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ABSTRACT

Six normal healthy males between the ages of 26 and 37 were confined to complete bedrest for 28 days. Prebedrest testing established ambulatory control data for each subject. Plasma volumes (PV) were a mean 89% of prebedrest controls at 13 days of bedrest (-375 ml) and 86% of controls at 27 days; blood volumes (BV) were 90% and 89% (-632 ml) of controls at these time intervals. No significant change in extracellular fluid (ECF) or total body water (TBW) was measured. Mean heart rates (HR) from weekly lower body negative pressure (LBNP) tests (-50 mmHg) increased linearly with duration of bedrest (r=.94). At the end of bedrest the legs showed a 600 ml decrease too large to be accounted for by the decrease in intravascular volume, suggesting redistribution in ECF and/or muscla atrophy. After both two and four weeks of bedrest, attempts were made to reverse the PV and HR changes. Each subject ingested one liter of isotonic saline, as commercial bouillon, during the application of -30 mmHg LBNP for 4 hours. This treatment was compared to saline ingestion alone and to no treatment in a crossover design on days 13, 14, and 15 of bedrest. The LBNP + saline treatment increased PV 186 ml, while saline alone increased PV 416 ml. Nonetheless, the increase in HR during LBNP stress was less after the combined treatment than after saline alone. Plasma volumes remained slightly elevated the morning after the LBNP + saline treatment, but HR response to LBNP had increased to pretreatment levels. After 4 weeks of bedrest, the LBNP + saline was again able to lower LBNP stressed HRs to prebedrest levels. The LBNP + saline treatment is an effective, though

transient counter measure against bedrest induced orthostasis, as measured by LBNP stress. This bedrest study is analyzed in the context of data collected from spaceflight and other bedrest studies, to assess the role of BV and other fluid compartments in the orthostasis post bedrest and postflight.

The Skylab experience has proven that man can work and live in a weightless environment for extended periods of time. Successive missions put man into space for 28, 59, and 84 days. Extensive medical experiments documented many of the changes effected by spaceflight. As in the previous manned missions, decrements in weight, plasma volume (PV), red cell mass (RCM), orthostatic tolerance, and exercise tolerance were seen postflight. Although it is difficult to compare the magnitude of change among the flights because of the small crews (3 men/flight) and individual variation, most changes did not seem to progress with increasing flight duration. Only PV seemed to have decreased the most after the longest mission. The greatest deficit in RCM was seen after the shortest mission; the greatest % weight loss after the middle mission; orthostatic intolerance was reportedly least in the crew of the longest mission; and the postflight decrement in exercise tolerance which was approximately equal in all crews. Any conclusions about the duration of flight and specific physical decrements must be drawn though with some caution, since the amount of personal exercise performed by the three successive crews increased, and this increase could have halted any progressive change which might otherwise have o urred. Skylab 3 crews exercised, on a daily basis, approximately twice the amount of time that the Skylab 2 crew exercised, and the Skylab 4 crew spent almost three times longer on daily exercise than did the Skylab 2 crew (5).

While no postmission physiological change has hindered dramatically the astronaut's ability to function, it would be advantageous to have a

countermeasure to protect future astronauts and lay crewmembers against orthostatic intolerance on return from orbit, particularly in the projected shuttle flights where the centrifugal forces during reentry will be along the Z (long) axis of the body, instead of the X axis (chest to back) as it was in Skylab and Apollo flights. In the past, several countermeasures have been tested in ground based studies including inflatable pressure cuffs around the extremities, hypoxia, anti-gravity suits, fluid replacement, plasma volume expanders such as 9-alpha flurohydrocortisone, and LBNP used for various periods. Most countermeasures have assumed that the decreased orthostatic, acceleration, and exercise tolerances are at least in part the result of decreased PV. It is not clear, however, whether hypovolemia alone is adequate to explain completely these intolerances.

In attempting to determine the etiology of spaceflight changes and to test proposed countermeasures, investigators have chosen bedrest as a convenient analog. Bedrest, like spaceflight, reduces gravitational force along the Z axis, although to a lesser degree than spaceflight, and produces physical confinement and muscular inactivity which were problematic for the early spaceflights. Like spaceflight, bedrest produces calcium loss, decreased PV and RCM, and orthostatic and exercise intolerances.

