catheter was introduced into an antecubital vein of the right arm for drawing venous blood samples immediately prior to and immediately after LBNP. Patency of the catheter was maintained by a continuous saline drip (approximately 1 ml/min) and by occasional flushing with one ml of heparinized saline (10 units/ml). Thus, multiple venous punctures were avoided.

The LBNP test started with a baseline control period consisting of 20 min resting in the supine posture followed by a 90-min period of 6° head-down tilt (HDT). We chose to employ a period of HDT before LBNP since previous experiments in our laboratory demonstrated shifts in cardiovascular response curves which simulate return to earth after a period of exposure to microgravity (Tomaselli et al. 1990). Following HDT, subjects were returned to the horizontal supine position and were exposed to graded LBNP with a protocol used in the Apollo program (Hoffler et al. 1974). Pressure within the LBNP chamber was decreased relative to ambient pressure to -8 mmHg for 1 min, -16 mmHg for 1 min, -30 mmHg for 3 min, -40 mmHg for 5 min, and -50 mmHg for 5 min. Test termination was based on the following criteria: a)

completion of 5 min at -50 mmHg; b) sudden onset of presyncopal symptoms such as a precipitous fall in systolic pressure greater than 25 mmHg between adjacent 1-min blood pressure readings and/or an abrupt bradycardia greater than 15 beats per min (bpm); c) a progressive diminution in systolic pressure to or below 80 mmHg; and/or d) symptoms of distress such as nausea, dizziness, or discomfort.

Blood pressures, heart rate, leg circumference and impedance data were recorded during the last minute of supine rest and HDT; continuously throughout the LBNP protocol; and at the end of the final stage of LBNP exposure prior to test termination (peak-LBNP). Heart rates were measured directly from a horizontal strip-chart recorder (Brush). Systolic and diastolic blood pressures were noninvasively measured from the left arm every 60 sec with an automated system. Korotkoff sounds superimposed upon a calibrated descending brachial cuff ramp were recorded on the horizontal strip-chart recorder. The first and fourth Korotkoff sounds identified systolic and diastolic pressure, respectively. Mean arterial pressure was calculated by dividing the sum of systolic pressure and twice diastolic pressure by three. Cardiac output was calculated as the product of heart rate and stroke volume. Systemic peripheral resistance was calculated by dividing mean arterial pressure by cardiac output.

Blood Analysis

Blood was collected rapidly and without stasis. A drape suspended across the arm prevented the subject from viewing the blood sampling. Microhematocrit values were determined in duplicate. Plasma osmolality was measured using the freezing point depression method. Plasma arginine vasopressin concentration was determined by the radioimmunoassay technique described by Keil and Severs (1977). Plasma renin activity was analyzed with the modified radioimmunoassay procedure of Haber et al. (1986) utilizing a New England nuclear kit. Plasma epinephrine and norepinephrine levels

were measured with a radioenzymatic technique described by Cryer et al. (1974) using the catechol-0-methyl transferase extraction method of Axelrod and Tomchick (1958). Hormones from all plasma samples were measured in duplicate, and an interassay coefficient of variation of 10 percent or less was required. In addition, just prior to the LBNP test, resting plasma volume was measured using an Evans blue dye dilution technique (Greenleaf et al. 1979), and blood volume was calculated from plasma volume and hematocrit. Percent change in plasma volume following HDT and LBNP was calculated from differences between hematocrit values from supine to HDT and LBNP values according to the method described by Greenleaf et al. (1979).

