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Loss of consciousness (LOC) during exposure to +Gz occurs in aircrew flying high-performance aircraft. This phenomenon is responsible for several USAF aircraft losses with accompanying loss of life. It has been recognized as a potential flying problem since 1938. Acceleration-induced LOC exhibits in 15- to 20-s periods of aircrew total incapacitation, amnesia, and clonic spasms. It can occur at relatively low G levels and without symptoms of loss of light (blackout). Operational anti-G equipment and methods are not completely effective in preventing LOC. A deficiency in the anti-G straining maneuver is considered to be the primary cause of G-induced LOC. Adequate G protection that would eliminate LOC as a serious problem for USAF high-performance aircraft operations must include supinating aircrew to a minimum seat back angle of 60° to 65°. Originator Supplied Key Words Include:

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SCIENCE NEWS NOTE

Operational G-Induced Loss of Consciousness: Something Old; Something New

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Loss of consciousness (LOC) during exposure to +G_z occurs in aircrew flying high-performance aircraft. This phenomenon is responsible for several USAF aircraft losses with accompanying loss of life. It has been recognized as a potential flying problem since 1938. Acceleration-induced LOC results in 15- to 20-s periods of aircrew total incapacitation, amnesia, and clonic spasms. It can occur at relatively low G levels and without symptoms of loss of light (blackout). Operational anti-G equipment and methods are not completely effective in preventing LOC. Considering the present status of G protection, a deficiency in the anti-G straining maneuver is considered to be the primary cause of G-induced LOC. This deficiency usually can be corrected with proper centrifuge training. Our present understanding of G protection suggests that the elimination of LOC as a serious problem for USAF high-performance aircraft operations must include the supination of aircrew to a minimum seat back angle of 60° to 65°.

TWO F-16 FATAL mishaps caused by G-induced loss of consciousness (LOC) occurred early in 1983. These tragedies have brought into focus the extreme dangers of flying aircraft capable of developing significant G forces. Accelerative forces of 3 to 4 G and greater sustained for more than a few seconds (3-5 s) can sufficiently reduce cerebral blood flow to produce severe brain tissue hypoxia with resulting LOC. Consequently, most tactical USAF and USN aircraft are potentially capable of rendering their aircrew unconscious. In a recent survey reported in "Flying

Safety" (36), a 12% rate of LOC had occurred in aircrew reporting incidents flying 12 different types of USAF aircraft (F-15, F-16, F-4, OV-10, T-33, A-37, etc.). LOC is not a specific high-G problem; some episodes had occurred at as low as 4 G. Interestingly, similar numbers of LOCs (30-40 each) had occurred in the F-15, F-16, and F-4, although aircraft losses due to LOC have been verified only in the F-16. Other types of USAF aircraft lost due to LOC have included the F-106, F-5, A-10, T-38, and T-37; and the Navy has lost two F-4s.

Loss of conscious is a function of both time at G and

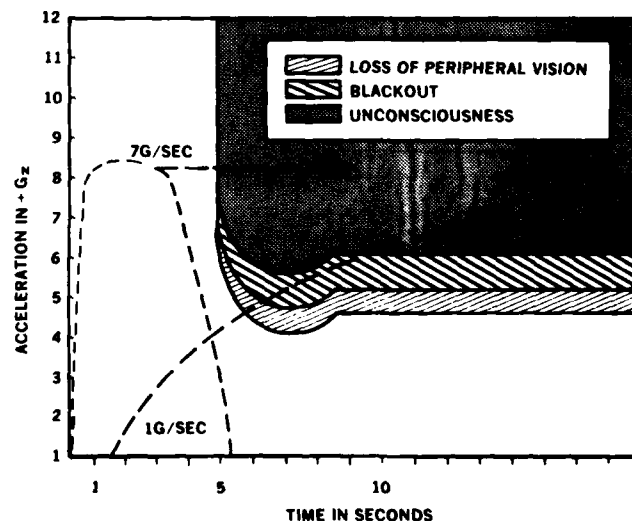


Fig. 1. Acceleration and time at maximum G required to produce visual symptoms and unconsciousness. Curves showing different rates of G onset demonstrate their importance for the occurrence of loss of peripheral vision, blackout, and unconsciousness—from White (47).

