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USE OF LOWER BODY NEGATIVE PRESSURE AS A COUNTERMEASURE TO NEGATIVE Gz ACCELERATION

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FOR THE COMMANDER

JAMES W. BRINKLEY
Director
Biodynamics and Bioengineering Division
Harry G. Armstrong Aerospace Medical Research Laboratory

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Lower body negative pressure (LBNP) was used at levels of 0, -50, and -100 torr as a countermeasure prior to centrifuge exposures of subjects to negative Gz at levels of -1.0, -1.5, and -2.0. EKG, echocardiographic, and subjective data were gathered. Physiological variables for end diastolic volume (EDV), end systolic volume (ESV), stroke volume (SV), heart rate (HR), and cardiac output (CO) were obtained at baseline values, then percent of baseline after LBNP was started, then during the -Gz exposure plateau. ANOVA revealed that the LBNP main effects were significant for EDV,ESV,SV, and HR ($p < .0027$). The negative Gz main effects were not significant for EDV,ESV, & SV ($p > .3821$), but were significant for HR and CO ($p < .0001$). Paired T-tests showed the LBNP of -50 and -100 torr lessened the HR changes during -Gz as compared to 0 LBNP ($p < .0199$). LBNP retained its effects for EDV,ESV, HR, and CO ($p < .0017$). The subjective results showed pairwise differences ($p = .0312$) in ratings between -2.0 Gz exposure unprotected and -2.0 Gz protected for facial congestion, sinus pain, and degree of comfort. Overall rankings of exposures also demonstrated that LBNP protected against negative Gz effects at $p = .028$.

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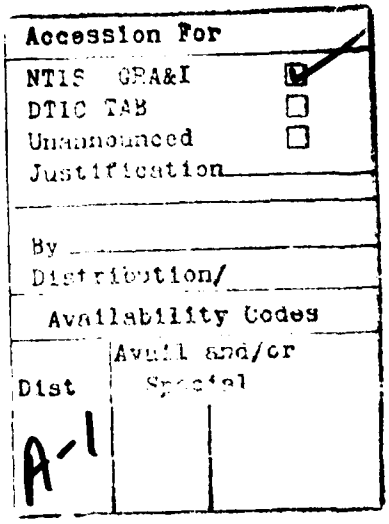
PREFACE

Support for this thesis was provided by the Acceleration Effects Branch of the Biodynamics and Bioengineering Division of the Harry G. Armstrong Aerospace Medical Research Laboratory at Wright Patterson AFB, Ohio 45433-6573. The dynamic portion of the research was carried out using the AAMRL human centrifuge, the Dynamic Environment Simulator, with engineering support from the Raytheon Service Company and scientific and technical support provided by the civilian and military members of the Acceleration Effects Branch. Development of the lower body negative pressure suit was funded by a FY-87 Laboratory Director's Fund grant. This work was accomplished under Project 7231, Task 35; Advanced Acceleration Protection Concepts.



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I. INTRODUCTION

The velocity and acceleration capabilities of aircraft developed in the years since the Wright brothers have truly set man free from the bonds of 1G earth. However, in achieving these capabilities, it has been discovered that man has certain physiological limits. G-induced loss of consciousness (GLOC) is a well documented effect that can occur during positive Gz (+Gz) acceleration of a certain duration and intensity that can produce incapacitation in pilots and can result in a fatal accident.

Ongoing work utilizing centrifuge studies is designed to develop methods to protect the occupant of high G, high speed aircraft from the adverse effects of positive Gz. Methods have included the formation and development of the G suit and valve, the development of the M-1/L-1 straining maneuver, seat tilt back, and more recently positive pressure breathing (8,64). However, as engineering development has progressed through these past four decades of supersonic flight it has become obvious the limiting factor to improved aircraft performance and capability will be physiological and not limited by the aircraft itself.

Most aircraft maneuvers and tactics involve turning so that force remains in a positive Gz (+Gz) or positive Gx (+Gx) direction. Negative Gz (-Gz) maneuvers have been specifically avoided because of unpleasant sensations and adverse physiological effects. As a consequence aircraft have been primarily built to withstand +Gx and +Gz forces. Aircraft

acceleration tolerances are now greater than human tolerances in most of the six degrees of freedom.

Relatively little work has been done in order to protect the pilot from -Gz acceleration. In an effort to try to expand the operational envelope of present fighter aircraft as well as to increase the operational and technological envelope of future designs in aircraft, this work was done to study -Gz acceleration, i.e. foot to head acceleration.

II. BACKGROUND

A. NEGATIVE Gz ACCELERATION

1. HISTORICAL

Negative Gz acceleration was first reported in use as a medical therapy for neurosis in Berlin in 1818. Patients were exposed to -4 Gz for an unknown length of time. The treatment results were a high subjective improvement rate and marked reddening and petechial hemorrhages about the face (71). Early authors described that -Gz acceleration may be accompanied by conjunctival hemorrhage, severe headache, mental confusion, muscle incoordination, unconsciousness, and staggering gait for several hours after exposure. Also reported were intense congestion in the head and face, a sensation of the eyes protruding and skull expanding, and a free flow of tears (27).

2. PHYSIOLOGICAL STUDIES

a. Comparison to Positive Gz Acceleration

During +Gz acceleration with a rate of onset from 1 - 2 G per second there is an immediate increase of blood pressure at the hip level, a reflex increase in heart rate, and decrease in blood volume measured at the ear. During exposures of greater than 30 seconds of +2 to +4 Gz the cardiac output diminishes, stroke volume diminishes, heart rate increases, the mean aortic pressure increases and the systemic vascular resistance increases. The effects increase as the magnitude of acceleration increases from 2 to 4. These effects are thought to be due to the decrease in hydrostatic pressures in the superior portion of the cardiovascular system due to the increase in the arterial pressure gradient that results from the increased G field (45).

Early +Gz studies demonstrated the effect of falling venous pressures measured in the jugular vein of the neck during 30 second +4 Gz exposures. Arterial and venous pressure both drop. However, the arterial-venous pressure difference is relatively maintained (28).

b. Negative Gz Studies

During the -Gz acceleration the direction of the increased hydrostatic pressure is reversed. The tendency under -Gz is for blood to pool in the blood vessels of the head and superior parts of the body. This has been used to explain the physiological effects.

Research was done at the Armstrong Aeromedical Lab in the 1950's involving dogs and subjecting them to -7 Gz. Observations revealed that venous pressure increased with the acceleration from 0 to approximately 60

mm Hg (measured at the external auditory meatus), and that arterial oxygen saturation fell to 60-65%. After the run both recover within fifteen to thirty seconds (19). Refer to Figure 1.

FIGURE 1 Physiological Data from -7 Gz Animal Experiment

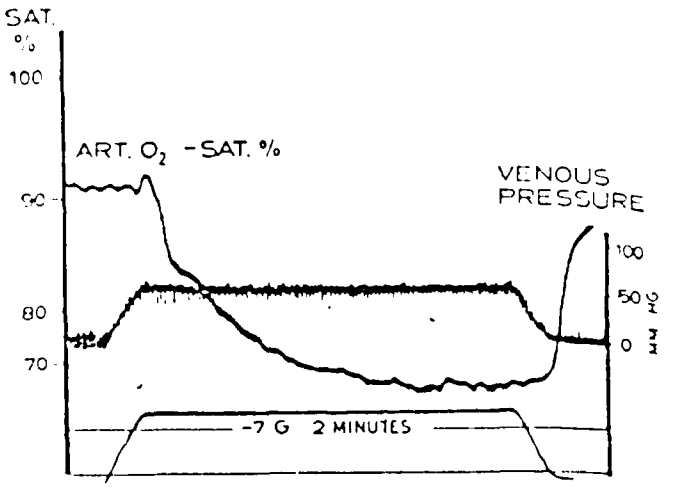


FIG. 2. Record obtained during exposure of an anesthetized dog to 7 negative G for 2 minutes. Venous pressure increases with the acceleration and arterial oxygen saturation falls to 60 to 65 per cent with recovery after the run. Pressures taken at the level of the external auditory meatus.

Reproduced from Gauer, Henry, et al (19).

Shaw and Henry did human experiments under -Gz acceleration to -3 Gz determining venous pressure changes in the forehead in seated subjects. The pressures in the forehead were found to vary linearly with the acceleration as one may find with a simple hydrostatic column of constant length. The position of the subjects was not varied. Chest X-rays were taken during the centrifuge runs. There was found to be no gross variation in the position of the heart. The diaphragm was found to remain at a constant level, but at -3 Gz the lateral portions were elevated minimally (65). Previous experiments had shown that under negative acceleration the

central venous system may receive a sudden autotransfusion of blood from the lower extremities and lower abdomen. There are estimates of about 800 cc of blood being available in the venous system of the legs (10). In addition reflex bradycardia interferes with the heart's ability to increase cardiac output to accommodate increased venous return. This combination results in a transient increase in right atrial pressures during these 15 second exposures (65).

Gauer and Henry did work in order to predict the venous pressures at various angles of tilt. It was discovered that venous pressure at the head depended not only on the distance from the heart to the head but the distance from the heart to the feet influencing the length of the venous column. Investigations with humans were performed subjecting them to -2 Gz with blood pressure cuffs inflated around the legs which effectively cut off the arterial and venous circulation to and from the lower extremities. Measured venous pressures were approximately 40 mm Hg which rose 10 mm Hg upon release of the cuffs. Further studies showed a change in pressures could be induced by bending the knees into a forward crouch position thereby reducing the effective length of the column and reducing venous pressures (19).

It was thought that, in human and animal experiments, the rise in central venous pressure during -Gz acceleration could be due to the over-distention of the cephalad venous reservoirs with blood. This hypothesis was studied by bleeding a number of dogs 20-30% of their estimated blood volume. Venous pressures at the level of the head during -Gz were measured before and after hemorrhage. Venous pressures were measured to be approximately 200 mm Hg during -10 Gz accelerations and were noted to drop to 150 mm Hg during the same acceleration after

phlebotomy. Removing 450 cc of blood reduced the venous pressure to 25-40 mm Hg (19).

Gauer used the principle of the hydrostatic indifference point (HIP) to explain what occurs in head down tilt studies. The HIP is the point where the hydrostatic pressure does not change with change in position; for the venous system this is approximately where the pressure equals 0. In the upright individual the HIP is measured to be 8-10 cm caudal to the heart. Wilkins found a decrease in right atrial pressures during 75 degrees of head down tilt compared to the supine (73) and Gauer hypothesized that the HIP, normally inferior to the heart, is shifted to a position superior to the heart. Other researchers noted that filling pressures of the right atrium were maximal when supine and lessened upon head up or head down tilt in the normovolemic subject (18). Early work by Jongbloed and Noyons noted a reduction in cardiac size with -Gz (34).

Other research examined volumes rather than pressure in a head down tilt study at Armstrong Aerospace Medical Research Laboratory and exhibited no change in left ventricular end diastolic, end systolic, and stroke volumes, and no change in cardiac output in head down tilts to 90 degrees. Subjects tilted at greater than 30 degrees demonstrated a line of plethora at approximately the second intercostal space anteriorly, possibly depicting the new HIP in the venous system (31).

c. Cerebrospinal Fluid Pressure

Since intracranial blood vessels are encased in a non-expansible skull and surrounded by cerebrospinal fluid (CSF), it is reasonable to expect the pressure in the intracranial vessels would be counteracted by the pressure in the fluid and tissues outside the vessels. Rushmer has determined that CSF

pressure increased linearly with venous pressures during -Gz exposures (60). However, the extracranial blood vessels are not supported in an enclosed cavity and under the influence of -Gz can readily distend and pool blood. Changes in pressure in the carotid sinus area can stimulate the vagal nerve and reflexly decrease the heart rate during -Gz acceleration.

Beckman in 1949 performed experiments with goats measuring CSF and venous pressures directly at the base of the skull near the foramen magnum. The studies showed that the CSF and venous pressures varied simultaneously and approximately the same amounts during various exposures to -Gz. Arterial pressures were also found to increase with the venous and CSF pressures and by comparable magnitudes. The experimental results seemed to indicate that within the described experimental limits of up to -9 Gz, the normal intracranial vessels are adequately protected by simultaneous changes in the CSF pressure (4).

d. Arterial Pressures

As noted previously in animal research, arterial pressures will vary in approximately the same magnitude initially as for venous and CSF pressures under -Gz acceleration (4).

Experiments done in the late 1940's described cardiovascular response and cerebral function during -Gz acceleration. Five subjects were given -3 Gz exposures for 15 seconds. Arterial and venous pressures were measured at the forehead level. Petechial hemorrhages were noted in the conjunctiva and actual subconjunctival extravasations of blood did occur. Transient diplopia was experienced twice and associated with mild periorbital edema. The venous blood pressure recordings indicated a rise

in venous pressures from approximately 0 to 70-90 mm Hg. Figure 2 shows the physiological recording.

FIGURE 2 Simultaneous Increase in Arterial and Venous Pressures with -Gz Acceleration.

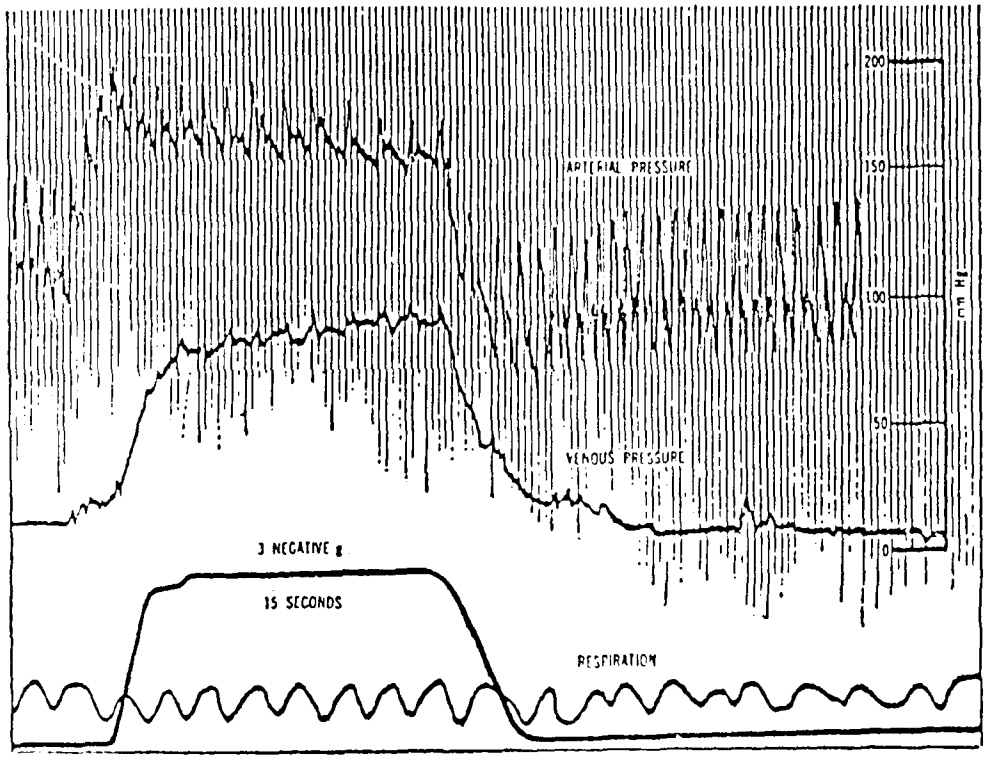


Figure 10. Arterial and venous pressures at head level during 3 negative G. With the rapid change of acceleration at the onset and at the end of acceleration both pressures rise and fall in a stepwise fashion by 75 mm Hg. This may indicate that the effective column lengths in both the venous and arterial systems are identical. The slopes of pressures at constant G are probably due to a reflex change of vascular tone and diminished cardiac output. Note reflex slowing of the heart.

Reproduced from Gamble, Shaw, Henry et al (17)

The recordings revealed a rapid parallel rise in the arterial and venous pressures. As the run progressed the heart rate slowed and there was a decrease in the arterial pressure and a steady rise in the venous pressure thought to be due to continued drainage of blood from the legs. Thus the arterial-venous pressure differential decreased with the increasing time of application of the acceleration. Further experiments with dogs under higher -Gz demonstrated that asystole could occur of a sufficient duration to result in a collapse of the arterial pressure and led to stagnant hypoxia and occasionally unconsciousness. Animal studies using electroencephalograms showed that immediately after -7 Gz acceleration abnormal EEG waves gave evidence of depression of the central nervous system (17).

The arterial-venous pressure difference diminishes gradually for prolonged -Gz exposures in man, approximately 30% at -5 Gz as venous pressures have a tendency to rise with the continuation of the exposure. The cardiac output falls probably as a result of the bradycardia. Arterial pressure in the femoral area falls to zero or becomes negative at -5 Gz and the peripheral vascular resistance decreases (59).

Henry performed a series of experiments with -Gz. Pressures in the arterial and venous system rose dramatically as measured in the carotid artery and jugular vein. The disturbances noted in experimental studies with men and animals seemed to result from excessive intravascular pressures in the superior portion of the body. The eyes were congested and the face became edematous. Conjunctival hemorrhages became frequent when accelerations were greater than -2.5 Gz of fifteen seconds or longer duration. Hemorrhages were massive when exposures of -5 Gz lasted for five to ten seconds. Pitting edema of the head and neck have been observed

in animals exposed to -10 Gz acceleration. The animal's tongue swelled two to three times its normal size. There was edema of the pharynx and upper trachea and death has resulted from respiratory obstruction and asphyxia. Occasional hemorrhage was seen in the anterior of the eye and the middle ear (27).

e. Bradycardia

Bondurant noticed while doing vectorcardiograms during various directions of acceleration that during his negative Gz work, bradycardia was consistently observed with heart rate decreasing to 38-45 beats per minute at -2 Gz. There were also noted occasional 3-5 second sinus pauses which persisted until the G exposure had ended (6). Ryan noted heart rates consistently below 50 for -3 Gz exposures (61). EKG abnormalities include prolonged PR interval, sinus arrest with junctional mechanisms, and PVC's at -3 Gz. Asystole of 7-10 seconds duration has been described (17). A study by Kennealy demonstrated sinus bradycardia during -2 Gz exposures. One individual developed a sinus arrest with a junctional rhythm in a -2 Gz thirty second exposure which resolved upon return to ambient 1 Gz except for a prolonged PR interval which normalized in two hours (39).

This bradycardia has led to a loss of arterial pressure due to prolonged asystole and resulted in loss of consciousness in research animals (17). This method of incapacitation has been implicated in inflight loss of consciousness and death when followed closely by +Gz acceleration (40,58).

Sicker's test subjects showed a slowing of the heart rate during -Gz acceleration and with the greater acceleration for a given individual usually producing a greater decrease in the heart rate. Four subjects noted some difficulty breathing at -2.5 to -3 Gz (66).

Lower vascular resistance was measured as a result of the baroreceptor reflex in animal studies by Rosenfield (59). The vagus nerve stimulation causes a decrease in heart rate, decreased contractility, and slowed AV node conduction (26). Sympathetic nerve impulses to the heart also decrease as a result of the baroreceptor stimulation (9).

Animal and human studies involving alternating -Gz and +Gz in a sinusoidal pattern showed that there are phase shifts and lags in cardiovascular responses. A peak resonance at 20-30 seconds per cycle has been shown and studies suggest a poor or possibly detrimental cardiovascular response at G oscillations in the frequency range encountered in aerobatic flight (23,44).

