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THE EFFECT OF SUSTAINED MUSCULAR CONTRACTION
ON TOLERANCE TO +Gz ACCELERATION

A DISSERTATION

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the Degree Doctor of Philosophy in the Graduate
School of the Ohio State University

By

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1970

Approved by

A handwritten signature in black ink, appearing to read "Robert C. Lusk". The signature is written in a cursive style with a horizontal line underneath the name.

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PREFACE AND ACKNOWLEDGEMENTS

In 1946, Drs. E. H. Lambert and E. H. Wood wrote, "...it can be predicted that with the advent of superspeed planes, the present antiblackout suits will be as obsolete as the planes in which they were designed to be used. Additional physiologic investigations are necessary before methods can be developed which will enable pilots to utilize fully the potentialities of these new aircraft" (1). More than two decades later, this statement remains remarkably true. The study presented in this dissertation is in the family of work which attempts to apply new physiological understanding to an old biomedical problem; in this case, the application of new understanding of the effects of isometric exercise to the old problem of G tolerance and blackout in aviators.

I wish to very gratefully dedicate this work to Professor Robert C. Little, my adviser. It was his wise counsel and continuing encouragement that led me to undertake and to persevere in the rigors of my Doctoral program.

Any research study involving a sophisticated environmental stress facility and computer-aided data reduction necessarily involves a considerable group of people beyond the investigator. I most gratefully acknowledge the support and cooperation of the following people in the conduct of this work: Dr. Alexander R. Lind, Chairman of the Department of Physiology, St. Louis University School of Medicine for introducing me to the physiology of sustained muscular contraction and for his thoughtful review of the manuscript; Leif A. Lohrbauer, Captain, USAF, MC and Ronald Wiley, Ph.D., for technical

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FIELDS OF STUDY

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Physiological Effects of Weightlessness

Physiological Effects of Sustained Acceleration

Biological Rhythms

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INTRODUCTION

The development of increasingly sophisticated manned aircraft and space systems continues to raise new problems of human performance in sustained acceleration environments. For example, low altitude, high speed flight involves the interaction of sustained acceleration and random vibration, for which human tolerances must be determined, performance limits established, and protective techniques developed. Advanced manned space systems have led to the requirement for data on man's performance capabilities in transverse or +Gx acceleration environments, definition of survivable acceleration levels for the design of space escape systems, and the study of human responses to short radius centrifugation for the development of an on-board centrifuge.

However, for the pilot of the modern fighter aircraft, the major operational problem involving sustained acceleration has remained unchanged; and it is that of protective techniques to prolong and increase man's tolerance of positive or +Gz accelerations. The measures and devices which are used or have been studied for their protection against sustained acceleration are well known and have been thoroughly reviewed (vide infra). This review will discuss recent studies which have extended our knowledge of the physiological mechanisms responsible for +Gz sustained acceleration tolerance and discuss proposals for new and improved G protective techniques. The first section will review briefly the physiological mechanisms of

tolerance to +Gz acceleration, discuss examples including direct photographic examination of the retinal circulation during acceleration, and describe recently developed electroretinographic methods for the measurement of retinal function during acceleration. In the following section, the physiological mechanisms of action of G protective techniques will be discussed, recent modifications of the standard U.S. Air Force CSU-3/P described, and the development of a new G protective technique discussed.

The Hydrostatic Model: Human tolerance in +Gz acceleration is defined by the occurrence of blackout and unconsciousness. Two cardiovascular mechanisms are involved: (1) the hydrostatic factor which reduces the effective perfusion pressure at the eye (retina) and brain and (2) the orthostatic factor or pooling of blood in dependent capacitance vessels and tissues which result in decrease venous return to the heart, a fall in cardiac output, and reduction in the effective perfusion pressure at the eye.

Blaise Pascal in the 17th century in his laws of hydrostatics showed in ideal fluids at rest under gravity that: (1) fluid pressure is equal in all directions; (2) pressures at all points in the same horizontal plane are equal and (3) pressure increases with depth. This increase is equal to $\rho g h$ dynes/cm² where ρ is the density of fluid in grams/cc, g is the gravitational constant 980 cm/sec², and h is the depth or height of the fluid column in cms. These laws may be applied to any fluid system including the human vascular system. When the body is horizontal, mean arterial pressures at the feet and the brain are approximately the same. When the body is upright, the

mean arterial pressure at the brain falls and increases at the feet. If the brain is 25 cm above the heart, the mean arterial pressure becomes approximately 80 mmHg and arterial pressure in the feet will be approximately 175 mmHg. Under increased acceleration, the resultant between the applied and the gravitational acceleration acts to increase the effective density of blood by increasing the g term in the $\rho g h$ equation. A dimensionless multiple of the gravitational constant, the term " G ", is used to indicate the interial resultant of body acceleration. At +5 Gz with a mean arterial pressure at the heart of 120 mmHg, the calculated pressure at the base of the brain is zero while at the feet it will be 370 mmHg and the subject would presumably be unconscious. The same hydrostatic factors apply in principle to the venous system. The hydrostatic component of vascular pressures was developed by Lindberg and Wood to illustrate the role of gravity in the systemic and pulmonary circulations (2). This model is adequate to explain intravascular pressure responses to short duration (10 sec) accelerations beyond which time circulatory reflex compensations occur. In addition during positive acceleration, blood pools in the dependent vessels and tissues, particularly of the leg, due to the increased venous transmural pressure secondary to the hydrostatic factor. Venous return to the heart is reduced and cardiac output falls. Cardiac output falls with little compensation, in apparent proportion with G load, in + Gz (2).

Acceleration and the Retinal Circulation: The accepted theory predicts that blackout in + Gz acceleration occurs at the moment that retinal artery pressure falls below intra-ocular pressure (~ 25 mmHg)

and retinal perfusion ceases. This explanation was first propounded by Andina in 1937, who pointed out that intra-ocular pressure is some 20 mmHg higher than intracerebral pressure and consequently blood supply to the retina fails before the cerebral circulation (3). As confirmation, Lambert showed that application of 30 to 40 mmHg negative pressure to the eyeball by means of suction goggles will raise the blackout threshold (4). Similarly, application of positive pressure to the eye will lower the threshold. Livingston pointed out that the retina has an oxygen requirement four times as great as that of any other body tissue, except the carotid body (5). Hence, even a minor reduction of blood supply should cause marked interference with vision.

Duane in 1954 examined the retina directly with a funduscope and drew illustrations at +Gz accelerations to blackout level (6). He measured arterial pressure in the radial artery with the arm so placed that the tip of the cannula was at eye level. A correlation between the subject's visual change and change in the fundus oculi was established as follows:

<u>Stage</u>	<u>Subjective</u>	<u>Objective</u>
I	Loss of peripheral vision	Arteriolar pulsation, i.e. recurrent exsanguination
II	Blackout	Arteriolar exsanguination and collapse
III	Return of central and peripheral vision	Return of arteriolar pulsation and temporary venous distension

Based on the fact that the inner retinal layers are sensitive to hypoxia, it was concluded that retinal arteriolar ischemia produced

hypoxia of these layers. It was shown by Lewis and Duane that the critical site for hypoxia was the junction of the ganglion and bipolar cells in the retina (7). In a later paper, Duane et al showed that when the hydrostatic pressure was such as to cause collapse of the arteriolar vessels during diastole and recovery in systole, a pulsation of the vessels may be observed which is associated with grayout, or reduction of the visual field to approximately 15° in all meridians (8). In addition, in those subjects in whom a photic drive of the EEG was observable at rest, loss of photic drive could be demonstrated at grayout level.

Leverett and his coworkers at the U.S. Air Force School of Aerospace Medicine (SAM) at Brooks Air Force Base, Texas have recently confirmed and extended Duane's hypothesis by direct photographic observation of the retinal circulation during +Gz acceleration. Three theories of blackout are defined as follows: (1) retinal theory - changes in vision follow changes in retinal hemodynamics caused by external compression of the globe or by decreased perfusion pressure; (2) central theory - blackout is first perceived in the visual cortex and this follows a change in electrical activity of the retina; (3) central compression - displacement of the cerebral contents causes either (a) mechanical compression of the optic chiasm or (b) overwhelming afferent impulses that take precedence over all of the information traveling on the visual pathways. Newsom, Leverett and Kirkland have collected direct photographic evidence for the retinal theory of blackout in human volunteer subjects during +Gz acceleration. Retinal photographs were taken by a scientist/observer riding

the centrifuge with the subject (9). Moment-to-moment changes in retinal circulation were presented in a series of retinal color photographs taken at the subject's blackout level. In rapid onset runs at blackout, the outstanding features were complete central retinal arterial collapse and exsanguination of the disc region, full veins and marked blanching of the nerve head. In most instances, venous pulsations were observed before the run and could be seen to occur intermittently at G. However, arterial pulsation occurred only during periods of disturbed vision. On several instances when the subject blacked out rapidly, the arterial pulsation did not have a chance to occur and complete collapse of the vessel occurred in a matter of a second after reaching peak G. During deceleration, immediate filling of the collapsed arteries occurred. A red hue occurred of the nerve head and reactive hyperemia was evident in the total retinal circulatory area.

These results were confirmed and further refined using the technique of retinal fluorescence angiography (9). Following the intravenous injection of 5% fluorescein, there is a fluorescence of the retinal vessels as the fluorescein passes through them. This fluorescence can be observed and photographed using appropriate filters for excitation and selection. With serial photographs, the phases of retinal circulation may be demonstrated. Serial photographs were taken at a predetermined rate, usually every 0.6 seconds, throughout the run by the instrument operator in the control room during centrifugation. These figures confirm that circulation to the retina is stopped or profoundly slowed during blackout but that

veins do not collapse. It may be postulated that this is due to occlusion of the central retinal vein at the cribriform plate when it exits the globe. The status of the coroidal-retinal circulation during acceleration as well as arterial and venous pressures in retinal vessels at blackout is unknown. At the present time, there is no pressure sensing device small enough to be placed in a vessel the size of the retinal circulation.

The Electroretinogram: Ward has described the effect of +Gz acceleration on the electroretinogram in dogs (10). The electroretinogram (ERG) is the gross electrical response of the retina to light stimulation. It is recorded between the anterior and posterior poles of the eyeball, i.e. across the retina. This bioelectrical light response was discovered independently last century by Holmgren and by Dewar and McKendrich (10). The wave's more prominent features were designated by the letters a, b, and c. While the subjective reporting of visual field changes is found to be one of the best indices of beginning or impending retinal ischemia in acceleration, it unfortunately requires a judgment by the subject at a time when it is not certain that his intellect is entirely unimpaired. The visual field changes of "grayout" to "blackout", generally found to precede unconsciousness, are attributed to retinal ischemia.

In Ward's studies, anesthetized beagles were exposed to graded +Gz stress on a short radius centrifuge. The ERG (response to a strobe flash) was recorded with an intracorneal electrode. The ERG a-wave decreased only at the higher Gz levels. Amplitude of the b-wave fell by 40-100 per cent at 1.5-3.0G. Transient increases were

noted in b-waves during early stages of acceleration stress and attributed to anoxia's effects. Mean blood pressure fell as low as 40 per cent above control. ERG recovery was frequently incomplete 10-15 minutes after 3-4 minute runs at the higher G levels.

If, as has been suggested, "blackout" does, in fact, occur with an initial failure at the ganglion cell level, with the ERG becoming involved only in a later (and perhaps dangerous) stage of retinal and cerebral ischemia, the b-wave amplitude may prove to be an unfruitful operational index of acceleration tolerance. However, work done on the tolerance of the various levels of the visual pathway, to anoxia, suggests that involvement of the ganglion cells should not precede bipolar failure by any great length of time. Recent unpublished work from the Royal Air Force Institute of Aviation Medicine would appear to bear this out (11). They noted beginning b-wave reduction, recorded from human subjects, in +Gz acceleration following subjective reports of visual field disturbances.