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level of G; so, theoretically, a relaxed "unprotected" individual could reach an extremely high G level and return to a sub-LOC-inducing G level within a very short period of time without losing consciousness. This observation was reported by Stoll (39), summarizing data obtained from various laboratories. Her report, which is considered a classic, generated several interesting G-tolerance curves including the information found in Fig. 1 that illustrates the time: G relationships relative to LOC (47). Of course, human G exposures necessary for these studies required a centrifuge with extremely rapid G-onset rates. The Naval Air Development Center (NADC) centrifuge, Warminster, PA, where most of these data were generated, is capable of $10 \text{ G} \cdot \text{s}^{-1}$ onset rates.

In 1979 our laboratory—Crew Technology Division, USAF School of Aerospace Medicine—reported on many episodes of LOC that had occurred on our centrifuge. These LOCs had been videotaped. Upon replaying the tapes we determined that the average duration of total incapacitation was 15.0 s, with a range of 9.0 to 20.5 s, in a population of 25 subjects (40). However, the problem is much greater than characterized by these few seconds of total incapacitation. The significant reduction in performance that both precedes and follows LOC is very important. Flying sophisticated fighter aircraft traveling at high speeds required high-performance aircrew capabilities. After an LOC occurrence, significant reductions in performance may well last for several minutes; the true recovery time has not yet been definitely determined. For this reason crewmembers of the Pacific Air Force (PACAF) are encouraged to terminate a mission after experiencing an in-flight G-induced LOC, return to home base, and report the occurrence to proper authorities. The aeromedical disposition of such cases is that they are reported as physiological incidents and not necessarily as conditions requiring medical disqualification (16).

Something Old

Loss of consciousness due to acceleration produced by aircraft is not new. The first suggestion that significant physiologic effects were produced by G while flying occurred in 1929 in propellered aircraft during the Schneider Trophy Air Race. During that race pilots reported loss of peripheral vision that neared total blackout while rounding pylons (28). Blackout, however, is not LOC. Recovery from total loss of vision is nearly immediate, with apparently no loss of performance except that which occurs during the 1- to 2-s blackout period and is due only to the loss of vision; that is, auditory and tactile sensations remain intact. To this day, the use of various degrees of loss of vision has remained a valuable physiologic indicator of G tolerance limits—a warning of a possible blackout or LOC. The validity of this visual cue, however, is directly proportional to its duration prior to a markedly decreased flow of blood to the brain and the occurrence of LOC. As the duration of the loss-of-vision phenomenon is reduced and becomes less than 1 s, its value as an operational LOC warning method is diminished. The duration of the visual-loss phenomenon

is rapidly reduced as the onset of G is increased to rates greater than $2 \text{ G} \cdot \text{s}^{-1}$ (Fig. 1). It is common for LOC to occur without any warning from loss of vision. The physiologic basis for blackout occurrence without LOC is the intraocular pressure causing retinal arteries to collapse at a higher eye-level blood pressure, and therefore lower G level, than that associated with LOC (33).