3. SUBJECTIVE

Negative Gz acceleration forces are experienced whenever a pilot/occupant of a plane undergoes an outside loop or spin or executes a maneuver involving such a motion. Kirkham and others have noted a significant problem with G incapacitation in aerobatic pilots. They mention that the most significant accelerations in aerobatic pilots are in the Gz axis (40). Positive Gz accelerations are encountered in turns, dive pull-outs, and upright banks; negative Gz accelerations are encountered in push-overs, outside loops and maneuvers during inverted flight.

Kirkham notes that human tolerance to +Gz has been well studied, however, tolerance to -Gz has been less well studied because the uncomfortable symptoms caused by these accelerations (40). Mohler notes that a pilot who performs an outside loop can experience -3.5 Gz for about one second. An inside aileron roll requires six seconds and imposes a maximum of up to +2.5 Gz. Studies done using accelerometers during aerobatic flying showed that in maneuvers such as the half vertical roll with negative pull out can attain levels of -3 to -4 Gz in the pull-out. The duration of -Gz was approximately 14 seconds. In an outside/inside vertical eight a pilot is subjected to levels of -5 Gz and within 5 seconds he is subjected to +5 Gz (49).

Whinnery commented that in military flying aerial combat maneuvering does not routinely involve high levels of -Gz, but may produce brief zero and -1 Gz exposures. Instead of using -Gz outside maneuvers in aerial combat it is found to be more efficient and pleasant for the pilot to roll over and use +Gz maneuvers. However, because of competition and the maneuvers in civilian aerobatics the pilots must tolerate both positive and negative Gz loads (72).

The subjective effects of negative Gz acceleration are described as follows:

-1 Gz: unpleasant but tolerable facial suffusion and congestion

-2 to -3 Gz: severe facial congestion, throbbing headache, progressive blurring, greying or occasionally reddening of vision after five seconds. Congestion disappears slowly, may leave petechial hemorrhages and edematous eyelids

-5 Gz: five seconds is the limit of tolerance, rarely reached by most subjects (13).

FIGURE 3 Graphs of Human Tolerances for Gx and Gz Acceleration

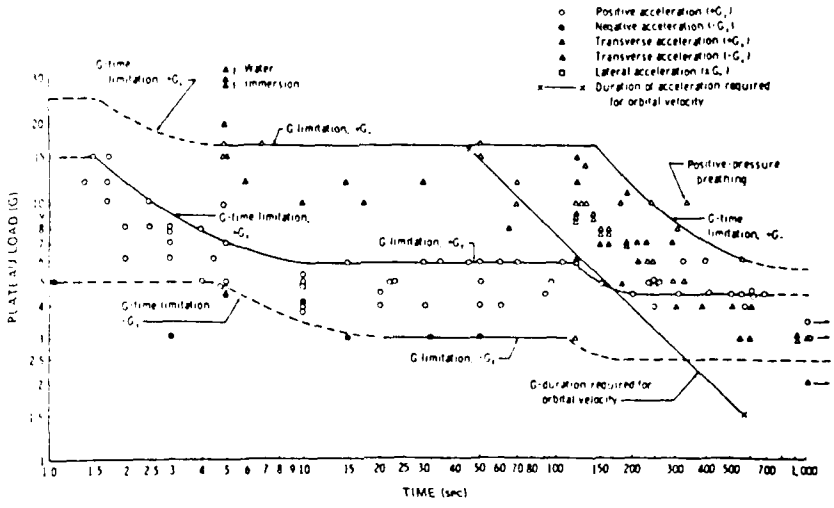


Figure 4–24. Human experience of sustained acceleration. Each datum point represents a plateau of acceleration, not an incidental peak. Curves estimate maximum voluntary tolerance in $+G_Z$, $-G_Z$ and $+G_X$ vectors using restraint harness, couches, or anti-G suits where applicable. Dotted lines are extrapolations on basis of other evidence. (After Fraser, 1966; data from many sources)

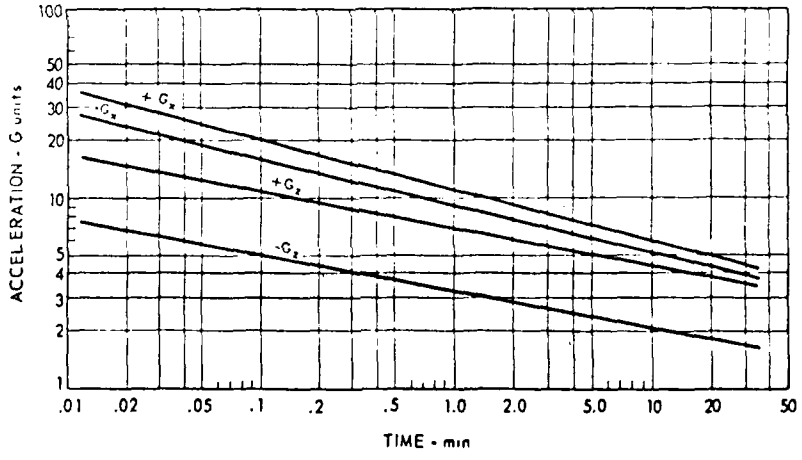


Figure 4–25. Comparison of average G tolerance in four vectors of sustained acceleration.

Reproduced from Fraser (13)

Figure 3 graphically represents human G limitations in the Gx and Gz directions. The tolerance to -Gz is shown to be less at any duration than +Gz, +Gx, or -Gx acceleration of the same magnitude.

Seiker did studies looking at symptoms in unprotected subjects utilizing 44 individuals exposed to -1, -2, and -3 Gz. The frequency of various complaints in objective findings were summarized in the following chart:

TABLE 1 Table of Frequency of Subjective Complaints during Negative Gz Acceleration

Acceleration	-1Gz	-2Gz	-3Gz
% of Subjects			
Uncomfortable fullness or pressure in head and neck	40	90	100
Bradycardia	30	90	100
Asystole	0	0	50
Headache	0	0	50
Conjunctival hemorrhage	0	0	40
Difficulty in breathing during acceleration	0	0	40
Diminished vision	0	0	40
Sinus pain	0	0	30
Ear pain	0	0	20

These studies were done with the subject in a seated posture lying on his side in a right lateral decubitus position (66).

The most frequent subjective finding under -Gz was fullness and flushing of the face and neck with headward shift of soft tissues of the cheeks and lower eyelids and lower lip. There was an increase in discomfort with increased acceleration. The unpleasantness of these symptoms was one of the factors which limited the tolerance in almost all subjects to 10 seconds at -2.5 Gz. Observers noted redness and fullness of

the face and neck and distortion of the face due to shift of soft tissues in all subjects during the centrifuge runs. Fifty percent of the individuals exposed to -3 Gz had a generalized headache that lasted one half to two days following the exposure. Twenty percent of the subjects noted pain in the frontal and temporal areas of the head. This pain increased in severity and became a limiting factor for tolerance in these persons. Three subjects complained of pressure or pain in the ears at -2.5 Gz which subsided after the acceleration. Exam of the ears showed only slight injection of the ear drum. Twenty-five percent of the individuals noted blurring of vision at -2.5 to -3 Gz. Tearing of the eyes and a tendency for the lower lid to cover the eye was also noted. Two individuals sustained conjunctival hemorrhages following exposure to -3 Gz for ten seconds. Fundoscopic exam following unprotected runs showed no changes in the retinal vessels. One person reported mental confusion during -3 Gz exposure in which he had cardiac asystole for nine seconds (66).

Visual symptoms of -Gz reported by Ryan were frequent and diverse. Blurring of vision was the most common complaint. Other subjects noted greying of the vision. Others experienced reddening of the vision at -2.5 and -3 Gz initially thought to be due to the squinting of the eyelids or possibly the phenomenon called "red-out." However, later authors have shown that this "red-out" phenomenon is either the result of the lower lid being pushed superiorly providing a red visual effect, or blood tinging of the lacrimal fluid (61).

At -3 Gz the pressure in the small vessels of the conjunctiva and sinus mucosa ranges from approximately 50-100 mm of Hg and the pain may become excessive (66).

A pilot exposed to -4 to -5 Gz for 30 seconds or more described his sensations as a severe expansion of the head with loss of hearing. The pilot said he could not swallow, had a choking sensation in his throat, and his vision was disturbed. The pain in his head was so severe that he felt it would burst and mental confusion developed. There have been comments that severe epistaxis also occurred with -Gz acceleration (27).

4. COUNTERMEASURES

Certainly, limiting the duration of the acceleration provides protection because arterial and venous pressures have been noted to take several seconds to increase and venous pressure increases only slowly thereafter (27). Inflatable leg cuffs have been recommended and have been proven to decrease venous return and possibly decrease venous pressures during -Gz (19). Use of the prone and semi-prone position may be of benefit in reducing the effective column heights and providing some protection (27,66).

Some of the early methods of increasing tolerance to negative Gz involved experiments utilizing mechanical pressure in the form of Ace bandages wrapped around the heads and necks of the animals. It was found to be an effective means of preventing local edema and bradycardia that occurred (17,27). Henry used a neck-sealing pressure helmet and found that if pressure was applied to the head and neck with the glottis closed during exposure to the negative acceleration, symptoms due to vascular engorgement of the head and neck were prevented or minimized. The effect of the countermeasure was minimized if the glottis remained open

because the resultant higher intrathoracic pressures increased cephalad venous pressures. The counterpressure about the carotid sinus area prevented the bradycardia found in unprotected subjects (27). It is interesting to note that head and neck suction has been suggested as a countermeasure to +Gz acceleration and there is some evidence of its efficacy (24).

Research has been performed by Sieker utilizing a positive pressure helmet from a high altitude suit. A graded pressure of 25 mm Hg per G was used. All the subjects tolerated -3 Gz with less discomfort and less pulse slowing than -2 Gz without protection. Higher levels of G seemed to show that the helmet provides about 2 Gz of protection. Subjective symptoms (fullness and pressure in the head and neck, ear pain, sinus pain, diminished vision, headache, and difficulty in breathing during acceleration) and physical signs (bradycardia, asystole, and conjunctival hemorrhage) all improved with use of the helmet. The results showed that in each case the bradycardia at any acceleration was less with the pressure helmet than without the helmet. The average percent fall of heart rate was greater in the unprotected than in the protected state and the average percent decrease was greater in -1 and -2 Gz accelerations without counterpressure than accelerations up to -5 Gz with counterpressure (66).

B. LOWER BODY NEGATIVE PRESSURE

1. HISTORICAL

The first major historical interest in lower body negative pressure (or LBNP) began in the early 1960's when it was discovered that LBNP procedures seemed to cause physiological changes similar to head-up tilt and orthostatic cardiovascular changes. Cardiovascular and aerospace researchers noted that these effects were independent of earth's gravity and had possible applications in physiological studies.

LBNP involves the application of reduced atmospheric pressure to the lower portion of the body surface usually in resting supine individuals. LBNP results in pooling of blood in the lower extremities and lower portion of the abdomen and a reduction in central venous blood volume. This stress imposed by this blood volume loss results in circulatory reflexes that attempt to compensate for these changes (74).

When the LBNP is applied to the lower body the vascular transmural pressure difference increases are directly related to the level of reduced atmospheric pressure. This elevation in pressure leads to expansion of capacitance vessels (or veins) and pooling of blood and also some plasma filtration into the extravascular spaces. Measurements of the amount of blood that pools during LBNP vary. Musgrave, et al, measured leg volume changes by water plethysmography using -40 mm Hg LBNP and reported pooling of 500 to 600 cc of blood in the legs (52). Brown et al measured the shift in the body's center of gravity with application of -70 mm Hg and these investigators estimated approximately 750 cc of blood was pooled in the lower body during this procedure (7). In general the studies have

shown that approximately 500 to 1,000 cc of blood is pooled during -30 to -50 mm Hg LBNP depending upon differences in individual physiology and experimental technique (74).

2. PHYSIOLOGICAL STUDIES

It has been shown that with the pooling of the blood to the lower body central venous pressure (CVP) falls 3 to 7 cm of H_2O depending upon the amount of lower body negative pressure. Even negative CVP values are recorded at greater than -40 mm Hg pressures. This reduction of CVP reflects the reduced venous return and diminished right heart filling and right heart output (74).

With LBNP there is a measured decrease in right ventricular function resulting in a reduction of pulmonary blood flow and reduction in the volume of the pulmonary capillary bed. A Soviet study by Katkov measured the effect of -30 mm Hg of LBNP. Right heart output, end-diastolic, and end-systolic volumes decreased by 22, 19, and 9 percent respectively which are similar to orthostatic measurements. Left ventricular function tests showed the left ventricular output decreased 26 percent, left ventricular end-diastolic volume decreased 22 percent, and end-systolic volume decreased 4 percent (38). Stroke volume of the left ventricle decreased by 47-50% for -50 torr LBNP exposures (14,25).

In experiments with anesthetized dogs Nutter showed that during -60 mm Hg LBNP there is an 85 percent reduction in diastolic pressure, 29 percent reduction in end-diastolic volume, and 25 percent reduction in end-

systolic volume (55). Cardiac output has been measured to decrease approximately 30 percent in humans with LBNP of -50 mm Hg (14).

Examining systemic arterial pressures in a series of studies Wolthius related that systolic pressures generally decrease; however, changes in diastolic pressure are variable. The pulse pressure was usually reduced and the reduction has been found to correlate approximately with the increase in heart rate. The heart rate was invariably increased but is variable among individuals at low levels of LBNP, but heart rate was seen to uniformly increase with increasing magnitude of LBNP. These changes in cardiovascular parameters with low levels of LBNP are similar to those that occur subsequent to hemorrhage: decreased venous return, reduced cardiac volumes, decreased pressures (especially systolic), and a reflex increase in peripheral resistance and heart rate (51,74).

Studies showed that similar changes in heart rate and blood pressure were found for -50 to -40 mm Hg LBNP, and for head-up tilt at 70 degrees in orthostatic studies (29,53). In fact LBNP has been used at the Civil Aeromedical Institute to simulate +Gz acceleration (42).

A Soviet study by Katkov, et al was designed to compare the effects of orthostasis and LBNP on central and coronary circulation. The experiment showed the greatest changes in CVP and pulmonary artery pressures at the low LBNP levels. When applied at a rate of 10 mm Hg per 1 to 2 minutes the CVP becomes negative dropping from 4.4 to -0.7 mm Hg with -30 mm Hg LBNP pressure. The systolic, diastolic, and mean pulmonary artery pressures all dropped significantly. The heart rate increased from 69 to 77 at -30 mm Hg but was statistically significant at -60 mm Hg at 92 beats per minute. The tilt test of 60 degrees and LBNP of -30 mm Hg were compared for effects on coronary and central circulation.

There were no significant differences in coronary circulation between the upright tilt test and LBNP. The central circulation differed in that the CVP pressure and heart rate were slightly higher for -30 mm Hg LBNP than at tilt, and at -60 mm Hg the heart rate and CVP were similar but the pulmonary artery pressures were slightly lower. This was thought to be due to the fact that in LBNP hypovolemia occurs without the hydrostatic pressure gradient that is present in tilt studies (37).

Frey, et al at Kennedy Space Center utilized women in studies with LBNP. Differences in heart rate and stroke volume, cardiac output and left ventricular ejection time showed very similar percent changes with LBNP of -50 mm Hg and other studies reporting changes with orthostasis. Comparison of cardiovascular parameters from this study and other studies involving men only, showed women may exhibit a higher increase in heart rate and less of an increase of peripheral resistance. The rest of the parameters were similar at this level of LBNP. The researchers raised the question of different cardiovascular compensatory mechanisms being used by women in comparison to men (14).

3. SUBJECTIVE

Some subjects reported sensations of tilting with onset and cessation of LBNP. Head up tilt sensations were reported with LBNP onset and head down tilt sensations with cessation of LBNP. It has been postulated that a decrease in CSF pressures with LBNP causes an endolymph flow with resulting vestibular stimulation that is interpreted in the semicircular canals as a change in body position (74). It is also possible that somatosensory sensations could add to the illusion of tilt.

Syncope or near-syncope was found in several experimental studies and was more frequent with LBNP exposures in excess of -40 mm Hg. Stevens and Lamb tested subjects at -25, -40, -60, and -80 mm Hg LBNP pressures and noted syncope in 0, 58, 70, and 100% of individuals tested at each level respectively (69). Epstein has developed a theory of two phases of LBNP-induced syncope. Presyncope, or Phase I, is a period of physiological instability characterized by a slow fall in arterial pressure, and variable increase in heart rate. Rapid cardiovascular decompensation occurs in Phase II when arterial pressure and heart rate falls and peripheral resistance decreases (11).

Factors that have been shown to decrease tolerance to LBNP stress and orthostasis are hypotension, low normal blood pressure, hemorrhage, dehydration, fever, pain, some medications, apprehension, prolonged bedrest, and weightlessness (74).

4. COMBINATION STUDIES

Many experiments have utilized LBNP as a stressor of the cardiovascular system to evaluate syncope, orthostasis, and weightlessness (74). Few experiments have been done using LBNP with head down tilt. The purpose of most of these was to evaluate orthostatic changes with bedrest simulation of weightlessness. Gazenko et al used LBNP in different regimens after 20 minutes of 20 degree head down tilt. LBNP of -30 torr (mm Hg) duplicated the cardiovascular parameters of 70 degrees head up tilt of for CVP, pulmonary and systemic pressures except the systemic diastolic pressure was slightly lower with LBNP. LBNP of -60 torr lowered pulmonary pressures significantly (22). Katkov showed changes

in cardiovascular parameters similar to the Gazenko experiment for LBNP performed both supine and at -15 degrees head down tilt (38).

C. RATIONALE

Efforts to protect against -Gz acceleration in the past have used pressure on the head and/or neck mechanically or pneumatically from a helmet as a method to decrease the baroreceptor response to the -Gz acceleration (27,66). Other methods employed have included the use of arterial occlusion cuffs on lower extremities in an effort to decrease cephalad venous pressures with -Gz. These have been somewhat successful in reducing measured venous pressures (19,73).

The negative physiological effects of -Gz include the increase in cephalad (or headward) venous pressures that cause many of the painful sensations of facial pressure and suffusion, and sinus pain or pressure. The increase in cephalad arterial pressures affects the carotid sinus and other baroreceptors in the wall of the aortic arch causing a reflex increase in vagus nerve impulses which decrease the heart rate, contractility, and peripheral resistance (59). The baroreceptors are very sensitive to large changes in arterial pressures especially from baseline values (26) and -Gz acceleration has been shown to cause periods of asystole, sinus arrest (39,66), and occasional incapacitation (17,40). Also, stimulation of the low pressure receptors in the atria and pulmonary artery help to sensitize the arterial baroreceptors to changes as well as elicit some vagal response directly themselves (26).

LBNP causes many cardiovascular changes which could counteract -Gz. LBNP lowers venous pressures, decreases right and left heart volumes, and shifts 500 to 800 cc of mostly venous vascular volume toward the pelvis and legs (74). Animal studies have shown the benefit of phlebotomy in reducing venous pressures in exposures to -Gz (19) and LBNP can be thought of as a "reversible" phlebotomy. Although lower body arterial volumes do not increase appreciably the systemic arterial pressure does decrease with increasing LBNP. This decrease in arterial pressures could protect against -Gz baroreceptor responses from the carotid sinus and arch baroreceptors. Unloading both atria and the pulmonary vasculature low pressure baroreceptors could decrease the -Gz effect on these physiologic receptors by decreasing the sensitivity of the arterial receptors.