Statistical Analysis

Descriptive statistics (mean ± SE) were obtained for all hemodynamic and plasma hormone response variables. These statistics were obtained for each combination of aerobic and strength profiles. Each of the physiologic responses measured during LBNP were analyzed using a three-way repeated measures ANOVA with aerobic and strength fitness profiles as between-subject factors and LBNP position as the within-subject factor. Tukey's Multiple Comparison procedure was used when any significant factor differences occured. Correlation coefficients were determined between each hemodynamic and hormonal response to peak LBNP with peak $\dot{V}O_2$ and leg strength. Subjects who became presyncopal were recognized as a subject subset and were not included in the general population for analysis purpose. Instead, two-sample t-tests were performed on peak $\dot{V}O_2$, leg strength, and hemodynamic response variables to test for any differences between tolerant and presyncopal subjects. Multiple regression using a stepwise procedure provided a systematic attempt to build a model for predicting orthostatic hypotension during LBNP using information on the change in mean arterial pressure on peak $\dot{V}O_2$, leg strength, and the twelve potential predictors listed in Table

2. Correlation coefficients were determined between the various responses of physiological variables to LBNP. An alpha level of 0.05 was used for all tests.

RESULTS

Since the fitness profiles were selected based on peak $\dot{V}O_2$ and leg strength criteria, subjects with high aerobic profiles had greater ($P < 0.05$) aerobic capacity than those with average aerobic profiles, while the subjects classified as high strength profiles had greater ($P < 0.05$) leg strength than those with low strength profiles (Table 1). There were no fitness profile differences in age, height, total body weight, or lean body mass.

The mean hemodynamic and hormonal responses of the four fitness profiles at supine, HDT and peak-LBNP are presented in Table 2. HDT resulted in reduced ($P < 0.05$) leg circumference and stroke volume and elevated ($P < 0.05$) mean arterial pressure. Compared to supine and HDT, peak-LBNP caused a reduction ($P < 0.05$) in stroke volume and, consequently, a reduction ($P < 0.05$) in cardiac output, despite a compensatory increase ($P < 0.05$) in heart rate (Table 2). Reduced stroke volume was associated with greater thoracic impedance ($P < 0.05$) at peak-LBNP. As a result of increased ($P < 0.05$) systemic peripheral resistance, mean arterial pressure remained unchanged compared to the supine posture and HDT. Peak-LBNP also increased ($P < 0.05$) leg circumference.

None of the hemodynamic, fluid shift and plasma hormone responses to HDT and LBNP were affected by strength profile (Table 2). In contrast, high aerobic profile was associated with lower ($P < 0.05$) arterial blood pressure, heart rate, and systemic peripheral resistance and greater ($P < 0.05$) stroke volume during HDT and LBNP compared to average aerobic profile.

Plasma norepinephrine and epinephrine increased ($P < 0.05$) with peak-LBNP compared to supine and HDT (Table 2) while plasma osmolality was not altered by any condition. Plasma renin activity was elevated ($P < 0.05$) by peak-LBNP, but plasma vasopressin was increased by peak-LBNP only compared to supine, but not HDT. These blood chemistries were not affected by fitness profile nor by moving from supine to HDT. Correlation coefficients of each hemodynamic and hormonal response, i.e., changes from pre-LBNP to peak LBNP, with peak $\dot{V}O_2$ and leg strength are presented in Table 3. Plasma norepinephrine and percent change in plasma volume at peak LBNP were the only physiological responses that were moderately correlated with either strength or peak $\dot{V}O_2$.

Seven of the 24 subjects demonstrated presyncopal symptoms which resulted in failure to complete the 15-minute LBNP protocol. The average time of LBNP exposure for these subjects was 9.6 ± 1.9 min. Two subjects each with the low strength/average aerobic capacity, high strength/high aerobic capacity, and high strength/average aerobic capacity profiles became presyncopal, while one subject with the low strength/high aerobic capacity profile reached presyncope. The number of presyncopal subjects with each fitness profile was not different (bivariate statistic); and the average time (minutes) of LBNP exposure was 13.4 ± 1.5 for low strength/average aerobic capacity, 12.9 ± 2.1 for high strength/average aerobic capacity, 13.8 ± 1.2 for low strength/high aerobic capacity, and 13.6 ± 1.4 for high strength/high aerobic capacity (ANOVA, $P = 0.90$). Presyncopal subjects had greater ($P < 0.05$) reduction in mean arterial pressure, smaller ($P < 0.05$) elevations in heart rate and systemic peripheral resistance, and greater ($P < 0.05$) increases in vasopressin and epinephrine at peak-LBNP (Table 4). This was manifested by significant bivariate correlations between reduction in mean arterial pressure and changes induced by LBNP in

vasopressin ($r = -0.65$, $P < 0.001$), heart rate ($r = 0.56$, $P < 0.005$), and systemic peripheral resistance ($r = 0.68$, $P < 0.001$).