The 28 day bedrest study reported here began as a simulation of Skylab II so that comparative ground based data could be accumulated using the same methodology. It gradually evolved into a more problem oriented study as tests were added which had been precluded by the mission constraints. Weekly PVs, bicycle ergometer tests and LBNP tests with a midpoint RCM

determination were performed so that the time course of physiological changes could be documented and the inter-relationships analyzed. Additionally, a counter measure involving bouillon ingestion during application of -30 mmHg LBNP, which had been used successfully by Hyatt et al., was evaluated in terms of its ability to restore PV and exercise and orthostatic tolerance to prebedrest levels (15).

METHODS

The subjects were healthy male paid volunteers between the ages of 26 and 37 who were physically active but not athletes. Their heights ranged from 1.68 to 1.83 meters and their weights from 62.7 to 76.3 kilograms; all were within 5% of ideal body weight. These subjects were selected from a large pool of applicants after being screened by a physician and a psychiatrist. The screening physical included a CBC, SMAC, urinalysis, exercise EKG and LBNP test. During a three week prebedrest control period, the subjects maintained normal activity schedules while eating the calorie and nutrient controlled Skylab diet. The Skylab flight diet was designed to allow each person to choose a palatable diet which could contain a range of nutrients and electrolytes. For example, dietary sodium could be chosen from 130-360 mEq per day, but after a sodium level was selected during a prestudy dietary trial, sodium was not allowed to vary more than 22 mEq/day. Other nutrients were handled similarly.

Prebedrest testing during the first three weeks established baseline control values for all test parameters. The subjects were confined for

28 days of absolute bedrest in The Methodist Hospital, Houston, Texas. Subjects were allowed free movement in the horizontal position but were not allowed to sit or elevate the knees. A two week recovery period with dietary control followed the bedrest period.

The saline solution countermeasure described by Hyatt was performed on days 13, 14, and 15 of the bedrest period utilizing a randomized three way crossover experimental design which confounded the treatment order. Saline was given as bouillon to make it more palatable to the subjects. Water was allowed ad libitum. PV and LBNP tests were performed before and after the following treatments:

(1) ingestion of 1 liter of bouillon per 70 K body weight during application for four hours of LBNP at -30 mmHg

(2) ingestion of the same amount of bouillon at the same rate as treatment #1

(3) water ad libitum for four hours.

The one liter of bouillon solution given (hereafter referred to as saline) was diluted to contain 155 mEq/L Na, 1.2 mEq/L K, and 151 mEq/L Cl. On the day the subjects arose from 28 days in bed, treatment #1 was repeated for all subjects after four hours of tests, including LBNP, supine and upright bicycle ergometry, and vestibular testing. LBNP and exercise responses were again tested following treatment.

Subjects were tested for orthostatic tolerance with LBNP (13) five times during the dietary control period, approximately weekly during bedrest, twice daily during the countermeasure study, twice on the day the

subjects arose, and 1, 2, and 8 days later (Post bedrest or Recovery (R)+1, R+2, and R+8). Tests for a given subject were performed at approximately the same time of day and at least one hour postprandial. The subjects' LBNP tests always preceded vestibular and bicycle ergometry testing. The LBNP protocol consisted of a 5 minute supine rest period, 1 minute at -8 mmHg, 1 minute at -16, 3 minutes at -30, 5 minutes at -40, 5 minutes at -50 and a 5 minute recovery period. Percentage change in leg volume during the LBNP experiment was measured by capacitance plethysomography (12). Resting leg volumes prior to the LBNP were estimated by measuring multiple circumferences manually using a modification of the Jobst tape.

Plasma volumes were determined utilizing ^{125}I human serum albumin (17) twice during the bedrest control period (Bedrest (BR)-40, BR-0), weekly during bedrest and daily during the countermeasure study days. AM PVs were performed after an overnight fast with the subject remaining supine in bed. During the countermeasure study PM PVs were measured during the last half hour of the three treatments and before the second LBNP test. This was four hours after eating. Red cell masses utilizing ^{51}Cr (17) were determined five times during this period (BR-40, BR-0, BR+13, BR+27, and R+14). Blood volumes were calculated from the RCM and PV. Three extracellular fluid (ECF) volumes (BR-40, BR-0, and BR+27) were measured from extrapolation to zero time of the serum $^{35}SO_4$ activity from the least squares fit of 5 samples obtained 30-240 minutes after injection of sodium sulfate (^{35}S) (17). Each time ECF was determined total body water (TBW) was measured using $^{3}H_2O$ specific activity of urine specimens collected

3-8 hours post injection of $^{3}H_2O$ (17). Water and food intake were allowed in the interim.