Protection Against Acceleration: Human tolerance of acceleration may be improved by the use of protective measures and devices. Straining and grunting in a tight turn was practiced by German pilots prior to World War II to improve their tolerance and culminated in the development of the M-1 maneuver (vide infra). Changing of posture had been practiced, particularly in World War II, when hunching forward in the cockpit was found to improve the tolerance of pilots in high acceleration maneuvers. The effects of posture, restraint, and body position on G tolerance have been well worked out and have been reviewed in detail (12). The history of G-suits and protective

devices dates from World War I and is well reviewed in recent monographs (2,13). The present wrap-around, cutaway type of anti-G suit presently used by the U.S. Air Force (designation CSU-3/P) improves blackout tolerance in the +Gz vector by about 2G (13). Analysis of contributions of portions of this suit show that the whole suit gives protection of approximately 2G in terms of blackout threshold while leg bladders alone give protection of about 0.2G. Abdominal bladders without leg bladders protect to about 1.2G while arm cuffs alone produce a hardly measurable protection. Arm cuffs added to a full suit will raise protection by a further 0.5G.

The effects of removing the leg bladders from the standard U.S. Air Force CSU-3/P suit (the Bikini Suit) to achieve improvements of comfort and convenience have recently been tested (14). No measurable difference in +Gz blackout threshold could be determined with the leg bladders removed in the domain of 5.5 to 6.5G. In addition, the majority of subjects tested were able to tolerate 25 seconds at 7-1/2G using this suit configuration. It was noted that peak G was achieved in 3 seconds while suit pressure took 5 seconds to reach maximum, suggesting once again that improvements in the G valve pressure regulator system could be made.

Hemodynamic Responses to G-Suit Inflation: The physiological mechanisms of action of anti-G suits were originally established on the Toronto centrifuge and at the Mayo Clinic and have been summarized by Wood and Lambert (15). It has been shown that inflation of a suit at 1G produces an initial increase in arterial pressure, followed by an almost immediate decrease in heart rate probably due

to a depressor reflex originating in the carotid sinus and aortic areas. The same pattern is seen when the suit is inflated under +Gz acceleration. With inflation during acceleration, the arterial pressure rises slightly above normal at the heart level but remains below normal at the head level. Since the pressure in the carotid sinus is not significantly elevated, slowing of the heart rate does not occur. Lindberg et al have pointed out that the decrease in cardiac output that occurs during +Gz acceleration is not significantly altered by the use of an inflated G-suit (16). Hence, the protection afforded by the suit is not associated with a relative increase in cardiac output. He observed that inflation of an anti-G suit under +Gz accelerations is associated with a relative increase in mean aortic pressure which acts to counteract the increased hydrostatic pressure. They propose that a G-suit is effective by increasing systemic vascular resistance and raising blood pressure as well as by reducing peripheral pooling.

Gray et al at the University of Pittsburgh have recently studied the acute and chronic effects of sudden G-suit inflation in the recumbent and passively tilted postures (17). Acute inflation to 80 mmHg in 3 seconds was associated with a transient increase in cardiac output, mean brachial artery pressure, central blood volume, and right atrial pressure with a transient decrease in pulse rate. Trends of these hemodynamic alterations were similar in both the recumbent and tilted groups. However, the changes were of greater magnitude in the tilted subjects who were at one +Gz acceleration, and might be expected to approximate those at higher G. Release of

the G-suit resulted in a drop in mean brachial artery pressure associated with tachycardia and increased cardiac output. Of particular interest is the transient nature of the hemodynamic responses to G-suit inflation. The cardiac index response, in particular, although increased significantly in the upright individuals, is brief and is returned near control levels by 45 seconds. The response is transient and probably of little importance in the prolonged use of the garment. Mean resting brachial artery pressure in the 18 healthy male subjects in these studies was 87 mmHg and increased approximately 3-1/2 mmHg on suit inflation. This pressor response was sustained throughout the G-suit application.

Straining Maneuvers: Present G protective techniques, principally the G-suit, attempt to prevent the consequences of the peripheral pooling of blood during acceleration by the application of a suitable counterpressure. A more logical approach would be to artificially raise mean systemic blood pressure. Since the early days of flying, the value of muscular straining as protection against the effects of +Gz acceleration have been recognized. Pilots observed that blackout or grayout could be postponed if they grunted or screamed and tensed the skeletal musculature during acceleration. The M-1 maneuver, defined as muscular straining with expiration against a partially closed glottis, is a most effective voluntary protection against the circulatory effects of +Gz acceleration (15,18). The physiologic basis for this protection has been correctly ascribed to its effect on increasing the arterial pressure at eye during acceleration. However, the major effect is, I believe, incorrectly

ascribed to the respiratory component of the maneuver, which is a forced expiration against a partially closed glottis over a period of 3 to 5 seconds. As forced expiration is instituted, the resulting increase in intrathoracic pressure is transmitted directly to the aorta. There is an immediate increase in peripheral arterial pressure which is only slightly less than the increase in intrathoracic pressure. During the period of forced expiration, intrathoracic pressure falls continuously while intra-abdominal pressure remains elevated creating a pressure gradient between the abdomen and thorax augmenting the return of blood to the heart. This maneuver affords approximately 2.4G protection against blackout (15). The circulatory mechanisms described here were identified by Rushmer in 1946 (19).

The tensing or isometric exercise of skeletal muscle has been mentioned as a G protective factor (2,20), but no attention has been given to the cardiovascular mechanisms involved. Muscular tensing has been alleged to decrease the amount of blood pooled in legs and abdomen and to increase peripheral arterial resistance. Muscle tensing alone has been reported to elevate blackout threshold by about 1G although the muscle groups used and the force exerted were not specified (13).

A logical approach to the problem of +Gz acceleration protection would be to artificially raise mean systemic blood pressure. A desirable method would be comfortable, convenient, non-irritating, effective repeatedly over a long duration and most importantly, physiological. Possible approaches to raising blood pressure are drugs, external counterpulsation, pain, cold, and carotid sinus

stimulation. The "pressure raising" approach has theoretical difficulties in that blood pressure is controlled within rather narrow limits by a number of effective servomechanisms, the baroreceptor system being the best known. Any disturbance in blood pressure is buffered or opposed by a mechanism seeking to restore it to its normal range. However, recent investigations by Lind and his coworkers on the effects of isometric exercise have identified rather striking cardiovascular responses to sustained muscular contraction, particularly the handgrip. In particular, 50% of a maximum voluntary handgrip contraction will raise mean arterial blood pressure up to 65 mmHg (21,22). The mechanism of the response is not established but there appears to be extensive peripheral vascular vasoconstriction, presumably in an attempt to maintain perfusion of muscles when the contraction threatens to restrict the muscle blood supply.

Whole body muscular straining is a well known and effective G protective maneuver (23,24,25,26). Straining maneuvers are Valsalva-like exercises that raise mean arterial blood pressure as a consequence of forcibly raising intrathoracic pressure; this pressure increase being directly transmitted to the great vessels. A reproducible form of physiological blood pressure elevation, without the problem of raised intrathoracic pressure, could offer a significant improvement to presently employed G protective maneuvers. The present study was designed to determine whether the blood pressure elevation achieved with fractional handgrip will extend man's G tolerance during both rapid and slow onset positive (+Gz) acceleration

on the human centrifuge, to define the interaction of handgrip and G-suit effects on +Gz acceleration tolerance and arterial blood pressure, and to demonstrate the independence of the pressor response to grip from respiratory or Valsalva-like straining maneuvers.

Other G protective techniques under active investigation in U.S. Air Force laboratories include various methods of cervical pressure to provide a hypertensive stimulus to the carotid sinus baroreceptors; counterpulsation techniques which will provide graded lower body counterpressure in relation to G load, synchronous with cardiac diastole and timed from the electrocardiographic R-wave; the application of hypothermic or cold pulses; and the use of constant wear counterpressure garments such as gradient elastic leotards.

METHODS AND MATERIALS

Subjects: Healthy U.S. Air Force personnel were chosen from a hazard panel roster. Subject characteristics are presented in Table 1. All passed a U.S. Air Force flying Class III physical examination and, in addition, had a normal 12 lead clinical electrocardiogram and Master's step test. All subjects were experienced riders of the U.S. Air Force School of Aerospace Medicine centrifuge and were knowledgeable in the use of the G-suit and other voluntary G protective maneuvers. All subjects performed the handgrip with their right hand irrespective of handedness. It has been shown that although the force of a maximal contraction is less in the non-dominant hand, the blood pressure response to any sustained fraction of that maximum is independent of handedness (18).

Centrifuge Facility: The accelerations used were produced by the School of Aerospace Medicine (Brooks Air Force Base, Texas) human centrifuge. This conventional, long arm (20 foot) centrifuge produces a low gradient +Gz acceleration environment typical of cockpit maneuvering accelerations (Figure 1). Tolerance was determined by the subject's response to peripheral and central lights. To determine +Gz acceleration tolerance, the subjects were exposed to two standard centrifuge profiles as seen in Figure 2. Each subject rode a series of rapid onset runs (ROR) at 1G per second onset to peak G with a plateau at peak of 10 seconds. Each run was followed by at least a 90 second rest period. Profiles were administered sequentially in

TABLE 1 SUBJECT CHARACTERISTICS.

SUBJECT #	AGE (YEARS)	HEIGHT (CMS)	WEIGHT (KILOS)	BODY SURFACE AREA (M ²)
1	35	176.53	90.90	2.08
2	34	181.61	61.36	1.80
3	34	182.88	85.00	2.08
4	22	184.15	70.90	1.94
5	25	175.26	62.27	1.76
6	28	180.34	84.09	2.04
7	24	182.88	88.63	2.12
8	23	180.34	71.81	1.92

FIGURE 1. United States Air Force School of Aerospace Medicine human centrifuge. This device has a twenty foot radius arm and an enclosed cabin which swings out passively to align with the resultant G vector.

FIGURE 2. The time history or acceleration profile of the force environments used in this study: rapid onset runs (ROR) with an onset of 1G/sec with a 10 second plateau and slow onset runs (SOR) with an onset of 0.07G/sec and no plateau.

0.2-0.3G increments until an endpoint was reached. One slow onset run (SOR) at 0.07G per second without a plateau was experienced by each subject until his endpoint was reached. As an endpoint for both runs, peripheral light loss (PLL) and/or central light dimming (CLD) were used. Light intensity, color and geometry were by standard techniques (27,28). All the subjects were instructed to relax during the +Gz exposure and under no circumstances to use a straining or any other self-protective maneuver. During the runs, they were monitored continuously by closed circuit television for safety purposes; this afforded an additional opportunity to monitor and control involuntary straining.

Dynamometer and Grip Technique: A simple lightweight hand-held dynamometer was used to record sustained handgrip contractions. The requirements of this study necessitated the design of a dynamometer with the following characteristics: (a) sensitive only to grip force and insensitive to the contraction of other muscle groups, (b) allow grip with minimal shortening of forearm flexor muscles, (c) lightweight (less than 500 grams) to be hand-held during positive acceleration, (d) insensitive to acceleration, (e) large signal output. The instrument fabricated specifically for this study is shown in blowup in Figure 3. The design and construction of the device are described in detail elsewhere (29). Two maximum voluntary handgrip contractions at an interval of one minute with duration of less than 2 seconds were performed by each subject at the start of each experiment. The mean of these two contractions was accepted as the maximum voluntary handgrip contraction (MVC). MVCs varied 2% or less in trained subjects and 4% or less in untrained subjects with absolute

FIGURE 3. The handgrip dynamometer used in this study is shown photographed intact (to the left) and in exploded view of its major components (to the right).

values varying from 40 to 70 kilograms in agreement with Lind (21). A voltage representing 30%, 40% or 50% of the dynamometer output at MVC was displayed to the subject on a needle dial such that, when subject effort on the dynamometer equaled, for example, 40% of his previous maximum, a zero or vertical needle position was given. The subject maintained the 40% MVC by holding the meter null with the grip. In the rapid onset runs, a 30 or 40% MVC was used. In the slow onset runs, a 50% MVC was held by all subjects. During the rapid onset runs which included handgrip, the grip was begun 60 to 90 seconds prior to the onset of acceleration and the 30-40% maximum voluntary contraction was maintained throughout the acceleration exposure. During the slow onset runs, the handgrip, at 50% MVC, was begun at the onset of acceleration and maintained throughout the run.