Since that 1929 air race, interest by the acceleration research laboratories has continued to focus primarily on the blackout phenomenon, with less concern directed toward LOC as a separate entity. Livingston (30) used the term "blackout" to identify the occurrence of either central light loss (blackout) or LOC, which he called blackout with fainting. He observed that this latter phenomenon "endured for 12–20 seconds." Similarly, Wood *et al.* (48) grouped data from blackouts with those from subjects suffering episodes of unconsciousness. However, Wood's group (48), as did Ham (28), warned that the greatest danger for the flyer from blackout or episodes of unconsciousness was the immediate loss of control of the aircraft. A distinction between the two types of incapacitation, thus warning pilots that unconsciousness was considerably more dangerous than simply an extended blackout, was never suggested. Two research articles (considered classics on the physiology of blackout) that suggest the importance of this phenomenon on the operational community are "Observation on the Fundus Oculi During Blackout," by Duane (17) and "Retinal Circulation in Man During Centrifuge Acceleration," by Newsom *et al.* (33). These detailed studies spanned nearly two decades; and it was not until the late seventies, 10 years after the Newsom article, that LOC was identified in the literature as a phenomenon *separate* from blackout (40), and its potential perils to pilots flying high-performance aircraft.

As soon as blackout and associated phenomena were identified, as being produced by high-performance aircraft, three methods were devised to improve the pilot's tolerance to accelerative forces—the pneumatic mechanical body-support system (anti-G suit/valve), the M-1 anti-G straining maneuver, and changing the position of the pilot's body relative to the G vector (basis for the reclining seat). Because the first two anti-G methods were extremely successful relative to aircraft performance capabilities, the reclined seat (the supined pilot) was never fully developed for operational use, mainly due to engineering complexities.

Something New

A new research direction regarding G tolerance was initiated in the early 1970s. Instead of directing physiologic research towards the blackout phenomenon and lower G levels associated with relaxed G tolerances, acceleration research began measuring and improving human tolerances to high sustained G (HSG) (34). These efforts were propelled by the addition of the F-15—the first USAF operational aircraft capable of routinely sustaining high G. This high-performance aircraft was developed without giving the aircrew any new G-protection systems. Thus, the pilots were protected with the same anti-G valve, anti-G suit,

and voluntary anti-G (M-1) straining maneuver used in earlier aircraft. All of these anti-G methods had been used without major modification since the 1940's, transitioning propellered aircraft and three generations of jet fighters.

Research in the 1970's in support of the F-15 was concerned with measuring physiologic stress (8,22), identifying potential high G-induced cardiac pathologies (6,7,31), evaluating medical conditions relative to G (25,43,46), and determining effects on aircrew performance and fatigue (9-11). Other high priority efforts included increasing tolerances to HSG by improving the M-1 through better training (29,34), improving physical conditioning (18), selecting aircrew for high G tolerances (25), and developing an improved anti-G valve (12) and anti-G suit (13,14,23). Simply, our directive was to increase the pilot's tolerance to the level of the F-15's maneuvering capability so that the aircraft could be used to its full potential.

The only new, practical anti-G system considered at that time for high-G operations was continuous 30- to 35- mmHg positive pressure breathing (PPB), both assisted (with chest counterpressure) and unassisted (15,37,38). Higher levels of assisted PPB (50 to 70 mm Hg) also have been considered, but only recently, and found to an effective anti-G method for higher G levels (5).

Renewed interest by our laboratory extended research (predictably, considering the effectiveness of the method) on the use of the reclining or tilted-back seat as an anti-G device (2,24). Those studies (2,24) and research by others (26,27,50) concluded that a seat reclined to 60° or more was the most effective of all anti-G devices and was the only system that "protected" the pilot against the hazard of LOC during HSG. This protection included increased G tolerances by significantly increasing light-loss levels, improved subject performance during high G, reduced fatigue and undesirable physiologic consequences, and even "prevented" possible cardiac pathologies which had been found in experimental animals subjected to HSG (6,7,31). At present, however, the probability of cardiac pathologies resulting from HSG exposures of man is considered remote (3,19,20). But improving aircrew tolerances to HSG and very high onset G was not an urgent issue until the LOC phenomenon was recognized as something far worse than a simple episode of a type of blackout (40,41).