Analysis of variance and linear correlations were performed to determine statistical differences which are defined as significant when $p<.05$. For N's less than 5 a normal distribution was not assumed and the Friedman 2 way analysis of variance using rank order was performed.

RESULTS

The results of the plasma volume (PV), extracellular fluid (ECF) and total body water (TBW) data are summarized in Table 1. Mean control PVs performed forty days apart, forty days before the beginning of bedrest and on the day the subjects went to bed, were not significantly different, varying only 32 ml. At the time of the BR-40 PVs, the subjects were not eating a controlled diet indicating that neither the Skylab diet nor climate changes in Houston between April and June produced a significant change.

Because of a natural disaster (a flood) which caused several days of electric power loss, only three subjects were tested on BR+7. These pre-breakfast PVs demonstrated a significant decrease at BR+7 which was equal to the mean decrease of the same subjects on BR+13. The mean percentage decrease for all six subjects was 89% of control at day 13, 84% at day 21, and 86% at day 27 and returning to normal two weeks post bedrest. Statistical analysis showed no significant difference among the ambulatory values on BR-40, BR-0, and R+13, but the bedrest PVs were statistically lower

than the ambulatory means on BR+13, BR+21, and BR+27 ($p<0.05$).

We measured ECF both 40 days prior to the beginning of bedrest and the day the subjects went to bed. These were meaned to obtain a control which was compared to the mean ECF after 27 days of bedrest. ECF decreased a mean 2.5% which was not statistically different. This decrease amounts to 8 ml/Kg body weight and is nearly completely accounted for by the statistically significant PV decrease of 6.8 ml/Kg body weight. No significant decrease in total body water was found.

Table 2 presents the mean blood volume data, heart rate data during LBNP, the left leg volumes and weights. The control BV represents the mean of the two prebedrest measurements. Blood volume decreased 10% during the first two weeks and an additional 1% during the second two weeks. The BV during bedrest like PV were statistically different from the ambulatory means.

For each subject a 5 minute mean resting heart rate and stressed heart rate during -50 mmHg were obtained on each LBNP test day. The mean of the four prebedrest tests was utilized as baseline value. Resting HR increased gradually during bedrest reaching a peak of 68 two hours after ambulation on day 28. The resting HRs were statistically different from the baseline values at 2, 24, and 48 hours post ambulation. Stressed HRs (-50 mmHg LBNP) increased linearly during bedrest ($r=.94$). After the day 7/11 tests and until two days postbedrest the stressed HR means were statistically different from the mean baseline.

The 7.8 liter left leg volume is the mean of four measurements during

the prebedrest control period. This mean was subtracted from the leg volumes measured during bedrest to obtain the change in leg volume. Resting leg volume decreased during bedrest. Statistical analysis indicated that the mean differences on BR+21 and BR+27 were different from a zero change. During the bedrest period there was a maximal decrease of 300 ml. We can assume that the contralateral leg decreased similarly indicating a 600 ml volume loss. Mean body weight showed no difference of statistical significance during the study period.

The data from the countermeasure study are reported in Table 3. When subjects received no treatment, HR increased 32 beats per minute (bpm) over the resting level. This increase is nearly identical to the 33 bpm increase on the morning of day 13, the first day of the countermeasure. Both saline and saline + LBNP lowered stressed HR significantly, with the combined treatment reducing HR to prebedrest levels. However, the protective effect was transient, and mean HR had increased to pretreatment levels by the next morning. PV increased 186 ml when saline was ingested during -30 mmHg LBNP, 416 ml for saline alone and 81 ml for no treatment. The three means were statistically different from zero but only the saline was different from the control mean. The following AM, a statistically significant increase in PV of 218 ml remained after the combined treatment only.

LBNP + saline treatment produced a statistically significant 246 ml increase in left leg size after 2 weeks of bedrest and a 323 ml increase after 4 weeks or approximately 500-600 ml increase in total leg volume. Neither of the other two treatments produced a statistically significant

increase. Since BV increased only 186 ml during the LBNP + saline treatment, additional fluid must have been sequestered in the legs, probably in the interstitial space.

DISCUSSION

When data from comparable periods of spaceflight and bedrest are examined we find that the BV losses are quite similar. After the 28 day Skylab II, BV was 92% of the control while after 28 days of bedrest BV was 89% of control (18). Although the decreases in BV seen postflight and postbedrest have been associated with decreased LBNP, orthostatic and exercise tolerance, the exact relationship among the changes has not been determined.