A multivariate stepwise regression model for prediction of change (Δ) in mean arterial pressure at peak LBNP included only four variables: Δvasopressin, $\%\Delta$plasma volume, Δperipheral resistance, and Δcardiac output, in that order of inclusion. The analysis of variance table resulting from the final step of the regression procedure with the intercept and weights from the prediction equation for each included predictor is reported in Table 5. A correlation coefficient of 0.946 was obtained for the multivariate regression model using changes in vasopressin, plasma volume, peripheral resistance, and cardiac output as the primary contributing factors for the prediction of change in mean arterial pressure, i.e., orthostatic hypotension, from pre-LBNP to peak LBNP. Leg strength and peak $\dot{V}o_2$ were not included in the regression model since their addition contributed less than 0.7% to the prediction of orthostatic hypotension.

DISCUSSION

When compared to the supine and HDT postures, LBNP increased heart rate, peripheral resistance, and leg circumference and decreased stroke volume and cardiac output. Lower stroke volume during LBNP probably reflected a reduced venous return and cardiac filling indicated by concommitant elevations in leg circumference and thoracic impedance. In addition, norepinephrine, epinephrine, vasopressin, and renin activity were elevated by LBNP. These data are consistent with previous observations during various forms of orthostatic challenge (Blomqvist and Stone 1982; Convertino 1987; Davies et al. 1976; Raven et al. 1984; Sather et al. 1986; Smith and Raven 1986). Tolerance to LBNP was evident by the ability to maintain arterial pressure (Table 2) whereas onset of presyncopal symptoms was associated with the inability of cardiovascular reflexes to maintain arterial pressure above 65 mmHg (Table 3).

We employed a multivariate analysis statistic to determine a "best" model for the prediction of hyptension during LBNP. Our model indicated that 95% of the reduction in mean arterial pressure during progressive LBNP could be explained by the magnitude of change in plasma vasopressin, systemic peripheral resistance, cardiac output and plasma volume. In other words, individuals in our study with the largest elevations in vasopressin, smallest elevations in systemic peripheral resistance, and largest reductions in cardiac output and plasma volume during LBNP had a greater predisposition for the development of orthostatic hypotension and syncope.

Since blood pressure varies as the product of cardiac output and systemic peripheral resistance, it is not surprising that these physiological parameters represented significant contributing factors to the prediction of hypotension during LBNP in our tolerant and intolerant (presyncopal) subjects. Our findings are not without precedent since previous investigations have demonstrated that tolerance to orthostatic challenge has been associated with a greater ability for compensatory increases in heart rate and peripheral resistance (Convertino et al. 1986, 1987; Sather et al. 1986). The results of our study are consistent with these findings since tolerant subjects demonstrated greater elevation in heart rate and peripheral resistance than intolerant subjects (Table 4). Our data are consistent with earlier findings (Convertino et al. 1990; Sather et al. 1986) that greater heart rate elevation in tolerant subjects may be associated with greater vagal withdrawal from the heart since their increase in epinephrine was less than, and norepinephrine was not different from, those of intolerant subjects. The inability of syncopal subjects to increase peripheral vascular resistance may reflect significant baroreflex impairment. Similar or greater circulating vasoactive hormones in syncopal compared to tolerant subjects may suggest that dysfunctional receptors may be one element of the reflex that contributed to orthostatic instability in these subjects.

However, inappropriate responsiveness of baroreceptors or neural regulatory integration may also contribute.