Tripp et al have used LBNP in a 45 and 90 degrees head down tilt study with echocardiography, and have demonstrated the efficacy of LBNP with 45 and 90 degrees head down tilts to pool blood caudally and qualitatively cause the same LBNP cardiovascular response as when supine. The measured cardiac volumes were decreased with LBNP at both levels of tilt. The increase in heart rate that occurs with LBNP was preserved at both levels of tilt. Although 90 degrees of tilt without LBNP demonstrated little effect on measured cardiovascular volumes per se, the unloading effect on the venous and arterial baroreceptors as well as the anecdotal improvement in subjective symptoms gave evidence that LBNP may protect against higher magnitude -Gz (70).

LBNP in many ways can be thought of as the opposite of the MAST (Military Anti-Shock Trousers) suit or G-suit. Both of these apparatuses utilize higher than ambient pressures on the lower body (legs and

abdomen). The theoretical effects were thought to provide an autotransfusion of blood and increase peripheral vascular resistance and support blood pressure and perfusion. In reality the MAST suit's primary cardiovascular effect seems to be an increase in peripheral vascular resistance, and possibly a mild increase in venous return. It serves as well as a splint for lower extremity and pelvic injuries (16,32). A MAST suit is utilized clinically in supine hypovolemic individuals.

In contrast to the MAST suit, the G-suit is used by healthy normovolemic upright pilots. Its mechanism of action to improve G-tolerance has been attributed to the increasing of peripheral vascular resistance that assists in the maintenance of adequate cerebral perfusion pressures, and the provision of counterpressure to oppose the extravasation of plasma into tissue. It may also facilitate increased venous return to the heart, and may support the diaphragm decreasing the heart-brain distance. This suit is useful in situations where the G-onset rates will not allow cardiovascular compensation to occur. G-suits usually improve G-tolerance by 1-2 Gz for an individual (43).

III. METHODS

A. SUBJECTS

Ten healthy young men (ages: mean 28.1 years , S.D. 3.84 years; weights: mean 69.9 kg, S.D. 14.2 kg; height: mean 170.1 cm, S.D. 6.46 cm), members of the Acceleration Stress Panel at Wright-Patterson Air Force Base, were used as subjects. The subjects were given detailed information and instructions concerning the experimental conditions before obtaining their consent. Each subject had been screened during training runs to insure that a good quality echocardiogram was obtainable.

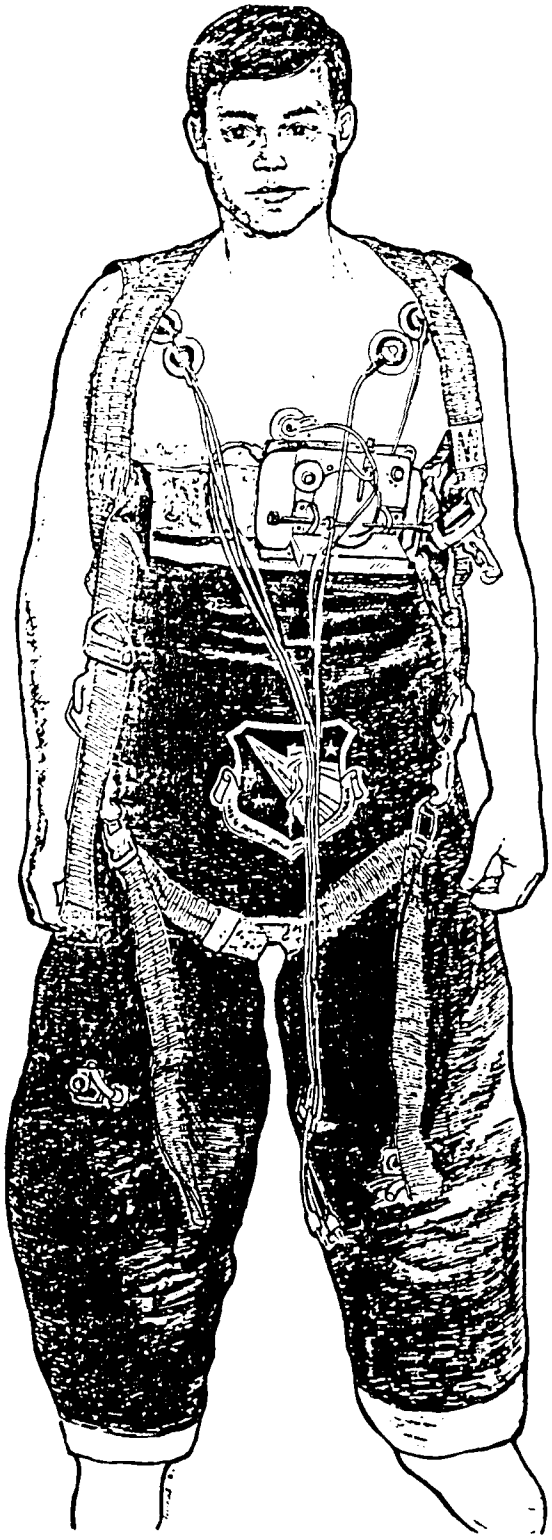
B. MATERIALS/EQUIPMENT

Echocardiograms were performed with a Hewlett-Packard Model 77020F two-dimensional echocardiograph and a 3.5 MHz transducer. Apical views of the left ventricle were obtained by placing the transducer over the subject's chest while the subject was seated in the centrifuge cab and the site marked. The subject then put on the LBNP suit and a chest harness was applied that held the transducer in place. The echocardiograph was mounted in the cab and a 90 degree sector scan was recorded at a rate of 30 frames per second. Video images and EKG images were stored on 1/2 inch high quality video tape in the medical monitor's control room.

During all of the -Gz exposures the subjects wore one of two different sized suits, both of which were constructed at the Armstrong Aerospace Medical Research Laboratory. The suit resembled a pair of

rigid oversized fisherman chest waders. The legs and lower abdominal section were made of a stiff polypropylene pipe that allowed some flexion of the knee joints. Neoprene-impregnated nylon material formed the outer surface of the suit as well as the abdominal seal. The abdominal seal was obtained by a superior continuation of the neoprene nylon. Rubber latex boots were used at the distal leg segments to provide an airtight seal for the feet. The suit was constructed with two evacuation ports of one inch diameter in the anterior thigh sections of the suit. See Figure 4 for an illustration of the suit.

FIGURE 4 Lower Body Negative Pressure Suit



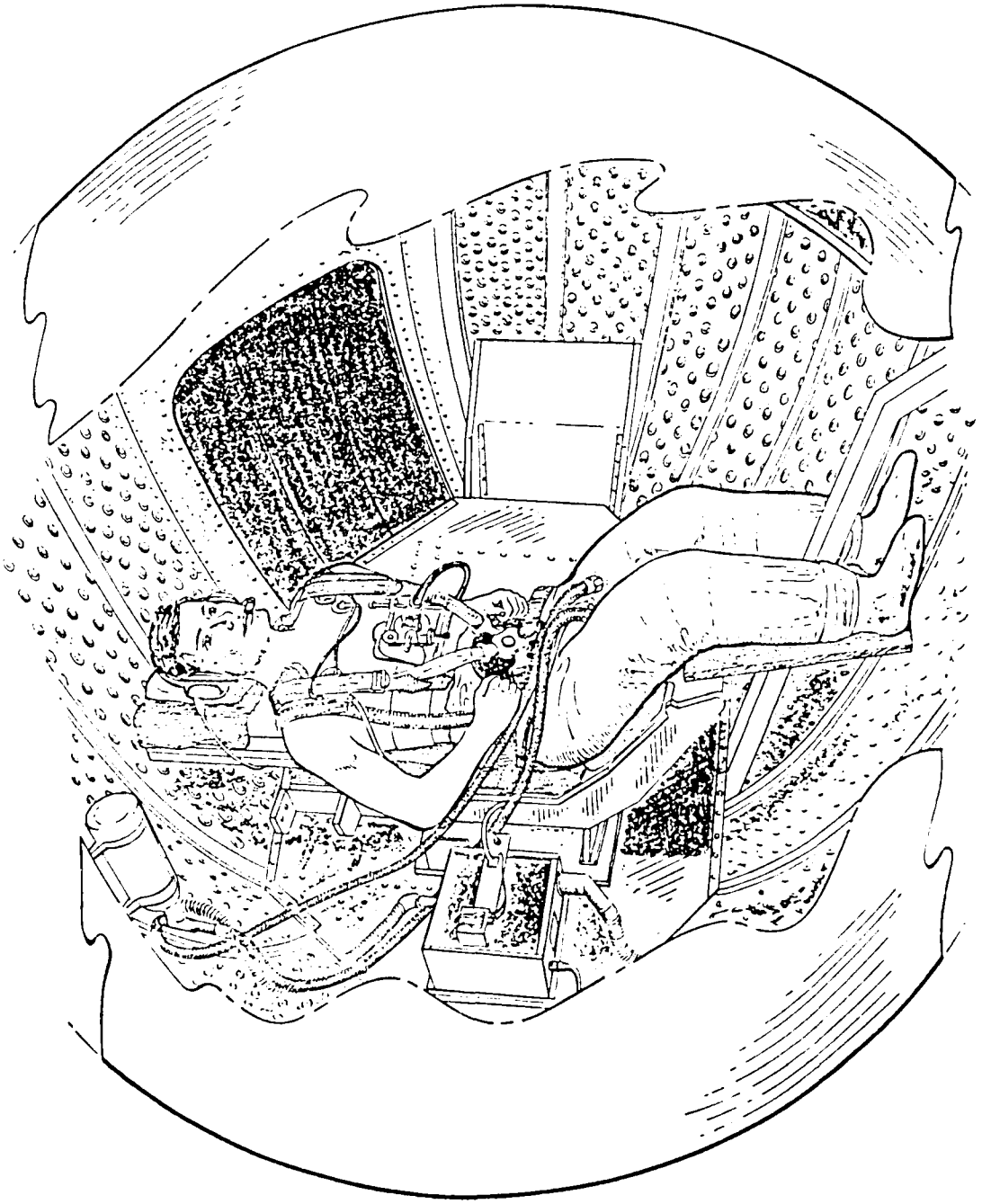
One inch diameter steel-reinforced hose connected each evacuation port to a 13 ampere commercial grade vacuum pump capable of 22 cubic feet per minute flow rate. Suit pressures were measured by an Omega pressure transducer and controlled by a 24V DC ASCO Model 8210 on/off electric solenoid valve controlling the flow from the hose. A Zenith 148 computer was used to monitor and control suit pressures by turning the valve on and off. A large metal cannister reservoir was also used as a source of low pressure in order to facilitate the beginning of suit evacuation and smooth airflow.

C. DESIGN

Each subject was exposed to -100, -50, and 0 mm Hg LBNP while accelerated at -1.0, -1.5, and -2.0 Gz. All levels of LBNP and -Gz were randomized into a 3 by 3 matrix. Each subject was limited to no more than three exposures per day. The randomization schedule is shown in Appendix A. A typical daily series of tests consisted of the subject entering the cab and sitting on the Stensel seat which has a seatback angle of 65 degrees. The gondola was then vectored so the seatback was in the horizontal position. See Figure 5. A trial echocardiogram was performed in the cab in order to determine the proper placement and angle of the transducer and the chest wall site was marked. The subject was then removed from the cab and the proper LBNP suit for his body size was put on. The waist was sealed with an elastoplast wrap applied over benzoin skin adhesive and then the chest mount harness was placed on the subject. The PCU 15 safety harness for quick emergency egress from the cab was then put on. The subject entered the cab and the echocardiogram transducer placed onto the site marked on the subject's chest at the predetermined

angle. Minor adjustments were made to the position of the transducer to insure proper image quality, and the transducer secured in the harness with the mount screws. The subject was visually monitored by video camera and voice communication maintained with headset and microphone. The EKG was monitored at all times, and the echocardiogram was monitored visually as well as being taped during the experimental exposures.

FIGURE 5 Diagram of Centrifuge Gondola



Direction of Acceleration ⟶

There was a fixed sequence of events for each exposure. The participant was seated and restrained in the cab while sitting with the seat back in horizontal position, his head outward with the subject's longitudinal axis aligned in the radius of the centrifuge arm. The vacuum pump was started while the valve to the LBNP suit was off. The valve was opened by the computer initiating the designated level of LBNP, which was then maintained by computer program. An 8 channel Clevite Brush recorder Model Mark 20C was used to directly record the drop in pressure in the suit. Within 10-15 seconds after the level of LBNP was insured, the subject was accelerated to the -Gz plateau with a 0.1 G per second onset rate, a thirty second plateau at the experimental level of -Gz chosen, and subsequent deceleration at 0.1 G per second back to baseline. LBNP at the selected pressure was maintained throughout the -Gz exposure. Echo and EKG data was collected throughout the exposure, and subjective questions were asked at the end of each testing series for each day. Each subject was exposed to each one of the three levels of -Gz conditions and to each one of the three LBNP conditions for each day of testing for a total of three exposures per day. LBNP was monitored both by continuous strip chart recordings and by sampling of pressures once per second by the computer program.

D. DATA ANALYSIS

Echocardiogram data were recorded throughout the exposure. If there was loss of quality of the echo image during the exposure to LBNP or -Gz the run was aborted and adjustments were made to the transducer to restore image quality. The image of the left ventricle was checked closely to insure visualization of the mitral valve and the left ventricular free wall. Adequate image quality was obtained for all test subjects that completed the experiment. However, there were subjects in the acceleration stress panel, in whom adequate image quality was not obtainable under preliminary experimental conditions, who then were dropped from this study prior to experimental runs.

Single plane ellipse method of echocardiogram analysis described by Kantrowicz et al (35) was used to determine end-diastolic volume (EDV) and end-systolic volume (ESV). Heart rate was determined by measuring the R-R interval on the EKG. Stroke volume (SV) was calculated by the difference in the EDV and ESV, and cardiac output determined by the product of the heart rate and the SV. These measurements were made by a single investigator who had been trained and had significant experience in the procedure. Tapes were subsequently reviewed by board-certified cardiologists and cardiovascular physiologists to verify clarity and adequacy of image quality.

The four data points obtained were: (1) at baseline just prior to beginning of LBNP ("BASELINE"), (2) after LBNP was instituted and the cardiovascular parameters had stabilized for 10-15 seconds ("LBNP ON"), (3) in the approximate middle of the 30 second -Gz plateau after the cardiovascular parameters had stabilized ("-Gz") and (4) after the subject had returned to ambient pressure and 0 Gz ("POSTBASELINE"). Three

measurements under each condition were obtained, and the Hewlett-Packard software was used to calculate the ESV, EDV, SV, heart rate (HR) and cardiac output (CO). The image of the left ventricle was checked closely for adequate visualization of the mitral valve. The largest left ventricular area corresponding to the EDV was chosen for measurement by a drawn ellipse, and the proper volume was calculated by the computer program. The image of the smallest left ventricular area was selected as the ESV, which occurred just prior to the opening of the mitral valve and correlated with the descending portion of the T wave.

A questionnaire which was used to evaluate subjective sensations and symptoms was completed by the subjects at the end of each day's experimental runs. After all experimental runs he was asked to rank the nine exposures from worst (1) to best (9). See Appendix B for the subjective questionnaire.

IV. RESULTS

A. PHYSIOLOGICAL

Testing was completed on 10 subjects and the data were reduced by one investigator. Upon data reduction it was noted that one subject who had completed all experimental runs had poor image quality on some echocardiograms during a few exposures and this individual was dropped from the data analysis.

Means were taken from the 3 replications averaging end diastolic volume, end systolic volume, and heart rate for each combination of subject (N=9), -Gz (-1.0, -1.5, & -2.0), pressure (0, -50,&-100 mm Hg) and order (baseline, LBNP ON, -Gz, & postbaseline) with coefficients of variation for the 3 replications of 1.3%, 2.1%, and 2.5% respectively. Stroke volume and cardiac output were then calculated from these means. Means and standard deviations for the 9 subjects' actual data are presented in Appendix C Table C. 1. Actual data converted to Percent of Baseline are presented on Appendix C Table C. 2.

Analyses of variance were performed in a repeated measures design with dependent variables: (1) percent of baseline for LBNP ON, and (2) percent of baseline for -Gz. Factors used were pressure, -Gz, and subject (random). Subject interactions were used as the residual error term for each F-test. See Table 2. If an F-test was significant, paired t-tests were

used forpairwise comparisons. T-tests were also used to determine whether changes were significantly different from 0. See Appendix D for the T-test table.

TABLE 2 ANALYSIS OF VARIANCE RESULTS

(I) PERCENT OF BASELINE FOR LBNP ON

SOURCE of ANOVA Sum of Squares

	DF
Between Subject(random)	8
Within Subject	
Pressure	1
Subject*Pressure	8
Total	17

DEPENDENT VARIABLE	EFFECT	DF	F-VALUE	P-VALUE
% EDV	PRESS	1,8	131.55	.0001
% ESV	PRESS	1,8	70.95	.0001
% HR	PRESS	1,8	18.37	.0027
% SV	PRESS	1,8	23.48	.0013
% CO	PRESS	1,8	0.13	.7302

TABLE 2 (CONT.) ANALYSIS OF VARIANCE RESULTS

(II) PERCENT OF BASELINE FOR -GZ

SOURCE of ANOVA Sum of Squares

	DF
Between Subject(random)	8
Within Subject	
Gz	2
Subject*Gz	16
Pressure	2
Subject*Pressure	16
Gz*Pressure	4
Subject*Gz*Pressure	32
Total	80

DEPENDENT VARIABLE	EFFECT	DF	F-VALUE	P-VALUE
% EDV	GZ	2,16	1.02	.3821
	PRESS	2,16	28.08	.0001
	GZ*PRESS	4,32	0.51	.7284
% ESV	GZ	2,16	0.27	.7695
	PRESS	2,16	33.48	.0001
	GZ*PRESS	4,32	0.38	.8198
% HR	GZ	2,16	24.37	.0001
	PRESS	2,16	9.72	.0017
	GZ*PRESS	4,32	1.84	.1447
% SV	GZ	2,16	0.87	.4393
	PRESS	2,16	1.63	.2269
	GZ*PRESS	4,32	1.18	.3364
% CO	GZ	2,16	23.72	.0001
	PRESS	2,16	14.40	.0003
	GZ*PRESS	4,32	1.79	.1544

Data summarizations and graphs have been made utilizing percent change from baseline for the cardiac parameters of EDV, ESV, HR, SV, and CO. See Tables 3-7 and Figures 6-10.

PERCENT OF BASELINE FOR LBNP ON

The effect of LBNP was dramatic prior to -Gz exposure. For EDV and ESV the LBNP main effects decreased EDV and ESV significantly more so for -100 mm Hg when compared to -50 mm Hg ($p<.0001$). LBNP effects on SV decreased SV significantly more for -100 mm Hg compared to -50 mm Hg ($p=.0013$). Effects on HR increased HR more for -100 mm Hg ($p=.0027$). CO increased significantly for each exposure of -50 and -100 mm Hg ($p<.0128$), but there was not a significant difference between -50 and -100 mm Hg ($p=.7302$). See Table 2 and Appendix D.