Analyzing postflight results, Hoffler found a correlation of statistical significance ($r=-0.54$) between percentage decrease in BV and percentage decrease in orthostatically stressed HR (11). Stevens et al. found a similar correlation between PV decrease and increase in orthostatically stressed HR after a hypoxic bedrest study (26). On the other hand, in the Chobanian study, PV change was greatest at one week, but orthostatically stressed HR increased progressively with bedrest duration (6). In this 28 day bedrest study we found a linear declind in orthostatic tolerance as assessed by the increase in HR during application of -50 mmHg LBNP stress ($r=.94$). BV, on the other hand, decreased 10% during the first two weeks and only an additional 1% during the second two weeks.

When the countermeasure was tested we found no quantitative correlation between PV increasea and lowering of HRs during LBNP stress. Both saline alone and the LBNP + saline treatments increased PV and orthostatic tolerance. PV increase, however, was greatest with saline alone (+416 ml), while LBNP + saline had a slightly but not significantly greater protective effect than saline alone. In contrast, Hyatt produced a 444 ml PV increase and a statistically significant lowering of LBNP stressed HR when LBNP treatment was combined with saline ingestion. Saline alone had a lesser effect, a 213 ml increase in PV and no statistically significant decrease in HR (15). Differences in the timing of the PV tests could explain the different results. In the Hyatt study PV determinations were performed two hours after LBNP testing instead of during the last 30 minutes of the 4 hour LBNP treatment. The additional 2 hours would have allowed time for diuresis of the saline solution and could explain Hyatt's lower PVs after only saline treatment. The additional time before PV measurement would have allowed interstitial fluid sequestered during the LBNP + saline treatment to return to the vascular space. We found approximately 500-600 ml of fluid sequestered in the legs during the saline + LBNP treatment. This fluid would return to the vascular system slowly. PV would be expected to increase after the saline + LBNP treatment. The two studies suggest it increases about 200 ml in two hours.

By the following morning, mean PV remained elevated about 200 ml in the saline + LBNP group showing delayed mobilization and diuresis of sequestered fluid, but both leg volumes and HRs have returned to pretreatment levels. Twenty-four hour urinary aldosterone excretion was found by

Leach to be elevated on the days when LBNP treatment was given and would help explain the residual PV elevation twenty hours after LBNP treatment (21).

At the end of the 28 bedrest days, all subjects received saline during a 4 hour LBNP treatment. Again this significantly lowered HRs during -50 mmHb LBNP testing to prebedrest levels. By the next day HR had increased to 26 bpm even though the subjects had been ambulatory in the interim, indicating that the LBNP + saline treatment is an effective, though transient, countermeasure.

Hyatt et al. at one time postulated that the deconditioning of bedrest might be largely the result of extravascular dehydration, defined by them as a decrease in the lower extremity interstitial fluid. After finding a negative water balance in bedrest subjects, they hypothesized that ECF volume was decreased and in a later study actually measured a 5% decrease using radioactive promide space as a measure of ECF (14,16). Theroetically extravascular dehydration might cause a larger extravasation of plasma during LBNP testing at a time when PV was already low, helping to explain the increased HR response of bedrested subjects during tilt or LBNP testing. We, however, have found an insignificant 2.5% ECF decrease in this 28 day study which was almost entirely accounted for by PV loss. ECF decreases were also small (-0.8%) after the 28 day Skylab II and were generally small after all spaceflights. The greatest mean decrease, which was still small (-216%) was noted after Apollo 15. In each testing sequence postflight orthostatic intolerance existed.

These bedrested subjects did not lose body weight and their ECFs remained unchanged. Yet within a week of the start of bedrest the leg volume showed a 3% mean decrease, seemingly too large to be accounted for by a BV change alone. A decrease in leg size without a comparable change in subject weight indicates a movement of ECF perhaps to the dependent portions of the body. Biostereometric analysis of body form confirmed this by showing an increase in the midriff volume when the subjects arose from bedrest on BR+28 (10).

