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Relaxed Tolerance Following HSG (High Sustained $+G_z$)

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INTRODUCTION. Changes in relaxed $+G_Z$ tolerance impacts the support required from G protective measures and may affect the capability of the aircrew to reengage following exposure to HSG. During and post-HSG, simultaneous physiological events which could increase or decrease G-tolerance are activated. METHODS. This study exposed 6 male centrifuge subjects to a GOR (Gradual Onset Rate = 0.067 G/second) acceleration to their subjective G-tolerance limit. Following a rest period, the subjects were exposed to a $7+G_Z$ ROR (Rapid Onset Rate = 1.0 G/second) acceleration for 60 seconds. Immediately after the ROR the subjects were reexposed to the GOR and taken to their relaxed tolerance limit. RESULTS. The subjects relaxed tolerance on the first GOR (pre-HSG) was $5.4 +G_Z \pm 0.7$ ($X \pm SD$) and their second GOR tolerance (post-HSG) was 4.5 ± 0.6. Statistical significance via Student's T Test (2 tailed) was $p=0.065$, but 5 of 6 subjects had a reduction in post-HSG tolerance. Electrocardiogram and heart rate evaluations provided an indicator of the cardiovascular response. CONCLUSIONS. This study suggests that relaxed $+G_Z$ tolerance is lowered following a high G exposure. Such a post-HSG reduction in relaxed tolerance would place a greater burden on the pilot's G protective systems thereby limiting one's ability to fully utilize the capability of the aircraft.

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RELAXED TOLERANCE FOLLOWING HSG (HIGH SUSTAINED +G_Z)

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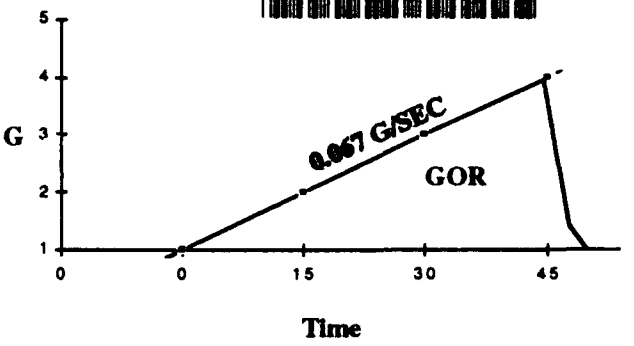
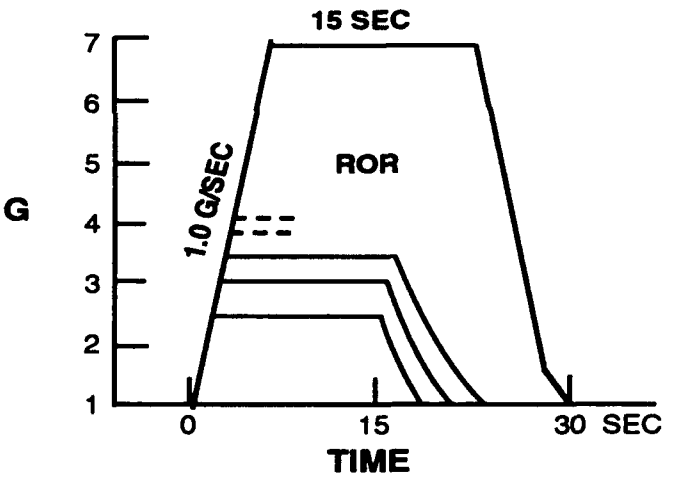
ABSTRACT *INTRODUCTION. Changes in relaxed +G_Z tolerance impacts the support required from G protective measures and may affect the capability of the aircrew to reengage following exposure to HSG. During and post-HSG, simultaneous physiological events which could increase or decrease G-tolerance are activated. METHODS. This study exposed 6 male centrifuge subjects to a GOR (Gradual Onset Rate = 0.067 G/second) acceleration to their subjective G-tolerance limit. Following a rest period, the subjects were exposed to a 7+G_Z ROR (Rapid Onset Rate = 1.0 G/second) acceleration for 60 seconds. Immediately after the ROR the subjects were reexposed to the GOR and taken to their relaxed tolerance limit. RESULTS. The subjects relaxed tolerance on the first GOR (pre-HSG) was 5.4 +G_Z ± 0.7 ($\bar{X}$ ± SD) and their second GOR tolerance (post-HSG) was 4.5 ± 0.6. Statistical significance via Student's T Test (2 tailed) was p=0.065, but 5 of 6 subjects had a reduction in post-HSG tolerance. Electrocardiogram and heart rate evaluations provided an indicator of the cardiovascular response. CONCLUSIONS. This study suggests that relaxed +G_Z tolerance is lowered following a high G exposure. Such a post-HSG reduction in relaxed tolerance would place a greater burden on the pilot's G protective systems thereby limiting one's ability to fully utilize the capability of the aircraft.*