PERCENT OF BASELINE FOR -Gz

Upon examining the ANOVA table (See Table 2) for the 5 dependant variables, the interactions between Pressure and -Gz were not significant for any of the variables ($p>.1447$). Main effects demonstrated significance ($p<.0017$) for the variables EDV, ESV, HR, and CO for the effect of pressure; for variables HR and CO for -Gz effects.

EDV

The effect of LBNP ("Pressure") on EDV prior to -Gz was significant (p=.0001). LBNP decreased baseline values significantly (p<.0001) to 90.1% for -50 and 81.9% with -100 mm Hg. During -Gz the effect of LBNP was lessened but still significantly different than baseline values at -1.0 Gz (p=.0041) and -1.5 Gz (p=.0004), but not for -2.0 Gz (p=.2141). The effect of -Gz acceleration was to increase EDV closer to baseline, averaging 97.0%, but the 3 levels of -Gz were not significantly different from each other (p=.3821). The effect of increasing the magnitude of -Gz seemed to normalize the effect of LBNP on the EDV. Pressure continued to exert a significant effect on EDV (p=.0001) and pairwise comparison of all three levels of of LBNP show significant differences (p<.0102), with greater negative pressure decreasing EDV. See Table 3 and Figure 6.

TABLE 3 END DIASTOLIC VOLUME RESULTS

LEGEND: * = SIGNIFICANTLY DIFFERENT FROM 100
| = NOT SIGNIFICANTLY DIFFERENT

(I) PERCENT OF BASELINE FOR LBNP ON

PRESS	MEAN (P=.0001)
-50	90.1*
-100	81.9*

(II) PERCENT OF BASELINE FOR -GZ

GZ	MEAN (P=.3821)
-1.0	97.4* |
-1.5	95.9* |
-2.0	97.8 |

PRESS	MEAN (P=.0001)
0	100.2
-50	98.0*
-100	93.0*

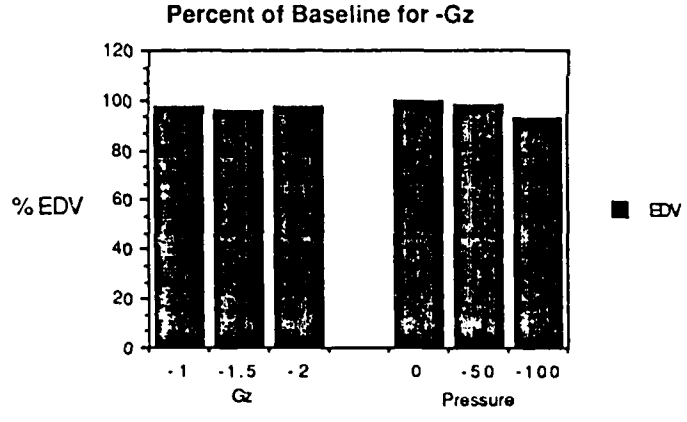
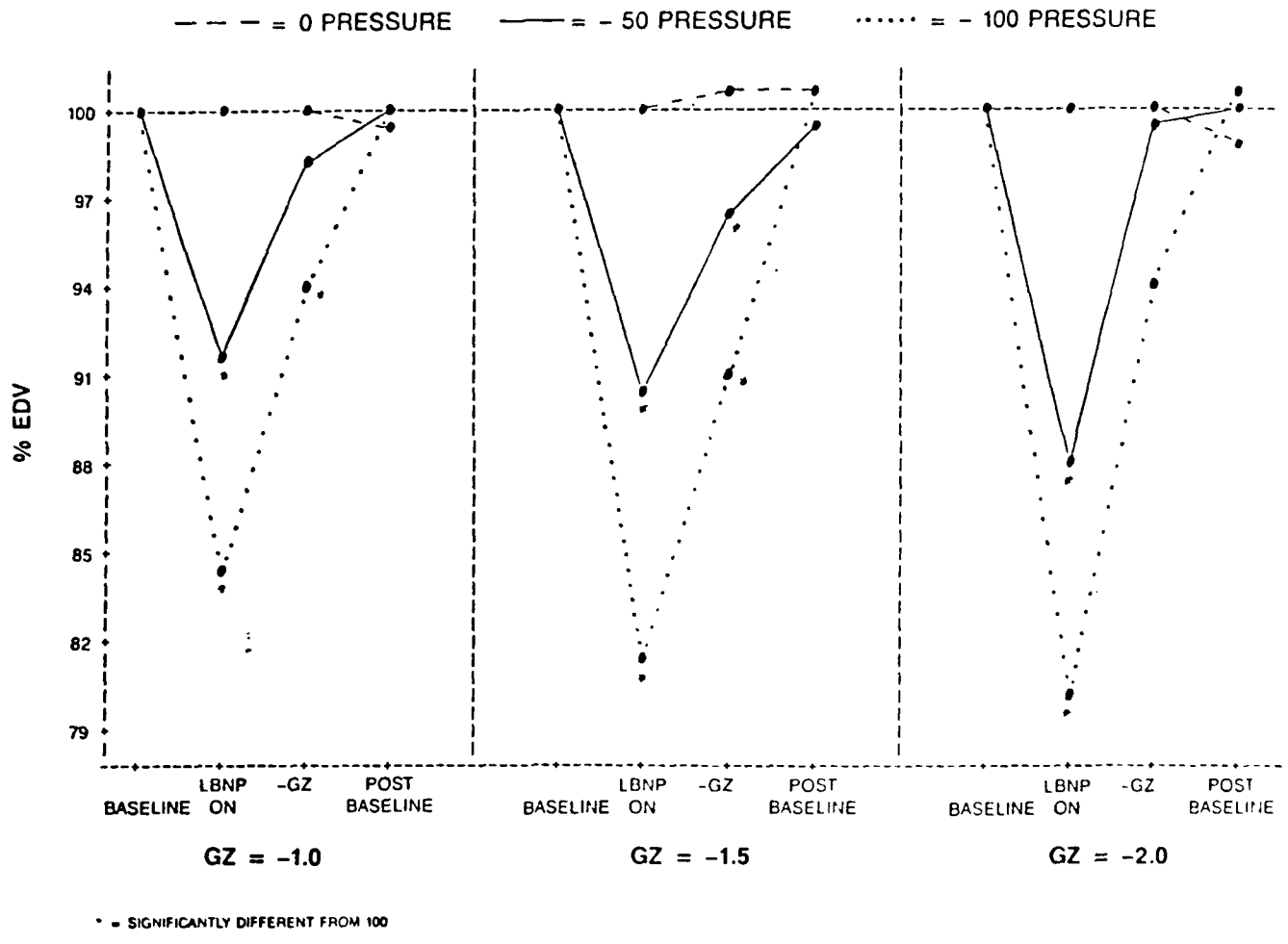


FIGURE 6. PERCENT OF BASELINE FOR END DIASTOLIC VOLUME



ESV

LBNP main effects on ESV were significant (p=.0001). LBNP decreased the ESV significantly (p<.0001) to 86.8% of baseline for -50 mm Hg and 79.0% of baseline for -100 mm Hg of pressure. During -Gz the pressure effects were significant (p=.0001) and were still significantly different than baseline values at -1.0 Gz (p=.0031) and -1.5 Gz (p=.0026). At -2.0 Gz the difference from baseline was nonsignificant (p=.2671). Pairwise comparisons of the three levels of pressure were all significant (p<.0025), and the individual levels were significantly lower than baseline (p<.0189) except for 0 mm Hg (p=.2935). Negative Gz effects were not significant (p=.7695). See Table 4 and Figure 7.

TABLE 4 END SYSTOLIC VOLUME RESULTS

LEGEND: * = SIGNIFICANTLY DIFFERENT FROM 100
| = NOT SIGNIFICANTLY DIFFERENT

(I) PERCENT OF BASELINE FOR LBNP ON

PRESS	MEAN (P=.0001)
-50	86.8*
-100	79.0*

(II) PERCENT OF BASELINE FOR -GZ

GZ	MEAN (P=.7695)
-1.0	96.2* |
-1.5	95.1* |
-2.0	96.8 |

PRESS	MEAN (P=.0001)
0	101.1
-50	96.6*
-100	90.4*

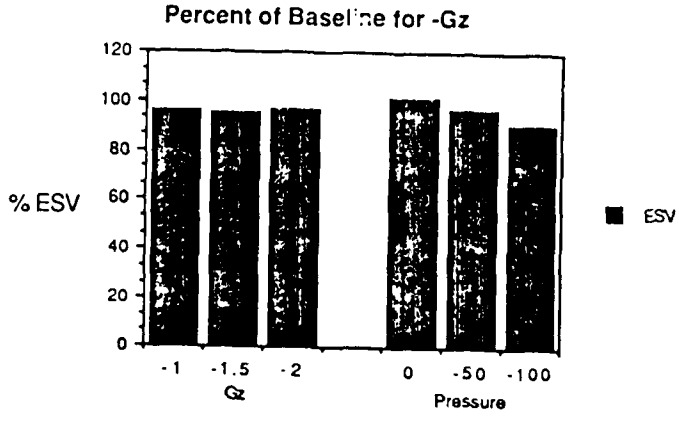
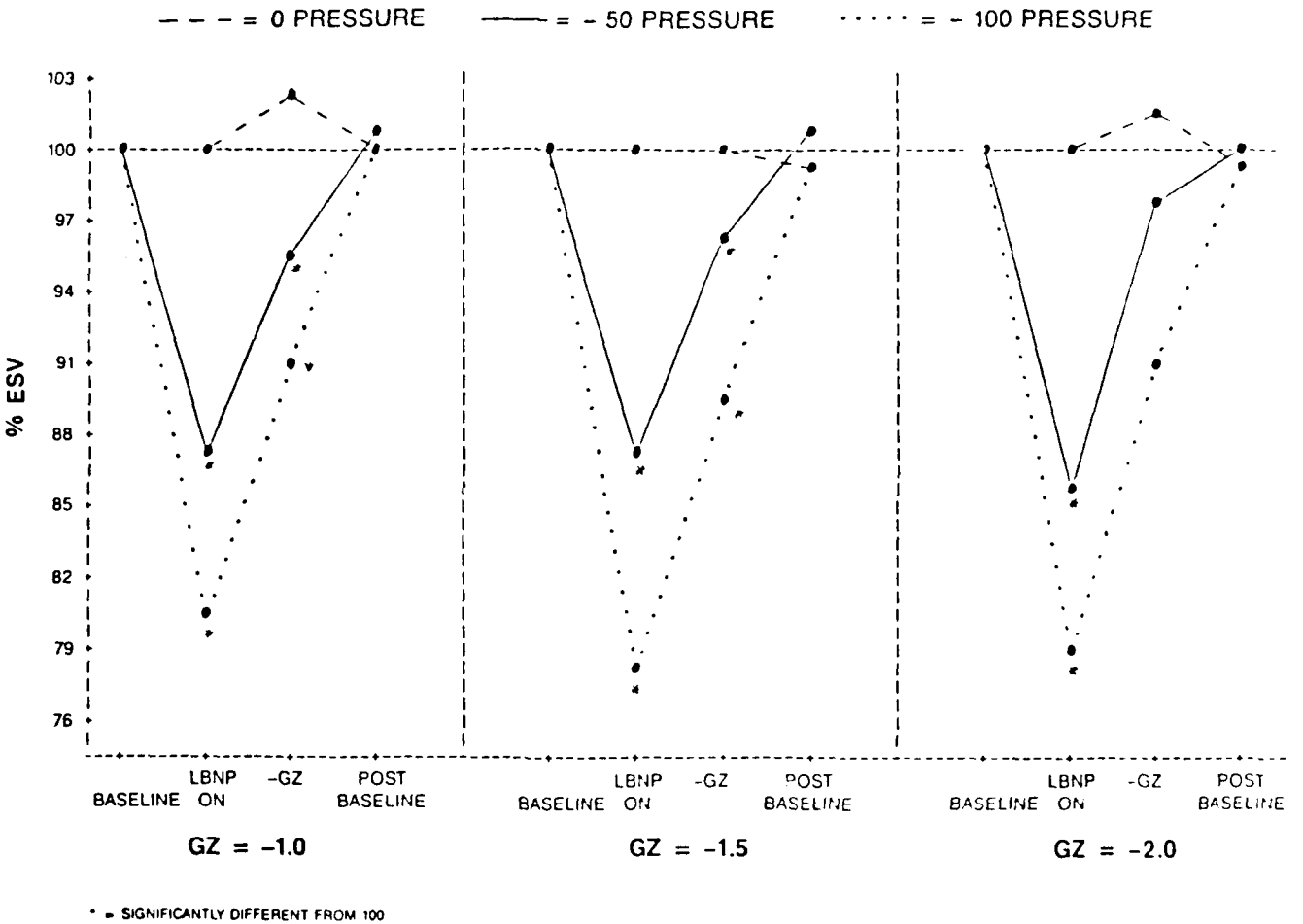


FIGURE 7. PERCENT OF PRE BASELINE FOR END SYSTOLIC VOLUME



* = SIGNIFICANTLY DIFFERENT FROM 100

SV

SV changes were significant with LBNP (p=.0013). SV values changed significantly from baseline (p<.0140) to 95.2% of baseline with -50 mm Hg and 86.1% of baseline with -100 mm Hg pressure. With -Gz acceleration both main effects become nonsignificant (p>.2269) demonstrating the ability of -Gz acceleration to normalize the effect of LBNP to approximately 98.7% of baseline values. See Table 5 and Figure 8.

TABLE 5 STROKE VOLUME RESULTS

LEGEND: * = SIGNIFICANTLY DIFFERENT FROM 100
| = NOT SIGNIFICANTLY DIFFERENT

(I) PERCENT OF BASELINE FOR LBNP ON

PRESS	MEAN (P=.0013)
-50	95.2*
-100	86.1*

(II) PERCENT OF BASELINE FOR -GZ

GZ	MEAN (P=.4393)	
-1.0	99.4	|
-1.5	97.2	|
-2.0	99.6	|

PRESS	MEAN (P=.2269)	
0	98.9	|
-50	100.5	|
-100	96.8*	|

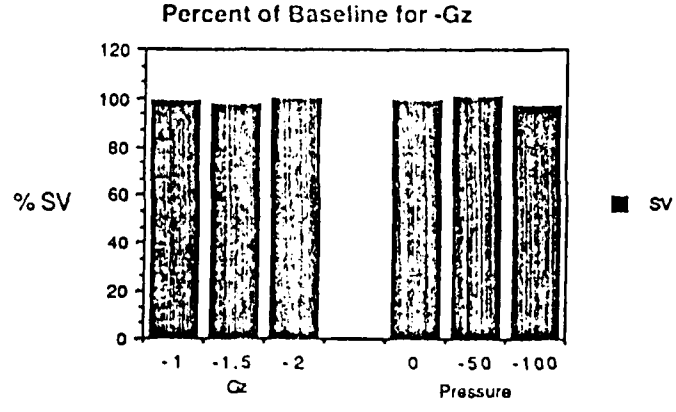
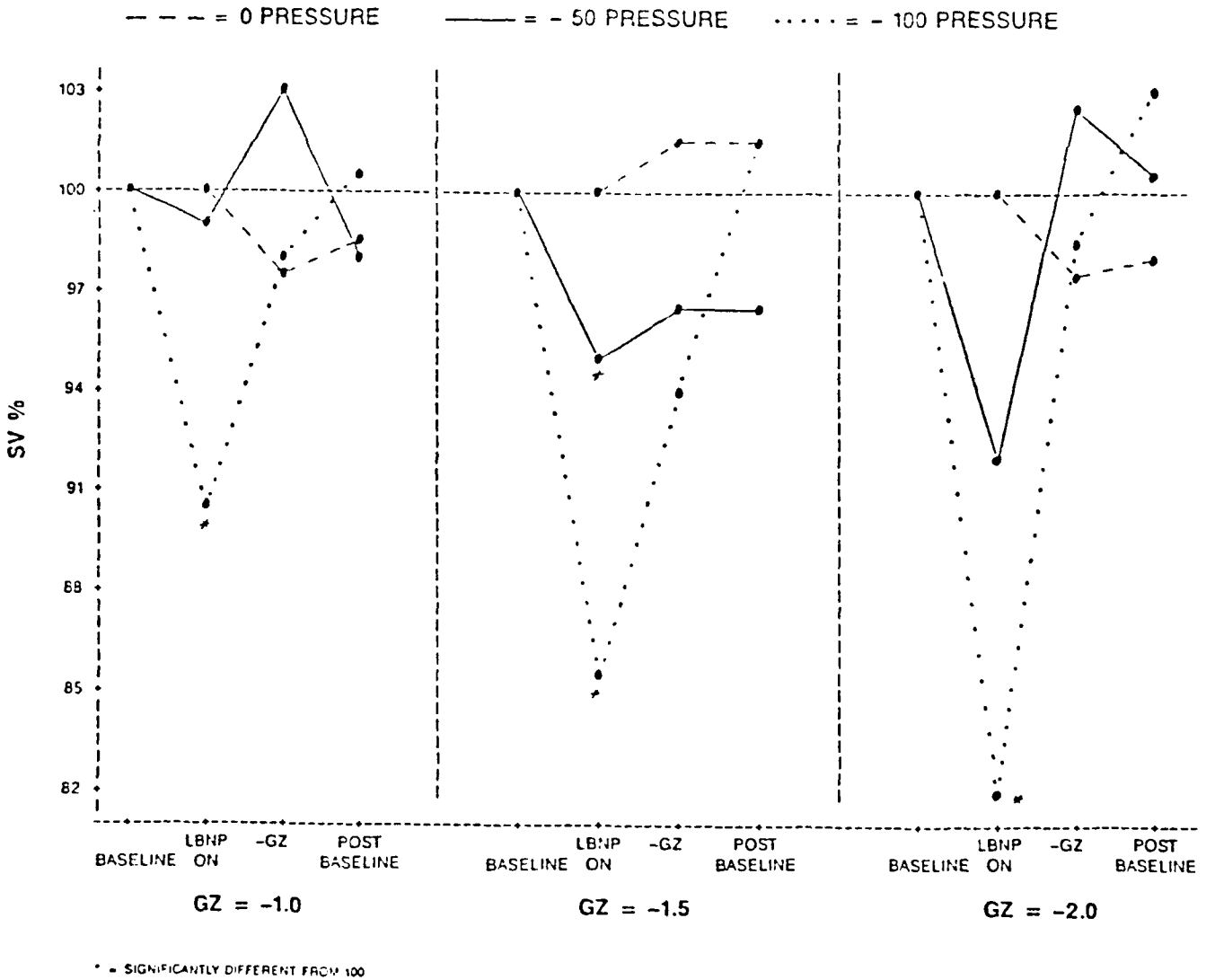


FIGURE 8. PERCENT OF BASELINE FOR STROKE VOLUME



HR

The main effects for the two levels of LBNP were significant (p=.0027). HR changes with LBNP show a significant increase in HR from baseline values (p<.0001) to 118.2% and 131.9% of baseline for -50 and -100 mm Hg LBNP respectively. With -Gz acceleration -Gz effects and pressure effects were both statistically significant (p=.0001, p=.0017). All of the pressures were significantly different than baseline (p<.0133) and both pairwise comparisons with 0 mm Hg were significant (p<.0199) but not the comparison between -50 and -100 mm Hg (p=.1502). Negative Gz effects on HR showed changes different than baseline values (p<.0004) and all pairwise comparisons significant (p<.0110), with increasing magnitude of the -Gz acceleration producing decreasing HR values. Negative acceleration had reversed the increase in HR that occurred with LBNP to levels below baseline values. LBNP pressure of -50 and -100 mm Hg had significantly decreased the effect of -Gz to 82.1% and 87.4% when compared to 0 pressure at 69.8% (p<.0199) across all levels of -Gz. See Table 6 and Figure 9.