Elevation of plasma vasopressin during LBNP occurred in the group of subjects who experienced presyncopal symptoms; the group of subjects who did not become presyncopal showed no alteration in vasopressin. The stimulation of vasopressin secretion may be a last defense against a failing blood pressure control since vasopressin demonstrates strong vasoconstrictive actions and sensitizes baroreceptors (Abboud et al. 1986; Davies et al. 1976). Vasopressin release is known to be induced by elevation of osmolality and/or reduction in blood volume. In our subjects, however, plasma osmolality did not change during LBNP and reduction in plasma volume was not related ($r = 0.14$, $P > 0.50$) to elevated vasopressin. Furthermore, presyncopal subjects did not suffer greater reduction in stroke volume and, thus, probably not a greater decrease in venous return. Therefore, a primary stimulus for vasopressin elevation in our subjects who experienced presyncopal symptoms may be the fall in arterial blood pressure.

We tested the hypothesis that strength and aerobic fitness profiles are determinants of cardiovascular reflex responses and orthostatic stability by measuring various hemodynamic and endocrine responses during HDT and LBNP and documenting episodes of presyncope in subjects with varying combinations of peak $\dot{V}O_2$ and leg strength. Our experimental design was unique in that it allowed us to systematically evaluate the interactions between varying strength and aerobic capacity profiles and their associations with blood pressure regulation during LBNP. We observed some differences in cardiovascular reflex responses between individuals with high and average aerobic profiles independent of strength profile. However, these differences were not associated with episodes of hypotension and syncope induced by LBNP. HDT, which was used to simulate an acute exposure to microgravity prior to LBNP, caused a headward fluid shift as

manifested by significant reduction in leg circumference. Compared to supine posture, 90 min of HDT did not induce significant changes in most resting hemodynamic and endocrine responses that were measured. Except for slight alterations in stroke volume and mean arterial pressure, our results are similar to those reported during the initial 120 min of 5° HDT (Nixon et al. 1982). More importantly, there were no interactions between responses provoked by HDT and aerobic or strength fitness profiles; therefore comparisons of responses to LBNP among groups were not influenced by prior exposure to HDT. Our experimental approach may be the first to demonstrate that various combinations of strength and aerobic capacity profiles do not influence hemodynamic responses and orthostatic stability following acute exposure to microgravity.

Several investigators have reported that high aerobic capacity was associated with orthostatic intolerance (Klein et al. 1977; Luft et al. 1976, 1980; Mangseth and Bernauer 1980). Others have suggested that these observations may reflect attenuated cardiovascular reflex responses in aerobically-trained subjects (Mack et al. 1987, 1991; Raven et al. 1984; Smith and Raven 1986; Smith et al. 1988; Stegemann et al. 1974; Tipton et al. 1982). Weight-trained subjects have demonstrated greater cardiovascular reflex response during LBNP compared to endurance athletes when exposed to LBNP protocols similar to ours (Smith and Raven 1986). These data raise the notion that aerobic fitness may reduce while strength fitness may enhance the effectivness of the blood pressure control system and orthostatic stability (Smith and Raven 1986; Smith et al. 1988; Stegemann et al. 1974). Contrary to this thesis, our data present little evidence to suggest that various combinations of strength and aerobic fitness profiles influence cardiovascular responses and orthostatic stability.

Endurance-trained athletes have also demonstrated less tachycardia response per mmHg decrease in systolic blood pressure during LBNP than weight-trained or nontrained subjects (Raven et al. 1984;

Smith and Raven 1986). This difference in heart rate increase during LBNP was eliminated by both metoprolol and atropine blockade, suggesting that lower cardiac "reactivity" in endurance athletes is due in part to differences in both sympathetic and vagal control of the heart (Smith et al. 1988). Although the subjects with high aerobic capacity in our study demonstrated lower resting heart rates, their higher stroke volumes compensated to maintain cardiac output, and the changes in heart rate and blood pressure during LBNP were not different in any of the fitness profiles. In this regard, our findings corroborate those of an earlier investigation (Raven et al. 1984). Furthermore, the elevation of norepinephrine, an index of sympathetic traffic (Eckberg et al. 1988), did not differ across profiles. Thus, we found little evidence to suggest that cardiac responsiveness was associated with fitness profiles in our subject population.