Loss of consciousness had occurred occasionally in experimental subjects on the USAFSAM centrifuge for many years. An episode of LOC was not a rare event, even though we had a well-developed G-training program that increased G tolerances in our subjects at their own rate of learning ability—our subjects have always been able to tolerate G as well as USAF pilots of high-performance aircraft (11,34,42). Since our centrifuge did not have G-onset rates higher than 1.0 to 1.5 $G \cdot s^{-1}$, all of the LOCs occurred during HSG or during moderate rates of G onset.

We had observed the symptoms and time of total incapacitation of LOC on our television monitor but considered these occurrences with little concern in-house, and only in the context of a laboratory/centrifuge

problem—not one particularly relevant to USAF operations. The importance of episodes of LOC to our laboratory and our desire to limit the occurrences were understandable; we considered the LOC event to be usually associated with some training problem—such as the subject's inability to perform an adequate anti-G straining maneuver or failure to recognize the light-loss warning criteria—not an operational problem. We now know that improper techniques in performing the anti-G straining maneuver are also important aircrew problems.

Also, we did not fully realize that aircrew had other problems that could induce LOC which our subjects did not experience on the centrifuge. Specifically, these were: 1.) rapid onset of G (above 1 $G \cdot s^{-1}$); 2.) unpreparedness for the G environment, such as an unexpected G maneuver as experienced by the back-seater, or experiencing more or faster onset G than planned by the pilot because the F-16 G-limiter allows the pilot to make full stick deflection and obtain maximum G-onset rate without running the risk of over-G'ing the airframe; 3.) the slow response of the operational anti-G suit inflation system which exacerbates the preceding problem; 4.) numerous human factors or medical-type problems that may unexpectedly reduce G tolerance; 5.) return to flying high-performance aircraft after a significant period of not flying—the loss of G-tolerance adaptation, which is not at present completely understood; and 6.) failure of the anti-G suit inflation system usually because of inadvertent anti-G suit hose disconnection. Since USAF operational high-performance aircraft (F-16 and F-15) routinely operate at HSG with rapid onset of G, most of the above situations would cause LOC without a preceding light-loss phenomenon as experienced and expected by most aircrew members (Fig. 1).

G-induced LOC was identified as an entity different from a long blackout and studied in some detail in our laboratory during 1976-78. The ability to conduct this study—not an experiment per se but an analysis of observations made over several years—was the acquisition of videotaping capability on the centrifuge. Using video tape recordings, which had been saved to use as G-training aids, Whinnery *et al.* (40) made incapacitation analyses and identified LOC as a physiologic research area of considerable importance and concern to the operational community.

For the first time, the uncontrolled clonic body movements were documented and reported along with the fact that frequently the subject was unaware of having been unconscious (41). Disorientation and confusion followed the episode of unconsciousness, so the subject remained totally incapacitated (unable to control an aircraft) for a mean of 15 s; the longest period in one individual was 20.5 s. An aircraft traveling at 500 miles $\cdot h^{-1}$ could travel 2 miles in 15 s or lose 10,000 ft altitude if in a dive. Loss of consciousness, because of its operational significance and differences from blackout, must therefore be considered as a specific acceleration physiologic problem. Also, because of its detrimental effects on performance, if LOC is to be addressed in toto it must be considered as a distinct psychophysiologic effort in the laboratory.

Prevention is the primary issue to be considered in

any research plan focusing on LOC. Prevention of a hazardous event, however, can be provided only through protection. Just reducing the probability of occurrence is not protection, not if a significant probability still remains.

Blood supply to the brain is the critical physiologic concern relative to LOC. Blood supply to organs above the heart is a function of a driving force (arterial blood pressure) and an opposing force to which gravity contributes—the product of G and heart to eye/brain vertical distance. As the accelerative forces increase, the opposing force also increases until blood flow to the upper body ceases. The human body is not always protected from this problem even at 1 G (normal gravity); fainting is a common occurrence. High G is not an absolute requirement for LOC in military aviation. Review of U.S. Air Force Air Training Command physiologic incident reports revealed that over a 4-year period approximately 2 LOC episodes per month were being reported. The mean G-level for LOC was +3.8 Gz with a range from 2.0 to 6.5 Gz (T-37 and T-38 aircraft).