TABLE 6 HEART RATE RESULTS

LEGEND: * = SIGNIFICANTLY DIFFERENT FROM 100
| = NOT SIGNIFICANTLY DIFFERENT

(I) PERCENT OF BASELINE FOR LBNP ON

PRESS	MEAN (P=.0027)
-50	118.2*
-100	131.9*

(II) PERCENT OF BASELINE FOR -GZ

GZ	MEAN (P=.0001)
-1.0	88.8*
-1.5	80.0*
-2.0	70.5*

PRESS	MEAN (P=.0017)
0	69.8*
-50	82.1* |
-100	87.4* |

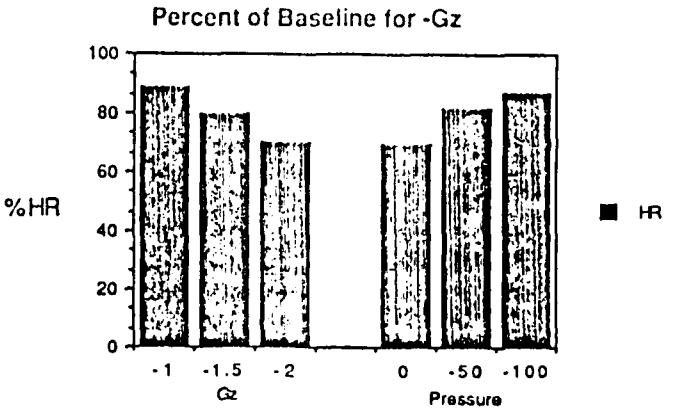
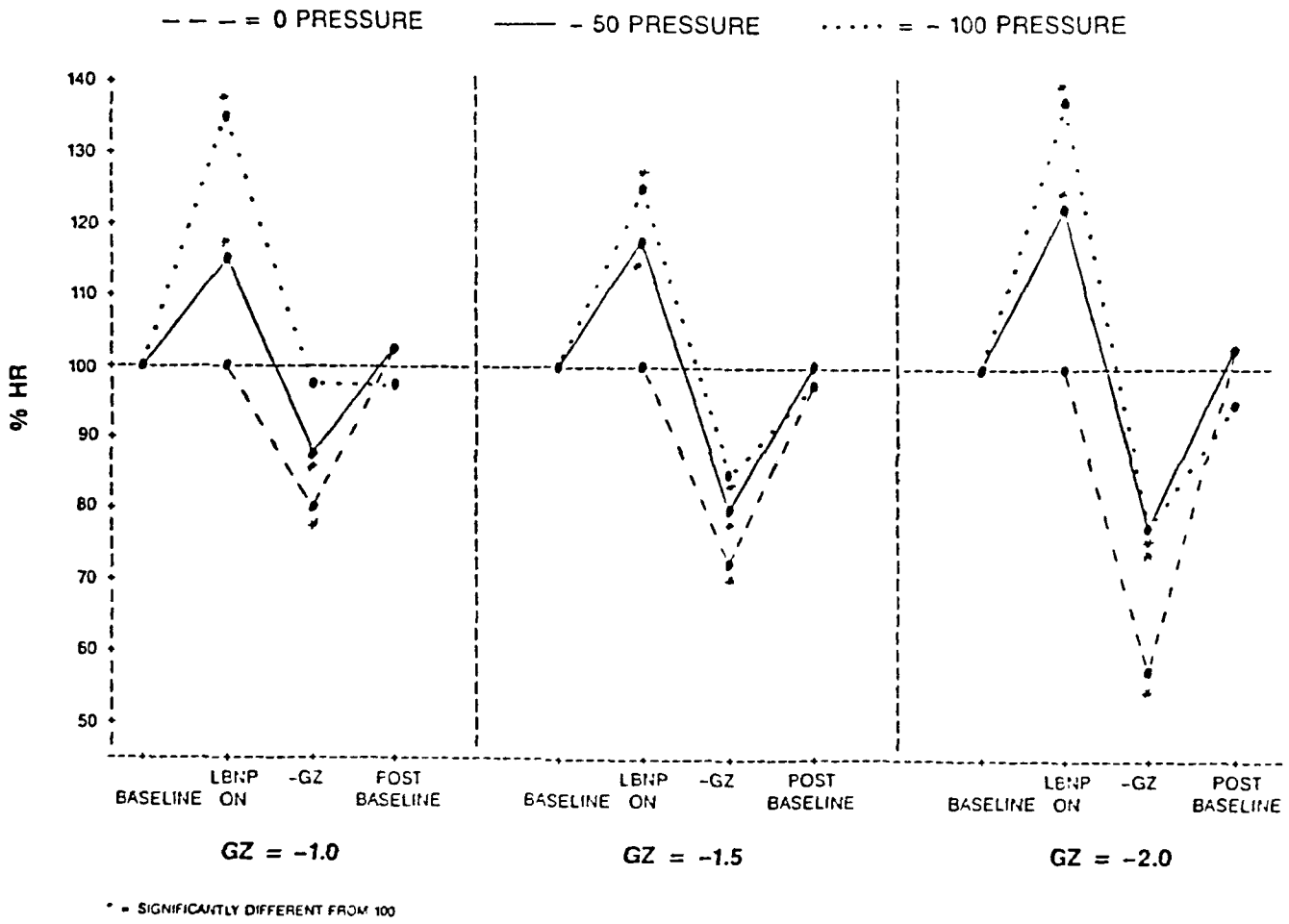


FIGURE 9. PERCENT OF BASELINE FOR HEART RATE



* = SIGNIFICANTLY DIFFERENT FROM 100

CO

Cardiac output (CO) is a combination of HR and SV values multiplied. CO increased significantly with LBNP to 112.1% and 113.5% of baseline for -50 and -100 mm Hg ($p<.0128$), but these changes were not significantly different from each other ($p=.7302$). With -Gz acceleration the main effects of Pressure and -Gz were both significant ($p=.0001$; $p=.0003$). Negative Gz acceleration decreased the values to an average of 78.7% of baseline. The main effects of -Gz show pairwise comparison of all three levels of -Gz to be significant ($p<.0156$), as well as each level being significantly different than baseline ($p<.0026$). Increasing the magnitude of -Gz decreased CO. The main effects of pressure show that all levels were different than baseline ($p<.0014$) and pairwise comparisons were significant for both comparisons with 0 mm Hg ($p<.0051$) but not the comparison between -50 and -100 mm Hg pressure ($p=.5033$). Increasing the magnitude of negative pressure increased the CO generally. See Table 7 and Figure 10.

TABLE 7 CARDIAC OUTPUT RESULTS

LEGEND: * = SIGNIFICANTLY DIFFERENT FROM 100
| = NOT SIGNIFICANTLY DIFFERENT

(I) PERCENT OF BASELINE FOR LBNP ON

PRESS	MEAN (P=.7302)
-50	112.1* |
-100	113.5* |

(II) PERCENT OF BASELINE FOR -GZ

GZ	MEAN (P=.0001)
-1.0	88.3*
-1.5	77.8*
-2.0	69.9*

PRESS	MEAN (P=.0003)
0	69.0*
-50	82.6* |
-100	84.3* |

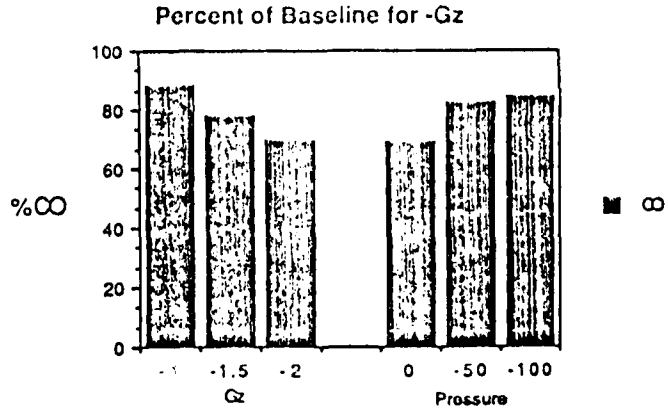
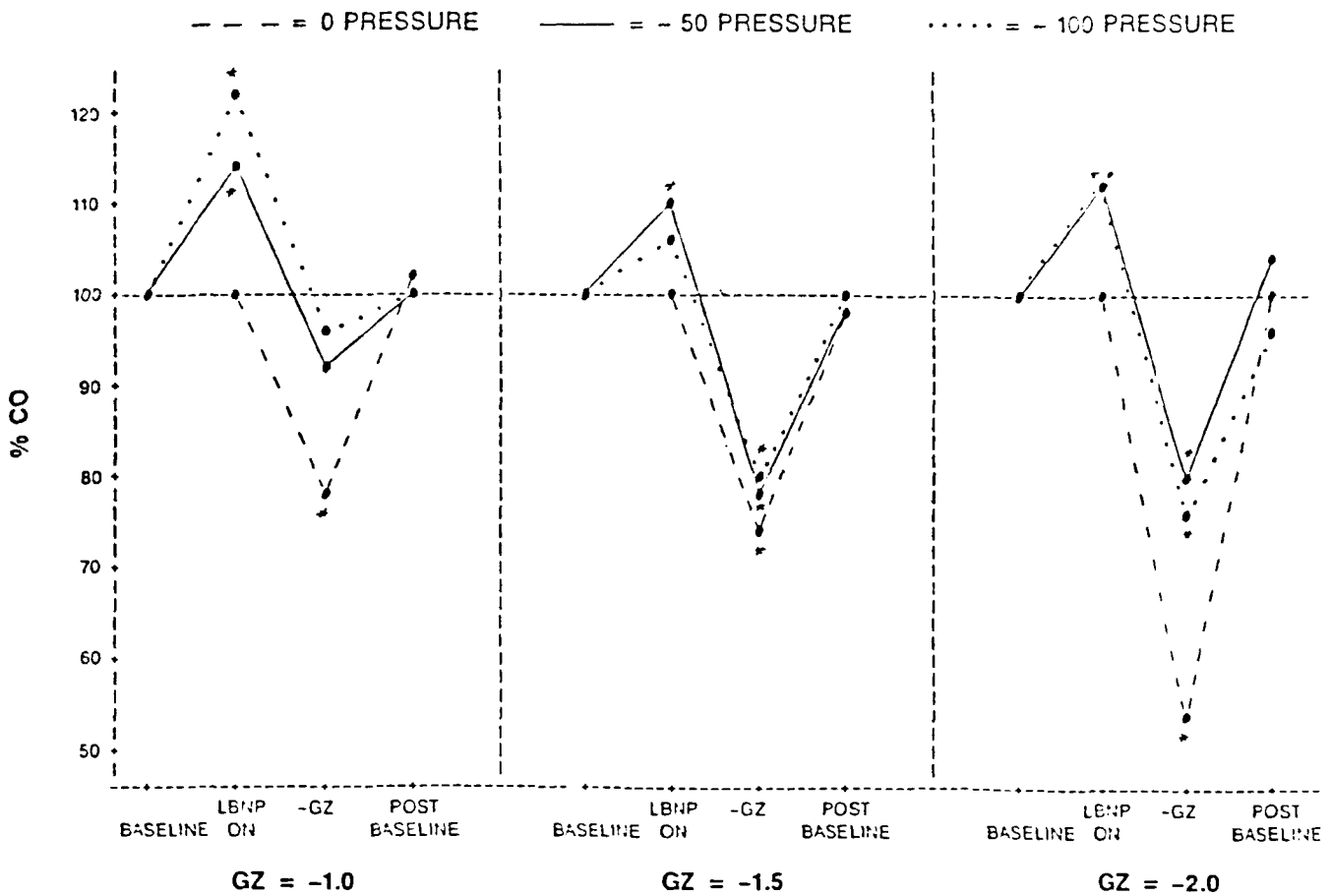


