解説

+G_z 加速度の生体への影響

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Effects of Positive G_z Acceleration on Human Body

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1. Introduction

Historically, Henry Head reported in 1918 that a test pilot suffered greyout and blackout in a Sopwith triplane pulling +4.5 Gz (60). Since that time, the effect of acceleration on the human body has been of great concern. Due to increased aircraft performance during World War II, many studies about human acceleration effects have been performed. Recently developed high performance fighter aircraft such as the McDonnell Douglas F-15 and General Dynamics F-16 are capable of producing high sustained Gz (HSG). HSG is defined as exposures to +6 Gz and greater for periods longer than 15 seconds. These aircraft are capable of rapid +Gz onsets exceeding 6 G/sec due to their greatly improved thrust-to-weight ratios. In the near future, moreover, aircraft capable of sideward acceleration may come into the Air Force inventory, exposing the pilot to increased Gy acceleration.

Many techniques to raise the tolerance to high sustained +Gz stress have been developed and used operationally, for example, the anti-G suit, the straining maneuvers (M-1 or L-1), and change of body position. Unfortunately, these definitely will not be adequate for aircraft with higher performance characteristics than the F-16. Therefore, many techniques have been recently re-evaluated and new techniques are being developed to match human physiologic performance in the air combat maneuvering capability of new generation fighter aircraft. We will have to develop more reliable anti-G procedures. Moreover, in order to extract the maximum performance of the man-aircraft system, we may need to monitor the pilot's physiological

state during flight.

It is extremely important, therefore, to understand the physiologic effects of acceleration along with current and proposed anti-G techniques. This study reviews classification, terminology, physiologic responses to +Gz acceleration, current protective methods against +Gz acceleration, and lastly suggests several methods which will be available in the future.

2. Classification

2.1 Linear Acceleration

When the linear velocity of a body changes over time, the difference in velocity, divided by the time required for the moving body to make the change, gives its mean linear acceleration (a).

The formula $a = v_2 - v_1 / \Delta t$

where v_1 = initial velocity

v_2 = final velocity

and Δt = elapsed time

is used to calculate the mean linear acceleration, which is a vector quantity with magnitude and direction. Acceleration is thus the rate of change of velocity. Appropriate units for the magnitude of linear acceleration are usually feet per second squared (ft/sec²) or meters per second squared (m/sec²). Consider an aircraft which accelerates from a dead stop to a velocity of 200 ft/sec in 10 seconds; the mean linear acceleration is (200-0)/10, or 20 ft/sec². The instantaneous linear acceleration, $\ddot{s}$ or $\dot{v}$, is the second derivative of displacement or the first derivative of velocity, d^2s/dt^2 or dv/dt , respectively.

A very useful unit of linear acceleration is the g , which is equal to g_0 , the amount of acceleration exhibited by a freely falling body near the earth surface. One g is equal to 32.2 ft/sec² or 9.81

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m/sec². In the previous example, in which an aircraft underwent a mean linear acceleration of 20 ft/sec², one divides 20 ft/sec² by 32.2 ft/sec² per g, to obtain 0.62 g.

2.2 Radial or Centripetal Acceleration

The radial or centripetal acceleration acts along the line represented by the radius of the curve and is directed toward the center of curvature. Its effect is a continuous redirection of the linear velocity, in this case called tangential velocity. Examples of this centripetal acceleration occur when an aircraft pulls out of a dive, or engages an enemy in a circular path in aerial combat. The value of the centripetal acceleration, a_c , can be calculated if one knows the tangential velocity, vt , and the radius, r , of the curved path followed:

$$a_c = vt^2/r.$$

As an example, we can calculate the centripetal acceleration of an aircraft traveling at 1000 ft/sec and having a radius of turn of 5000 ft: $(1000)^2/5000$ gives a value of 200 ft/sec² for the centripetal acceleration. This value, when divided by 32.2, comes out to 6.2 g.

2.3 Angular Acceleration

The rate of change of angular velocity is angular acceleration, α . The mean angular acceleration is

$$\alpha = \omega_2 - \omega_1 / \Delta t$$

where ω_1 = initial angular velocity

ω_2 = final angular velocity

and Δt = time interval over which the angular velocity changes.

$\ddot{\theta}$, $\dot{\omega}$, $d^2\theta/dt^2$, and $d\omega/dt$ can all be used to symbolize instantaneous angular acceleration, the second derivative of angular displacement or the first derivative of angular velocity with respect to time. If a figure skater is spinning at 6 revolutions per second (2160 degrees/sec) and then takes 2 seconds to come to a complete stop, the rate of change of angular velocity is $(0 - 2160)/2$, or -1080 degree/sec², which is a mean angular deceleration of 1080 degrees/sec².

The other commonly used unit of angular acceleration is radian per second squared (rad/sec²).

3. Terminology

When the main interest is the effect of acceleration on man, the direction in which an acceleration or inertial force acts is described by the use of a three axis coordinate system (X, Y, and Z), in which the vertical (Z) axis is parallel to the long axis of the body. Considerable confusion can arise if a clear distinction is not made between the applied acceleration and the resulted inertial force as these, by definition, always act in diametrically opposite directions. Thus, a headward acceleration tends to displace tissues such as viscera and the eyes footward and the resultant force is termed positive G, +Gz. The standard aeromedical terminology for indicating the direction of accelerations and inertial forces acting on man is listed in Table 1 and illustrated in Figure 1.