Instrumentation: Direct radial arterial blood pressure at eye level was recorded through a Teflon catheter connected to a Statham type P-37 miniature pressure transducer mounted on the wrist. The strain gauge membrane was oriented in the same plane as the +Gz acceleration vector and mounted at the same level as the catheter tip. The transducer was calibrated in situ with a mercury manometer and the zero reference level arterial pressure was taken as the outer canthus of the left eye. A fully instrumented subject with the dynamometer in his hand is seen in Figure 4. With the hand strapped to the head and the elbow supported in this fashion, the relative position of the catheter tip and the eye did not change during acceleration, as indicated by continuous TV monitoring. Pressures so recorded represented the radial artery pressure at the catheter

FIGURE 4. A fully instrumented subject seated in the centrifuge cabin. Note the taped forearm supporting the radial artery catheter and pressure transducer assembly at eye level, the nasal thermistor for transducing respiration, the hand-held dynamometer, and the G-suit (uninflated). The subject is visually fixating on the dynamometer null indicator and monitoring peripheral lights (not visible in this photograph).

tip without artifact due to the applied acceleration acting on the transducer or on the fluid filled tubing. In some experiments, indirect blood pressure was also determined automatically with an electrospphygmomanometer using techniques developed by Rogge et al (30). This technique superimposes the electronically recorded Korotkoff sounds on a trace of the decaying pressure in the arterial cuff (see Figure 16 for example). With proper calibration, the indirect blood pressure can be read accurately to the nearest 5 mmHg. A three lead vectorcardiogram, using the Frank lead system, was recorded in addition to respiration, using a nasal thermistor. Electromyogram from the dorsal surface of the contracting forearm was recorded using surface electrodes and displayed as the raw EMG signal. The handgrip dynamometer output was monitored and subjects were under continuous television observation and voice communication. A typical direct paper chart tracing of the recorded variables is seen in Figure 5.

Protocol: Two studies were performed, separated by approximately five months, the first consisting of the rapid onset profile and the second, the slow onset profile. In each study, G tolerance was determined in four conditions: (1) +Gz (control, no protection), (2) +Gz + fractional voluntary handgrip (grip), (3) +Gz + G-suit and (4) +Gz + grip + G-suit. The order of treatment or condition was assigned by the use of a simple Latin squares experimental design as seen in Table 2. At the conclusion of the slow onset run study, measurements were made with subjects seated in the centrifuge cab at 1G during the following conditions: (1) G-suit alone, (2) G-suit + grip and (3) grip with an arterial occlusive cuff (300 mmHg) applied

TABLE 2 EXPERIMENTAL DESIGN

DAY	SUBJECT	ORDER OF CONDITIONS a.			
1	1,2	1	2	3	4
2	3,4	4	1	2	3
3	5,6	3	4	1	2
4	7,8	2	3	4	1

a. CONDITIONS:

1. +Gz (control, no protection)
2. +Gz + grip (fractional handgrip)
3. +Gz + G-suit
4. +Gz + grip + G-suit

at the onset of the grip and continued 60 seconds after the offset of the grip and (4) grip + occlusive cuff applied for 60 seconds at 5 seconds prior to offset of grip. In addition, three subjects were studied in whom ventilation, and esophageal and intragastric pressures were recorded during handgrip, Valsalva and M-1 maneuvers, Gastric and esophageal pressures were recorded with tandem balloon catheters filled with 1/2 cc of air and connected to Statham strain gauge manometers according to the technique of Meade (31). Respiratory flow patterns were recorded by use of a calibrated mass flowmeter (Technology Incorporated Model 150) with the subject connected to a one way, open breathing system.

Data Analysis: All signals were simultaneously recorded on paper strip chart (Figure 5) and on 14 channel magnetic tape. The analog tape was digitized, off-line, at a rate of 250 times per second. Systolic, diastolic and mean arterial blood pressure were computed for each heart beat, recorded on digital tape and punched into cards. The respiratory signal was inadequate for mechanical processing. Instantaneous heart rate was computed manually on a desk-calculator from the arterial blood pressure pulse interval and averaged over five beats. The vectorcardiographic signals were analyzed using a computer program developed by Cohen et al (32). The results of an analysis of the effects of positive acceleration, grip and G-suit on the Frank lead electrocardiogram are presented in a separate report (33). An analysis of variance was performed using two main effects, conditions and subjects. Interactions between these two effects determined the residual term. Differences between subject means for conditions were tested by use of the Duncan Multiple Range

FIGURE 5. A typical paper chart tracing of the recorded variables: EKG, EMG, respiration, direct arterial blood pressure, dynamometer output and accelerometer, in slow (SOR) and rapid (ROR) onset runs. The EMG and respiration were not recorded during rapid onset runs.

Test and the single-tailed Student's "T" test of paired differences. The statistical practice involved is described by Crow (34) and the computations used are listed in Bruning and Kintz (35).

RESULTS

Fractional Voluntary Contractions and Times to Fatigue: In pilot studies in four subjects, blood pressure responses were evaluated during 20, 30, 40 and 50% maximum voluntary contractions held to fatigue. Typical results are presented in Table 3 and Figure 6 and confirm the previously published results of Lind et al (21,22). Note that the time-to-fatigue, that point at which the subject under maximal effort and with positive encouragement can no longer maintain the fractional contraction, decreases sharply as one progresses from a 20% to a 50% contraction. A 20% contraction can be held approximately 6 minutes while a 50% contraction is held for 1-1/2 minutes. The increase in mean arterial blood pressure in mmHg determined indirectly with an electrospgymomanometer in one typical subject is seen in Figure 6. Typically, a 45 mmHg increase in mean arterial blood pressure is seen with a 50% maximum voluntary contraction held to fatigue. Note also that the slope of the blood pressure increase rises progressively as one increases the per cent contraction. It is also interesting to note that the maximum blood pressure response achieved is relatively similar in the four fractions of maximal grip. Note also that an approximately 25 mmHg increase in mean arterial blood pressure occurs in a 40% contraction during the first 90 seconds.

+Gz Tolerance, Rapid Onset Runs: Tolerance was defined as the acceleration or G level at which the subject reported loss of peripheral lights (PLL) or central light dimming (CLD) during rapid

FIGURE 6. Mean arterial blood pressure response, recorded indirectly, during 20, 30, 40, and 50% of maximum voluntary handgrip contractions (MVC) held to fatigue by a typical subject.

onset (0.75G per second), 10 second peak profiles of +Gz (positive) acceleration. The ROR tolerance G levels are presented in Table 4 and Figure 7 and the statistical analysis of these values in Tables 5 and 6.

The analysis of the ROR tolerance data will be developed in detail as an example of the statistical procedures used in this paper. The hypothesis tested was that the average tolerance in G in control, grip, G-suit and G-suit + grip conditions do not differ significantly. The method used to test this hypothesis was a one-way classification analysis of variance (ANOVAR). If the average tolerance levels for the four conditions differed significantly (F ratio <0.05 probability), then confidence statements about the differences between them were made by the use of Student's "T" statistic for paired variables (34), or the Duncan Multiple Range Test. To test the hypothesis that the G tolerance in four conditions does not differ significantly, the data of Table 4 were used. The results of the analysis of variance are seen in Table 5. The analysis uses two main effects: (a) conditions (control, grip, G-suit and G-suit + grip) and (b) subjects, using the interactions between the two to arrive at a residual term for calculating the F values presented in the table. The ANOVAR used conventional equations (35) and was computed on an IBM 7094 system. The F value for the main effect treatment, namely the conditions, was 43.7, which is significant at the $p \leq 0.005$ level. Thus the hypothesis that the tolerance levels of the four conditions is the same is rejected at the 1% level of confidence, and we conclude that significant differences between these values exists. Differences between the

G LEVEL AT PERIPHERAL LIGHT LOSS (ROR)

FIGURE 7. +Gz acceleration tolerance as measured by G level at the instant of peripheral light loss during rapid onset runs (ROR), mean values for eight subjects, $\pm$ the standard error of the mean (S.E.M.).

mean tolerance for the four conditions were tested by Student's "T" test of paired samples. Results of these tests are seen in Table 6. Each condition was different from every other (six possible pairs) except grip vs G-suit which was not significantly different at the 5% level.

In the control or unprotected position, mean tolerance was 3.6 ± 0.4 (S.D.) G. This value agrees well with handbook values for unprotected tolerance to positive acceleration (36,37). With grip (30-40% of a maximum voluntary handgrip) G tolerance was 4.5 ± 0.8 G which is significantly greater than control ($p < 0.0025$). Note that the grip was begun 90 seconds prior to the onset of G and maintained throughout the run. The G-suit tolerance was 4.8 ± 0.3 G which is significantly greater than control ($p < 0.0005$) but not different from the grip condition. With grip and G-suit in simultaneous combination, tolerance was 5.4 ± 0.3 G which is significantly greater than control ($p < 0.0005$), grip ($p < 0.0025$) and G-suit ($p < 0.0025$) conditions.

Mean Arterial Blood Pressure - Rapid Onset Runs: Mean arterial blood pressure (MABP) in mmHg at eye level for eight subjects in control, grip, G-suit and grip + G-suit conditions is presented in Table 7 and Figure 8. For each subject, a series of five beats was summed at each of seven periods during each run: (1) rest or 0 time, (2) 30 seconds, (3) 60 seconds, (4) initial or 90 seconds, just prior to the onset of the acceleration pulse, (5) low, the five sequential lowest beats irrespective of their time of occurrence, (6) compensation, the last five beats before offset of the acceleration pulse, and (7) rebound or recovery, the five

FIGURE 8. Mean arterial blood pressure in millimeters of mercury during +G_z acceleration in rapid onset runs in four conditions: control, G-suit, grip and G-suit plus grip, mean for eight subjects. Note that the initial value is taken just before the onset of acceleration. The "low" value is the nadir of the blood response regardless of its exact time of occurrence (approximately 6-7 seconds after the onset of the acceleration). In the grip and G-suit plus grip conditions, a 30-40% MVC is held from zero time until the end of the acceleration profile.

sequential highest beats after the acceleration offset. Measurements were taken from the record of the run just prior to the tolerance or endpoint run, as the latter run was variably terminated at each subject's endpoint.

In the initial analysis of variance (Table 8), the hypotheses that there were no significant differences in mean arterial blood pressure due to either condition or period were tested. In both cases, the F is significant and the hypothesis is rejected, leading to the conclusion that both time or period, during the acceleration profile and condition, effect the blood pressure response. The results of the Duncan Multiple Range Test of significance between means of the six possible pairs of conditions at the five periods during the acceleration profile is seen in Table 9. All blood pressures returned to rest values before the initiation of the next run. A typical tracing of the mean arterial blood pressure during a grip run as recorded directly is seen in Figure 5. The resting blood pressures were the same for all conditions (range 77-79 mmHg). After 90 seconds, that is at the initial period, MABP was 81 and 83 mmHg in the control and G-suit conditions respectively. These conditions are identical, up to this period, as the G-suit is inflated during the G profile only (inflation begins at 2.5G and precedes at 1 psi/G). In the grip and grip + G-suit conditions, the initial MABP was 105 and 103 mmHg respectively, both values significantly greater than control ($p < 0.01$). Note that in these two conditions, a 30-40% MVC grip has been held for 90 seconds. The MABP increase in the grip and G-suit + grip conditions was 24 and 20 mmHg respectively. The low MABP was 10, 17, 7 and 19 mmHg in control, grip, G-suit and G-suit

+ grip conditions respectively. No one of these values is significantly different from any other with the exception of G-suit vs G-suit + grip ($p < 0.01$). Irrespective of the initial or starting MABP, all blood pressure values at peripheral light loss were statistically from the same population with the one exception, as would be expected if PLL is determined by an eye level blood pressure which is less than intra-ocular pressure. The rebound MABP was 91, 102, 87 and 94 mmHg in the control, grip, G-suit and G-suit + grip conditions respectively. Note also (Tables 8 and 9) that the blood pressure during the period of reflex compensation is significantly higher in the two grip conditions than in the non-gripping conditions.