FIGURE 10. PERCENT OF BASELINE FOR CARDIAC OUTPUT



* = SIGNIFICANTLY DIFFERENT FROM 100

FIGURE 11. SUMMARIZATION OF CARDIAC VOLUMES ACROSS NEGATIVE GZ GRAPH

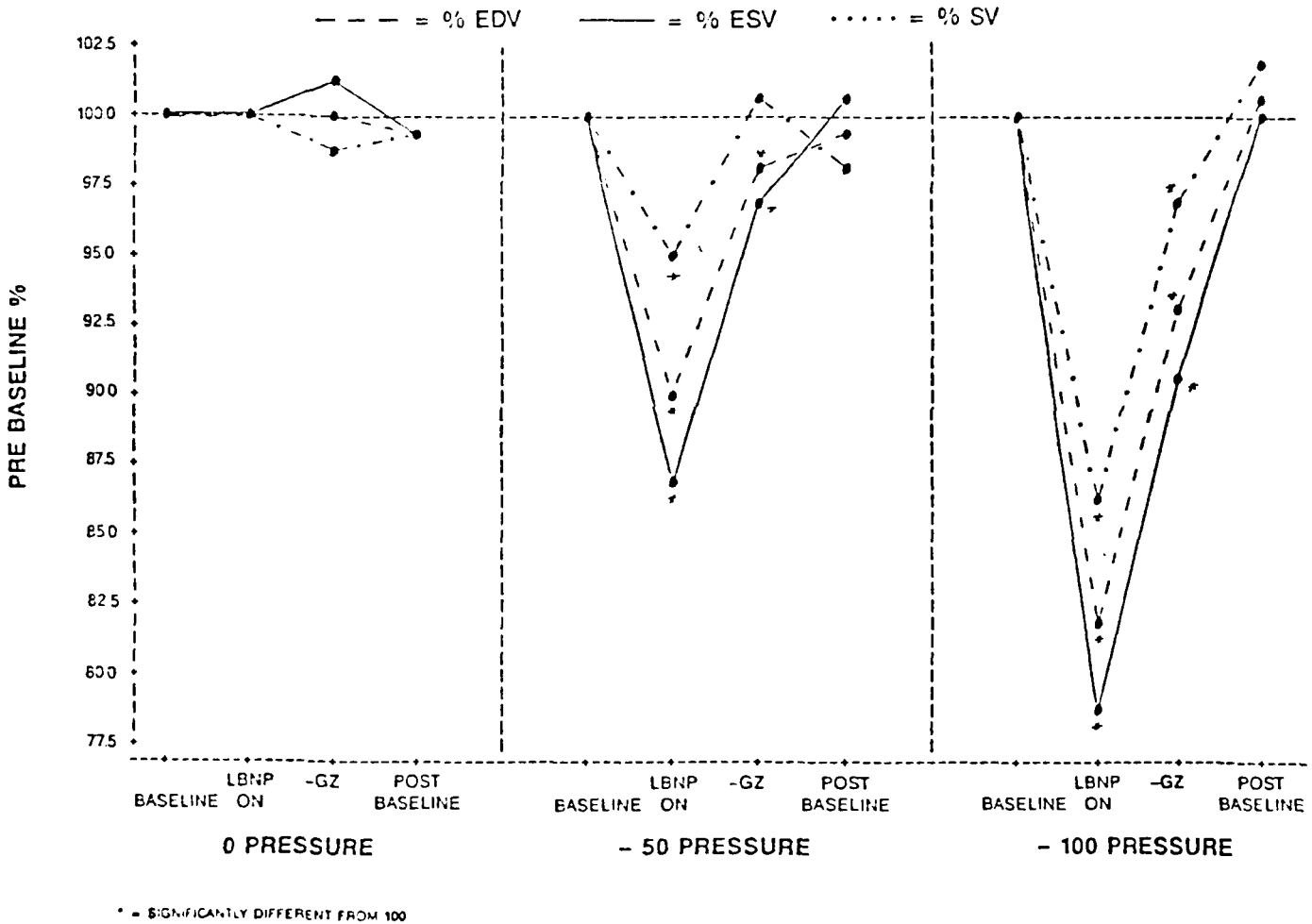


FIGURE 12. SUMMARIZATION OF EDV, ESV, HR, AND CO GRAPH

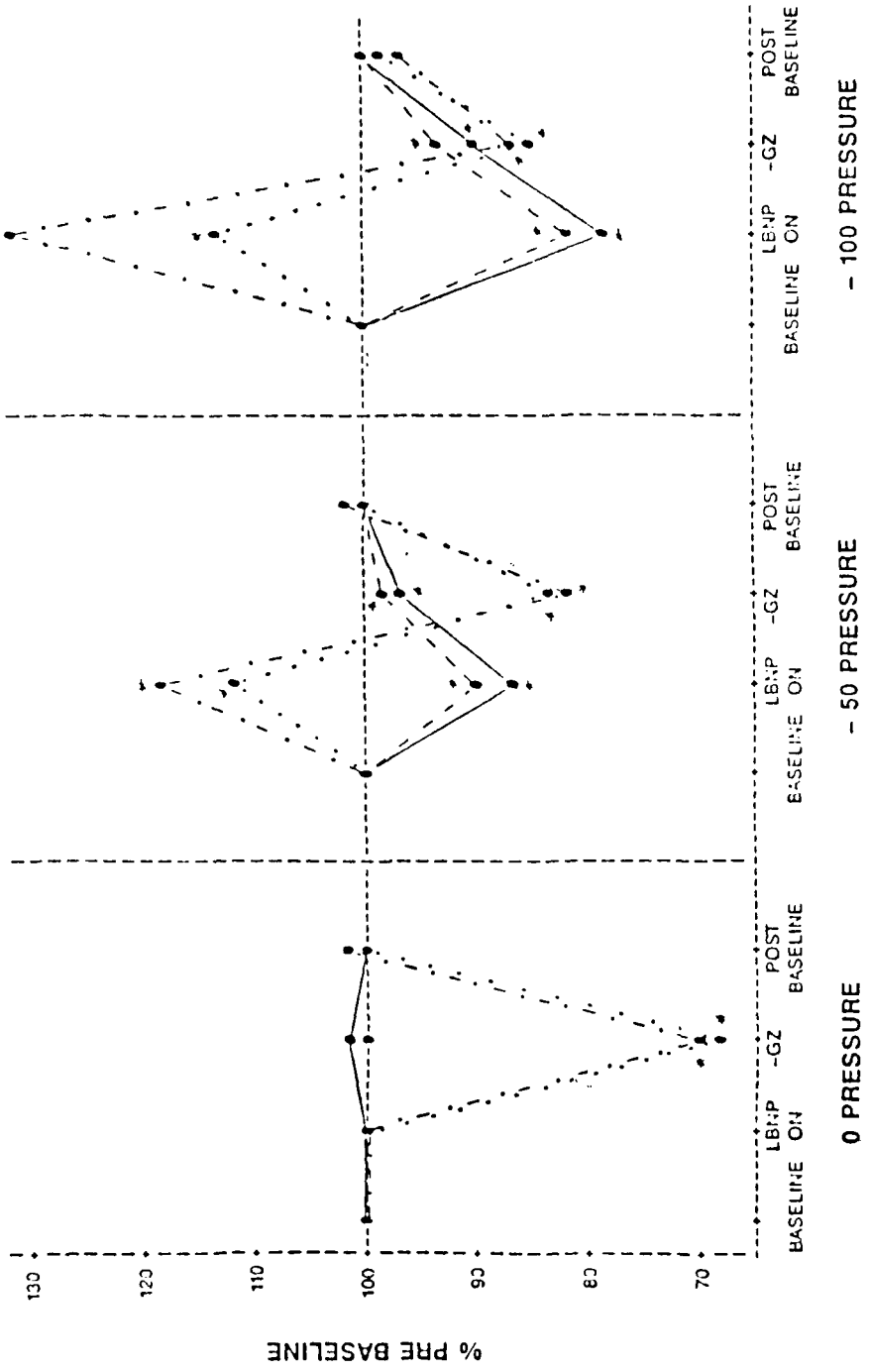
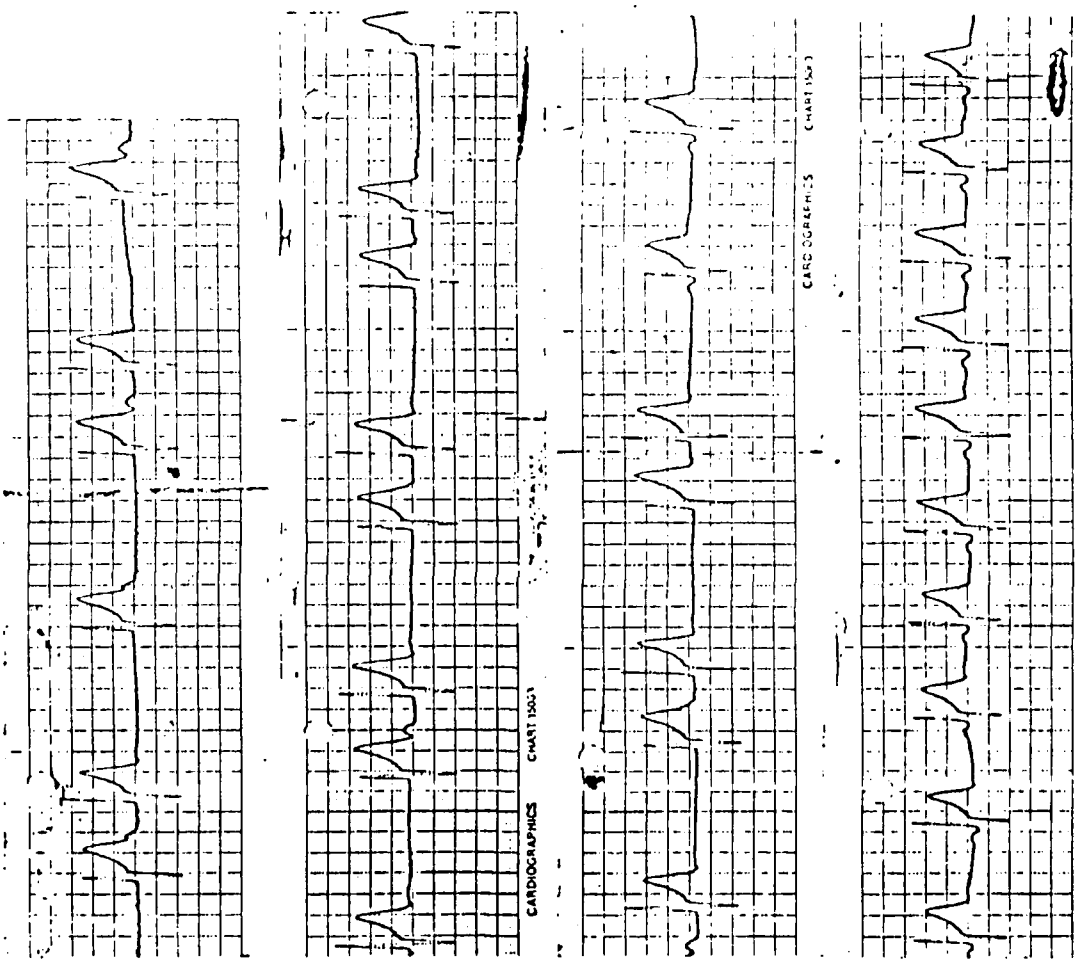


FIGURE 13. EKG OF ONE SUBJECT AT -2.0 GZ UNPROTECTED



Data were summarized for all the cardiac volumes and are shown in Figure 11. The data for all the variables except SV are shown in Figure 12. Figure 13 shows the EKG of one test subject during a -2.0 Gz run without LBNP and shows prolonged sinus pauses, a junctional rhythm, and some evidence of transient third degree heart block.

B. SUBJECTIVE

The results of the subjective questions are shown in Appendix E. Significant results are shown in Table 8.

The sign test was used to determine pairwise differences among the 9 combinations of -Gz and Pressure for the responses to the subjective questionnaire. Only three of the questions showed significant differences. See Table 8.

The question for facial congestion showed differences (p=.0156) in the ratings between -2.0 Gz and 0 mm Hg (abbreviated to -2.0/0) and all other exposures except -1.5/0 . The -1.5 Gz /0 mm Hg exposure was rated higher than -1.5/-100 (p=.0312), -1.0/-50 (p=.0156), and -1.0/-100 (p=.0156). Negative 2.0 Gz and -50 mm Hg was rated higher than -1.0/-100 (p=.0312) and -1.0/-50 (p=.0156).

Sinus pain ratings were higher in -2.0/0 than in all the other -Gz/LBNP exposures at p=.0312.

The degree of comfort ratings demonstrated the exposure of -2.0/0 less comfortable than all exposures (p=.0312) except -2.0/-100.

The overall ratings done after all the runs were significant when evaluated with Freidman's nonparametric test (p<.01). Using Freidman's

test for individual effects reveals the rankings are significant for each effect of -Gz and LBNP at p=.028. Using the sign test at -2.0 Gz, -50 and -100 mm Hg LBNP was tolerated better than 0 mm Hg at p=.0312, but there was no significance in the ratings between -50 and -100. At -1.5 and -1.0 Gz the same differences were found among the -100, -50, and 0 pressures. Examining the ranks across pressures reveals pairwise comparison at 0 mm Hg among -2.0, -1.5 and -1.0 shows that greater magnitude of -Gz consistently ranked lower (or, were rated worse) at p=.0312. At -50 mm Hg LBNP -2.0 was ranked lower than -1.5 and -1.0 (p=.0312), but the rankings between -1.5 and -1.0 were not significantly different. At -100 mm Hg the pairwise comparisons were all significant at p=.0312 and rankings followed the magnitude of -Gz as in the 0 mm Hg rankings. See Table 8.

TABLE 8

Subjective Questions Results

Using a 2-tailed sign test, pairwise mean ratings (i.e. comparing two Gz-Pressure conditions) were significantly different (p=.0156) if all 7 subjects had one of the conditions rated higher than the other condition or (p=.0312) if 6 subjects had one of the conditions rated higher than the other condition and one subject had them rated the same. The condition ratings that demonstrated pairwise significant differences (p<.0312) are shown by two-ended arrows below.

QUESTION	GZ	PRESSURE	MEAN RATING
FACIAL CONGESTION	-1.0	-50	1.6
FACIAL CONGESTION	-1.0	-100	1.7
FACIAL CONGESTION	-1.5	-100	1.7
FACIAL CONGESTION	-1.5	-50	2.1
FACIAL CONGESTION	-1.0	0	2.3
FACIAL CONGESTION	-2.0	-100	2.3
FACIAL CONGESTION	-2.0	-50	2.9
FACIAL CONGESTION	-1.5	0	3.3
FACIAL CONGESTION	-2.0	0	4.3
SINUS PAIN	-1.0	-100	1.1
SINUS PAIN	-1.0	-50	1.4
SINUS PAIN	-1.5	-100	1.4
SINUS PAIN	-1.0	0	1.6
SINUS PAIN	-1.5	-50	1.6
SINUS PAIN	-2.0	-100	1.9
SINUS PAIN	-1.5	0	2.0
SINUS PAIN	-2.0	-50	2.1
SINUS PAIN	-2.0	0	3.1
DEGREE OF COMFORT	-1.0	-100	1.4
DEGREE OF COMFORT	-1.0	-50	1.6
DEGREE OF COMFORT	-1.5	-100	1.7
DEGREE OF COMFORT	-1.0	0	1.9
DEGREE OF COMFORT	-1.5	-50	2.1
DEGREE OF COMFORT	-1.5	0	2.7
DEGREE OF COMFORT	-2.0	-50	2.7
DEGREE OF COMFORT	-2.0	-100	3.0
DEGREE OF COMFORT	-2.0	0	4.3

TABLE 8 (CONT.)

OVERALL RATING RESULTS

QUESTION	GZ	PRESSURE	MEAN RATING
OVERALL RATING	-2.0	0	1.0
OVERALL RATING	-1.5	0	2.3
OVERALL RATING	-2.0	-50	3.0
OVERALL RATING	-2.0	-100	4.5
OVERALL RATING	-1.5	-50	5.0
OVERALL RATING	-1.0	0	6.0
OVERALL RATING	-1.5	-100	6.5
OVERALL RATING	-1.0	-50	7.8
OVERALL RATING	-1.0	-100	8.8

The Friedman nonparametric Two Way Analysis of Variance was used to determine if the overall rating was significant.

Calculated S'= 39.5 (n=6,k=9) with $S_{\alpha=.01}$= 21 for 8 d.f.

Therefore the overall ratings were significant with $p<.01$.

In order to test for the pressure effect and -Gz effect within the rankings the mean ratings were ranked across each effect. These rankings were used to calculate the S'. For both -Gz and pressure:

Calculated S'= 6.0 (n=3,k=3) p=.028 for these parameters.

The rankings were significant across each effect. Pairwise comparisons can be used to examine pairwise differences. See text for the description of the differences.

V. DISCUSSION

A. PHYSIOLOGICAL

Accepted normal values of EDV, ESV, SV, and CO are 120-130 cc for EDV, 50-65 cc for ESV, 70 cc for SV and CO of 3.5-5.0 liters per minute (26). Experimental values as measured by single plane ellipse method from echocardiographs are lower than these normal values. Studies using the same technique found that this method often underestimated cardiac volumes that were definitively measured by angiography (12,35,63). EDV is underestimated by 30% by one estimate (35), bringing the baseline values of 87.7-90.3 cc observed in this experiment to normal ranges. The same study calculated ESV to be underestimated by 19%. SV is therefore underestimated because of the underestimation of EDV. CO calculated in this matter is dependent upon the SV also. Taking into account these underestimations, the data obtained for these cardiac variables seem to fall within the range of accepted cardiac physiological limits. Erbel noted that this error is mainly due to visualization of a tangential cross-section of the cardiac chamber leading to shorter axes being used for the ellipse determination. Also, trabeculation of the ventricle caused the volume to be underestimated because of low resolution (12).

Even with the magnitude of the underestimation, the average coefficient of variations for the three data points of 1.3%, 2.1%, and 2.5%for the variables EDV, ESV, and HR demonstrate the reproducibility of the data measurements. The data were analyzed as the percent change from baseline values, so the magnitude of the variables was not as important as the relative changes from baseline. Any bias in volume measurements would have been consistent across all measurements, and using the change from baseline values would help reduce this bias as a factor in our experimental results. There was no inter-observer variability as all measurements were performed by a single investigator.

The experimental results with LBNP alone showed cardiovascular changes somewhat consistent with previous studies of LBNP (69,74), although cardiac volumes were measured in few studies. A decrease in filling volumes of the heart (EDV, ESV, and SV) occurred with a reflex increase in HR.

The change in EDV, ESV, and SV measured in other studies with LBNP of -40 to -50 torr show a decrease of 19-26% for EDV (1,38,54), of 22% for ESV (38), SV changes of 22-35% for -40 torr (67) and 47-50% with -50 torr (14,15) measured using various techniques. The changes of 10% for EDV, 13% for ESV, and 5% for SV obtained for -50 torr in this -Gz study were notably less. In previous work at AAMRL utilizing echocardiography EDV, ESV, and SV changes with LBNP during static head down tilt showed decreases averaging 26% for EDV, 25% for ESV, and 28% for SV at -50 torr. In the head down tilt study the echo technique was accomplished by the same observer and data gatherer and these data obtained (70) were consistent with previous LBNP studies. Other factors in

this dynamic study must account for the differences. Some reasons can be postulated to account for the lower volume changes:

1. LBNP was instituted for only 10-15 seconds prior to -Gz acceleration. The duration of LBNP in other studies is usually several minutes and often includes a staircasing of negative pressures giving a total exposure of 15-20 minutes (14,15,29,33,57,67). Usually changes in volumes of lower extremities takes 1-2 minutes for 80-90% of the blood volume to initially pool, then extravasation occurs slowly as a result of the increased gradient in transmural pressures (52,53).
2. These subjects started their LBNP exposure in a seat with hip and knee flexed approximately 90 degrees. Some vascular shifts headward may have already occurred and stabilized prior to experimental LBNP. This factor cannot be changed readily because of the configuration constraints and the necessity to keep operational factors intact.
3. LBNP has not been used in any published experiment for a supine individual in a seated position. LBNP may not have the same degree of cardiovascular effects because of the vertical portion of the thighs.
4. Shifting or movement of the echo image may have occurred but this is not likely because of the quality of the images that was maintained.

These are all problems that cannot easily be changed in this design because of the desire to test LBNP in the environment of a cockpit configured to simulate operational conditions in order to evaluate protection of -Gz.

With other LBNP research HR changes were consistent with this study. LBNP of -50 mm Hg produced increases of HR of 15-40% (14,15,29,33,50,67), usually in the higher ranges for women subjects (14).

This experiment showed HR increases of 18% for -50 torr and 32% for -100 torr. These HR values were obtained using direct beat-to-beat interval measurements from the echo rather than the estimating techniques used for the volumes. These HR changes were consistent with those of the static head down tilt study (70).

CO increased in this study but CO usually decreased in other LBNP studies similarly but not identically to orthostasis (14,37,69). This could be due to the relatively small measured decrease in SV that occurred with the large increase in HR that is the other necessary factor in CO calculation. A Russian experiment in space showed similar increases in CO measured by two dimensional echo (2). It could be that the barorecepter stimulation of HR was greater than the decrease in SV after 10-15 seconds. A steady state after another 1-2 minutes could easily reveal a measured decrease in CO. Also the seated posture could have influenced the baroreceptors and made them more sensitive to the decrease with LBNP.

A main purpose of this study was to determine if LBNP could protect against -Gz acceleration. There was qualitative evidence that the data graphs showed a reversal of the LBNP effects in all variables toward the baseline values when the -Gz acceleration was applied. See Figures 11 and 12. This demonstrated that -Gz reversed the effects of LBNP. Also since -Gz exposures were performed without LBNP pressure comparisons can be made directly to exposures of -Gz with LBNP. Figure 12 reveals that -Gz's greatest percent effects with 0 mm Hg LBNP were with HR changes. LBNP of -50 and -100 mm Hg lessened these HR changes, -100 more so (but not statistically significant). LBNP had the same effect for CO changes probably because this variable was dependant on HR changes.

Since -Gz effects were not significantly different for EDV, ESV, and SV these exposures were averaged across -Gz exposures and an additional graph made comparing what happens to these variables across different levels of LBNP, see Figure 11. It seems evident that with 0 mm Hg pressure (i.e. no LBNP) there is little change in cardiac volumes (EDV, ESV, SV) with the magnitude of -Gz used in this study. This is consistent with the head down tilt study findings from Jennings, et al (31). Since this study used -2.0 as a maximum and the head down tilt was a -1 Gz maximum, the data suggest that from -1 to -2 there is little change in the measured left heart volumes.

The effect of -50 and -100 torr on the volume variables is obvious in Figure 11 when the exposures are averaged across -Gz. Increasing LBNP pressure caused greater decreases in all three measurements. With -Gz all the volumes returned closer to baseline values. In fact there was no significant difference in EDV, ESV, and SV between the baseline and -2 Gz exposures with LBNP.

Looking at Figure 12, when LBNP of -50 mm Hg was performed first, the cardiac volume changes with LBNP are dramatic. The heart volumes EDV, ESV, and SV decreased as a result of blood shift and venous pooling. Filling pressures decreased as reflected in other experiments (37,38,74). Peripheral resistance increased approximately 50% as measured in other studies (14,74). HR increased reflexly in order to maintain perfusion. In this stressed condition -Gz reversed all measured cardiac variables. EDV, ESV, and SV all returned to closer to baseline levels. HR changes reversed from significantly greater than 100% to significantly below 100%. Negative Gz seemed to normalize LBNP effects on volumes, showing that LBNP could possibly protect against -Gz effects.

The cardiac variable that was most obviously effected is HR, as shown in Figure 12 along with EDV, ESV, and CO. With no LBNP protection the HR percent decreases were large. With the -50 and -100 torr LBNP the HR changes with -Gz were lessened considerably and both pairwise comparisons between 0 and -50 torr and 0 and -100 torr level of LBNP were significant.

Other studies (27) show that -Gz acceleration increased venous and arterial cephalad pressures, but this study implies that the cardiac volumes do not change appreciably with up to -2.0 Gz under this experimental configuration and in these subjects. There are no available data on cardiac volumes at a greater magnitude than -1 Gz. The increase in pressure that occurs seems to effect the HR and CO data. We may assume that the subjects were in a homeostatic state and well hydrated, and that intravascular volumes were adequate. These initial pressure increases that occurred in the superior portion of the body were probably accompanied by a volume of blood that was not reflected in the EDV and ESV measurements because these are left heart measurements and initial blood pooling would be more dramatic in the venous portion because it is a low pressure reservoir. Since these data did not measure right heart volumes directly it may be postulated that they increased at least transiently. The heart rate (HR) slowed as a reaction to these cephalad pressures on the baroreceptors and peripheral vasodilation occurred. More blood volume was present cephalad because of -Gz acceleration but left ventricular volumes were not changed as measured by these experimental data. Research has shown that apical pulmonary congestion increased dramatically with -Gz acceleration, and it makes sense that the low pressure pulmonary vasculature and the low pressure systemic venous system will

dilate and act as the reservoir for the headward blood shift (5,65). It is evident from the experimental data that these volumes were not appreciably shifted to the left side of the heart.

With -100 torr the effects on EDV, ESV, SV, and HR with LBNP on were all dramatic and were greater in magnitude than -50 torr ($p<.0027$). The comparisons between -50 and -100 for CO was not statistically significant. When the -Gz acceleration was added, the pressure of -50 and -100 decreased HR more for -100 mm Hg, but not significantly more. With the -Gz effect the EDV and ESV were brought closer to baseline, and actually reversed the changes measured in HR and CO. The unloading of these cephalad pressures is possibly the key to an effective countermeasure for -Gz.