4. Physiological Effects of +Gz Acceleration

4.1 Cardiovascular Effects

A. Blood Pressure Response

The physiologic effects of acceleration are primarily due to the increased hydrostatic pressures developed in the vascular system. Burton et al. (9) explain the effect of +Gz by the hydrostatic column effect in seated pilot at +1 Gz and at +6 Gz. The

Fig. 1 Inertial G forces in acceleration (Reproduced from Aviation Medicine, Vol. 1, P.217, Tri Med Books, London. 1978)

Table 1 Equivalent Terms for Directions of Linear Accelerations and Gravito-Inertial Forces

Acceleration	Gravito-inertial force	Related aircraft motions
+az	+Gz	Level flight
Headward	Footward	Coordinated turn
	Positive	Pull-up from dive
	Eyeballs-down	"Inside" maneuvers
-az	-Gz	Inverted flight
Footward	Headward	Push-over into dive
	Negative	"Outside" maneuvers
	Eyeballs-up	
+ax	+Gx	Increasing forward velocity
Forward	Backward	(e.g., application of after burner)
	Positive transverse	
	Chest-to-back	
	Supine	
	Eyeballs-in	
-ax	-Gx	Decreasing forward velocity
Backward	Forward	(e.g., application of speed brakes)
	Negative transverse	
	Back-to-chest	
	Prone	
	Eyeballs-out	
-ay	+Gy	Left slip
To right	To left	Left skid
	Left lateral	
	Eyeballs-left	
+ay	-Gy	Right slip
To left	To right	Right skid
	Right lateral	
	Eyeballs-right	

effective hydrostatic column height from head to eye level (30 cm at 1 Gz) will be elongated to 180 cm at 6 Gz (Figure 2). If the arterial pressure at heart level is 120 mmHg, the arterial pressure at eye level will be 98 mmHg at +1 Gz and -12 mmHg at +6 Gz theoretically. If the physical hydrostatic pressure influence could not be modified, then man would lose consciousness at +6 Gz. Of course, cerebral perfusion of related individuals during exposures to +Gz cannot be accurately predicted using this model because of the cardiovascular compensatory adjustments which occur approximately 6–10 sec after acceleration onset (28).

During the application of +Gz forces the increasing hydrostatic pressure produces decreasing arterial pressure at head level and blood pooling in the venous capacitance vessels below the heart. Krutz et al. (39) demonstrated the sequence of hemodynamic events during +Gz exposure, resulting in the rapid fall in eye-level blood pressure with the onset of acceleration (Figure 3). The decrease in blood pressure and circulatory blood volume initiates a series of reflex responses of the circulatory system. Stretch receptors in both the high-pressure and low-pressure system play an important role in the maintenance of functional homeostasis.

Fig. 2 Hydrostatic pressure and systolic arterial pressure of a man seated in the upright position at +1 Gz and +6 Gz. (From Burton et al. (9))

Fig. 3 Cardiovascular responses in an individual exposed to his +Gz tolerance level. Note that the fall in eye-level blood pressure ($\bar{P}_a$) and mean eye-level blood pressure ($\bar{P}_a$) is accompanied by a decrease in temporal artery blood flow velocity ($\dot{Q}_{ta}$). (From Krutz et al. (39))

Two areas of the high pressure system are equipped with stretch receptors: the carotid sinus and the aortic arch. When the arterial pressure falls, the diameter of these elastic vessels decreases resulting in the shortening of their walls. The inhibitory

discharge from these receptors decreases and the brain stem reticular formation is activated, which, by sympathetic outflow, produces vasoconstriction as well as an increased heart rate and force of contraction. Of these two high-pressure-system areas, the primary input is from the carotid sinus. The atrial stretch receptors perform a similar function on the low pressure side of the cardiovascular system during +Gz. Very small decreases in filling volume will lead to a sharp decrease in cardio-inhibitory atrial receptor afferent discharge rate. The fall in atrial pressure works in synergy with the high-pressure receptors during +Gz to increase heart rate and force of contraction. Moreover, the following three responses play a progressively more important role as the duration of the +Gz stress is increased. Sympathetic discharge of renal nerves promptly increases and subsequently increases aldosterone secretion resulting in fluid retention. In addition, vagal afferents to the hypothalamus increase the secretion of ADH, also leading to fluid retention. Finally, the decreased stretch of the atrial receptors mediates the sympathetic discharge which shifts renal blood flow from the outer cortex to the medulla, thus promoting sodium retention.

Fig. 4 The mean maximum heart rates from a group of nine men are compared at various +Gz levels of 45-s duration, with their mean pre-G heart rates. (From Burton et al. (9))

B. Heart Rate

One of the most consistent effects of acceleration is a change in heart rate. At low G levels, heart rate is known to be directly correlated with the level and duration of +Gz exposure (42, 49, 54, 61, 72). At high sustained G (HSG), however, a G vs. heart rate relationship is less discernible; for maximum heart rate appears to be obtained at relatively low HSG intensities; namely +7 Gz (Figure 4) (42, 49, 54).

Occasionally, during exposure to HSG, a decrease in heart rate occurs. This decrease was first noted by Shubrooks (54) who termed it "high G bradycardia." This may be attributable to a reflex baroreceptor response to the high arterial pressure resulting from the M-1 maneuver.

C. Arrhythmias

Cardiac arrhythmias frequently occur during exposure to HSG (Figure 5). Shubrooks et al. (42, 54) found only one of 14 men exposed to HSG did not have premature ventricular contractions (PVC). These premature beats tend to increase during the latter portion of the exposure and appear to be directly correlated with the inspiratory phase of the M-1. Also, it seems that PVC's occur with increased frequency at the higher HSG levels. Other ECG changes such as AV dissociation, ventricular tachycardia, and sino-atrial block, are reported (71, 73, 74, 75, 76).

Fig. 5 Recording (sternal electrodes) of five types of cardiac arrhythmias observed during and after exposure to HSG. (From Leverett et al. (42))

D. S-T and T changes

Because of ECG artifacts due to muscular tensing and electronic filtering, S-T segment and T-wave changes which occur during HSG are difficult to assess. However, a decrease in T-wave amplitude and some S-T segment depression of the ischemic

type has been observed during exposure to HSG (9, 15, 16, 57, 65). This has been attributed to a simple increase in sympathetic tone (16, 57), or to changes in heart position.

E. Heart Pathology

In the past 10 years, much time, effort and G exposure have been spent on investigations aimed directly or indirectly at determining the possible risk of cardiac pathology due to high-G exposure in man (5, 10, 20, 26, 27, 41, 46). Burton and Mackenzie (11) exposed six adult miniature swine, wearing inflated anti-G suits to a series of +5, 7, and 9 Gz runs of 45 seconds duration. Immediately after these runs, the pigs were autopsied. The endocardium of the left ventricles showed evidence of recent hemorrhage of varying severity. Histologically, heart hemorrhage was limited to the subendocardial area. In Burns' study, however, he suggests that the injury observed during +Gz exposures is the result of the emotional stress of handling, restraint, and the unfamiliar environment of the centrifuge and +Gz.