INTRODUCTION Acceleration research has defined human tolerance to various types of acceleration profiles; however, changes in relaxed tolerance following HSG exposure have not been evaluated. Whinnery and Jackson conducted a study on the reproducibility of +G_Z tolerance testing using the * USAF School of Aerospace Medicine medical evaluation protocol (Figure 1). Their evaluation showed no differences in relaxed GOR (Gradual Onset Rate) tolerance between profiles 1 and 3; however, the intervening profile, a ROR (Rapid Onset Rate) series is conducted relaxed and rest time is allowed following each centrifuge run (4). The Whinnery and Jackson study showed that basic acceleration tolerance testing is repeatable when the subject is allowed to rest between centrifuge runs and is in a physiologically stable condition. If a rest period is not allowed and the subject is reexposed to a relaxed GOR acceleration immediately after a HSG exposure, a number of physiological factors may be present to change acceleration tolerance.

* The acceleration research program is now part of the Armstrong Laboratory, Crew Techology Division, Brooks AFB, TX.



GOR (Gradual Onset Rate) Profiles (1) and (3)
+G_Z tolerance for Profile (1) = 4.38
+G_Z Tolerance for Profile (3) = 4.37



ROR (Rapid Onset Rate) Profile (2)
+G_Z tolerance for Profile (2) = 3.51

Figure 1: Acceleration Profiles and +G_Z Tolerance from Whinnery and Jackson, "Reproducibility of +G_Z Tolerance Testing".

Opposing physiological principles may be involved in determining relaxed +G_Z tolerance following exposure to HSG. Table I is a summary listing of the cardiovascular response mechanisms that may be involved. Certainly, an exposure to 7+G_Z or higher will trigger the carotid and aortic baroreceptor reflexes to increase heart

rate, increase systemic arterial resistance and cause venoconstriction. The baroreceptor reflex is known to have a phase delay of approximately 5 - 7 seconds; thus, the reflex can be expected to lag behind G onset and offset for a certain time. Increased arterial CO_2 is also a known agent to increase $+G_Z$ tolerance (2). The proposed cardiovascular mechanism for this increase in tolerance is the CO_2 stimulated dilation of cerebral blood vessels. Very mild arterial hypoxia may have a similar affect on cerebral vessels. Interestingly, this same vasodilation principle may be in effect in the exercised muscle and reduce $+G_Z$ tolerance by causing peripheral pooling.

Table I: Cardiovascular Response Mechanisms Potentially Affecting Post-HSG Tolerance

INCREASE TOLERANCE	DECREASE TOLERANCE
Baroreceptor Reflex	Muscle Metabolites (pH, CO_2, Lactic Acid)
Increased Heart Rate	Increased Venous Compliance
Increased Arterial Resistance	Capillary Filtration
Venoconstriction	
Increased CO_2	
Hypoxia	

The effect of heat buildup, increased CO_2, decreased pH, hypoxia, lactic acid buildup and the presence of other metabolites from the working muscle will cause vasodilation and, particularly in the post-HSG period, may contribute to a reduction in $+G_Z$ tolerance. The hydrostatic effect of HSG will cause blood pooling in the great veins of the legs and may also cause increased vessel compliance further enlarging the vein. In addition, the increased hydrostatic pressure in the legs will tend to increase capillary filtration leaking blood plasma into the tissues. The anti-G suit will tend to resist the pooling and increased capillary filtration during HSG but, during the post-HSG period, the anti-G suit is not inflated. In the immediate post-HSG period, a rush of blood from the periphery may reach the heart and stimulate atrial receptors causing vasodilation and a slowed heart rate. If this reflex remains effective for 10 to 20 seconds, the impact would be to reduce $+G_Z$ tolerance. While not listed in Table I, the stress of HSG will cause an increase in circulating catecholamines. The general effect of these circulating hormones will be to increase blood pressure and potentially increase $+G_Z$ tolerance.