So with LBNP the effects of -Gz were lessened for the variables of HR and CO. Negative Gz reversed effects on ESV, EDV, SV, and HR for LBNP. It may have been better to perform the -Gz acceleration first and then attempt to protect with LBNP. This was not done in this experiment for several reasons. LBNP was instituted first so that the drop in suit pressures could be validated as complete. Also the drop in pressure often took 5-10 seconds. Since the -Gz plateaus were only 30 seconds in length, if LBNP was started at the -Gz plateau only 20-25 seconds of cardiovascular response could be monitored, and uniform parameters could have been difficult to ascertain. Discussions were made concerning instituting LBNP on the G rise, but differences in suit evacuation profiles existed between the two different suits and with 10 subjects the variability could not be accurately assessed. Also, this study was considered to be a preliminary study. LBNP operationally would probably be used in a stepwise fashion as G suit pressures are raised according to G magnitude. Also, since the 0 torr

control was used as a comparison, we thought the differences would be apparent.

A valid criticism may involve the lack of a control for the -Gz acceleration. The study could have used 0, -1.5 and -3.0 as easily and therefore provided a 0 Gz control. This may have improved the differences among the -Gz levels on the variables. It was hypothesized that the -Gz effect might be significant on the cardiac volumes from -1 to -2 but this was only true for HR.

The present study clearly shows that once blood was pooled with LBNP the -Gz acceleration results in lessening of the effects of LBNP. Work done in the opposite order, -Gz first then LBNP, would be an interesting followup and should be considered if the other technical problems mentioned could be solved. It may be easier or more difficult to unload the cephalad pressures of -Gz with LBNP later.

Another point to consider is the -Gz magnitude in the experiment was not in actuality pure -Gz. Pure -Gz could have been accomplished if the cab were allowed to swing freely, however for this experiment the cab was locked in place. This was done because of technical difficulties with overshooting the -Gz magnitude when the cab was free. Also the subject would be tilted at -1 Gz in between exposures possibly biasing results. Therefore all subjects experienced +1 Gx throughout exposures. It was not thought to be a contributing factor to the results because it was consistent. However the resulting G vector for the -1 Gz exposure was actually 1.41 G in a 45 degree direction from horizontal. For the -1.5 Gz the resultant vector was 1.80 G in magnitude 34 degrees from horizontal, and for -2 Gz the magnitude was 2.23 G at 27 degrees from horizontal. As the magnitude

of -Gz in this experimental configuration increases the resultant G vector becomes closer to pure -Gz.

B. SUBJECTIVE

These subjective questions were pooled from several -Gz articles (27,66). The results from subjective questionnaires were in agreement with physiological findings despite the fact only 7 subjects completed the questionnaires and one of these did not perform the rankings. For facial congestion, where the most differences were noted, both the -1.5 and -2 Gz exposure without pressure were rated the worst and the ratings were significantly higher than most of the other ratings. Negative 2 Gz/-50 torr also rated higher than the -1 Gz/-50 and -1 Gz/-100 runs.

For the sinus pain question, the ratings for -2/0 were significantly higher than all other exposures. For the degree of comfort, the ratings for -2/0 were significantly higher than all other combination of conditions except one. The subjective ratings were handled in a different way than in Sieker's study (see Table 1), but there is a similar preponderance of ratings for facial congestion at these levels of -Gz (66). Interestingly, his experiments showed no complaints of sinus pain up to -2 Gz where in our experiment the symptom was noted at a moderate degree at -2 Gz in many subjects.

Examining the mean ratings for almost all questions one can easily note that the -2 Gz/0 torr run was rated consistently worst if there was a difference. The -50 and -100 torr LBNP runs were rated usually better as one would expect if LBNP was providing some protection. Although not

all questions gave significant results, at higher -Gz levels these signs and symptoms may be more pronounced and differences may emerge. Having more subjects in the study complete the questionnaire might have allowed differences to emerge.

The overall rating was a forced ranking scale that seemed to give similar results to the other questions. A problem with these data was that the rating could not be completed until the 9 combinations were done, often taking 8-14 days. The memory of each exposure could certainly have faded. Also the ranking that was used (1 being worst, 9 being best) was opposite the ranking of the rest of the questions. Although this could have been confusing to the subjects there was no obvious "opposite" ranking noticed, however the scale should be reversed for further work.

Using the Friedman nonparametric rank test the effect of -Gz and the effect of LBNP pressure on ranking were significant at p=.028. See Table 8. This demonstrates the consistency in these ratings for each effect. The -Gz was consistently rated less desirable as the magnitude increased. The LBNP was preferred across the increasing pressures. The pairwise findings are fairly consistent and remarkable in that there were only 6 completed rankings yet there was consistency in the all the rankings for -Gz, and consistency for the extremes of LBNP pressure, i.e. 0 and -100 torr. One could argue that the rankings were not blinded, but this might be possible in further studies. These subjective findings are not to be minimized. Often the limitation of -Gz acceleration is more subjective than physiologic and subjective improvement will be a vitally important aspect of providing a countermeasure to -Gz.

The echocardiographic data obtained for -Gz was in some ways disappointing. No definitive work on estimates of cardiac volumes had been done at greater than -1 Gz. A tilt experiment done at 90 degrees head down tilt showed essentially no changes in cardiac volumes (31). The pressure changes documented in the early -Gz studies in the cephalad arterial and venous studies in animals and in venous studies in humans are not apparently accompanied by measurements of cardiac volume changes

Negative Gz increases cephalad blood volume and increases headward arterial and venous vascular pressures. The effect on arterial volumes is probably minimal due to the elasticity of the arterial system. The venous volumes increase markedly due to their distensibility. The increased venous volumes and pressure probably provide most of the subjective symptoms of facial swelling, sinus pressure, and discomfort. The arterial baroreceptor reflex is activated by the higher arterial pressures. The low pressure baroreceptors are also stimulated and increase the sensitivity of the arterial reflex (26). The reflex gives vagal stimulation and sympathetic inhibition, resulting in a decrease in heart rate, a decrease in contractility, and peripheral vasodilation. Afterload will decrease. Despite the possibility of transient right heart volume increases, the left heart volume changes were unremarkable at least at these tested levels of -Gz. Higher -Gz levels may demonstrate some effect.

It is interesting to note the hydrostatic indifference point, or HIP that Gauer discusses (21) was measured to be 8-10 cm caudal to the heart. Head down tilt studies of 5-6 degrees fairly consistently exhibited increases in central and right heart volumes when measured after 15-10 minutes (41,54). Knitelius and Stegeman noticed an increase of total heart volume of 5.2% in a 6 degree head down tilt after 5 minutes (41). In a 6 degree

head down tilt study by Lollgen, right atrium and pulmonary artery pressures increased significantly after 15 minutes after tilt was initiated but left ventricle volumes only increased after 40 minutes (47). These findings are consistent with no change in volumes found in the short term with -Gz in our echo study.

Tilts of 15 degrees head down with CVP and pulmonary artery pressures measurements were usually unchanged in the first several hours (38). This was seen to differ with head down tilt of 75-90 degrees so that filling pressures and volumes at the right side of the heart were unchanged in some studies (3,20,73), but other observations indicated a slight increase in right heart pressures and volumes (37,62). The variability is probably the result of subject variation, differences in baselines, technique, and/or measurements. There is evidence of the HIP may shift to near the second intercostal space with 90 degree head down tilt (20,31).

If -1 Gz acceleration causes this shift in the pressure gradient then -Gz of greater magnitude may shift this HIP even more cephalad. This can be considered analogous to the HIP shifting caudally in +Cz acceleration. Therefore during -Gz of a greater magnitude the venous pressure and volume and the arterial pressure at the head are measured to be much higher (as measured by Gauer, Henry, and Rosenfield) but the filling pressures at the heart itself are probably decreasing, as they do with +Gz accelerations (28,30,45). Cardiac volumes were only determined in this present study at up to -2 Gz and there was little change in volumes. Further work at greater levels of -Gz may illuminate a reduction in these cardiac volumes.

The image quality of the echocardiographs was satisfactory for technical analysis and although the technique has been shown to

underestimate volumes it proved to be a consistent measure in these experimental conditions. The average coefficients of variation for the duplicate measurements are very good and probably reflect the consistency in placement and direction of the ultrasound transducer for each subject. Improvements in measuring volumes with the two dimensional echocardiograph (56) are on the horizon and may be of use in the future.

LBNP did not cause syncope or near syncope in this study. This could be due to the population pool. Only males were utilized in order to be consistent, and males may have less syncope with LBNP than females (50), although this has been questioned by other authors (14). Probably the most important reason for the lack of syncopal symptoms in our subjects was that the duration of the LBNP was only 40-50 seconds total and for all exposures at least -1 Gz was used which partially reversed the LBNP effects. Also, the subjects' legs were elevated into the seated position and this may have lessened the LBNP effects. Indeed the changes in cardiac volumes were less than seen in supine LBNP studies for reasons postulated earlier, although the heart rate changes were similar. For whatever reason, subjective tolerances for LBNP or for -Gz were not exceeded in this study.

One point that may be useful to discuss is the importance of the operational nature of the experiment. The subjects in our study were seated in a cockpit seat so that the backrest was horizontal. The participant's knees and hips were flexed to approximately 90 degrees. The suit design which allowed this was fairly unique and was made so in order to evaluate LBNP in an operational environment, i.e. seated as a pilot/occupant would experience it. LBNP caused quantitative changes in cardiovascular parameters in our seated subjects that were similar to those in studies of supine subjects if one accounts for the variations due to the underestimation

of heart volumes. Also, it seems that the vertical upper leg portion would decrease the LBNP effect and indeed further testing may prove this as true. Possibly greater magnitudes of LBNP could counteract any negative posture effect. It remains to other studies to examine this.

Venous pressures measured by Henry, Gauer, Rosenfield and others during -Gz showed a lessening of the pressures measured near the head when hydrostatic column length was changed by arterial occlusive cuffs on the lower extremities or by a change in posture. Certainly the change in posture for our seated subjects could lessen the -Gz effect because the upper legs were orthogonal to the -Gz field and would not contribute to the arterial/venous vascular loading. However one must remember that the resultant G vectors would partially effect vertical leg volumes because of the +1 Gx component. We kept the +1 Gx constant to control this. It is more difficult to consider how these vertical portions effect the combination of LBNP and -Gz.

Certainly this study needs to be performed at higher -Gz levels in order to prove efficacy at operational levels. The -2 Gz used in this study was shown tolerable in other studies (27,66) and was fairly well tolerated in this study despite sinus bradycardia into the 30's and petechiae about the head in some subjects. Figure 13 shows an EKG in one subject during a -2.0 Gz with no LBNP run. The transient sinus pauses and junctional rhythm with heart rate of 30-40 per minute demonstrate the high vagal response from the -Gz acceleration.

C. FURTHER STUDIES

Later studies could utilize subjects protected at -3 Gz and perhaps higher and results could be evaluated by the same methods. Since the AAMRL Generic Sustained Acceleration Protocol limits exposure to -2.5 Gz (unprotected), accelerations above this limit must have protection in force. LBNP therefore must be instituted prior to the -Gz plateau. A balanced design such as in this experiment using -Gz levels of -1.0, -2.5, and -4.0 could be combined with 0, -50, and -100 or possibly 0, -60 and -120 in order to evaluate the LBNP protection at higher -Gz. The -4.0 Gz exposure with 0 torr LBNP would need to be dropped because of the Generic Protocol limits. Increasing the LBNP pressures to greater than -100 torr has not been routinely practiced experimentally but since the exposure is of short duration and the LBNP is counteracted by -Gz, it should not be dangerous.

Neck pressurization may provide a synergistic effect in further -Gz experiments by decreasing the stimulation of the carotid sinus. A neck pressurization experiment by Mancia showed more of an effect on arterial afterload reduction than on the bradycardia (48), however; there may be some beneficial effect on the venous pressures with -Gz.

VI. CONCLUSION

The pressure effects of -50 and -100 torr LBNP demonstrated protection for HR changes. LBNP also revealed persistent unloading of the cardiovascular volumes despite -Gz acceleration. Subjective symptoms of -Gz that were noted at higher -Gz levels were improved with LBNP. There is evidence that LBNP may serve as a countermeasure to the adverse effects of -Gz acceleration.

VIII. APPENDICES

APPENDIX A
RANDOMIZATION MATRIX

Legend:

	Pressure	Gz
A =	0 mm Hg	1= -1.0 Gz
B =	-50 mm Hg	2= -1.5 Gz
C =	-100 mm Hg	3= -2.0 Gz

DAY

	1			2			3		
Run	1	2	3	1	2	3	1	2	3
Subject									
1	A2	C3	B1	B3	A1	C2	C1	B2	A3
2	B3	C1	A2	A1	B2	C3	C2	A3	B1
3	C3	A1	B2	B1	C2	A3	A2	B3	C1
4	A1	B2	C3	C2	A3	B1	B3	C1	A2
5	C1	A2	B3	B2	C3	A1	A3	B1	C2
6	B2	A3	C1	C3	B1	A2	A1	C2	B3
7	A3	B1	C2	C1	A2	B3	B2	C3	A1
8	C2	B3	A1	A3	C1	B2	B1	A2	C3
9	B1	C2	A3	A2	B3	C1	C3	A1	B2

If more than 9 subjects are used, reverse the rows of the design and assign subject 10 to row 1 , subject 11 to row 2 , etc.

APPENDIX B SUBJECTIVE QUESTIONNAIRE

Use of LBNP as a Protection for -G_Z acceleration
QUESTIONS

Subject # ____
-Gz profile ____
LBNP level ____

Please answer in regard to the last centrifuge run...

On a scale from 1 to 5,(1 being none, 5 being very severe) rate the amount of facial congestion felt
1 2 3 4 5

On a scale from 1 to 5, (1 being none, 5 being very severe) rate the degree of headache felt
1 2 3 4 5

Please rate the degree of sinus pain felt(1= none, 5 being very severe)
1 2 3 4 5

Did you notice any change or difficulties in vision ?
Yes No

Rate any blurring of vision noticed
1 2 3 4 5

Rate any degree of "graying" of vision noticed
1 2 3 4 5

Did any degree of double vision occur?
Yes No

If yes, please rate the degree
1 2 3 4 5

Rate any loss or change in hearing noted (1 being none and 5 being a very severe change or loss)
1 2 3 4 5

Rate your degree of comfort, (1 being very comfortable, 5 being very uncomfortable)
1 2 3 4 5

Any ringing in your ears? Yes No
If yes, please rate the degree
1 2 3 4 5

Are there any more comments you'd like to make concerning this run?

(After all the runs)
Please rate the combination of conditions from the best to the worst, 1 to 9.

APPENDIX C

TABLE C. 1 ACTUAL DATA

MEANS AND STANDARD DEVIATIONS OF SUBJECTS (N=9)

ORDER	MEAN EDV	MEAN ESV	MEAN HR	MEAN SV	MEAN CO	STD EDV	STD ESV	STD HR	STD SV	STD CO
Gz PRESSURE										
-1.0 0										
BASELINE	90.3	52.6	71.1	37.6	2.64	10.6	4.2	8.7	8.2	0.52
LBNP ON	90.3	52.6	71.1	37.6	2.64	10.6	4.2	8.7	8.2	0.52
-GZ	90.5	53.8	56.1	36.7	2.03	12.7	5.8	6.6	8.5	0.36
POST BASE	89.5	52.5	73.6	37.0	2.70	9.5	3.4	10.5	7.8	0.66
Gz PRESSURE										
-1.0 -50										
BASELINE	88.7	53.0	72.5	35.7	2.53	8.9	3.9	11.3	6.7	0.32
LBNP ON	81.1	46.0	82.9	35.1	2.86	7.8	2.4	13.3	6.3	0.39
-GZ	87.2	50.6	63.8	36.7	2.33	10.0	3.9	11.4	8.6	0.70
POST BASE	88.5	53.5	72.6	34.9	2.51	9.0	4.4	7.8	6.8	0.37
Gz PRESSURE										
-1.0 -100										
BASELINE	87.6	50.9	71.6	36.8	2.59	9.6	5.2	10.5	7.2	0.44
LBNP ON	73.6	40.6	94.7	33.0	3.10	7.1	3.5	9.3	5.5	0.45
-GZ	82.0	46.2	70.7	35.8	2.48	8.5	4.3	14.3	6.3	0.41
POST BASE	87.8	50.9	69.9	36.8	2.55	8.6	4.2	7.3	6.2	0.34

TABLE C. 1(CONT.)

ORDER	MEAN EDV	MEAN ESV	MEAN HR	MEAN SV	MEAN CO	STD EDV	STD ESV	STD HR	STD SV	STD CO
Gz PRESSURE										
-1.5 0										
BASELINE	89.3	52.2	73.9	37.1	2.71	7.8	3.6	11.2	7.2	0.51
LBNP ON	89.3	52.2	73.9	37.1	2.71	7.8	3.6	11.2	7.2	0.51
-GZ	89.7	52.0	54.5	37.6	2.00	8.5	3.8	13.5	7.4	0.48
POST BASE	89.7	52.0	71.6	37.7	2.66	9.2	3.6	10.0	7.9	0.46
Gz PRESSURE										
-1.5 -50										
BASELINE	88.2	51.3	71.1	36.9	2.58	9.9	4.4	10.5	6.6	0.33
LBNP ON	79.7	44.8	82.8	34.8	2.84	7.9	4.4	12.2	5.3	0.32
-GZ	84.8	49.4	57.0	35.5	1.99	9.3	4.0	7.4	6.4	0.27
POST BASE	87.4	51.6	70.9	35.8	2.50	9.7	3.9	7.5	7.2	0.38
Gz PRESSURE										
-1.5 -100										
BASELINE	88.5	52.6	76.0	35.9	2.70	8.8	3.8	8.8	6.0	0.39
LBNP ON	71.8	41.0	94.1	30.8	2.88	7.5	2.9	7.7	6.1	0.51
-GZ	80.4	46.9	65.6	33.5	2.18	6.7	3.3	13.4	5.2	0.52
POST BASE	88.8	52.3	74.5	36.5	2.71	9.4	3.4	6.8	7.1	0.54

TABLE C. 1(CONT.)