In the studies of serum enzyme changes, creatine phosphokinase (CPK), lactic acid dehydrogenase (LDH), and serum glutamic oxaloacetic transaminase (SGOT) levels from the coronary sinus blood were found to be elevated after HSG (13, 19). Most of the LDH elevation was attributed to skeletal muscle stress, but the cardiac isoenzyme #5 was also found to be elevated (20, 62).

In Laughlin's review (41) and Burns' study (5) of cardiac damage during +Gz stress, they conclude that the lesions in swine probably result from very high levels of both sympathetic adrenergic tone to the heart and circulating plasma catecholamines acting on the cardiac cells. And they also mention that +Gz exposure poses no significant risk for cardiac damage in human. To date, fortunately, no clinical findings have implicated +Gz as the causative agent in permanent cardiovascular dysfunction in man (26).

4.2 Retinal and Cerebral Effects

In addition to the reflex cardiovascular compen-

satory mechanisms, the cerebral circulation is protected by a fall in jugular venous pressure which helps maintain the arteriovenous pressure differential (36) and the decrease in cerebrospinal fluid pressure which reduces intracranial resistance to blood flow.

The hemodynamics of the retinal circulation are generally considered analogous to those of the cerebral circulation. However, during +Gz exposure, visual disturbances (greyout and blackout) occur before unconsciousness occurs. This is attributed to the 20 mmHg intraocular pressure which must be overcome for retinal perfusion to occur.

Leverett et al. (43) showed the gradual collapse of retinal circulation and the correlation between eye-level blood pressure changes and retinal circulation during +Gz (Figure 6).

Berkhout et al. (2) recorded the EEG under +4.5, 6.0, and 7.0 Gz exposure and found it to be altered during +Gz exposure. These EEG changes, however, were not pathological in nature and were noncumulative with repeated +Gz exposure. They concluded that human performance levels at HSG are not compromised by the physiology associated with CNS changes which occur at G. The only serious pathology attributed to high G which has been reported in the literature is in an abstract in which a +Gz exposure, in an uncontrolled aircraft, is related to some form of temporary brain damage, but symptoms of that disappeared within 6 months (50).

4.3 Respiratory Effects

The lung, being a semi-elastic structure which is supported primarily by its basilar surface, demonstrates a gradient of pressure from the apex to the base. Even at 1 Gz, the effect of gravity creates marked regional variations in both ventilation and perfusion. At increased G loads the ventilation/perfusion ratio (V/Q) is increased in the upper portion of the lung, whereas in the lower lung this ratio is decreased. As a result of the disturbance of the V/Q ratio during acceleration it was assumed by Gauer (24) and at a later date experimentally shown

Fig. 6 Changes in retinal circulation and eye-level blood pressure during blackout on the human centrifuge. (From Leverett et al. (43))

(35, 36) that a decrease of oxygen saturation takes place, indicating that respiratory gas exchange fails progressively.

The desaturation of arterial blood which occurs during +Gz was demonstrated by Barr (1) in 1962. He observed an arterial desaturation (SaO_2 81–95%) in man exposed to 4.5–5.0 Gz and suggested that the hypoxemia was the result of both physiologic and anatomic right-to-left shunting. Leverett et al. (42) examined the effect of exposure time at peak +8 Gz on PaO_2 and the effect of various levels of HSG on PaO_2 . They noticed that a rapid reduction in oxygen tension occurs during the first 20 seconds of exposure to +8 Gz. However, continued acceleration exposure for an additional 40 seconds caused little additional reduction in PaO_2 . These PaO_2 values are quite similar to those from Michaelson's study in regard to G effect (Figure 7).

From previous studies reviewed by Glaister (30), subjects breathing air did not show a significant fall in VC up to levels of +6 Gz. These findings suggested that exposure to levels of +7 to 9 Gz for 45 seconds combined with anti-G suit inflation is

Fig. 7 The effect of exposure time at +8 Gz on PaO_2 in subjects performing the M-1 Maneuver of while positive-pressure breathing. (From Burton et al. (9))

sufficient to produce regional compression atelectasis and/or airway closure without having to invoke

the effect of rapid absorption of oxygen distal to occluded airways. Of course, this atelectatic condition would be magnified at +Gz if 100% oxygen was breathed while wearing anti-G suit. It is generally agreed that to develop atelectasis exposures of short duration three factors are necessary: a. +Gz directed acceleration exposure, b. the subject must be wearing an inflated anti-G suit, and c. breathing 100% oxygen.

In summary, the major physiologic effects of +Gz on the pulmonary system are airway closure, right-to-left shunting, atelectasis, and altered distribution of ventilation and perfusion resulting in venous admixture and hypoxemia.

4.4 Human Gz Tolerance

A measure of man's capability at +Gz is usually determined while the subject is relaxed and is quantified by the G level and duration at which specific physiologic systems are significantly altered. Criteria used in quantifying acceleration tolerance commonly are of visual origin, i.e., various stage of light loss such as peripheral light loss, central light dimming, and central light loss.

In a recent study, Parkhurst et al. (49) reported human tolerance to HSG at slow onset rates, in which a mean relaxed +Gz tolerance was +3.7 Gz with range of +3.3 to 4.3 Gz and a mean straining Gz tolerance with anti-G suit of +8.8 Gz with a range of +7.5 to 9.0 Gz in 14 subjects. Of the total group (14 subjects) tested, 9 subjects tolerated the total planned exposure period of 9 Gz for 45 seconds without loss of vision. Edelberg et al. (21) also reported similar data previously.

In conclusion, current positive Gz tolerance capability is usually considered to be the followings for gradual onset rates (less than 1 G/sec):

Condition	Mean Gz Tolerance*	Range
Relaxed	3.7	3.3–4.3
+ M-1	+1.5	1.5–3.0
+ Proper Fitting Garment	+0.3	
+ Anti-G Suit & Valve	+1.2	1.0–2.0
Total :	6.7**	5.8–9.5

* Gz tolerance endpoints are peripheral light loss.

** Gz tolerance is not necessary additive since there is some evidence of synergistic action between M-1 and anti-G garments.