Heart Rate - Rapid Onset Runs: The heart rate responses are seen in Figure 9 and Tables 10, 11 and 12. Heart rate was calculated over a 5 beat period from the interbeat interval of the arterial blood pressure pulse in the same periods: rest, initial, low, compensation and recovery, as used for blood pressure. The heart rate increased during the period of grip prior to both the grip and grip + G-suit runs. It rose from a mean of 84 to 100 during grip preceding the +Gz + grip run and from 78 to 112 during grip preceding the +Gz + grip + G-suit run. There was a further increase in rate during acceleration with both conditions, rising to maximum of 114 and 115 during compensation for grip and grip + G-suit runs respectively. The heart rate during control and G-suit runs rose during acceleration to a maximum of 102 and 105 respectively during compensation. Note that during the initial and low periods, the heart rate is significantly higher in the conditions in which grip is present.

Respiration - Rapid Onset Runs: In the ROR, the nasal thermistor

FIGURE 9. Heart rate in beats/minute during +G_Z acceleration in rapid onset runs in four conditions: control, G-suit, grip and G-suit plus grip, mean for eight subjects. Heart rate was calculated from the beat to beat time interval and meaned over five beats. Note that the initial value is taken just before the onset of acceleration. The "low" value is taken at the nadir of the blood pressure response regardless of its exact time of occurrence (approximately 6-7 seconds after the onset of the acceleration).

system failed and respiratory patterns during ROR in the four conditions can only be described for two subjects. In these subjects, the pattern of respiration was only slightly disturbed during acceleration and there was no evidence of breathholding or respiratory straining maneuvers (vide infra, respiration - slow onset runs).

G Tolerance - Slow Onset Runs: All subjects experienced a single slow onset run (SOR) at 0.1G per second onset to subjective endpoint, peripheral light loss or central light dimming. Individual subject and mean data for G level at peripheral light loss is presented in Tables 13, 14 and 15 and Figure 10. Note that there was a progressive increase in acceleration tolerance from control to grip to G-suit and to the G-suit + grip conditions comparable to that seen with the rapid onset runs. The acceleration tolerances for slow onset runs are approximately 1G higher than for rapid onset runs in all conditions. Control Gz tolerance (SOR) was 4.61 ± 1.1 (SEM) with a range of 3.4 to 6.7. The range of tolerance for +Gz + grip was 3.9 to 7.0G with a mean of 5.6 ± 1.1 , significantly different ($p < 0.01$) from control. The G-suit provided protection comparable to grip alone. The G-suit mean was 5.8 ± 1.0 G with a range from 3.9 to 7.0G, significantly different from control ($p < 0.025$) and G-suit + grip ($p < 0.05$). As in the rapid onset runs, tolerance, when G-suit and grip were used in combination, surpassed that with grip or G-suit alone. In the grip + G-suit condition, tolerance ranged from 5.0 to 7.0G with a mean of 6.3 ± 1.0 G, significantly ($p < 0.01$) different from all other conditions. In all studies, an upper limit of 7G was used as a maximum allowable stress for subject safety considerations. A total of 6 subjects, one during Gz + grip, two during Gz + G-suit and three during Gz + grip + G-suit

FIGURE 10. +Gz acceleration tolerance as measured by G level at the instant of peripheral light loss during slow onset runs (SOR), mean values for eight subjects $\pm$ the standard error of the mean.

had their runs terminated at 7G prior to the occurrence of peripheral light loss. Thus the degree of G protection in the three test conditions is in fact greater by an unknown amount than the recorded mean values.

Mean Arterial Blood Pressure at Eye Level - Slow Onset Runs: Mean arterial blood pressure responses at eye level for the 8 subjects in control, grip, G-suit and G-suit + grip conditions are presented in Table 16 and Figure 11. Note that the fall in blood pressure during the SOR in the grip condition is less steep than the control and is significantly different from the control at 3, 4 and 5G (Tables 17 and 18). The presence of the G-suit affords a similar and significant improvement of the blood pressure response to acceleration. Note that at 1-3G, there are 8 subjects in the control condition, but that the number of subjects decreases with increasing G level as subjects begin to reach their endpoints. The blood pressure decay (see Figure 11) in the grip + G-suit condition is significantly different from either grip or G-suit at the 3 and 4G levels, and a clearly additive effect is seen. There is no significant difference between the blood pressure responses at 1, 2 and 3G for any of the conditions.

Respiration - Slow Onset Runs: A typical pattern of respiration, as recorded with a nasal thermistor during a slow onset run, is seen in the first panel of Figure 5. This data has not been reduced for rate or volume. Note, however, that rate in depth appears to increase slightly and the pattern of respiration becomes more uniform, in agreement with previous descriptions of respiration during positive acceleration (12). There is no evidence of breathholding, Valsalva or other respiratory straining maneuver.

FIGURE 11. Mean arterial blood pressure in millimeters of mercury during +G_Z acceleration in slow onset runs in four conditions: control, grip, G-suit and grip plus G-suit, mean for eight subjects. Note that this figure presents mean values of five beats taken every ten seconds for the individual subject blood pressure data seen in Figures 14 and 15 (vide infra). Although all eight subjects are present at 1, 2 and 3G, the "n" or numbers of subjects decreases as individual tolerances are reached. The last value during acceleration, therefore, represents only one to three subjects.

Heart Rate - Slow Onset Runs: The heart rate responses in the slow onset runs are seen in Figure 12 and Tables 19, 20, and 21. The mean heart rate at rest (1G) was similar for all four conditions and rose progressively with increasing G in each case. The increase in rate with grip alone was greatest at each G level, whereas the increase in heart rate with grip + G-suit was intermediate between grip + G-suit and control conditions. The heart rate at each G level during G-suit and control runs was similar. Statistically significant differences are presented in Table 21.

Linear Regression of Heart Rate and Blood Pressure: Inspection of Figures 11 and 12 suggests an inverse relationship between heart rate and blood pressure during slow onset +Gz acceleration. Table 22 and Figure 13 present the results of a linear regression of heart rate on mean eye level arterial blood pressure by the method of least squares fit. The fits are surprisingly good with the correlation coefficients for the four conditions ranging from $r = -0.996$ to -0.894 .

Best-Fit Functions for MABP in SOR: The mean arterial blood pressure responses during slow onset runs for the eight individual subjects in the four conditions are seen in Figures 14 and 15. The hydrostatic model of the cardiovascular mechanisms of blackout proposed by Wood (2) and Howard (12) predicts that tolerance to +Gz acceleration occurs at or near an eye level blood pressure of 20 mmHg. Examination of Figure 11, the subject-and-time-meamed, MABP response to SOR for G level at MABP of 20, shows control about 4.2G, grip 6.1, G-suit 6.4 and G-suit + grip >7G. These values do not agree particularly well with the observed mean G tolerance levels of 4.6, 5.6, 5.8 and 6.3 respectively. Therefore, best-fit linear and exponential functions

FIGURE 12. Heart rate in beats/minute during +G_z acceleration in slow onset runs in four conditions: control, grip, G-suit, and G-suit plus grip, mean for eight subjects. Heart rate was calculated from the beat to beat time interval and meaned over five beats. Although all eight subjects are present at 1, 2, and 3G, the "n" or number of subjects decreases as individual tolerances are reached. The last heart rate value in each condition, therefore, represents only one to three subjects.

FIGURE 13. Linear regression of mean eye level arterial blood pressure on heart rate for +Gz acceleration slow onset runs.

FIGURE 14. Continuous (beat by beat) mean arterial blood pressure (millimeters of mercury) response for each of the eight individual subjects during +G_z acceleration slow onset runs in control and grip conditions. Note that the acceleration ramp slants at time = 4 seconds and ends for a given subject when his pressure is seen to rise to a rebound or recovery value higher than the pre-acceleration value. Note the prominent respiratory variation in blood pressure in some of the subjects.

FIGURE 15. Continuous (beat by beat) mean arterial blood pressure (millimeters of mercury) response for each of the eight individual subjects during +G_Z acceleration slow onset runs in G-suit and G-suit plus grip conditions. Note that the acceleration ramp slants at time = 4 seconds and ends for a given subject when his pressure is seen to rise to a rebound or recovery value higher than the pre-acceleration value. Note the prominent respiratory variation in blood pressure in some of the subjects.

were determined for the decreasing portion of all the individual data points (Figures 14 and 15) for each condition, by the method of least squares.

Mean Arterial Blood Pressure Response to Grip and Vascular

Occlusion: The mean arterial blood pressure response in four subjects to a 50% MVC grip when a venous occlusive cuff (300 mmHg) is applied either at the onset or the end of the 90 second grip period is seen in Figure 16 and Table 23. When the cuff is applied simultaneously with the onset of the grip, the rise in blood pressure is 28 mmHg in 90 seconds. The grip is left off but the cuff remains on and the blood pressure falls 9, 6 and 2 mmHg at 30, 60, 90 seconds respectively. In other words, if applied at the onset of grip, an occlusive tourniquet sustains the blood pressure elevation produced by grip, which drops on the average only 6 mmHg out of 28 or about 20%. When the cuff is not applied until 5 seconds before the grip is let off (grip end), the blood pressure elevation produced by grip (16 mmHg) falls approximately 10 mmHg or about 60%.

Mean Arterial Blood Pressure Responses to G-suit and G-suit +

Grip: The effects of mean arterial blood pressure (MABP) of static G-suit inflation at three pressures (80, 160 and 240 mmHg) and G-suit + grip in combination are seen in Table 24. At 80 mmHg, the G-suit has essentially no sustained effect on MABP. A small (8-10 mmHg) transient rise in MABP is seen in the first few seconds on the strip chart of the raw data but is not sustained and has disappeared in 30 seconds. A similar transient MABP increase is noted at suit pressure of 160 mmHg but, at this level, a small (~5 mmHg) sustained pressure rise is seen at 30 seconds. Only one subject was studied

FIGURE 16. Mean arterial blood pressure (millimeters of mercury) response to 50% maximum voluntary contraction (MVC) handgrip, mean values for four subjects. In the upper set, an occlusive cuff (300 millimeters of mercury) has been applied to the arm above the gripping forearm at the onset of grip (rest or zero time) and maintained for 180 seconds. In the lower set, the cuff is inflated five seconds prior to the end of grip (85 seconds) and is maintained to 180 seconds.

at 240 mmHg. When G-suit (160 mmHg) and grip in combination produce a partially additive MABP response, the G-suit provides most of the combined effect at 30" and the grip most of the effect at 90".

Intrathoracic and Intra-gastric Pressure During Valsalva, M-1 and Grip Maneuvers: In three subjects, intrathoracic (P_{eso}), intra-gastric (P_{gas}) and expiratory flow were measured before, during and after 40% MVC grip, a 40 mmHg Valsalva maneuver and an M-1 maneuver as seen in Figure 17. Figure 17 represents a typical subject. The grip was held for approximately 20 seconds. Note that the respiratory pattern as indicated by expiratory flow is unaltered by the grip maneuver. No change is seen in the level or pattern of either intrathoracic or intra-gastric pressure. This is in contrast to the necessary cessation of respiration and increase in P_{eso} and P_{gas} seen during Valsalva and M-1 maneuvers. In Figure 18, the same signals are recorded at the grip onset (upper panel) and grip offset (lower panel) of a 40% MVC grip held to fatigue. Note that even at the point of fatigue where the subject can no longer maintain the grip, the intrathoracic and intra-gastric pressures are not increased. A brief transient in P_{eso} was seen at the grip offset representing a weak cough.