ORDER	MEAN EDV	MEAN ESV	MEAN HR	MEAN SV	MEAN CO	STD EDV	STD ESV	STD HR	STD SV	STD CO
Gz PRESSURE -2.0 0										
BASELINE	88.7	51.5	73.3	37.2	2.69	7.9	2.3	7.9	6.0	0.26
LBNP ON	88.7	51.5	73.3	37.2	2.69	7.9	2.3	7.9	6.0	0.26
-GZ	88.7	52.3	42.0	36.4	1.46	9.1	4.3	13.8	6.5	0.34
POST BASE	87.4	51.1	74.9	36.3	2.69	7.5	2.5	9.5	5.9	0.40
Gz PRESSURE -2.0 -50										
BASELINE	88.2	50.8	71.6	37.3	2.62	10.1	4.3	12.0	8.7	0.52
LBNP ON	77.5	43.6	87.3	33.9	2.93	6.3	3.0	10.5	6.1	0.51
-GZ	87.8	49.8	54.9	38.0	2.09	13.0	6.3	9.2	8.2	0.59
POST BASE	88.1	50.8	72.9	37.2	2.70	9.1	4.4	7.5	7.7	0.54
Gz PRESSURE -2.0 -100										
BASELINE	87.7	51.7	75.7	36.1	2.71	7.8	3.3	5.9	6.0	0.37
LBNP ON	70.3	40.7	103.3	29.6	3.04	5.2	2.2	5.7	5.4	0.49
-GZ	82.7	46.9	58.5	35.8	2.04	11.0	6.8	12.0	8.1	0.43
POST BASE	88.4	51.4	71.0	37.0	2.60	8.4	3.6	7.9	6.0	0.35

TABLE C. 2 ACTUAL DATA-PERCENT OF BASELINE

ORDER	MEAN % EDV	MEAN % ESV	MEAN % HR	MEAN % SV	MEAN % CO	STD % EDV	STD % ESV	STD % HR	STD % SV	STD % CO
Gz PRESSURE										
-1.0 0										
LBNP ON	100.0	100.0	100.0	100.0	100.0	0.0	0.0	0.0	0.0	0.0
-GZ	100.2	102.2	79.5*	97.5	77.2*	3.3	4.7	9.0	6.7	7.5
POST BASE	99.2	99.9	103.5	98.5	102.1	1.7	3.3	7.6	5.6	11.0
Gz PRESSURE										
-1.0 -50										
LBNP ON	91.5*	87.0*	114.6*	98.8	113.0*	3.3	3.3	8.3	8.4	10.4
-GZ	98.4	95.4*	88.4*	102.8	91.2	5.9	2.5	11.4	15.0	19.5
POST BASE	99.8	101.0	101.4	98.0	99.5	2.2	2.0	11.4	6.2	14.2
Gz PRESSURE										
-1.0 -100										
LBNP ON	84.3*	80.2*	133.9*	90.5*	121.3*	5.2	4.9	16.6	7.8	19.8
-GZ	93.7*	91.1*	98.6	98.0	96.4	4.0	5.2	11.0	6.1	11.0
POST BASE	100.3	100.4	98.6	100.7	99.2	1.6	3.1	10.2	4.5	8.7

* = SIGNIFICANT CHANGE FROM 100 (BASELINE)

TABLE C. 2 (CONT.)

ORDER	MEAN % EDV	MEAN % ESV	MEAN % HR	MEAN % SV	MEAN % CO	STD % EDV	STD % ESV	STD % HR	STD % SV	STD % CO
Gz PRESSURE										
-1.5 0										
LBNP ON	100.0	100.0	100.0	100.0	100.0	0.0	0.0	0.0	0.0	0.0
-GZ	100.4	99.7	73.5*	101.5	74.8*	1.9	4.0	11.9	4.6	13.7
POST BASE	100.3	99.6	97.4	101.7	98.9	2.5	2.7	7.4	6.0	6.2
Gz PRESSURE										
-1.5 -50										
LBNP ON	90.4*	87.4*	116.7*	94.8*	110.8*	2.4	3.5	8.1	4.1	10.3
-GZ	96.2*	96.3*	80.6*	96.3	77.6*	2.9	4.4	6.2	4.9	7.9
POST BASE	99.1	100.8	100.8	96.7	97.5	1.5	4.1	10.6	6.2	11.6
Gz PRESSURE										
-1.5 -100										
LBNP ON	81.1*	77.9*	124.4*	85.7*	106.7	2.7	3.2	8.9	6.3	11.8
-GZ	91.1*	89.4*	85.9*	93.9	80.9*	5.1	5.9	11.8	8.4	15.6
POST BASE	100.3	99.4	98.4	101.6	99.9	1.5	2.0	7.0	5.8	8.9

* = SIGNIFICANT CHANGE FROM 100 (BASELINE)

TABLE C. 2 (CONT.)

ORDER	MEAN % EDV	MEAN % ESV	MEAN % HR	MEAN % SV	MEAN % CO	STD % EDV	STD % ESV	STD % HR	STD % SV	STD % CO
Gz PRESSURE										
-2.0 0										
LBNP ON	100.0	100.0	100.0	100.0	100.0	0.0	0.0	0.0	0.0	0.0
-GZ	100.0	101.5	56.3*	97.7	54.9*	4.1	5.7	14.1	6.4	13.6
POST BASE	98.7	99.3	102.3	97.9	100.4	2.8	1.9	10.8	7.1	14.9
Gz PRESSURE										
-2.0 -50										
LBNP ON	88.3*	86.1*	123.4*	91.9	112.5*	4.4	4.9	14.6	10.7	10.0
-GZ	99.4	98.0	77.2*	102.5	79.0*	4.5	8.9	9.6	8.8	11.7
POST BASE	100.0	100.0	103.0	100.4	103.2	2.4	2.8	9.7	7.3	9.8
Gz PRESSURE										
-2.0 -100										
LBNP ON	80.2*	79.0*	137.4*	82.0*	112.6*	2.6	4.5	14.3	6.9	14.1
-GZ	94.1	90.9	77.8*	98.7	75.7*	7.9	12.6	18.5	9.6	13.8
POST BASE	100.8	99.6	94.1	02.8	96.9	2.4	3.8	11.2	6.7	14.1

* = SIGNIFICANT CHANGE FROM 100 (BASELINE)

APPENDIX D

T-TESTS OF PHYSIOLOGIC DATA

PERCENT OF BASELINE FOR LBNP ON

(I) T-TEST P-VALUES FOR MAIN EFFECT PRESSURE

VARIABLE	-50 PRESSURE	-100 PRESSURE	-50 PRESSURE MINUS -100 PRESSURE
% EDV	.0001	.0001	.0001
% ESV	.0001	.0001	.0001
% HR	.0001	.0001	.0027
% SV	.0140	.0001	.0013
% CO	.0001	.0128	.7302

PERCENT OF BASELINE FOR -GZ

(I) T-TEST P-VALUES FOR MAIN EFFECT -GZ

VARIABLE	-1.0 GZ	-1.5 GZ	-2.0 GZ	-1.0 GZ MINUS -1.5 GZ	-1.0 GZ MINUS -2.0 GZ	-1.5 GZ MINUS -2.0 GZ
% EDV	.0041	.0004	.2141	.1503	.8038	.2736
% ESV	.0031	.0026	.2671	.4847	.8307	.5489
% HR	.0026	.0001	.0001	.0063	.0001	.0120
% SV	.7867	.0542	.8014	.2600	.9319	.1515
% CO	.0026	.0001	.0001	.0047	.0002	.0156

(II) T-TEST P-VALUES FOR MAIN EFFECT PRESSURE

VARIABLE	0 PRESSURE	-50 PRESSURE	-100 PRESSURE	0 PRESSURE MINUS -50 PRESSURE	0 PRESSURE MINUS -100 PRESSURE	-50 PRESSURE MINUS -100 PRESSURE
% EDV	.8153	.0050	.0006	.0102	.0001	.0036
% ESV	.2935	.0189	.0006	.0021	.0001	.0025
% HR	.0001	.0001	.0133	.0199	.0050	.1502
% SV	.5198	.8063	.0257	.4973	.2651	.1196
% CO	.0001	.0002	.0014	.0051	.0017	.4033

NOTE: T-TESTS INVOLVED 2 HYPOTHESES: (1) MEANS WERE EQUAL TO 100, (2) MEAN DIFFERENCES WERE EQUAL TO 0.

APPENDIX D(CONT.)

T-TESTS OF PHYSIOLOGIC DATA

PERCENT OF BASELINE FOR -GZ

(III) T-TEST P-VALUES FOR PRESSURE WITHIN -GZ

GZ	VARIABLE	0 PRESSURE	-50 PRESSURE	-100 PRESSURE	0 PRESSURE MINUS -50 PRESSURE	0 PRESSURE MINUS -100 PRESSURE	-50 PRESSURE MINUS -100 PRESSURE
-1.0	% EDV	.8920	.4402	.0016	.4470	.0058	.1615
	% ESV	.2007	.0006	.0009	.0113	.0002	.0642
	% HR	.0001	.0162	.7466	.1734	.0132	.0024
	% SV	.2870	.5914	.3440	.2672	.1739	.4210
	% CO	.0001	.2155	.3537	.1161	.0064	.3565
-1.5	% EDV	.5623	.0043	.0008	.0031	.0018	.0180
	% ESV	.8272	.0357	.0006	.0705	.0033	.0036
	% HR	.0002	.0001	.0073	.1579	.0006	.2548
	% SV	.3406	.0512	.0604	.0885	.0030	.5392
	% CO	.0006	.0001	.0061	.6110	.0386	.6356
-2.0	% EDV	.9954	.6873	.0561	.6908	.0112	.0391
	% ESV	.4507	.5207	.0623	.1174	.0107	.0667
	% HR	.0001	.0001	.0069	.0145	.0438	.9187
	% SV	.3235	.4242	.7007	.2912	.8368	.3518
	% CO	.0001	.0007	.0008	.0048	.0191	.5040

(IV) T-TEST P-VALUES FOR -GZ WITHIN PRESSURE

PRESSURE	VARIABLE	-1.0 GZ	-1.5 GZ	-2.0 GZ	-1.0 GZ MINUS -1.5 GZ	-1.0 GZ MINUS -2.0 GZ	-1.5 GZ MINUS -2.0 GZ
0	% EDV	.8520	.5623	.9954	.8653	.8806	.8210
	% ESV	.2007	.8272	.4507	.3487	.6329	.4879
	% HR	.0001	.0002	.0001	.1786	.0015	.0032
	% SV	.2870	.3406	.3235	.0922	.9069	.0518
	% CO	.0001	.0006	.0001	.6141	.0032	.0013
-50	% EDV	.4402	.0043	.6873	.4274	.7419	.1461
	% ESV	.0006	.0357	.5207	.5179	.4674	.5855
	% HR	.0162	.0001	.0001	.1062	.0482	.3883
	% SV	.5914	.0512	.4242	.3795	.9461	.0927
	% CO	.2155	.0001	.0007	.1203	.1221	.7626
-100	% EDV	.0016	.0008	.0561	.1843	.8980	.3123
	% ESV	.0009	.0006	.0623	.4879	.9778	.7008
	% HR	.7466	.0073	.0069	.6108	.0020	.1309
	% SV	.3440	.0604	.7007	.2479	.8603	.3733
	% CO	.3537	.0061	.0008	.0190	.0036	.3970

NOTE: T-TESTS INVOLVED 2 HYPOTHESES: (1) MEANS WERE EQUAL TO 100, (2) MEAN DIFFERENCES WERE EQUAL TO 0.

APPENDIX E. SUBJECTIVE QUESTIONS-ACTUAL DATA

This table includes the individual ratings for each subject with mean ratings for each condition. For each question the scale was from 1 (none) to 5 (severe). The overall rating scale (last question) was a ranking of each exposure from (worst) to 9 (best). Within each question, data is ranked by mean rating. Using a 2-tailed sign test, pairwise mean ratings (i.e. comparing two Gz-Pressure conditions) were significantly different (p=.0156) if all 7 subjects had one of the conditions rated higher than the other condition or (p=.0312) if 6 subjects had one of the conditions rated higher than the other condition and one subject had them rated the same.

	CONDITIONS		MEAN	SUBJECT #						
QUESTION	GZ	PRESSURE	RATING	830013	860005	860012	860016	860017	870002	870005
FACIAL CONGESTION	-1.0	-50	1.6	1	2	1	1	2	2	2
FACIAL CONGESTION	-1.0	-100	1.7	1	2	1	2	2	2	2
FACIAL CONGESTION	-1.5	-100	1.7	1	2	1	2	1	3	2
FACIAL CONGESTION	-1.5	-50	2.1	1	2	2	2	3	3	2
FACIAL CONGESTION	-1.0	0	2.3	2	2	2	3	2	3	2
FACIAL CONGESTION	-2.0	-100	2.3	1	2	3	2	2	3	3
FACIAL CONGESTION	-2.0	-50	2.9	2	4	3	2	3	3	3
FACIAL CONGESTION	-1.5	0	3.3	2	4	3	4	3	3	4
FACIAL CONGESTION	-2.0	0	4.3	4	5	4	4	4	5	4
HEADACHE	-1.0	-100	1.0	1	1	1	1	1	1	1
HEADACHE	-1.0	-50	1.3	1	2	1	1	1	2	1
HEADACHE	-1.5	100	1.3	1	2	1	1	1	2	1
HEADACHE	-1.0	0	1.4	1	2	1	1	1	3	1
HEADACHE	-1.5	0	1.6	1	3	1	1	1	3	1
HEADACHE	-1.5	-50	1.6	1	2	1	1	2	3	1
HEADACHE	-2.0	-100	1.7	1	3	1	1	1	4	1
HEADACHE	-2.0	-50	1.9	1	4	1	1	1	4	1
HEADACHE	-2.0	0	2.9	4	4	3	1	2	5	1

APPENDIX E. (CONT.)

QUESTION	CONDITIONS GZ	CONDITIONS PRESSURE	MEAN RATING	SUBJECT # 830013	860005	860012	860016	860017	870002	870005
SINUS PAIN	-1.0	-100	1.1	1	1	1	1	1	2	1
SINUS PAIN	-1.0	-50	1.4	1	2	1	1	2	2	1
SINUS PAIN	-1.5	-100	1.4	1	2	1	1	1	3	1
SINUS PAIN	-1.0	0	1.6	1	2	1	1	2	3	1
SINUS PAIN	-1.5	-50	1.6	1	2	1	1	2	3	1
SINUS PAIN	-2.0	-100	1.9	1	3	1	1	2	4	1
SINUS PAIN	-1.5	0	2.0	1	4	1	1	3	3	1
SINUS PAIN	-2.0	-50	2.1	1	4	1	1	3	4	1
SINUS PAIN	-2.0	0	3.1	3	5	2	2	4	5	1
VISION BLURRING	-1.0	0	1.0	1	1	1	1	1	1	1
VISION BLURRING	-1.0	-50	1.0	1	1	1	1	1	1	1
VISION BLURRING	-1.0	-100	1.0	1	1	1	1	1	1	1
VISION BLURRING	-1.5	0	1.0	1	1	1	1	1	1	1
VISION BLURRING	-1.5	-50	1.0	1	1	1	1	1	1	1
VISION BLURRING	-1.5	-100	1.0	1	1	1	1	1	1	1
VISION BLURRING	-2.0	-100	1.0	1	1	1	1	1	1	1
VISION BLURRING	-2.0	0	1.3	1	2	1	1	2	1	1
VISION BLURRING	-2.0	-50	1.3	1	3	1	1	1	1	1
VISION GRAYING	-1.0	0	1.0	1	1	1	1	1	1	1
VISION GRAYING	-1.0	-50	1.0	1	1	1	1	1	1	1
VISION GRAYING	-1.0	-100	1.0	1	1	1	1	1	1	1
VISION GRAYING	-1.5	0	1.0	1	1	1	1	1	1	1
VISION GRAYING	-1.5	-50	1.0	1	1	1	1	1	1	1
VISION GRAYING	-1.5	-100	1.0	1	1	1	1	1	1	1
VISION GRAYING	-2.0	0	1.0	1	1	1	1	1	1	1
VISION GRAYING	-2.0	-100	1.0	1	1	1	1	1	1	1
VISION GRAYING	-2.0	-50	1.1	1	2	1	1	1	1	1

APPENDIX E. (CONT.)

QUESTION	CONDITIONS GZ	CONDITIONS PRESSURE	MEAN RATING	SUBJECT # 830013	860005	860012	860016	860017	870002	870005
DOUBLE VISION	-1.0	0	1.0	1	1	1	1	1	1	1
DOUBLE VISION	-1.0	-50	1.0	1	1	1	1	1	1	1
DOUBLE VISION	-1.0	-100	1.0	1	1	1	1	1	1	1
DOUBLE VISION	-1.5	0	1.0	1	1	1	1	1	1	1
DOUBLE VISION	-1.5	-50	1.0	1	1	1	1	1	1	1
DOUBLE VISION	-1.5	-100	1.0	1	1	1	1	1	1	1
DOUBLE VISION	-2.0	0	1.0	1	1	1	1	1	1	1
DOUBLE VISION	-2.0	-50	1.0	1	1	1	1	1	1	1
DOUBLE VISION	-2.0	-100	1.0	1	1	1	1	1	1	1
HEARING LOSS	-1.0	-50	1.0	1	1	1	1	1	1	1
HEARING LOSS	-1.5	-100	1.0	1	1	1	1	1	1	1
HEARING LOSS	-1.0	-100	1.1	1	1	1	2	1	1	1
HEARING LOSS	-1.5	-50	1.1	1	1	1	2	1	1	1
HEARING LOSS	-1.0	0	1.3	1	1	2	2	1	1	1
HEARING LOSS	-2.0	-100	1.3	1	2	1	2	1	1	1
HEARING LOSS	-1.5	0	1.7	1	3	1	4	1	1	1
HEARING LOSS	-2.0	-50	1.7	1	4	1	3	1	1	1
HEARING LOSS	-2.0	0	2.1	2	3	2	4	1	1	2
DEGREE OF COMFORT	-1.0	-100	1.4	1	1	1	1	2	2	2
DEGREE OF COMFORT	-1.0	-50	1.6	1	2	1	1	2	2	2
DEGREE OF COMFORT	-1.5	-100	1.7	1	2	1	2	1	2	3
DEGREE OF COMFORT	-1.0	0	1.9	1	2	2	1	2	3	2
DEGREE OF COMFORT	-1.5	-50	2.1	1	2	2	1	3	3	3
DEGREE OF COMFORT	-1.5	0	2.7	1	4	3	3	2	3	3
DEGREE OF COMFORT	-2.0	-50	2.7	1	4	3	2	2	4	3
DEGREE OF COMFORT	-2.0	-100	3.0	2	3	4	1	2	4	5
DEGREE OF COMFORT	-2.0	0	4.3	4	5	4	4	4	5	4

APPENDIX E. (CONT.)

QUESTION	CONDITIONS GZ	CONDITIONS PRESSURE	MEAN RATING	SUBJECT # 830013	860005	860012	860016	860017	870002	870005
RINGING IN EARS	-1.0	0	1.0	1	1	1	1	1	1	1
RINGING IN EARS	-1.0	-50	1.0	1	1	1	1	1	1	1
RINGING IN EARS	-1.0	-100	1.0	1	1	1	1	1	1	1
RINGING IN EARS	-1.5	0	1.0	1	1	1	1	1	1	1
RINGING IN EARS	-1.5	-50	1.0	1	1	1	1	1	1	1
RINGING IN EARS	-1.5	-100	1.0	1	1	1	1	1	1	1
RINGING IN EARS	-2.0	0	1.0	1	1	1	1	1	1	1
RINGING IN EARS	-2.0	-50	1.0	1	1	1	1	1	1	1
RINGING IN EARS	-2.0	-100	1.0	1	1	1	1	1	1	1
OVERALL RATING	-2.0	0	1.0	1	1	1	1	-	1	1
OVERALL RATING	-1.5	0	2.3	2	2	3	2	-	3	2
OVERALL RATING	-2.0	-50	3.0	3	4	2	3	-	2	4
OVERALL RATING	-2.0	-100	4.5	4	7	4	5	-	4	3
OVERALL RATING	-1.5	-50	5.0	5	5	5	4	-	5	6
OVERALL RATING	-1.0	0	6.0	6	3	7	6	-	6	5
OVERALL RATING	-1.5	-100	6.5	7	8	6	7	-	2	4
OVERALL RATING	-1.0	-50	7.8	8	6	8	8	-	8	9
OVERALL RATING	-1.0	-100	8.8	9	9	9	9	-	9	8

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