5. The Factors Influencing +Gz Tolerance

5.1 Rate of Onset

Human +Gz tolerance is greatly influenced by the rate of onset. Edelberg (21) compared human tolerance to slow and rapid onset acceleration. In his study of 32 subjects with anti-G suits who underwent a total of 80 centrifuge runs, average +Gz tolerance to gradual onset rates (0.07 G/sec for 70 seconds) was +5.6 Gz compared to +3.7 Gz for rapid onset rates (1 G/sec 15 seconds) in spite of the longer duration of the gradual onset rate exposure. Stoll (59) observed that human tolerance to positive G is determined by physiological end points under various onset rates. Her curves relate human acceleration tolerance to acceleration onset rates (Figure 8). As shown in Figure 8, the time to end point is shorter with higher onset rates. This is attributed to the fact that rapid onset rate acceleration does not allow for compensatory cardiovascular responses as do gradual onset rates.

Fig. 8 Human acceleration intensity/duration blackout tolerance curves compared with various acceleration onset rates. Note that acceleration-intensity tolerance is a function of both duration and onset rates. (From Stoll (59))

5.2 Motivation

Burton mentions in his review (9) that motivation is an important factor in tolerating a higher G level. Experienced high-G personnel who are highly motivated can rapidly learn how to increase their HSG tolerance; i.e., approximately 100 Tactical Air Command fighter pilots using anti-G suits have readily attained +8 to 8.5 Gz for 30 seconds after a brief oral and visual training session on the technique of straining maneuvers.

5.3 Blood Glucose Level

Experimentally, hypoglycemia has been clearly shown to reduce tolerance to positive Gz acceleration. Using enough insulin to lower the blood sugar to about 50% of the resting value Browne (3) observed a reduction of approximately +0.6 Gz during the brief period when hypoglycemia was maximal. Decreased Gz tolerance due to hypoglycemia can result from failure to eat before flying or from the body's over-reaction to eating high carbohydrate foods such as a "lunch" of a candy bar and a soft drink 1–2 hours before flying. Raichle et al. (51) reported such a case of a student pilot with a classical past history of functional hypoglycemia who lost consciousness during spins producing accelerative forces of approximately +3.5 Gz. His subsequent centrifuge evaluation revealed a 24% decrement in rapid onset and a 19% decrement in slow onset +Gz tolerance with dietary induced hypoglycemia.

5.4 Age and Clinical Parameters

Hull et al. (38) studied for the first time the relationship between +Gz tolerance and age in healthy men. In a study of 53 healthy U.S. Air Force aircrewmembers between the age of 25 and 55, there was a significant positive relationship between age and +Gz tolerance ($P < 0.05$ for both gradual and rapid onset run) (Figure 9). They concluded that increasing age probably confers some protection against the effects of unresisted G forces and certainly causes no decrement in G tolerance. In a later study, Whinnery (72) and Epperson et al. (22) also showed that the relaxed +Gz tolerance in

Fig. 9 Relationship between +Gz tolerance and age. Above, ROR G tolerance, $r = 0.27$, $P < 0.05$, $Y = 3.18 + 0.013X$, $SEE = 0.38$. Below, GOR G tolerance, $r = 0.31$, $P < 0.05$, $Y = 3.60 + 0.028X$, $SEE = 0.68$. (From Hull et al. (38))

healthy men were positively correlated with age for rapid onset acceleration and with age and weight for gradual onset acceleration. Certainly as age increases it is usual for weight, experience (flying hours), blood pressure, rigidity of blood vessels, and percent body fat to increase.

Overall, the high +Gz prototype subject would seem to be the older, shorter, heavier, more experienced (more flying hours and more fighter pilot experience) individual with a higher blood pressure, higher hematocrit, higher cholesterol and triglycerides, hypertensive response to tilt-table testing, and higher blood pressure response to maximum treadmill exercise testing. On the other hand, the low +Gz prototype subject was demonstrated to be a taller, thinner, (less flying hours and less fighter pilot experience), individual with low blood pressure, lower hematocrit, and more arrhythmias.

5.5 Other Factors Decreasing +Gz Tolerance

- (a) Chronic and acute hypotension.
- (b) Conditions leading to hypovolemia (25, 31, 32, 33, 40, 47, 48); i.e., dehydration, dietary restriction of sodium, heat stress, prolonged bed rest, and sunburn.
- (c) All types of hypoxia.
- (d) Environmental or self-imposed stress factors (60) such as fatigue, lack of sleep, smoking etc.
- (e) Any acute infectious process leading to a febrile condition (105), for example, colds and viral infections.
- (f) Alcohol ingestion also affects +Gz tolerance. The tracking performance at +Gz after alcohol ingestion was studied by Burton et al. (7). They found an overall reduction in task performance of 79% in subjects at +5Gz after having consumed 3 oz of 95% ethyl alcohol (Figure 10). Several other physical abnormalities such as varicose vein, hemorrhoids, hernia, high myopia, and glaucoma also decrease +Gz tolerance (28).

6. Current Methods to Increase +Gz Tolerance

6.1 Straining Maneuvers (M-1 and L-1)

The M-1 and L-1 maneuvers are straining techniques used by pilots to improve G tolerance. The M-1 maneuver, often referred to as the "grunt" maneuver since it approximates the physical effort required to lift a heavy weight, consists of pulling the head down between the shoulders, slowly and forcefully exhaling through a partially closed glottis, and simultaneously tensing all skeletal muscles (arms, legs, and abdomen). Pulling the head downward gives some degree of postural protection (shortening the vertical eye-to-heart distance); intrathoracic pressure is increased by strong muscular expiratory efforts against a partially closed glottis; and the contraction of abdominal and peripheral muscles raises the diaphragm and externally compresses capacitance vessels. For long-duration +Gz exposures, this maneuver must be repeated every 4–5 seconds. Wood et al. found the M-1 maneuver

Fig. 10 The relationship of percent reduction in task performance per percent blood alcohol concentration with acceleration exposure. (From Burton et al. (7))

resulted in a maintained increase in eye-level systemic arterial pressure (Psa) and a consistent increase in tolerance ranging from +0.4 to 5.2 Gz, with an average increase of +2.56 Gz (77, 78). When properly executed, the exhalation phase of the M-1 maneuver results in an intrathoracic pressure of 50 to 100 mmHg, which raises the arterial blood pressure at head level and thereby increases +Gz tolerance at least +1.5 Gz (74). Psa responses, which are fluctuating with breathing efforts, are similarly seen while performing both the M-1 and L-1 maneuvers as shown in Figure 11.