Current operational methods used to increase G tolerance only act upon the driving force and fail to consider the opposing forces which must be addressed if real "G protection" is to be accomplished. Theoretically, the only approaches available in protecting aircrew against LOC are to eliminate G (weightlessness) or reduce the heart-eye/brain column vertical distance. Since G cannot be eliminated, the arterial column must be shortened relative to the G vector. This reduction in vertical column length can be achieved only by supinating the aircrew. The abdominal bladder of the anti-G suit does prevent this arterial column from elongating during G onset, but probably does not significantly reduce the column length—the anti-G suit prevents the problem from becoming worse during G.

The idea to recline the aircrew member has been considered in the laboratory (2,15,24,26,27,49) and in aircraft design concepts. Because of numerous engineering complexities, however, the tilted seat has never been seriously considered for incorporation into operational aircraft. The F-16 has a 30° permanently back-slanted seat which begins to adopt this concept, but operationally significant G protection to reduce the incidence of LOC does not occur until at least a 45° angle is attained—with greater angles offering greater benefits (2,11,24,26). Laboratory and operational experiences have shown that the 30° seat of the F-16 will not prevent LOC, although some pilots have perceived it as very protective against G—possibly offering a false sense of security and potentially contributing to the F-16 LOC problem (11).

The seat back angle that would eliminate the LOC problem in operational high-performance aircraft would be (including aircraft angle of attack) not less than 75°, with anti-G suit support and a headrest angle of not greater than 45° (2,49,50). The headrest angle is important relative to G protection associated with various seat back angles—greater headrest angles significantly reduce G protection. On the other hand, a relatively large headrest angle is required for the pilot to see the instrument panel—the minimum is

considered to be not less than 45° (4). Seat back angles greater than 75° in fighter aircraft probably would not be practical; high angles of attack could result in effective back angles greater than 90°, resulting in an unacceptable -Gz component. Glaister and Lisher (26,27) reported excellent G protection at a level "which is in more accord with the performance of modern military aircraft," using a 65° seat back angle, inflated anti-G suit, and 35 mm Hg PPB unassisted.

Our laboratory plans to examine combinations of level of assisted PPB with various seat back angles as improved methods of G protection. It may be possible to develop an effective LOC prevention method with seat back angles less than the presently preferred optimum of 60 to 65° using some level of assisted PPB. A reduction in the seat back angle necessary to prevent LOC would improve the acceptability of this approach as an operational option.

Since the U.S. Air Force will continue to operate high-performance aircraft without the G protection of the reclining seat, G-induced LOC must be addressed relative to all aspects of this complex problem. At best, LOC can only be reduced, not eliminated, through the use of several approaches now or soon to be available to operational units. These methods to increase G tolerance, in lieu of offering G protection, include adequate G training (21) and frequent G exposures (both can be accomplished on a pilot training centrifuge), properly functioning and fitting and anti-G suit/valve systems¹, a muscle-strength physical conditioning program, appropriate medical standards, responsible social habits and diet, and a specifically designed aerobics fitness program.

The U.S. Air Force now realizes that G-induced LOC in aircraft is not an unusual event, nor does it necessarily have a medical basis in asymptomatic aircrew. All medical conditions investigated to date in asymptomatic medically evaluated aircrew have failed to show any predisposition to G-induced LOC. Most individuals with a demonstrated predisposition to G-induced LOC have been completely healthy, with an altered physiologic rather than an anatomic basis for their reduced G-tolerance or LOC (43,46). Our laboratory has found that certain individuals with markedly enhanced parasympathetic (vagal) tone, perhaps accentuated by a high degree of endurance training, were predisposed to marked cardiac-rate and rhythm irregularities and LOC (35). G-induced LOC is usually a physiological problem and, as such, could be treated in operations as a physiological incident. This approach has been instituted at PACAF and may well become a USAF regulation (1,16).