FIGURE 17. Respiratory flow (expiration) and esophageal (P_{ESO}) and gastric (P_{GAS}) pressures during 15 second 40% handgrip (upper panel), 40 mmHg Valsalva (middle panel), and an M-1 maneuver (lower panel). Note the typical cessation of respiration and increases in gastric and esophageal pressures in the Valsalva and M-1 maneuvers, (gastric approximately 38 mmHg which is typically slightly higher than esophageal pressure, approximately 30 mmHg) and note the unaltered pattern and level of these pressures during the brief, approximately 15 second, handgrip.

FIGURE 18. Respiration flow (expiration) and esophageal (P_{eso}) and gastric (P_{gas}) pressures during a 40% maximum voluntary handgrip held to fatigue. The blood pressure (electrosphygmomanometer) trace is incidentally seen. The onset of grip is noted by an arrow in the upper panel and the release or grip off, approximately 90 seconds later, is seen in the lower panel. Note that the patterns and levels of esophageal and gastric pressures are unchanged. An incidental transient, probably a cough, is seen in P_{eso} at grip off.

DISCUSSION

Since the early days of World War II, it has been clear that a tactical advantage in air combat operations goes to the pilot with the highest tolerance to positive (+Gz) acceleration. In the decade 1939 to 1949, there was an intensive program of research to develop effective G protective techniques. This work produced the present antigravity counterpressure garment or G-suit (38,39,40,41,42). Although research on the problem has continued in the subsequent two decades, the G-suit is still the standard acceleration protection technique and no significant improvement has been made in its design or performance. The development of new higher performance air superiority fighters by the United States Air Force has lead to a requirement for techniques to significantly improve human tolerance to positive acceleration.

Cardiovascular Response to Positive Acceleration: Human tolerance to +Gz acceleration is defined by the occurrence of blackout and unconsciousness. Two cardiovascular mechanisms are involved: (1) the hydrostatic factor which reduces the effective perfusion pressure at the eye (retina) and brain and (2) the orthostatic factor or pooling of blood in dependent capacitance vessels and tissues which results in decrease venous return to the heart, a fall in cardiac output, and, finally, reduction in the effective perfusion pressure at the eye. These vascular mechanisms are adequately explained by consideration of simple hydrostatics. A hydrostatic model of the hemodynamic responses to positive acceleration has been elegantly worked out by Lindberg and Wood (2) and by

Howard (12) (vide supra). However, these mechanisms pertain only to exposures of less than 10-12 seconds. Beyond this time, cardiovascular reflex compensations to the acceleration load occurs: blood pressure is reflexly elevated, heart rate increases and cardiac output is redistributed and neither the blood pressure response or tolerance time can be predicted by a simple hydrostatic model.

The presently accepted theory predicts that blackout in +Gz acceleration occurs at the moment that retinal artery pressure falls below intra-ocular pressure (~ 20 mmHg) and retinal perfusion ceases (12). This explanation was first propounded by Andina, who was the first to point out that intra-ocular pressure is some 20 mmHg higher than intracerebral pressure and consequently blood supply to the retina fails before the cerebral circulation (3). As confirmation, Lambert showed that application of 30 to 40 mmHg negative pressure to the eyeball by means of suction goggles will raise the blackout threshold (4). Similarly, application of positive pressure to the eye will lower the threshold. Livingston pointed out that the retina has an oxygen requirement four times as great as that of any other body tissue, excepting the carotid body (5). Hence, even a minor reduction of blood supply should cause marked interference with vision.

Duane examined the retina directly with a funduscope and drew illustrations at +Gz accelerations at blackout levels and measured arterial pressure in the radial artery with the arm so placed that the tip of the cannula was at eye level (6). A clear correlation between the subject's visual change and change in retinal perfusion was established. Leverett and his coworkers at the U.S. Air Force School of Aerospace Medicine (SAM) at Brooks Air Force Base, Texas, have

recently confirmed and extended Duane's hypothesis by direct photographic observation of the retinal circulation during +Gz acceleration (7).

Protective Techniques: As Wood has suggested, there are three general methods that can be used to increase man's ability to tolerate the reactive forces produced by positive (+Gz) acceleration: (1) limiting the duration of the acceleration exposure, (2) assuming postures or positions that reduce the length of vertical hydrostatic vascular distances and (3) the use of procedures or devices that increase blood pressure (2). The study and development of G protective devices and techniques has been well reviewed (2,12,13). In this paper, I have demonstrated that isometric exercise, a simple 50% of a maximum handgrip contraction, gives about 1G protection in both rapid and slow onset acceleration profiles. The modern G-suit gives protection ranging from 1-2G depending on the endpoint used, suit pressurization profiles and subject factors (2,13). In the present study, G protection of 1.2G in ROR and 1.1G in SOR was demonstrated in good agreement with the literature. The G-suit and grip, in combination, give protection of 1.9 and 1.7 in ROR and SOR respectively. As noted above, however, the mean G tolerance in the SOR G-suit + grip condition is artificially low as the runs were arbitrarily stopped at 7G for safety reasons and 3 subjects had not yet reached tolerance at this level.

Human tolerance to acceleration may be improved by the use of a variety of protective measures and devices. Straining and grunting in a tight turn was practiced by pilots, prior to World War II, to improve their tolerance and culminated in the development of the M-1

maneuver (18,43). Changing of posture had been practiced, particularly in World War II, when hunching forward in the cockpit was found to improve the tolerance of pilots to high acceleration maneuvers. The effects of posture, restraint and body position on G tolerance have been well worked out and have been reviewed in detail (13). The history of G-suits and protective devices dates from World War II and is well reviewed in recent monographs (2,12,13,41). The wrap-around CSU-3/P cutaway-type of anti-G suit, presently used by the U.S. Air Force, improves blackout tolerance in the +Gz vector by about 2G (13). Analysis of contributions of portions of this suit show that the whole suit gives protection of approximately 2G in terms of blackout threshold while leg bladders alone give protection of about 0.2G. Abdominal bladders without leg bladders protect to about 1.2G while arm cuffs alone produce a hardly measurable protection. Arm cuffs added to a full suit will raise protection by a further 0.5G.

The physiological mechanism of action of anti-G suits was originally established on the Toronto centrifuge and at the Mayo Clinic and have been summarized by Wood and Lambert (15). They have shown that inflation of a G-suit at 1G produces an initial increase in arterial pressure, followed by an almost immediate decrease in heart rate probably due to a depressor reflex originating in the carotid sinus and aortic areas. The initial pressure increase decays during the bradycardia. A similar pattern is seen when the suit is inflated under +Gz acceleration. With inflation during acceleration, the arterial pressure rises slightly above normal at the heart level but remains below normal at the head level. Since the pressure in the carotid sinus is not significantly elevated, slowing of the

rate does not occur. Lindberg et al have pointed out that the decrease in cardiac output that occurs during +Gz acceleration is not significantly altered by the use of an inflated G-suit (16). Hence, the protection afforded by the suit is not associated with a relative increase in cardiac output. They observed that inflation of an anti-G suit under +Gz acceleration is associated with a relative increase in mean aortic pressure which acts to counteract the increased hydrostatic pressure. In Wood and Lambert's studies, blood pressure was measured at head level during G-suit inflation at suit pressures used at 2, 4, 6, and 8G (approximately 40, 140, 240, 340 mmHg respectively) but only for 15 seconds (15). The only data they present is one figure from one subject. The magnitude and time history of the blood pressure response to graded (50-350 mmHg) levels of G-suit counterpressure are apparently not available in the literature. The data presented in Table 24 (vide supra), although from a small set of subjects, suggests that 80 mmHg counterpressure has no sustained effect on blood pressure and that 160 mmHg has a slight (~ 5 mmHg) sustained effect.

Gray et al have recently studied the acute (5 second) and chronic (5 minute) effects of sudden G-suit inflation in the recumbent and passively tilted postures (17). Acute inflation to 80 mmHg in 3 seconds was associated with a transient increase in cardiac output, mean brachial artery pressure, central blood volume and right atrial pressure with a transient decrease in pulse rate. Trends of these hemodynamic alterations were similar in both the recumbent and tilted groups. However, the changes were of greater magnitude in the tilted subjects who were at one +Gz acceleration (and might be expected to

approximate those at higher G). Of particular interest is the transient nature of the hemodynamic responses to G-suit inflation. Cardiac index response, in particular, although increased significantly in the upright individuals, is brief and is returned near control levels by 45 seconds. The response is transient and probably of little importance in the prolonged use of the garment. Mean resting brachial artery pressure in the 18 healthy male subjects in these studies was 87 mmHg and increased approximately 3-5 mmHg on suit inflation. This pressor response, however, was sustained throughout the five minute G-suit application.

I would propose, from an analysis of this and similar data, that: (1) an increase in mean arterial pressure is the single most significant hemodynamic factor in improving G tolerance, (2) that the principle mechanism of action of a G-suit is its sustained elevation of systemic blood pressure, (3) that the effect of G-suit inflation at pressures of less than 100 mmHg on blood pressure, though probably sustained, is minimal (5 mmHg or less), and (4) other physiological pressor agents or techniques should be both G protective and additive with G-suit protection.

Straining Maneuvers: Present G protective techniques, principally the G-suit, attempt to prevent the peripheral pooling of blood during acceleration by the application of a suitable counterpressure. No significant attempt has been made to develop techniques to prevent the fall in mean arterial blood pressure at head level. A logical approach would be to artificially raise mean systemic blood pressure. Vasoconstrictive drugs have been tried as G protective agents but irregular, delayed and relatively unpredictable dose-response relations

limit their utility (44). The hypertensive effects of breathing increased concentrations of CO_2 have been tested and shown by Gauer to improve +Gz tolerance approximately 10%. The relatively small effect and the associated headache have limited the application of this technique (24).

Since the early days of flying, the value of muscular-respiratory straining as protection against the effects of positive acceleration (+Gz) has been recognized. Pilots, German WWII aviators in particular, observed that they could delay grayout and blackout if they grunted, screamed and strained their muscles. The M-1 maneuver or muscular straining, with a series of forced expirations against a partially closed glottis, is probably the most effective voluntary protection against the circulating effects of +Gz acceleration. G protection of 2-2.5G is afforded by a properly performed M-1 maneuver (15,18). The physiological basis of this protection has been correctly ascribed to its effect of increasing arterial pressure at eye level (19). The hypertensive and hence G protective effect of straining, however, clearly has two components: respiratory and muscular. Rushmer described the respiratory or thoraco-abdominal mechanism by which the M-1 maneuver raised arterial blood pressure: forced expiration against the partially closed glottis raises the intrathoracic pressure which is transmitted directly or hydraulically to the intrathoracic great vessels. There is an immediate rise in arterial pressure which is only slightly less than the rise in intrathoracic pressure. During the remainder of the maneuver, intrathoracic pressure falls continuously while intra-abdominal pressure remains elevated, creating a pressure gradient between the

abdomen and thorax augmenting the return of blood to the heart (19).