The L-1 maneuver is similar to the M-1 maneuver except that the crewmember forcefully attempts to exhale against a completely closed glottis while tensing all peripheral skeletal muscles. Proper execution of the L-1 maneuver is at least equal to the M-1 maneuver in increasing tolerance to +Gz (58).

6.2 Anti-G Suit

Extensive research has been conducted in designing mechanisms to externally support the cardiovascular system. Different techniques which have been developed and used vary from merely taping the lower part of the body and limbs, as the Japanese did in World War II, to complicated pulsating pneumatic systems.

In 1942, the U.S. Navy developed a pneumatic anti-G suit, supplying three gradients of air pressure—high, intermediate, and low—to the calves, ab-

Fig. 11 Eye-level blood pressure response while performing the M-1 and L-1 straining maneuvers at high sustained G. (From Burton et al. (9))

dominal area, and thighs, respectively. It was named the Gradient Pressure Suit, AAF Type G-1 suit. It afforded visual protection of approximately 1.5 Gz above the blackout threshold (25). It was standardized by the U.S. Army Air Force in 1943 and next year modified to single pressure and cutaway model called the G-3A, which is the prototype of the anti-G suit used at present.

The protective value of inflated G-suits against +Gz acceleration is well documented and has been frequently reviewed (34, 37, 45). Simply wearing a well-fitted, uninflated G-suit has been shown to increase +Gz tolerance by 0.2 to 0.3 Gz in rapid onset accelerations (12). In one study in which the G-suit increased relaxed +Gz tolerance by 1.2 G, it was found that the abdominal bladder alone accounted for over 50% (0.7 G) of the increase in relaxed +Gz tolerance (8). The uninflated suit accounted for 0.3 G increase and pressurization of the legs accounted for only 0.2 G increase in tolerance. Generally, the properly fitting anti-G suit gives about 1.5 Gz increased tolerance above normal relaxed values.

6.3 Physical Conditioning

Until recently, there was no established consensus on the influence of physical training to +Gz tolerance. It appears, however, that endurance training does not improve +Gz tolerance but weight

training does. Cooper et al. (17) showed no significant difference between a group of runners and a control group in their ability to tolerate positive Gz during either gradual or rapid onset centrifuge acceleration. A later study by Epperson et al. (23) gave similar data for these 2 groups but reported a significant ($P < 0.005$) increase in tolerance time to HSG in weight trainers. They suggested the reasons why weight trainers can tolerate a longer +Gz acceleration are as follows: individuals exposed to high-G environments must perform an M-1 or L-1 maneuver in order to resist the effects of +Gz. Since the straining maneuver is very hard physical activity, it by itself can be extremely fatiguing and is probably a major contributor to G fatigue. Weight-trained individuals can perform a straining maneuver more easily. They are, therefore, less fatigued by HSG. Epperson et al. concluded that a physical conditioning program of weight training significantly improves human +Gz tolerance.

6.4 Positive Pressure Breathing (PPB)

PPB has been reported in several studies to increase tolerance to HSG (52, 53, 56). The relative effectiveness of PPB as an anti-G technique has been compared to the M-1 maneuver at HSG (+3, 6, and 8 Gz for 60 seconds) while subjects were wearing a standard 5-bladder anti-G suit. PPB was found to be as effective as the M-1 maneuver in increasing +Gz

Fig. 12 Eye-level systemic arterial pressure (Psa) response during PPB and during M-1 maneuver at +8.0 Gz for 60 seconds. (From Shubrooks (56))

tolerance. Less fluctuation in arterial pressure occurred with PPB than with the M-1 maneuver which should decrease the risk of LOC. The majority of the subjects preferred PPB to the M-1, because of less fatigue associated with PPB. In later studies, Shubrooks et al. (55, 56) and Shaffstall et al. (52) have reported similar results. In Shubrooks' study of 5 subjects, the +Gz tolerance increased 0.3 to 1.5 Gz with PPB, equal to the M-1 in two subjects and greater than M-1 by 0.3 to more than 0.5 G in three. Measurement of Psa demonstrated the effectiveness of PPB in maintaining an elevation of Psa during +Gz (Figure 12). Obviously, PPB with ambient air offers equivalent protection at HSG to the M-1 with less subject fatigue. However, current pressure demand oxygen regulators can only deliver 100% oxygen in the pressure settings. Accordingly, the probability of an increased incidence of atelectasis associated with 100% oxygen presently prevents the use of PPB as an operational anti-G technique.

6.5 Postural Change and Tilt Back Seat

The simplest and most effective means of increasing +Gz tolerance is to change the position of the body's long axis relative to the inertial force. This reduces the height of the hydrostatic column of blood extending from the heart to the head. The shortening of the eye-to-heart distance can be obtained by backward tilting of the seat. As shown in Figure 13 the eye-to-heart distance progressively

Fig. 13 Decreased vertical heart-to-eye distances obtained by tilting backward. (From Burns (4))

decreases with backward tilt of the seat beyond 30 degrees. Interestingly various authors have found significant increases in tolerance limits with various seatback angles greater than 45 degrees from the vertical. Seat back angle of 0–30 degrees give no significant increase in tolerance values (14, 29, 44, 63, 70).

Crossley and Glaister (18) report that the relationship between seatback angle and G tolerance is a cosine function. The protection afforded by this position not only increases G tolerance, but also

FLOW CHART A - SMART G - SUIT

FLOW CHART B - SMART OXYGEN VALVE

decreases the heart rate, fatigue, stress, and work (M-1 effort decreased) (4), and increases comfort (6). Relaxed +Gz tolerance actually doubles as the seat back angle is increased from 13 degrees to 75 degrees (4). However, it is very difficult to apply this to the seat of actual aircraft, because the visual field in the cockpit will be extremely limited. The seat which is developed by the U.S. Navy to solve this vision restriction problem is an adjustable pelvis and leg elevating (PALE) seat (Figure 14). Von Beckh and Voge (64, 66, 67, 68, 69) reported that the mean G tolerance for all subjects with 75 degrees seat back angle with the PALE seat, with M-1 maneuver and anti-G suit, is 11.5 Gz, and several individual can tolerate 14 Gz for 45 seconds without PLL. They found that the PALE seat gives excellent G protection without vision restrictions. This seat could be used in advanced fighter aircraft in the future if the problems related to the escape system, the cockpit display and controls, and pilot acceptance are solved.