Another mechanism that has been proposed to contribute to the prevalence of orthostatic intolerance in aerobically-conditioned individuals is an inappropriate regulation of systemic peripheral resistance since attenuated vasoconstriction is associated with low orthostatic intolerance (Convertino et al. 1986, 1987; Sather et al. 1986). In one study (Mangseth and Bernauer 1980), nonrunners demonstrated 38% elevation in peripheral resistance during tilt compared to only 14% increase in runners. This notion is supported by the observation that cardiopulmonary baroreflex control of forearm vascular resistance was attenuated in aerobically-trained individuals (Mack et al. 1987, 1991). We observed less peripheral resistance during rest and at peak LBNP in subjects with high aerobic capacity (Table 2), but with no adverse effects on blood pressure regulation during or tolerance to LBNP. Our results may reflect that less elevation in vascular resistance during cardiopulmonary baroreflex stimulation in aerobically-trained subjects is associated with their larger resting blood volumes (Mack et al. 1987) and elevated resting central venous pressure (Convertino et al. 1991; Shi et al. 1992) rather than a compromised blood pressure regulation.

Greater leg venous compliance and associated venous pooling may contribute to prevalence of orthostatic instability in individuals with high aerobic fitness since endurance-trained runners have demonstrated significantly greater leg compliance during LBNP compared with both weightlifters and sedentary controls (Luft et al. 1976, 1980). However, when muscle mass of the leg is accounted for, leg venous compliance was independent of aerobic and/or strength fitness profiles (Convertino et al. 1988). The results of the present study support this notion since we observed little difference in leg circumference changes during LBNP between aerobic and strength fitness profiles.

The reasons for the discrepancies between the finding that fitness profiles are not associated with cardiovascular reflex responses or orthostatic tolerance (present study, Convertino et al. 1986, 1987; Greenleaf et al. 1985; Klein et al. 1969; Lansimies and Rauhala 1986; Ludwig et al. 1987) and results from other investigators indicating a prevalence of orthostatic instability in endurance trained athletes (Klein et al. 1977; Luft et al. 1976, 1980; Mangseth and Bernauer 1980; Raven et al. 1984) are unclear. It is possible that the specificity of weight training compared to endurance training has dictated physical and physiological adaptations that influence orthostatic stability. If this notion holds true, than combinations of varying strength and aerobic profiles should be associated with varying orthostatic responses. Our results do not support this hypothesis. A more likely explanation may be that the relatively 'high' aerobic fitness levels of our subjects were lower than the 62-70 ml/(kg•min) of endurance-trained runners used in other studies. Taken together, the available data support the notion that an aerobic capacity above a critical threshold of approximately 60 ml/(kg•min) may be a prerequisite for reduced cardiovascular reflex responses and prevalence to orthostatic hypotension in highly trained endurance athletes (Convertino 1987). Since

genetic factors have been shown to influence $\dot{V}o_2max$ (Klissouras et al. 1973), it is possible that the apparent predisposition to orthostatic instability in endurance-trained athletes may represent inherent hemodynamic characteristics associated with support of high aerobic demands. Recent analyses of multivariate data support the hypothesis that genotype predisposition may contribute significantly to cardiovascular reflex control during orthostatic challenges (Convertino et al. 1986; Ludwig et al. 1987). However, further research is needed to test this thesis.

CONCLUSIONS

The major finding of the present investigation was that compensatory hemodynamic, hormonal, and vascular volume responses to head-down tilt and lower body negative pressure were not associated with aerobic capacity or muscular strength. Our findings are consistent with the hypothesis that fitness characteristics and blood pressure regulation during an orthostatic challenge equal to or less than that induced by terrestrial gravity are essentially independent phenomena.