The muscular component of the hypertensive effect of straining has not previously been clearly separated from the respiratory maneuver. Wood, in his most recent review of acceleration effects, states that the respiratory maneuver during the M-1 increases the blood pressure, while the tensing of muscles of the extremities prevents the peripheral pooling of blood, implying that only the respiratory part of the maneuver effected blood pressure (2). In the present study, I have clearly demonstrated that muscle tensing has a hypertensive and G protective effect; but that this is not related to an M-1 or Valsalva-like maneuver. The increase in mean arterial pressure is seen in the absence of any significant change in rate or depth of respiration (Figure 5), and with normal, resting intrathoracic and intra-abdominal pressures (Figure 15). The only information concerning the respiratory effects of handgrip has been presented by Wiley and Lind who noted mild hyperventilation during 50% maximum voluntary handgrip contractions held to fatigue (45). They too noted no increase in intrathoracic pressure during the grips, even when held to fatigue.

Lambert, Wood and Baldes reported that pulling against a force of 19 pounds per G applied to a mock aircraft control stick increased G tolerance 0.7G, which agrees well with the 0.9G (ROR) and 1.0G (SOR) protection afforded by grip in this study (20). As in the present study, the protection from pulling on the stick was additive to that of a G-suit. No information is given concerning respiration or blood pressure; and the response cannot be attributed to either Valsalva-like maneuver or isometric muscular effort.

Blood Pressure Response to Handgrip: The mechanism of the respiratory component of straining maneuvers is well appreciated, as described above. However, the mechanisms of the pressor response to handgrip or sustained muscular contractions are less well understood. Cardiovascular responses to sustained isometric or non-dynamic exercise have been studied recently in a series of papers by Lind and his coworkers (21,22,46,47). These workers have used sustained handgrip as their model of isometric exercise and studied responses to percentage fractions of maximum voluntary contractions. They have demonstrated, "...the existence of a powerful cardiovascular reflex causing an unexpectedly high rise in blood pressure response to sustained contractions" (47). This pressor response is a 40-60 mmHg increase in mean arterial blood pressure during a sustained 50% maximum handgrip contraction (22). At contractions of less than 15% of maximum blood pressure, response is small and plateaus or steady state pressures were reached. At greater than 20% contraction, the blood pressure continues to rise for as long as the contraction can be held and no steady state is reached. The pressor response seen is apparently independent of the size of the muscle group involved in the contraction, provided that the relative tension is constant. In addition, it is independent of the number of muscle groups involved. That is, the blood pressure response to a single forearm contraction is not increased by simultaneous contraction of the other forearm or of another muscle group (46).

The duration of the contraction that can be voluntarily held, or the "time-to-fatigue", appears to vary inversely with tension. The blood pressure response depends both on duration and on tension.

Blood flow through the contracted forearm increases at low levels of tension and then decreases as the per cent of maximum tension increased. On release of the contraction, the blood pressure falls immediately to control levels. The peripheral blood flow response is sustained or temporarily increases before falling.

These investigators considered three possible mechanisms underlying the cardiovascular response to sustained contraction: (1) humoral, (2) central nervous drive, and (3) reflex activity. It was found that complete circulatory occlusion of the contracting forearm actually increased the rate of development of the blood pressure response to the contraction, suggesting that the pressor response is not caused by release into the general circulation of materials produced in the active muscles (22). In addition, the abrupt offset of the pressor response at the end of the grip would not favor a humoral mechanism. The experiments with occlusive cuffs in the present study confirm these observations. The presence of the occlusive (300 mmHg) cuff, in place prior to the grip, increased the subsequent MABP response to the grip (Figure 14). Further evidence against a central drive theory and in favor of a local response is presented in the experiments of sustained contractions carried out with the circulation occluded and with the occlusion continued after the exercise ended. In such studies, the blood pressure remains elevated at the end of the contraction until the occlusion was released (Figure 14). When the occlusion is maintained after the grip ends, blood pressure falls to a level between 20 and 80% of the increase found at the end of the contraction (depending on when occlusion is begun) and returns to

control values only when the cuff is released, suggesting that sustained pressor response is caused by the continued action of a reflex due to aerobic metabolites or other products of muscular contraction remaining trapped in the arm.

Further evidence for the reflex nature of the blood pressure response to grip was obtained by Lind et al from the study of a patient with syringomyelia who had sensory involvement in one arm with minimum sensory involvement in the other (48). Sustained handgrip contraction in the uneffected arm resulted in a typical pressor response, but there was no increase in blood pressure when the same degree of tension was developed within the effected arm. Recent studies by this group has suggested that potassium may be the metabolic product collecting in the exercising tissue and stimulating the pressor response (22,49).

There is apparently only a single study of the central hemodynamic response to sustained handgrip contraction (21). In this study, the blood pressure rise was shown to be associated with a rise in cardiac output which, in turn, depended upon an increase in heart rate but not stroke volume. "Surprisingly", to the authors, systemic vascular resistance showed little change or slight reduction in most cases. Stroke volume showed no systematic change at 10-20% of maximum voluntary contraction but a consistent reduction was seen in all cases of 50% MVC when cardioacceleration was pronounced. After beta sympathetic blockade with propranolol, the blood pressure response to grip was still present, even though the cardiac output increase was markedly reduced. In the same study, subjects with essential hypertension were given guanethidine after a

control handgrip. Doses were sufficient to lower resting arterial blood pressure significantly and the pressor response to the Valsalva maneuver was abolished. However, the pressor response to sustained handgrip was unimpaired. The authors conclude that "these mechanisms add little to the illucidation of the mechanisms involved in the pressor response but do indicate that the response is fundamentally powerful enough to occur even after a Valsalva response is abolished". That the pressor response to grip is a locally initiated reflex would seem to be established. However, the mechanisms are not clearly identified. Specific receptor and effector mechanisms remain to be identified.

From the observations of the present study and from the literature, it seems likely that the pressor response to grip involves a reflex mechanism initiated by metabolites accumulating in the exercising muscle stimulating an undefined nervous system receptor. Although blood flow increases in statically contracting muscle at tensions of up to 70% of maximum, consideration of the post-exercise hyperemia seen after contractions of greater than 20% MVC lead Lind et al to the conclusion that, during such static contractions, blood flow is never sufficient for the muscles' metabolic needs and that a "blood debt" occurs which is only partially repaid by the post-grip hyperemia. In Figure 14, it was noted that the pressor response to a 90 second, 40% MVC was 75% (16 vs 28) greater when arterial occlusion was present during the grip. In addition, the pressor response is maintained at about 80% of the final grip value if the venous occlusion is maintained at cessation of grip. This is consistent with the hypothesis that

some locally accumulating factor, trapped by the occluding cuff, produces or contributes to the pressor response to grip.

In conclusion, this latter demonstration may also have a useful application. A pilot, having gripped once to elevate his blood pressure, could, by appropriate cuffing, maintain most of the pressor effect and its G protection for periods of 1-2 minutes or more. As shown in these results, this protection should be additive to that of his standard G-suit. The maximum length of time that the cuff alone can sustain the pressor response without discomfort and optimum cuff cycles and pressures remain to be determined. A schema for such a G protective assembly based on this concept has been prepared to support a patent application.

SUMMARY

Muscular straining is a well known and effective G protective maneuver. A reproducible form of this technique without the problem of raised intrathoracic pressure could be a significant improvement in the presently employed G protective maneuvers. This study was designed to determine whether the blood pressure elevation achieved with fractional handgrip contractions will extend man's G tolerance during both rapid (ROR) and slow (SOR) onset runs on the human centrifuge. Eight healthy Air Force volunteers were used as subjects. Direct radial arterial blood pressure at eye level, the vector-cardiogram using the Frank lead system, respirations using a nasal thermistor and handgrip dynamometer output were recorded. G tolerance as indicated by peripheral light loss or central light dimming was determined in the ROR (1.0G/second onset) during 10 second exposures to progressively increasing G levels (0.3G increments). During the SOR, the 8 subjects were exposed in a single run to a progressively increasing G stress (0.1G/second onset) until peripheral light loss. G tolerance during the rapid onset runs was increased in all subjects when the run was preceded by 60-90 seconds of grip, increasing from 0.3 to 1.8G with a mean of 0.95G (n=8). The use of a standard G-suit alone increased tolerance 0.7-1.25G with a mean of 1.13 (n=6). G-suit and grip increased tolerance 1.4-2.4G over control values with a mean of 1.86G (n=7). In the 5 subjects in which G-suit and G-suit + grip were studied, grip increased G-suit tolerance from

0.4-1.0G with a mean increase of 0.76G (n=5). During the slow onset runs, there was an increase in G tolerance with grip, G-suit and G-suit + grip comparable to that seen with the rapid onset runs. The control +Gz mean was 4.6G (3.4-6.7) compared to 5.6 for Gz + grip (3.9-7.0), 5.8 for Gz + G-suit (4.8-7.0) and 6.31 for Gz, grip and G-suit (5.0-7.0). The mean blood pressure under each condition was evaluated both prior to, during, and after the rapid onset runs. In the two instances when grip is applied prior to acceleration (grip, and grip + G-suit), the blood pressure at the onset of acceleration is elevated approximately 20 mmHg when compared to control values. In order to evaluate the possibility that a forearm contraction using the handgrip dynamometer involved a modified Valsalva or M-1 maneuver, intrathoracic and intra-abdominal pressure and ventilation were monitored in 3 subjects during a maximum forearm voluntary contraction. The characteristic increase in intra-abdominal and intrathoracic pressure and the ventilation characteristic of a Valsalva or M-1 maneuver were not seen during 50% MVC handgrip held to fatigue. CONCLUSIONS: (1) 30% or 40% maximum voluntary handgrip contraction increases +Gz acceleration tolerance approximately 1G under both rapid and slow onset conditions; (2) the protection offered by grip is additive to that of the standard G-suit; (3) the increased Gz tolerance obtained with handgrip is not related to having performed an M-1 or Valsalva-like maneuver; (4) mean blood pressure elevation correlates directly with increased Gz acceleration tolerance.

APPENDIX

RESULTS IN TABULAR FORM

(Tables 3-24)

TABLE 3 TIME TO FATIGUE, MEAN ARTERIAL BLOOD PRESSURE, AND RATE OF INCREASE OF BLOOD PRESSURE DURING FRACTIONS OF MAXIMAL VOLUNTARY HANDGRIP CONTRACTIONS.

Time to fatigue, in seconds, of four subjects holding 20, 30, 40 and 50% of a maximum voluntary handgrip contraction while supine.

<u>SUBJECT</u>	<u>% MAXIMUM VOLUNTARY CONTRACTION</u>			
	<u>20%</u>	<u>30%</u>	<u>40%</u>	<u>50%</u>
1	562	290	130	91
2	620	260	118	90
3	765	305	208	110
4	350	240	145	75
MEAN	574.2	273.8	150.2	91.5

Increase in mean arterial blood pressure in mmHg (determined indirectly with electrospgymomanometer) in four subjects holding 20, 30, 40 and 50% of a maximum voluntary handgrip contraction while supine.

<u>SUBJECT</u>	<u>% MAXIMUM VOLUNTARY CONTRACTION</u>			
	<u>20%</u>	<u>30%</u>	<u>40%</u>	<u>50%</u>
1	46	50	*39	*34
2	45	57	57	43
3	25	38	52	49
4	38	36	60	48
MEAN	38.5	42.2	52.0	43.5

*Last B.P. measurement not available.

Rate of increase of mean arterial blood pressure in mmHg/second (determined graphically) in four subjects holding 20, 30, 40 and 50% of a maximum voluntary handgrip contraction.

<u>SUBJECT</u>	<u>% MAXIMUM VOLUNTARY CONTRACTION</u>			
	<u>20%</u>	<u>30%</u>	<u>40%</u>	<u>50%</u>
1	0.080	0.176	0.362	0.549
2	0.065	0.250	0.483	0.489
3	0.032	0.131	0.226	0.400
4	0.109	0.146	0.379	0.613
MEAN	0.072	0.176	0.362	0.513

TABLE 4 G LEVEL AT PERIPHERAL LIGHT LOSS IN EIGHT SUBJECTS IN CONTROL, GRIP, G-SUIT AND G-SUIT + GRIP CONDITIONS DURING RAPID ONSET RUNS.