6.6 Future Integrated Anti-G System

This system will employ flight status sensors linked by computer which sends an appropriate signal to both the electronically controlled anti-G valve and oxygen regulator. Consequently, it will make the anti-G suit and oxygen regulator work more efficiently. For example, as shown in Flow Chart A, it starts with combat status or not. If it is No, it works at normal cruise inflation schedule although it goes to combat inflation schedule if Gz is more than +3.0 Gz. If it is Yes, the combat inflation schedule starts with pre-inflation and the anti-G suit is inflated through the electronically controlled valve according to the Gz. Meanwhile, as shown in Flow Chart B, the anti-G oxygen regulator system is also linked with the computer which properly controls the pressure and concentration of oxygen presented to the pilot as necessary. It automatically becomes a combat gas mixture and PPB mode under combat status or normal cruise flight over 1.5 Gz, and sending 6% carbon dioxide in oxygen with positive pressure, which is able to

PALE (PELVIS AND LEGS ELEVATING) SEAT

Fig. 14 Tilt-back seat compared with the PALE seat. (From von Beckh (67))

improve the Gz tolerance of pilot. Moreover, if an objective physiological signal which will be able to foresee pilot incapacitation is available, the computer controlled physiological monitoring system will send a warning signal to pilot and the integrated anti-G system, resulting in stronger G-protection. This signal acquisition must be unobtrusive and unencumbering to the pilot. At present, we don't have such physiological signals capable of foreseeing pilot incapacitation. We will, therefore, have to define those signals which reliably signify incapacitation. If we can get these systems operative, the G tolerance capability will be increased by +3.0 to 4.0 Gz allowing the pilots Gz tolerance to match the G capability of the new generation of fighter aircraft.

Bibliography

- 1) Barr, P. O.: Hypoxemia in man induced by prolonged acceleration, *Acta Physiol. Scand.*, **54**, 128-137 (1962)
- 2) Berkhout, J., McDonnell, R. D. & Leverett, S. D., Jr.: Changes in electroencephalogram spectra during repeated exposure to +Gz acceleration, USAF-AMRL-TR-72-123 (1973)

- 3) Browne, M. K.: Report No. 1044, Flying Personnel Research Committee, Air Ministry, London, 1958.
- 4) Burns, J. W.: Re-evaluation of a tilt-back seat as a means of increasing acceleration tolerance, *Aviat. Space Environ. Med.*, 46, 55-63 (1975)
- 5) Burns, J. W., Laughlin, M. H., Witt, W. M., Young, J. T. & Ellis, J. P., Jr.: Pathophysiologic effects of acceleration stress in the miniature swine, *Aviat. Space Environ. Med.*, 54, 881-893 (1983)
- 6) Burton, R. R., Iampietro, P. F. & Leverett, S. D., Jr.: Physiologic affects of seatback angles < 45 degrees (from the vertical) relative to G, *Aviat. Space Environ. Med.*, 46, 887-897 (1975)
- 7) Burton, R. R. & Jaggars, J. L.: Influence of ethyl alcohol ingestion on a target task during sustained +Gz centrifugation, *Aerospace Med.*, 45, 290-296 (1974)
- 8) Burton, R. R. & Krutz, R. W., Jr.: G tolerance and protection with anti-G suit concepts, *Aviat. Space Environ. Med.*, 46, 119-124 (1975)
- 9) Burton, R. R., Leverett, S. D., Jr. & Michaelson, E. D.: Man at high sustained +Gz acceleration: a review, *Aerospace Med.*, 45, 1115-1136 (1974)
- 10) Burton, R. R. & Mackenzie, W. F.: Cardiac pathology associated with high sustained +Gz: subendocardial hemorrhage, *Aviat. Space Environ. Med.*, 47, 711-717 (1976)
- 11) Burton, R. R. & Mackenzie, W. F.: Heart pathology associated with exposure to high sustained +Gz, *Aviat. Space Environ. Med.*, 46, 1251-1253 (1975)
- 12) Burton, R. R., Parkhurst, M. J. & Leverett, S. D., Jr.: +Gz protection afforded by standard and preacceleration inflations of the bladder and capstan type G suits, *Aerospace Med.*, 44, 488-494 (1973)
- 13) Burton, R. R.: Physiologic response to repeated high G simulated aerial combat maneuvers, Preprints, Aerospace Med. Assoc. Annual Scientific Meeting, 84-85 (1977)
- 14) Burton, R. R. & Shaffstall, R. M.: Human tolerance to aerial combat maneuvers, Preprints, Aerospace Med. Assoc. Annual Scientific Meeting, 56-57 (1978)
- 15) Chimoskey, J. E.: Coronary blood flow and electro-cardiogram during headward acceleration in unanesthetized dogs, *Aerospace Med.*, 41, 1028-1030 (1970)
- 16) Cohen, G. H. & Brown, W. K.: Changes in ECG contour during prolonged +Gz acceleration, *Aerospace Med.*, 40, 874-879 (1969)
- 17) Cooper, K. H. & Leverett, S. D., Jr.: Physical conditioning versus +Gz tolerance, *Aerospace Med.*, 37, 462-465 (1966)
- 18) Crossley, R. J. & Glaister, D. H.: Effect of posture on tolerance to positive (+Gz) acceleration, AGARD CP-82, Item 6 (1971)
- 19) Dowell, R. T. & Stone, H. L.: Specificity of heart biochemical changes resulting from +Gz acceleration, Preprints, Aerospace Med. Assoc. Annual Scientific Meeting, 79-80 (1978)
- 20) Dowell, R. T., Stone, H. L. & Sordahl, L. A.: Myocardial energy requirement and coronary blood flow response during +Gz acceleration, Preprints, Aerospace Med. Assoc. Annual Scientific Meeting, 154-155 (1977)
- 21) Edelberg, R., Henry, J. R., Maciolek, J. A., Salzman, E. W. & Zuidema, G. D.: Comparison of human tolerance to accelerations of slow and rapid onset, *J. Aviat. Med.*, 27, 482-489 (1956)
- 22) Epperson, W. L., Burton, R. R. & Bernauer, E. M.: The effects of physical conditioning on +Gz tolerance, Preprints, Aerospace Med. Assoc. Annual Scientific Meeting, 82-83 (1977)
- 23) Epperson, W. L., Burton, R. R. & Bernauer, E. M.: The influence of differential conditioning regimens on simulated aerial combat maneuvering tolerance, *Aviat. Space Environ. Med.*, 53, 1091-1097 (1982)
- 24) Gauer, O. H.: Die Atemmechanik unter Beschleunigung, *Luftfahrtmed.*, 2, 291-294 (1938). Also in Dept. of the Air Force, German Aviation Medicine, World War II. Vol. I. Respira-