Our results have important implications for the development of selection criteria for exercise programs for astronauts who undergo significant orthostatic challenge upon return from spaceflight or pilots who are exposed to high gravitational forces while flying high performance aircraft. Several cross-sectional comparisons of syncopal episodes and cardiovascular reflex responses during orthostatic challenges between highly trained endurance athletes and nonathletes have raised the concern that endurance exercise training should be minimized and de-emphasized in these populations (Klein et al. 1977; Tipton 1983). The results of the present study, as well as previous investigations (Convertino et al. 1984, 1986, 1987, Klein et al. 1969; Ludwig et al 1987), indicate that maintaining or obtaining a $\dot{V}o_2max$ with regular endurance-type exercise in the range of 30-60 ml/(kg•min)

should not contribute to orthostatic instability. Since the $\dot{V}O_2max$ of our high aerobic fit subjects was 15%-25% above that of the average astronaut (Berry et al. 1980) or high performance aircraft pilot (Banta and Grissett 1987), we find no reason for these individuals to refrain from such exercise activity.

TABLE 1. Subject descriptive data.

VARIABLES	AEROBIC			STRENGTH		
	HIGH		AVERAGE	HIGH		LOW
Age, yr	30 ± 1		32 ± 1	30 ± 1		32 ± 1
Height, cm	179 ± 2		178 ± 1	178 ± 1		180 ± 1
Weight, kg	78.2 ± 2.0		83.4 ± 3.6	79.7 ± 3.1		81.9 ± 2.8
Lean Mass, kg	66.4 ± 1.6		65.6 ± 2.4	67.1 ± 2.3		65.0 ± 1.6
Body Fat, %	15.2 ± 1.2	*	20.5 ± 1.7	15.7 ± 1.1	*	20.0 ± 1.9
Plasma Volume, ml/kg	44.8 ± 0.7	*	38.7 ± 1.3	42.5 ± 1.4		40.9 ± 1.4
Blood Volume, ml/kg	73.6 ± 1.1	*	64.1 ± 2.5	69.4 ± 2.4		68.3 ± 2.4
Peak VO2, ml/kg/min	54.8 ± 1.3	*	40.5 ± 1.1	48.9 ± 2.8		46.3 ± 1.9
Leg Strength, % body weight	93 ± 4		101 ± 6	112 ± 4	*	82 ± 2