METHODS AND MATERIALS TABLE II contains a summary of the methods and materials used for this study. The subjects were instrumented with a 2-lead electrocardiograph, fitted with standard USAF CSU-13B/P anti-G suit and placed in the centrifuge gondola. The subjects were seated in an F-15 type ejection seat. The standard light bar was adjusted to the subjects height and maintained at approximately 76 cm (30 in.) from the subjects face. Subjects held a positive pressure centrifuge control switch (dead man switch) and were instructed to stop the centrifuge anytime they lost either 100% of their peripheral vision or 50% of their central vision i.e., 100% Peripheral Light Loss (PLL) and/or 50% Central Light Dim (CLD).

Table II: Methods and Materials

SUBJECTS: Six Experienced Male Centrifuge Subjects
- Age: 22-37 yrs.
- Height: Range 1.65-1.88 M (66-74 in.) Mean 1.72 M
- Weight: Range 61.4-89.4 KG (135-190 lbs.) Mean=76.1 KG

ARMSTRONG LABORATORY, CREW TECHNOLOGY DIVISION
- 6.1 M (20 Ft) CENTRIFUGE

EXPERIMENTAL CONDITIONS:
- CSU-13B/P Anti-G Suit
- ALAR Anti-G Suit Inflation Valve
- F-15 (13 degree) Seat Back Angle
- Standard Light Bar (Central Red Light, 50 degree Peripheral Green Light

Centrifuge profiles are shown in Figure 2. The GOR (1G per 15 sec.) centrifuge runs were made with the G-suit worn but not inflated. Subjects were instructed to stay relaxed during the GOR accelerations. The standard anti-G suit inflation valve and inflation rate was used for the ROR (1G/sec.) acceleration to 7 G. A 3 G warm up run was conducted prior to the first GOR. The subjects were allowed to rest as long as they wished between the first GOR and the $7+G_Z$ ROR (usually 2-5 min.). Following the 7G - 60 sec. ROR, the centrifuge was slowed to 1 G and the GOR was started as quickly as the operator could reengage the centrifuge controls. The total lapsed time from 7G to the start of the GOR

averaged 14 sec. For the ROR, the subjects were instructed to perform anti-G straining maneuvers with sufficient intensity to maintain vision. The subjects were reminded to relax again for the second GOR.

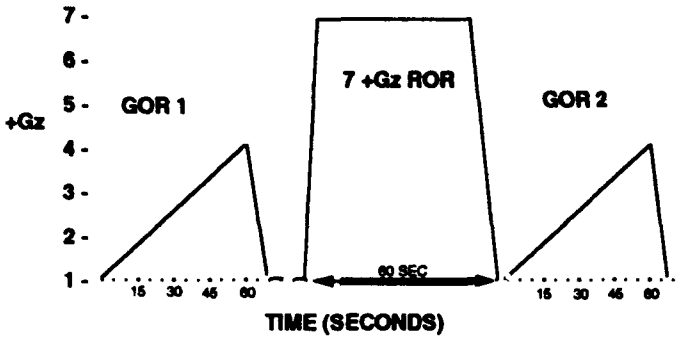


Figure 2: Acceleration Profiles

Resting heart rates were obtained after the subjects were seated in the gondola and resting comfortably. Heart rates were recorded throughout all centrifuge runs and for a 30-second post run period. All heart rate data shown on Figure 3 are 10-sec averages.

RESULTS A comparison of the relaxed GOR tolerances are shown in Table III. With one exception, all subjects had reduced G-tolerance on GOR 2 with the mean difference (GOR 2 - GOR 1) being - 0.9 G. While the mean difference in GOR tolerances was large for a relaxed tolerance study, the one subject who had an increase in GOR 2 tolerance caused the p value (two tailed T Test) to be statistically not significant (p=0.065). The mean $+G_Z$ tolerance level found for GOR 1 (Table III) was approximately $1+G_Z$ higher than Whinnery reported in his study. While this difference may be due in part to the large variation in the $+G_Z$ tolerances reported in both studies, a portion of the increase in the GOR 1 tolerances reported in Table III was due to the increase in $+G_Z$ tolerance caused by wearing a tight-fitting but uninflated anti-G suit (1).