SUBJECT	CONTROL	GRIP	G-SUIT	G-SUIT + GRIP
1	3.5	4.2	a---	5.4
2	3.7	4.1	4.8	5.6
3	3.3	3.8	a---	5.1
4	3.1	3.4	3.8	4.5
5	3.2	4.5	4.2	4.6
6	4.3	5.2	5.6	6.7
7	3.7	5.3	5.3	a---
8	3.8	5.6	5.0	6.0
<hr/> x	<hr/> 3.57	<hr/> 4.51	<hr/> 4.78	<hr/> 5.41
SEM	0.14	0.28	0.27	0.29
SD	0.39	0.78	0.68	0.78

^a Subject not able to complete the run due to fatigue.

TABLE 5 ANALYSIS OF VARIANCE FOR G LEVEL AT PERIPHERAL LIGHT LOSS DURING RAPID ONSET RUNS.

SOURCE OF VARIANCE	DEGREES OF FREEDOM (df)	SUM OF SQUARES (S.S.)	MEAN SQUARE (M.S.)	F	LEVEL OF SIGNIFICANCE (P<)
TOTAL	31	25.3	---	---	
CONDITION A ^a	3	14.0	4.6	43.7	0.005
CONDITION B					
SUBJECTS	7	9.01	1.3		
INTERACTIONS					
A x B					
A x S	21	2.2	0.12	---	
B x S					
A x B x S					

^a The four conditions were: control, grip, G-suit, G-suit + grip.

TABLE 6 STUDENTS "T" TEST (PAIRED SAMPLES) OF DIFFERENCES
BETWEEN MEAN G VALUES AT PERIPHERAL LIGHT LOSS IN FOUR CONDITIONS
IN RAPID ONSET RUNS.

<u>CONDITIONS</u>	<u>T</u>	<u>df</u>	<u>P</u>
1. Control vs Grip	4.6680	7	<0.0025
2. Control vs G-suit	9.3380	5	<0.0005
3. Control vs G-suit + Grip	13.1544	6	<0.0005
4. Grip vs G-suit	0.5000	5	NS
5. Grip vs G-suit + Grip	4.8878	6	<0.0025
6. G-suit vs G-suit + Grip	6.5320	4	<0.0025

TABLE 7 MEAN ARTERIAL BLOOD PRESSURE (MM HG) AT EYE LEVEL MEAN VALUES $\pm$ SEM FOR EIGHT SUBJECTS IN CONTROL, GRIP, G-SUIT AND GRIP + G-SUIT CONDITIONS AT REST, 30", 60", INITIAL (90"), LOW, COMPENSATION AND RECOVERY PERIODS FROM THE FINAL OR TOLERANCE RAPID ONSET RUN.

PERIOD	MEAN ARTERIAL BLOOD PRESSURE (MM HG)			
	CONTROL	GRIP	G-SUIT	GRIP + G-SUIT
1. Rest	79 $\pm$ 3.1	79 $\pm$ 2.5	77 $\pm$ 2.3	77 $\pm$ 3.7
2. 30"	---	86 $\pm$ 2.3	---	90 $\pm$ 4.2
3. 60"	---	99 $\pm$ 4.4	---	93 $\pm$ 1.3
4. Initial	81 $\pm$ 3.4	105 $\pm$ 5.1	83 $\pm$ 3.6	93 $\pm$ 12.5
5. Low	7.8 $\pm$ 5.0	14 $\pm$ 7.1	1.6 $\pm$ 6.4	17.4 $\pm$ 6.2
6. Compensation	20 $\pm$ 7.1	41 $\pm$ 7.4	22 $\pm$ 5.8	38 $\pm$ 6.8
7. Recovery	92 $\pm$ 3.2	102 $\pm$ 3.8	87 $\pm$ 4.3	94 $\pm$ 5.5

TABLE 8 ANALYSIS OF VARIANCE OF MEAN ARTERIAL BLOOD PRESSURE IN RAPID ONSET RUNS (ROR).

SOURCE OF VARIANCE	DEGREES OF FREEDOM (df)	SUM OF SQUARES (S.S.)	MEAN SQUARE (M.S.)	F d.	LEVEL OF SIGNIFICANCE (P<)
TOTAL	95	121,083	---	---	---
a. CONDITION A (Conditions)	3	2,499	833	12.3	0.05
b. CONDITION B (Periods)	3	107,112	35,704	563	0.001
c. SUBJECTS	5	5,461	1,092	26	0.001
INTERACTIONS					
A x B	15	2,198	244	5.9	0.005
A x S	15	1,018	67	1.6	NS
B x S	45	949	63	1.5	NS
A x B x S		1,842	40	---	---
RESIDUAL					

- a. The four conditions were: control, grip, G-suit and G-suit + grip.
b. Four time periods are used in this analysis: rest, initial, low and recovery.
c. Only four of the eight subjects were used in this analysis - those four that had no missing data, i.e. a measure was made at 4 times (periods) and 4 conditions - to avoid the problems of missing entries in the analysis.
d. $df_{\text{error}} = 95 - 5 - 3 - 3 = 84$

TABLE 9 SIGNIFICANCE LEVELS (P =) OF A DUNCAN MULTIPLE RANGE TEST OF DIFFERENCES BETWEEN MEANS OF MEAN ARTERIAL BLOOD PRESSURE IN FOUR CONDITIONS AT REST, INITIAL, LOW, COMPENSATION AND RECOVERY PERIODS IN RAPID ONSET RUNS.

<u>CONDITIONS</u>	<u>PERIODS</u>				
	REST	INITIAL	LOW	COMPENSATION	RECOVERY
1. Control vs Grip	NS ^{a.}	0.01	NS	0.05	NS
2. Control vs G-Suit	NS	NS	NS	NS	NS
3. Control vs G-Suit + Grip	NS	0.01	NS	0.05	NS
4. Grip vs G-Suit	NS	0.01	NS	0.05	0.05
5. Grip vs G-Suit + Grip	NS	NS	NS	NS	NS
6. G-Suit vs G-Suit + Grip	NS	0.01	0.05	0.05	NS

a. NS = not significant

TABLE 10 MEAN HEART RATE (BEATS/MIN) $\pm$ 1 STANDARD DEVIATION FOR EIGHT SUBJECTS IN CONTROL, GRIP, G SUIT AND GRIP + G SUIT CONDITIONS AT REST, 30", 60", INITIAL (90"), LOW, COMPENSATION AND RECOVERY TIMES FOR THE FINAL OR TOLERANCE RAPID ONSET RUN.

<u>PERIOD</u>	<u>CONDITIONS</u>			
	CONTROL	GRIP	G SUIT	G SUIT PLUS GRIP
1. Rest	82 $\pm$ 12	84 $\pm$ 19	86 $\pm$ 15	78 $\pm$ 12
2. 30"	---	91 $\pm$ 21	---	93 $\pm$ 22
3. 60"	---	99 $\pm$ 23	---	96 $\pm$ 16
4. Initial	82 $\pm$ 12	100 $\pm$ 25	86 $\pm$ 15	99 $\pm$ 22
5. Low	98 $\pm$ 16	113 $\pm$ 21	98 $\pm$ 11	110 $\pm$ 22
6. Compensation	102 $\pm$ 18	114 $\pm$ 21	105 $\pm$ 12	115 $\pm$ 21
7. Recovery	81 $\pm$ 14	88 $\pm$ 12	86 $\pm$ 14	95 $\pm$ 18

TABLE 11 ANALYSIS OF VARIANCE OF HEART RATE DURING RAPID ONSET RUNS.

SOURCE OF VARIANCE	DEGREE OF FREEDOM (df)	SUM OF SQUARES (S.S.)	MEAN SQUARE (M.S.)	F	LEVEL OF SIGNIFICANCE (P=)
TOTAL	99	44357	---	---	---
CONDITION A	3	5308	1769	6.0	<0.01
CONDITION B	4	10818	2704	12.7	<0.01
SUBJECTS	4	16598	4149	61.8	<0.01
INTERACTIONS					
A x B	12	1507	125	1.8	---
A x S	12	3512	292	4.3	---
B x S	16	3391	211	3.1	---
A x B x S	48	3220	67	---	---
RESIDUAL					

TABLE 12 SIGNIFICANCE LEVELS (P=) OF A DUNCAN MULTIPLE RANGE TEST OF DIFFERENCES BETWEEN MEANS OF HEART RATE IN FOUR CONDITIONS AT REST, INITIAL, LOW, COMPENSATION AND RECOVERY PERIODS IN RAPID ONSET RUNS.

<u>CONDITIONS</u>	<u>PERIODS</u>				
	REST	INITIAL	LOW	COMPENSATION	RECOVERY
1. Control vs Grip	NS	0.05	0.01	0.05	NS
2. Control vs G-suit	NS	NS	NS	NS	NS
3. Control vs G-suit + Grip	NS	0.05	0.05	0.05	0.05
4. Grip vs G-suit	NS	0.05	0.01	NS	NS
5. Grip vs G-suit + Grip	NS	NS	NS	NS	NS
6. G-suit vs G-suit + Grip	NS	0.05	0.05	NS	NS

TABLE 13 G LEVEL AT PERIPHERAL LIGHT LOSS IN EIGHT SUBJECTS IN CONTROL, GRIP, G-SUIT, AND G-SUIT + GRIP CONDITIONS DURING SLOW ONSET RUNS.

SUBJECTS	CONTROL	GRIP	G-SUIT	G-SUIT + GRIP
1a	3.4	3.9	5.0	5.0
2a	3.5	4.0	4.8	5.1
3	5.4	5.9	5.7	7.0 ^a
4	4.0	5.7	4.9	5.7
5a	5.4	6.0	5.1	7.2
6a	3.7	6.1	7.0 ^a	7.0 ^a
7	6.7	7.0 ^a	7.0 ^a	7.0 ^a
8	4.8	6.4	6.5	6.5
$\bar{x}$	4.61	5.63	5.75	6.31
SD	1.11	1.17	0.95	0.91

^a Tolerance endpoint not reached. Run terminated at 7G as a safety consideration.

TABLE 14 ANALYSIS OF VARIANCE FOR G LEVEL AT PERIPHERAL LIGHT LOSS DURING SLOW ONSET RUNS.

SOURCE OF VARIANCE	DEGREES OF FREEDOM (df)	SUM OF SQUARES (S.S.)	MEAN SQUARE (M.S.)	F	LEVEL OF SIGNIFICANCE (P<)
TOTAL	31	42.2	---	---	
a. CONDITION A (Conditions)	3	12.0	4.0	12.0	0.005
CONDITION B					
SUBJECTS	7	23.2	3.3	---	---
INTERACTIONS					
A x B					
A x S	21	6.9	0.33	---	---
B x S					
A x B x S					

a. The four conditions were: control, grip, G-suit, and G-suit + grip.

TABLE 15 STUDENTS "T" TEST (PAIRED SAMPLES) OF DIFFERENCES
BETWEEN MEAN G LEVELS AT PERIPHERAL LIGHT LOSS IN FOUR CONDITIONS
DURING SLOW ONSET RUNS.

CONDITIONS	T	df	P
1. Control vs Grip	3.7910	6	<.01
2. Control vs G-suit	2.8882	6	<.025
3. Control vs G-suit + Grip	8.0841	6	<.0005
4. Grip vs G-suit	0.4619	6	NS
5. Grip vs G-suit + Grip	4.0657	6	<.005
6. G-suit vs G-suit + Grip	2.2100	5	<.05

TABLE 16 MEAN ARTERIAL BLOOD PRESSURE (MM HG) AT EYE LEVEL MEAN VALUES $\pm$ SEM FOR EIGHT SUBJECTS IN CONTROL, GRIP, G-SUIT AND G-SUIT + GRIP CONDITIONS AT 1 (REST), 2, 3, 4, 5, 6, 7G AND RECOVERY IN SLOW ONSET RUNS.