Values are mean ± SE
* $P < 0.05$ between Groups

TABLE 2. Hemodynamic and plasma hormone responses at supine, HDT and peak LBNP

VARIABLES		AEROBIC HIGH	AEROBIC LOW	STRENGTH HIGH	STRENGTH LOW
Mean Arterial Pressure, mmHg			†		
supine	A	84 ± 2	87 ± 3	86 ± 3	84 ± 2
HDT	B	85 ± 2	95 ± 2	90 ± 3	90 ± 1
peak LBNP	AB	84 ± 4	91 ± 3	88 ± 4	86 ± 3
Cardiac Output, l/min					
supine	A	4.5 ± .2	4.2 ± .4	4.5 ± .3	4.2 ± .3
HDT	A	4.4 ± .3	3.4 ± .2	4.1 ± .2	3.8 ± .3
peak LBNP	B	3.0 ± .3	2.7 ± .3	3.0 ± .2	2.9 ± .3
Stroke Volume, ml			†		
supine	A	93 ± 4	68 ± 5	83 ± 6	80 ± 7
HDT	B	83 ± 6	52 ± 4	70 ± 7	67 ± 8
peak LBNP	C	44 ± 4	30 ± 3	36 ± 3	39 ± 6
Heart Rate, bpm			†		
supine	A	48 ± 2	62 ± 2	55 ± 4	55 ± 3
HDT	A	53 ± 2	68 ±	60 ± 3	59 ± 6
peak LBNP	B	72 ± 3	94 ± 3	86 ± 5	80 ± 6
Peripheral Resistance, mmHg/l/min			†		
supine	A	19 ± 1	31 ± 8	29 ± 8	21 ± 2
HDT	A	20 ± 2	28 ± 2	23 ± 2	25 ± 2
peak LBNP	B	28 ± 2	35 ± 3	31 ± 3	32 ± 3
Thoracic Impedance, ohms					
supine	A	20.7 ± .4	21.4 ± .6	20.8 ± .5	21.2 ± .6
HDT	A	20.6 ± .4	21.4 ± .6	20.8 ± .5	21.1 ± .6
peak LBNP	B	21.5 ± .6	22.1 ± .7	21.6 ± .5	22.0 ± .7
Leg Circumference, %Δ					
supine	A	—	—	—	—
HDT	B	-0.8 ± .1	-1.8 ± .1	-0.8 ± .1	-0.8 ± .1
peak LBNP	C	0.9 ± .1	0.9 ± .4	0.7 ± .1	1.0 ± .3
Norepinephrine, pg/ml					
supine	A	194 ± 23	187 ± 21	216 ± 26	170 ± 14
HDT	A	228 ± 30	222 ± 28	254 ± 38	202 ± 18
peak LBNP	B	412 ± 36	501 ± 47	502 ± 41	412 ± 41
Epinephrine, pg/ml					
supine	A	25 ± 6	31 ± 9	31 ± 9	24 ± 6
HDT	A	35 ± 9	47 ± 20	56 ± 22	29 ± 8
peak LBNP	B	55 ± 13	94 ± 14	91 ± 17	57 ± 11
Vasopressin, pg/ml					
supine	A	.3 ± .1	.2 ± .1	.1 ± .1	.3 ± .1
HDT	AB	.4 ± .1	.5 ± .4	.1 ± .1	.7 ± .3
peak LBNP	B	.5 ± .2	.7 ± .4	.4 ± .2	.8 ± .3
Renin Activity, ng Ang I/ml/hr					
supine	A	3.0 ± 0.5	2.2 ± 0.4	2.7 ± 0.6	2.6 ± 0.5
HDT	A	3.3 ± 0.5	2.0 ± 0.4	2.5 ± 0.6	2.8 ± 0.5
peak LBNP	B	4.7 ± 1.2	4.7 ± 1.0	4.8 ± 1.5	4.6 ± 0.7
Plasma Volume, %Δ			*		
supine		—	—	—	—
HDT		-1.4 ± 1.5	-1.9 ± 0.9	-2.4 ± 1.2	-1.1 ± 1.2
peak LBNP		-5.2 ± 1.4	-10.4 ± 1.8	-9.2 ± 1.4	-6.2 ± 2.0

Values are mean ± SE.

A, B, C: denotes differences ($P < 0.05$) between stages; same letters are not different

† denotes $P < 0.05$ for high vs low aerobic values

* denotes interaction ($P < 0.05$) between stage and level of aerobic capacity

TABLE 3. Pearson product correlation coefficients (r) describing relationships between aerobic capacity (peak VO2), and leg strength with changes (Δ) in hemodynamics, hormones, and fluid shifts during LBNP.

VARIABLES	Peak VO2	Leg Strength
Δ Mean Arterial Pressure	0.00	0.11
Δ Systolic Pressure	0.02	0.02
Δ Diastolic Pressure	-0.01	0.17
Δ Heart Rate	-0.28	0.09
ΔStroke Volume	-0.22	0.06
Δ Cardiac Output	-0.16	0.09
Δ Peripheral Resistance	-0.10	-0.03
Δ Leg Circumference	-0.08	-0.05
Δ Norepinephrine	-0.30	0.44 *
Δ Epinephrine	-0.09	0.22
Δ Vasopressin	0.08	-0.04
Δ Renin Activity	-0.15	0.21
Δ Plasma Volume	0.44 *	-0.44 *