- tion and Centrifuge Forces. p. 576. U.S. Government Printing Office, Wash., D.C., 1950, 9.
- 25) Gauer, O. H. & Zuidema, G. D.: Gravitational Stress in Aerospace Medicine, Little, Brown & Co., Boston, 1961.
 - 26) Gillingham, K. K.: Absence of high-G stress cardiopathy in a human centrifuge rider, SAM-TR-78-17, USAF School of Aerospace Medicine, Brooks AFB, TX, 1978.
 - 27) Gillingham, K. K. & Crump, P. P.: Changes in clinical cardiologic measurements associated with high +Gz stress, *Aviat. Space Environ. Med.*, 47, 726-733 (1976)
 - 28) Gillingham, K. K. & Krutz, R. W., Jr.: Aero-medical review: effects of the abnormal acceleratory environment of flight, SAM-TR-74-57 (1974)
 - 29) Gillingham, K. K. & McNaughton, G. B.: Visual field contraction during G stress at 13 degrees, 45 degrees, and 65 degrees seatback angles, *Aviat. Space Environ. Med.*, 48, 91-96 (1977)
 - 30) Glaister, D. H.: The effects of gravity and acceleration on the lung, AGARDograph 133, Technivision Services, U.K., 1970.
 - 31) Graybiel, A., Patterson, J. L. & Packard, J. M.: Sunburn as a cause of temporary lowering of blackout threshold in flyers, *J. Aviat. Med.*, 19, 270-278 (1948)
 - 32) Greenleaf, J. E., van Beaumont, W., Bernauer, E. M., Haines, R. F., Sandler, H., Staley, R. W., Young, H. L. & Yusken, J. W.: Effects of rehydration on +Gz tolerance after 14 days' bed rest. *Aerospace Med.*, 44, 715-722 (1973)
 - 33) Greenleaf, J. E., Brock, P. J., Haines, R. F., Rositano, S. A., Montgomery, L. D. & Keil, L. C.: Effect of hypovolemia, infusion, and oral rehydration on plasma electrolytes, ADH, renin activity, and +Gz tolerance, *Aviat. Space Environ. Med.*, 48, 693-700 (1977)
 - 34) Hallenbeck, G. A.: Design and use of anti-G suits and their activating valves in World War II, USAF Tech.
 - 35) Henry, J. P.: Studies of the physiology of negative acceleration, A. F. Tech. Rept., 5953, 59, USAF, Wright-Patterson AFB, Ohio., 1950.
 - 36) Henry, J. P., Gauer, O. H., Ketty, S. S. & Kramer, K.: Factors maintaining cerebral circulation during gravitational stress, *J. Clin. Invest.*, 30, 292-300 (1950)
 - 37) Howard, P.: The physiology of positive acceleration, In: Gillies, J. A. (Ed.), *A Textbook of Aviation Physiology*, Chapter 23, Pergamon Press, New York, 1965.
 - 38) Hull, D. H., Wolthuis, R. A., Gillingham, K. K. & Triebwasser, J. H.: Relaxed +Gz tolerance in healthy men: effect of age, *J. Appl. Physiol. Exercise Physiol.*, 45 (4), 626-629 (1978)
 - 39) Krutz, R. W., Jr., Rositano, S. A. & Mancini, R. E.: A comparison of techniques for measuring +Gz tolerance in man, *J. Appl. Physiol.*, 38 (6), 1143-1145 (1975)
 - 40) Lambert, E. H.: Comparison of the physiological effects of positive acceleration on a human centrifuge and in an airplane, *J. Aviat. Med.*, 20, 308-335 (1949)
 - 41) Laughlin, M. H.: An analysis of the risk of human cardiac damage during +Gz stress, A review, *Aviat. Space Environ. Med.*, 53, 423-431 (1982)
 - 42) Leverett, S. D., Jr., Burton, R. R., Crossley, R. J., Michaelson, E. D. & Shubrooks, S. J., Jr.: Human physiologic responses to high, sustained +Gz acceleration, USAFSAM-TR-73-21 (1973)
 - 43) Leverett, S. D., Jr. & Newsom, W.: Photographic observations of the human fundus oculi during +Gz blackout on the USAF School of Aerospace Medicine centrifuge, 19th International Astronautical Congress of Bio-Astronautics, Book 4, Pergamon Press, Oxford, 1971.
 - 44) Lindberg, E. F., Marshall, H. W., Sutterer, W. F., McGuire, T. F. & Wood, E. H.: Studies of cardiac output and circulatory pressures in human beings during forward acceleration, *Aerospace Med.*, 34, 81-91 (1962)
 - 45) Link, M. H. & Coleman, H. A.: Research and