Heart rate evaluations (Figure 3) provide an indication of the cardiovascular reactions to the acceleration stress. During GOR 1, the mean heart rate increased from the resting rate of 93 to 126 beats/min. The recovery back to near normal rates was quite rapid, less than 30 sec. The mean heart rates during the ROR to $7+G_Z$ very quickly reached high values, 175 beats/min.,

Table III: $+G_Z$ Tolerance (G Levels at Vision Loss Criteria)

SUBJECT	GOR 1	GOR 2	(GOR 2-GOR 1)	
1	5.9	4.7	-1.2	
2	6.3	4.2	-2.1	
3	4.6	5.0	+0.4	
4	5.8	5.3	-0.5	
5	4.5	4.2	-0.3	
6	5.4	3.6	-1.8	
$\bar{X} \pm SD$	5.4 ± 0.7	4.5 ± 0.6	-0.9 ± 0.9	(p=.065)

indicating the very high degree of stress and the intensity of the straining maneuver. The heart rate recovery following the $7+G_Z$ ROR and prior to the G level of GOR 2 reaching approximately $3+G_Z$ was rapid; however, as the G level of GOR 2 increased above $3+G_Z$ the mean heart rate started to increase. This increase in mean heart rate at increased G level would indicate that cardiovascular control mechanisms, probably baroreceptors, were reacting to the acceleration stress.

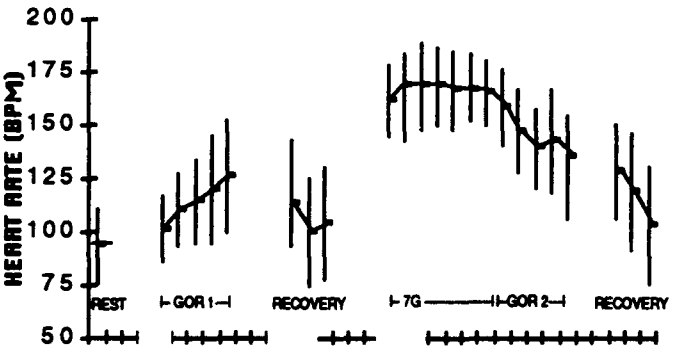


Figure 3: Heart Rates

CONCLUSIONS The results of this study did not prove that relaxed $+G_Z$ tolerance immediately following a HSG exposure is significantly reduced; however, a number of factors would seem to indicate that the post-HSG tolerances are lower. All but one of the subjects had a lower tolerance post-HSG. The single subject who did not show a decrease had a relatively low tolerance during the first GOR indicating that he may not have reached his light loss tolerance level during

this centrifuge run. An assessment of the cardiovascular control mechanisms tends to support the concept that post-HSG tolerance is reduced. During the first few seconds, post-HSG the relatively high arterial pressure sensed at the baroreceptors may initiate a parasympathetic response which will decrease G-tolerance. At HSG, the combination of the anti-G straining maneuver and the anti-G suit generally decreases the blood volume in the portions of the body covered by the anti-G suit. When the G level is returned to $1+G_Z$, the blood volumes return toward normal values (3). The decrease in blood volume in the dependent portions of the body during acceleration occurs even though the acceleration induced hydrostatic forces act in an opposite manner. If one assumes that a decrease in lower body blood volume equates to a decrease in blood flow, it is reasonable that metabolite buildup will occur in the large muscles of the lower body. This metabolite buildup will cause reactive hyperemia when the anti-G suit is deflated and blood flow is not restricted. This post-HSG shift in blood volume away from the heart would reduce $+G_Z$ tolerance. As mentioned in the results section, the heart rate (Figure 3) did increase near the end of the GOR 2 showing that a baroreceptor response was present, but in 5 of 6 subjects the post-HSG tolerance was still decreased.

From an operational prospective, the statistical significance of this study is not particularly important. Operationally, the important aspect of this study is that the cardiovascular control and recovery mechanisms seem to be causing or allowing a decrease in post-HSG tolerance. Aircrew need to understand that a period of potentially lowered G-tolerance will force them to strain harder to reach a given G level and will place greater demands on their G protective equipment.

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The voluntary informed consent of the subjects used in this research was obtained as required by AFR 169-3.

BIOGRAPHY: Robert M. Shaffstall, Col, USAF, Ret, is the Senior Acceleration Scientist for KRUG Life Sciences, San Antonio, TX. Prior to his retirement from the Air Force, Colonel Shaffstall's 26 year career included assignments in physiological training, and research and development in acceleration, life support equipment and chemical defense. In addition, he served as Chief of the Aeromedical/Human Factors Division, Air Force Operational Test and Evaluation Center and as Chairman of the Aerospace Physiology Department, USAF School of Aerospace Medicine. He has a BS Degree in zoology from Fort Hays Kansas State College, an MS in physiology from the University of Southern California and is board certified in aerospace physiology. He has authored over 25 scientific articles and abstracts, is a long time member of SAFE and is a Fellow of the Aerospace Medical Association.