* $P < 0.05$

TABLE 4. Comparisons of Peak VO2, leg strength, and changes (Δ) in hemodynamic and hormone responses from HDT to Peak LBNP between tolerant and presyncopal subjects

VARIABLES	PRESYNCOPAL (n = 7)		TOLERANT (n = 17)
Peak VO2, ml/kg/min	48.0 ± 2.5		47.5 ± 2.2
Leg Strength, ft-lb	101 ± 10		95 ± 4
Δ Mean Arterial Pressure, mmHg	-24 ± 5	*	-2 ± 2
Δ Heart Rate, bpm	8 ± 6	*	23 ± 2
ΔStroke Volume, ml	-17 ± 8		-32 ± 2
Δ Cardiac Output, l/min	-0.9 ± .3		-1.0 ± .2
Δ Peripheral Resistance, mmHg/l/min	-2.3 ±3.1	*	7.3 ± 1.5
Δ Leg Circumference, %	1.0 ± .2		1.6 ± .2
Δ Norepinephrine, pg/ml	281 ± 80		228 ± 31
Δ Epinephrine, pg/ml	74 ± 29	*	36 ± 7
Δ Vasopressin, pg/ml	5.6 ± 3.1	*	0.2 ± .1
Δ Renin Activity, ng Angl/ml/hr	1.3 ± .7		2.3 ± .6
Δ Plasma Volume, %	-4.8 ± 1.9		-5.7 ± 1.1

Values are mean ± SE
* P < 0.05 between Tolerant and Presyncopal subjects

TABLE 5. Analysis of variance and parameter estimates from "best" model for predicting the change in mean arterial pressure (ΔMAP) from rest to peak LBNP.

Variables	df	Std. Error	Sum of Squares	F Value	Prob > F	Increment to R-squared
Δ Vasopressin (ΔVP)	1	0.19	281.0	41.0	0.000	.570
Δ Cardiac Output (ΔQ)	1	1.60	205.3	29.9	0.001	.155
Δ Plasma Volume (%ΔPV)	1	0.16	435.3	63.5	0.000	.096
Δ Peripheral Resistance (ΔSPR)	1	0.17	361.4	52.7	0.000	.125
Total Model	4		1552.7	56.6	0.000	.946 *
Error	13		89.1			

* - R-squared for the total model

Model Equation: ΔMAP = 4.9 - 1.2 (ΔVP) + 8.7 (ΔQ) + 1.2 (%ΔPV) + 1.3 (ΔSPR)

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V.A. Convertino[1], K.L. Mathes[2], M.L. Lasley[3], C.M. Tomaselli[4], M.A.B. Frey[5], G.W. Hoffler[1]

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Hemodynamic and hormonal responses to lower-body negative pressure (LBNP) were examined in 24 healthy men to test the hypothesis that responsiveness of reflex control of blood pressure during orthostatic stress is associated with strength and/or aerobic capacity. Subjects underwent treadmill tests to determine peak oxygen uptake (peak VO2) and isokinetic dynamometer tests to determine leg strength. Based on predetermined criteria, the subjects were classified into one of four fitness profiles of six subjects each matched for age, height, and weight: (a) low strength/low aerobic fitness; (b) low strength/high aerobic fitness; (c) high strength/low aerobic fitness; and (d) high strength/high aerobic fitness. Following 90 min of 6° head-down tilt (HDT), each subject underwent graded LBNP through -50 mmHg or presyncope, with maximal duration 15 min. All groups exhibited typical hemodynamic, hormonal, and fluid shift responses during LBNP, with no intergroup differences except for catecholamines. Seven subjects, distributed among the four fitness profiles, became presyncopal. Subjects who showed greatest reduction in mean arterial pressure (MAP) during LBNP had greater elevations in vasopressin and lesser increases in heart rate and peripheral resistance. Peak VO2 nor leg strength were correlated with fall in MAP or with syncopal episodes. We conclude that neither aerobic nor strength fitness characteristics are good predictors of responses to LBNP stress.

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