<u>G LEVEL</u>	<u>MEAN ARTERIAL BLOOD PRESSURE</u>							
	CONTROL		GRIP		G-SUIT		GRIP + G-SUIT	
1	74 $\pm$ 3.6	(8)	73 $\pm$ 3.5	(8)	72 $\pm$ 2.6	(7)	77 $\pm$ 4.0	(7)
2	48 $\pm$ 3.5	(8)	53 $\pm$ 4.6	(8)	50 $\pm$ 4.0	(8)	50 $\pm$ 5.2	(7)
3	30 $\pm$ 4.9	(8)	42 $\pm$ 3.7	(8)	41 $\pm$ 4.6	(8)	51 $\pm$ 5.5	(7)
4	24 $\pm$ 6.1	(5)	36 $\pm$ 5.3	(7)	29 $\pm$ 4.5	(8)	44 $\pm$ 7.0	(7)
5	9 $\pm$ 3.7	(3)	27 $\pm$ 2.9	(6)	22 $\pm$ 7.0	(6)	32 $\pm$ 7.6	(7)
6	8	(1)	21 $\pm$ 4.3	(4)	27 $\pm$ 8.6	(3)	36 $\pm$ 6.6	(4)
7	---	(0)	13	(1)	13 $\pm$ 1.0	(2)	25 $\pm$ 11.1	(3)
Recovery	75 $\pm$ 5.4	(8)	84 $\pm$ 6.5	(8)	68 $\pm$ 3.1	(8)	80 $\pm$ 7.6	(7)
Term	15 $\pm$ 4.1	(8)	14 $\pm$ 2.8	(8)	16 $\pm$ 4.4	(8)	25 $\pm$ 7.8	(6)

NOTE: () = numbers of subjects reaching the particular G level.

TABLE 17 ANALYSIS OF VARIANCE OF MEAN ARTERIAL BLOOD PRESSURE DURING SLOW ONSET RUNS AT THREE G.

SOURCE OF VARIANCE	DEGREES OF FREEDOM (df) ^{c.}	SUM OF SQUARES (S.S.)	MEAN SQUARE (M.S.)	F	LEVEL OF SIGNIFICANCE (P=)
TOTAL	27	5473	---	---	
a. CONDITION A (Conditions)	3	1198	399	15.2	<u>≤0.025</u>
b. CONDITION B (G Level)	0	0	0	---	
SUBJECTS	6	3803	633		
INTERACTIONS					
A x B	0	0	0		
A x S	18	471	26		
B x S	0	0	0		
A x B x S	0	0	0		
RESIDUAL					

- a. The four conditions were: control, grip, G-suit and G-suit + grip.
- b. Because of the fact that subject SOR tolerance varied (as expected), seven subjects got to 3G in all 4 conditions while only one subject got to 6G in all conditions. Because of the varying degrees of freedom (numbers of subjects), the main effects of condition and G level cannot be tested, and only effects of condition at 3G are tested.
- c. $df_{\text{error}} = 27 - 6 - 3 = 18$.

TABLE 18 SIGNIFICANCE LEVELS (P=) OF A DUNCAN MULTIPLE RANGE TEST OF DIFFERENCE BETWEEN MEANS OF MEAN ARTERIAL BLOOD PRESSURE IN FOUR CONDITIONS AT 1, 2, 3, 4, 5, 6, and 7G, AND TERMINAL (i.e. TOLERANCE ENDPOINT) AND RECOVERY PERIODS.

CONDITIONS	<u>"G"</u>								TERM.	REC.
	1	2	3	4	5	6	7			
1. Control vs Grip	NS ^{a.}	NS	NS	0.05	0.01	-- ^{b.}	--		NS	NS
2. Control vs G-suit	NS	NS	0.01	NS	0.05	--	--		NS	NS
3. Control vs G-suit + Grip	NS	NS	0.01	0.01	0.01	--	--		NS	NS
4. Grip vs G-suit	NS	NS	NS	NS	NS	--	--		NS	0.05
5. Grip vs G-suit + Grip	NS	NS	0.01	0.01	0.01	--	--		NS	NS
6. G-suit vs G-suit + Grip	NS	NS	0.01	0.01	0.01	--	--		NS	0.05

a. NS = not significant.

b. df = 0 for these G levels (i.e. at 6G, one, and at 7G, no subjects completed all four conditions). Therefore not computed.

TABLE 19 MEAN HEART RATE IN BEATS PER MINUTE FOR EIGHT SUBJECTS +Gz ACCELERATION IN SLOW ONSET RUNS AT 1-7G. "FINAL" HEART RATE IS THAT RATE PRESENT AT THE SUBJECT'S TOLERANCE OR HIGHEST G LEVEL REACHED.

ACCELERATION "G"		CONDITION							
		CONTROL		GRIP		G-SUIT		G-SUIT + GRIP	
(Rest)	1	87	(8)	87	(8)	86	(8)	84	(7)
	2	97	(8)	103	(8)	96	(8)	100	(7)
	3	101	(8)	111	(8)	104	(8)	105	(7)
	4	98	(5)	118	(7)	100	(8)	104	(7)
	5	109	(3)	127	(6)	104	(6)	114	(7)
	6	132	(1)	130	(3)	115	(3)	126	(3)
	7	-----		157	(1)	143	(2)	136	(2)
	Final	112	(6)	141	(6)	118	(7)	120	(6)
(Recovery)	1	91	(7)	105	(8)	109	(7)	116	(7)

NOTE: () = numbers of subjects to reach a particular G level.

TABLE 20 ANALYSIS OF VARIANCE OF HEART RATE DURING SLOW ONSET RUNS AT THREE G.

SOURCE OF VARIANCE	DEGREE OF FREEDOM (df) ^{c.}	SUM OF SQUARES (S.S.)	MEAN SQUARE (M.S.)	F	LEVEL OF SIGNIFICANCE (P _≤)
a. CONDITION A (Conditions)	3	567	189	3.28	NS
b. CONDITION B (G Level)	0	0	0	0	
SUBJECTS	6	7075	1179	0	
INTERACTIONS					
A x B	0	0	0	0	
A x S	18	1036	57	0	
B x S	0	0	0	0	
A x B x S	0	0	0	0	
RESIDUAL					
TOTAL	27	8678			

- a. The four conditions were: control, grip, G suit and G suit + grip.
- b. Because of the fact that subject SOR tolerance varied (as expected), seven subjects got to 3G in all 4 conditions while only one subject got to 6G in all conditions. Because of the varying degrees of freedom (numbers of subjects), the main effects of condition and G level cannot be tested, and only effects of condition at 3G are tested.
- c. $df_{\text{error}} = 27 - 6 - 3 = 18$

TABLE 21 SIGNIFICANCE LEVELS (P=) OF A DUNCAN MULTIPLE RANGE TEST OF DIFFERENCES BETWEEN MEANS OF HEART RATE IN FOUR CONDITIONS AT 1, 2, 3, 4, 5, 6, AND 7G, AND TERMINAL (i.e. TOLERANCE ENDPOINT) AND RECOVERY PERIODS.

<u>CONDITIONS</u>	<u>"G"</u>							TERM.	REC.
	1	2	3	4	5	6	7		
1. Control vs Grip	NS ^{a.}	0.05	0.05	0.05	0.01	-- ^{b.}	-- ^{b.}		
2. Control vs G-suit	NS	NS	NS	NS	NS	--	--		
3. Control vs G-suit + Grip	NS	NS	NS	NS	0.05	--	--		
4. Grip vs G-suit	NS	0.05	NS	0.05	0.01	--	--		
5. Grip vs G-suit + Grip	NS	NS	NS	NS	0.01	--	--		
6. G-suit vs G-suit + Grip	NS	NS	NS	NS	0.01	--	--		

a. NS = not significant.

b. df = 0 for these G levels

TABLE 22 LINEAR REGRESSION OF HEART RATE (X) ON MEAN EYE LEVEL ARTERIAL BLOOD PRESSURE (Y) AT 1, 2, 3, 4, 5, AND FINAL (TOLERANCE) G LEVELS FOR EACH OF FOUR CONDITIONS: CONTROL, GRIP, G SUIT AND G SUIT + GRIP. THE VALUES USED ARE MEAN FOR THE TOTAL NUMBER OF SUBJECTS WHO REACHED EACH G LEVEL, i.e. N=8 AT 1G, N=3-5 AT 5G .

		CONTROL		GRIP		G SUIT		G SUIT + GRIP	
		<u>Y</u>	<u>X</u>	<u>Y</u>	<u>X</u>	<u>Y</u>	<u>X</u>	<u>Y</u>	<u>X</u>
(Rest)	1	74	87	73	87	72	86	77	84
	2	48	97	53	103	50	96	50	100
	3	30	101	42	111	41	104	51	103
	4	24	98	36	118	29	100	44	104
	5	--	--	27	127	22	104	32	114
	FINAL	15	112	14	141	16	118	25	120
REGRESSION EQUATION ($Y = mx + b$) AND CORRELATION COEFFICIENT (r)									
CONTROL		$Y = -2.339 x + 269$				$r = -.898$			
GRIP		$Y = -1.089 x + 166$				$r = -.996$			
G SUIT		$Y = -1.749 x + 216$				$r = -.894$			
G SUIT + GRIP		$Y = -1.437 x + 197$				$r = -.985$			

TABLE 23 MEAN ARTERIAL BLOOD PRESSURE (MM HG) AT EYE LEVEL IN FOUR SUBJECTS DURING A 90 SECOND 40% MAXIMUM VOLUNTARY HANDGRIP CONTRACTION WITH AN OCCLUSIVE (250 MM HG) CUFF APPLIED AT (A) THE ONSET OF GRIP AND (B) THE 5 SECONDS PRIOR TO END OF THE GRIP.

<u>A. CUFF AT ONSET OF GRIP</u>								
SUBJECT	TIME (SECONDS)							
	0	30	60	90	120	150	180	210
2	55	59	65	69	63	62	65	58
5	93	102	113	136	123	129	127	114
7	80	79	93	108	97	102	109	92
8	100	105	122	128	121	123	130	110
$\bar{x}$	82	86	98	110	101	104	108	94
SD	19.8	21.5	25.3	29.9	27.9	30.3	29.9	25.5
<u>B. CUFF AT END OF GRIP</u>								
2	72	79	70	80	74	71	78	73
5	77	81	87	104	90	87	79	73
7	80	78	88	91	82	86	91	79
8	90	90	97	100	92	92	93	93
$\bar{x}$	80	82	86	94	85	84	85	79
SD	7.6	5.4	11.2	10.7	8.2	9.0	7.8	9.0

TABLE 24 THE EFFECTS ON MEAN ARTERIAL BLOOD PRESSURE $\pm$ SEM OF G-SUIT AT THREE PRESSURES (80, 160, 240 MM HG), GRIP (50% MVC) AND G-SUIT PLUS GRIP IN COMBINATION.

	PRESSURE (MM HG)	NUMBER OF SUBJECTS	REST	30"	60"	90"	RECOVERY
G-Suit	80	2	78 $\pm$ 22	79 $\pm$ 10	80 $\pm$ 17	80 $\pm$ 18	73 $\pm$ 21
G-Suit	160	3	71 $\pm$.3	76 $\pm$ 4	69 $\pm$ 11	68 $\pm$ 2	57 $\pm$ 1
G-Suit	240	1	68	67	74	72	55
G-Suit	160	3	71 $\pm$.3	76 $\pm$ 4	70 $\pm$ 1	68 $\pm$ 2	57 $\pm$ 1
Grip		6	79 $\pm$ 2.5	86 $\pm$ 2.3	99 $\pm$ 4.4	105 $\pm$ 5.1	---
G-Suit (160) + Grip		3	74 $\pm$ 3	86 $\pm$ 4	89 $\pm$ 4	103 $\pm$ 7	65 $\pm$ 6

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