An even greater redistribution of ECF seems to occur during spaceflight. Dramatic leg volume changes (-6%) have been measured as early as 6 hours into flight (11). These reached 14% the day prior to reentry. The first postflight leg volume measurements showed a 9% decrease in leg volume. This is more than four times as large as the 2% changes found in bedrested subjects at a comparable measurement period (R+2 hours) (10, 11). Astronauts complain of "head fullness" and nasal stuffiness, and photographs showed facial plethora and distended neck and scalp veins indicating venous redistribution which could be associated with an ECF redistribution. Astronauts, unlike bedrested subjects lose weight. Since postflight ECF measurements showed non-significant changes, the weight loss of spaceflight is more likely the result of deficient food and water intake rather than a diuresis of interstitial fluid.

During and after bedrest relatively consistent shifts in PV seemed to occur during LBNP but the results do not indicate a tendency toward increased extravasation or venous pooling per se. Leg volume increases with LBNP calculated from capacitance plethysmography were about 3% even when plasma volume was expanded with saline during the countermeasure study.

Several investigators including Menninger, Bartok, and Hoffler have attempted to demonstrate more dramatic fluid shifts postbedrest than prebedrest, but the % change in leg volume during LBNP or tilt testing are similar (1,22). No change may actually occur or the techniques used may not have been sensitive enough to measure the small change. These findings differed from those during spaceflight where the increase in leg volume caused by maximum LBNP was 83% greater than on earth (19). Vascular fluids must be far more labile in the zero gravity environment making fluid shifts into the legs more dramatic.

Van Beaumont et al., who reduced PV by dehydration or bedrest, found an inverse relationship between plasma extravasation and absolute PV using centrifugation as the stress (2). They found a twofold greater loss of PV following centrifugation prebedrest than postbedrest, although acceleration tolerance was reduced postbedrest. Their studies would indicate that absolute PV or BV rather than enhanced extravasation of the BV into fluid deficient legs is more important in determining the amount of fluid lost from the vascular space.

Techniques used to increase orthostatic tolerance have been directed at increasing PV and ECF or changing its distribution. For example, in the previously mentioned hypoxic bedrest study, LBNP was used for 10 hours a day for two consecutive days to restore PV (27). Bohnn et al. have also used 9-alpha flurohydrocortisone to correct bedrest induced PV decreases and to prevent the orthostatic instability of bedrest (4). Stevens et al. used both drugs and occlusive cuffs but found that while both restore PV, neither had any significant effect on the HR response to tilt (26). In our study a treatment using LBNP and saline ingestion reduced the HR response to LBNP stress while increasing PV and ECF in the legs.

Also important to the deconditioning of bedrest and of at least the early spaceflights is the relative physical inactivity of subjects or crewmembers. In most of the early missions the very athletic astronauts who had trained assiduously preflight became abruptly inactive during flight. Muscle groups went unused, and the long bones were no longer needed to support the body's weight; muscle and osseous atrophy resulted. Heart size calculated from upright chest films is smaller both postflight and postbedrest (13, 23) and one investigator found that the decrease persisted even in the supine position postbedrest (25). Significant decrements in upright exercise tolerance have been seen after every flight and in both supine and upright exercise after bedrest (14,22,25,26,29).

In normal subjects, however, who were thermally dehydrated to produce PV deficits of up to 25%, increased HRs were seen only in the upright position (24). Saltin's study indicates that in the supine position reduced BV alone could not produce increased HR response to exercise. In this bedrest study Sawin found slight lowering of supine exercise HRs at the beginning of bedrest, indicating that the lowered BV was adequate or even more suitable to the supine position (26). Only later (BR+21) did the HRs increase during supine exercise. In the Stevens hypoxic study HR response to a given exercise load was much higher if the PV had not been re-expanded by LBNP (27). However, maximum oxygen consumption was decreased in all subjects following bedrest indicating that maximum cardiac output remains below the prebedrest levels even after correction of BV.

There appears, therefore, to be at least two factors contributing to the so-called cardiovascular deconditioning of bedrest, one relating to

BV loss and possible ECF redistribution and one relating to inactivity. When exercise levels are high during spaceflight, the inactivity factor would become less important. This would possibly explain why some spaceflight induced physical changes were not progressive during Skylab. In bedrest fluid volume deficits and redistribution are probably more important in the early stages in that PV decreases rapidly during bedrest reaching a 10% deficit in a few days. Later in bedrest, non BV related factors may become important. Even though PV and BV slowed their rate of change after two weeks, the LBNP stressed HR increased linearly with increasing bedrest duration ($r=.94$).