- Development, Acceleration, Chapter IV, Medical Support of the Army Air Forces in World War II, Dept. of Air Force, Wash., D.C., 283-295, 1955.
- 46) MacKenzie, W. F., Burton, R. R. & Butcher, W. I.: Cardiac pathology associated with high sustained +Gz: II. Stress cardio-myopathy, *Aviat. Space Environ. Med.*, 47, 718-725 (1976)
 - 47) Martin, E. E. & Henry, J. P.: The effects of time and temperature upon tolerance to positive acceleration, *J. Aviat. Med.*, 22, 382-390 (1951) Sustained +Gz, Preprints, Aerospace Med. Assoc. Annual Scientific Meeting, 89-90 (1951)
 - 48) Nunneley, S. A. & Stribley, R. F.: Heat and dehydration effects on human acceleration responses, Preprints, Aerospace Med. Assoc. Annual Scientific Meeting, 67-68 (1978)
 - 49) Parkhurst, M. J., Leverett, S. D., Jr. & Shubrooks, S. J., Jr.: Human tolerance to high, sustained +Gz acceleration, *Aerospace Med.*, 43, 708-712 (1972)
 - 50) Phillips, P. B. & Zarriello, J. J.: Central nervous system injury from high radial G force, *Aerospace Med.*, 30, 847-851, 39, 1246-1258 (1959)
 - 51) Raichle, M. E. & King, W. H.: Functional hypoglycemia: a potential cause of unconsciousness in flight, *Aerospace Med.*, 43, 76-78 (1972)
 - 52) Shaffstall, R. M. & Burton, R. R.: Evaluation of assisted positive-pressure breathing on +Gz tolerance, *Aviat. Space Environ. Med.*, 50, 820-824 (1979)
 - 53) Shaffstall, R. M., Morgan, T. R. & Burton, R. R.: The effect of assisted positive pressure breathing on +Gz tolerance, Preprints, Aerospace Med. Assoc. Annual Scientific Meeting, 80-81 (1977)
 - 54) Shubrooks, S. J., Jr.: Changes in cardiac rhythm during sustained high levels of positive (+Gz) acceleration, *Aerospace Med.*, 43, 1200-1206 (1972)
 - 55) Shubrooks, S. J., Jr.: Positive pressure breathing as a +Gz protective maneuver, Preprints, Aerospace Med. Assoc. Annual Scientific Meeting, 94-95 (1971)
 - 56) Shubrooks, S. J., Jr.: Positive-pressure breathing as a protective technique during +Gz acceleration, *J. Appl. Physiol.*, 35, 294-298 (1973)
 - 57) Shubrooks, S. J., Jr., Burns, J. W. & Erickson, H. H.: Coronary hemodynamics during positive +Gz acceleration, *Aviat. Space Environ. Med.*, 46, 413-418 (1975)
 - 58) Shubrooks, S. J., Jr. & Leverett, S. D., Jr.: Effect of the valsalva maneuver on tolerance to +Gz acceleration, *J. Appl. Physiol.*, 34, 460-466 (1973)
 - 59) Stoll, A. M.: Human tolerance to positive G as determined by the physiological end points, Report No. NADC-MA-5508, 1955.
 - 60) USAF Flight Surgeon's Guide, AFR 161-18, 27 Dec. 1968.
 - 61) Vettes, B., Vieillefond, H. & Auffret, R.: Cardiovascular responses of man exposed to +Gz acceleration in a centrifuge, *Aviat. Space Environ. Med.*, 51, 375-378 (1980)
 - 62) Voge, V. M.: Acceleration forces on the human subject, *Aviat. Space Environ. Med.*, 51, 970-980 (1980)
 - 63) Voge, V. M.: Comparison of several G tolerance measuring methods at various seat back angles, *Aviat. Space Environ. Med.*, 49, 377-383 (1978)
 - 64) Voge, V. M.: Psycho-physiological assessment of acceleration induced changes in various seat configurations, *Aviat. Space Environ. Med.*, 48, 527-538 (1977)
 - 65) Voge, V. M. & von Beckh, H. J.: Psycho-physiological assessment of acceleration induced changes in human positioned in conventional and in P.A.L.E. seat configurations, Preprints, Aerospace Med. Assoc. Annual Scientific Meeting, 51-52 (1975)
 - 66) Voge, V. M., von Beckh, H. J. & Bowman, J. S.: Psycho-physiological and physio-chemical assessment of acceleration induced changes (Gx-Gz) in human positioned in various seat back angle configurations, AGARD conference pro-

- ceedings No. 189 (The pathophysiology of high sustained +Gz acceleration, limitation to air combat maneuvering and the use of centrifuges in performance training, A3-1 ~ A3-9 (1976)
- 67) von Beckh, H. J.: Biomedical considerations for the development of a P.A.L.E. (Pelvis And Legs Elevating) G-protective aircrew seat, Preprints, Aerospace Med. Assoc. Annual Scientific Meeting, 57-58 (1973)
- 68) von Beckh, H. J., Voge, V. M. & Bowman, J. S.: Centrifuge evaluation of the G-protective P.A.L.E. (Pelvis And Legs Elevating) seat concept, Preprints, Aerospace Med. Assoc. Annual Scientific Meeting, 49-50 (1975)
- 69) von Beckh, H. J., Voge, V. M. & Bowman, J. S.: Dynamic testing of various 15-G rated acceleration protective seat assemblies using the P.A.L.E. (Pelvis And Legs Elevating) position at onset rates of 3.5G/s, Preprints, Aerospace Med. Assoc. Annual Scientific Meeting, 31-32 (1976)
- 70) von Diringshofen, H.: "Long chair" position for fighter pilots, J. Aviat. Med., 26, 467-470 (1955)
- 71) Whinnery, J. E.: Acceleration induced A-V dissociation: hemodynamic consequence, Aviat. Space Environ. Med., 53, 432-434 (1982)
- 72) Whinnery, J. E.: +Gz tolerance correlation with clinical parameters, Aviat. Space Environ. Med., 50, 736-741 (1979)
- 73) Whinnery, J. E. & Gillingham, K. K.: Medical standard for experimental human use in acceleration stress research, Aviat. Space Environ. Med., 54, 241-245 (1983)
- 74) Whinnery, J. E. & Gondek, M. R.: Medical evaluation of G-sensitive aircrewman, Aviat. Space Environ. Med., 49, 1009-1013 (1978)
- 75) Whinnery, J. E., Laughlin, M. H. & Hickman, J. R., Jr.: Concurrent L.O.C. and sino-atrial block during +Gz stress, Aviat. Space Environ. Med., 50, 635-638 (1979)
- 76) Whinnery, J. E., Laughlin, M. H. & Uhl, G. S.: Coincidence LOC and ventricular tachycardia during +Gz stress, Aviat. Space Environ. Med., 51, 827-831 (1980)
- 77) Wood, E. H. & Hallenbeck, G. A.: Voluntary (self-protective) maneuvers which can be used to increase men's tolerance to positive acceleration (abstract), Federation Proc., 5:115 (1946)
- 78) Wood, E. H. & Lambert, E. H.: Some factors which influence the protection afforded by pneumatic anti-G suits, J. Aviat. Med., 23, 218-228 (1952)

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