The countermeasure which adequately replaced lost fluid volume also returned HRs during LBNP stress to prebedrest levels but was only transiently effective. During the countermeasure study after two weeks of bedrest, no improvement in the HR response to supine exercise was produced by either LBNP + saline or saline alone. On R+1 even though the subjects had been ambulatory after the previous day's testing, HRs were again elevated during LBNP testing. Postbedrest HRs remained elevated during bicycle ergometry even after BV had returned to control levels and were elevated during supine exercise.

Even larger PV deficits (up to 25%) in non-bedrested subjects fail to produce elevated HRs. This indicates that lost fluid volume could not be the only cause of the elevated supine exercise HRs. Greenleaf was also unable to explain acceleration intolerance merely on the basis of decreased BV. He found that blood transfusion and oral isotonic fluid consumption (11 ml/Kg) restored acceleration tolerance in hypovolemic ambulatory individuals bled 500 ml (7). However, after 14 days of bedrest a twofold greater

consumption (23 ml/Kg) of a hypertonic electrolyte drink (620 osm/l) restored only two-thirds of a 35% loss in +Gz tolerance (8). It appears, however, that bedrest and spaceflight produce physiological changes other than a BV loss which contribute to orthostatic and acceleration intolerance.

Nonetheless, whether fluid loss totally explains these intolerances, fluid replacement has proven effective in eliminating or helping to alleviate them temporarily. The LBNP + saline treatment, in particular, has proven effective in increasing BV and reducing LBNP stressed HR increases. Hyatt has tested the countermeasure using both 2 and 4 hours of LBNP after 7 days of bedrest (15) and we have tested the countermeasure using four hours of LBNP after 14 and 28 days of bedrest. In each case the treatment was able to reduce LBNP stressed HRs to prebedrest levels. The LBNP treatment also increased leg volume, presumably by sequestering fluid in the interstitial space, and caused an increased aldosterone secretion. The changes in spaceflight are viewed as homeostatic in nature rather than pathological since they only cause problems early during the postflight interval. A countermeasure which would effectively prevent acceleration intolerance and syncope during the early postflight period is perhaps all that is called for, since all the physiologic changes mentioned here, have been found to be reversible even without treatment. Preflight physical status is usually accomplished by about a month postflight. When exercise levels are high, as they were in the 84 day Skylab IV mission, changes produced by inactivity may be precluded, so that the dominant problem in that type of mission would be the more easily correctable postmission hypovolemia.

Table 1

Plasma Volume, Extracellular Fluid, and Total Body Water During 28 Days of Bedrest

	PV		ECF		TBW	
	ml	% of Control	Liters	% of Control	Liters	% of Control
BR-40	3420		14.0		45.6	
BR-0	3388		14.1		45.5	
BR+13	3015	89				
BR+21	2844	84				
BR+27	2918	86	13.7	98	45.9	101
Post BR+13	3434	101				
Overall SE	69	2	0.2	2	0.3	1

Table 2

Mean Blood Volume, Heart Rates, and Leg Volumes
During 28 Days of Bedrest

	BV ml	BV % of Mean Control	HR (bpm) Resting	HR (bpm) -50 mmHg	Δ Leg Volume ml	Weight Kilograms
Mean Control	5570		57	73	7804	70.0
BR+7/11			60	90	-204	70.3
BR+13	5113	90	62	95	-145	70.1
BR+14			59	94	-142	70.3
BR+15			60	92	-198	70.3
BR+21			65	102*	-245	69.6
BR+27	4938	89	64	107	-300	69.8
Up 2 Hrs			68	104	-184	
Post BR+1			69	95	-143	70.7
Post BR+2			66	90	- 40	70.1
Post BR+8			61	79	- 57	70.2
Post BR+13	5488	96				69.7
Overall SE	71	2	2	4	47	2.0

* Five subjects

Table 3

Plasma Volume and LBNP Results During the Saline Countermeasure Study

	Δ Heart Rate (bpm)		Δ Plasma Volume (ml)		Δ Resting Leg Volume (ml)
	-50 LBNP	Next AM	AM-PM	AM Next AM	AM-PM Same Day
Pre Bedrest	15				
BR+13	33				
Countermeasure					
Control	32	36*	81	-66	101
Saline	22	35*	416	69	59
Saline + LBNP	18	30*	186	218	264
BR+27	43				
Post BR+2 hours	35				
PM Saline + LBNP	18				323
Overall SE	3	4	86	115	71

*Five subjects

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