We do not know the total time of diminished pilot performance related to LOC. Cognitive processes that are required for flying sophisticated operational aircraft may require several minutes for complete recovery following an LOC incident. In addition to the length of time required for this recovery, methods to increase the recovery rate should be determined; e.g., PPB breathing

¹An advanced anti-G valve that will reduce the time required to inflate the anti-G suit is undergoing flight tests and could be operational by 1985 (12).

100% oxygen, increase in inhaled CO₂, and/or the use of auditory or tactile stimulations. These methods may also be effective in reducing or eliminating the amnesia that is frequently found after an episode of LOC.

Fig. 1 identifies an area of high G for short durations (1–2 s) that is symptom free—presumably a function of “aerobic energy stores” in the central nervous system. This “safety region” has not been completely studied although Stoll (39) identified the occurrence of “greyout” during a 2.5-s +15 Gz epoch. It may be possible to increase this energy store. Also, this area of “physiologic protection” from LOC should be more accurately determined as it related to the time necessary to apply the anti-G system. Benefits of G-suit inflation may not be improved by increasing G-suit inflation rates beyond a specific time limit; e.g., an anti-G suit that inflates within 1 s may be as effective in increasing G-tolerance as a suit that inflates in 0.25 s after the onset of G.

Unlike blackout, the effects of which are reversible within 2–3 s and without significant sequelae, LOC results from severe brain hypoxia, with occasional seizure-like (clonic) activity and an average total incapacitation time of 15 s. Potentially pathologic effects of repeated LOC must be considered, and it is questionable that human experimentation policies would permit deliberate production of LOC in studies involving humans as subjects. Consequently, much of the research into this LOC phenomenon will require the use of animals. An animal model from which reasonably sophisticated performance data can be obtained will have to be developed. However, acceleration studies involving human subjects in which LOC has occurred accidentally can be developed so that useful recovery performance data are obtainable.

The recent LOC survey by Pluta (36) found approximately equal total numbers (in parenthesis) of LOC occurrences in the F-15 (44), F-16 (34), and F-4 (42), yet fatal USAF mishaps attributed to confirmed LOC most commonly occur in the F-16. Several reasons could be advanced for this situation: F-16 differences in speed, maneuverability, mission requirements, and aircraft-handling characteristics. Also, LOC mishaps in the F-15 and F-4 may have occurred but escaped detection by the safety investigation boards, as increased awareness of G-induced LOC problems is a recent phenomenon. An additional concern is that the F-16 has not been an operational aircraft for very long, yet already the number of LOC incidents is near those of the F-4 and F-15 which have been in the operational inventory for many years. One observation about the F-16 which sets it apart from other aircraft is the comparative quietness of its cockpit. This quietness has been considered as a possible reason for mishaps due to pilot spatial disorientation (32). Loss of auditory stimulation during LOC episodes may prolong the unconsciousness to a point where pilot recovery of the aircraft is impossible. Certainly, the relationship between LOC recovery times and auditory stimulation should be investigated.

Since LOC will be a continuing problem, technology may become available that allows an aircraft to safely fly itself when the pilot is incapacitated. This technology

may be available now in the F-16 without verification that the pilot is incapacitated; e.g., the aircraft goes on automatic pilot once certain preprogrammed criteria are met or exceeded by the aircraft—regardless of the condition of the pilot. Useful criteria would be aircraft minimum altitudes, control stick input algorithms, etc. If the pilot's condition is to be considered, an automated system to detect LOC in the pilot will have to be developed. Several methods are being investigated in the laboratory which would detect LOC and may be adaptable for aircrew operational use. Detection methods to warn aircrew that LOC is imminent, either through cardiovascular or central nervous system changes, might be useful in developing preventive LOC systems. These physiologic changes usually occur, however, with very little time available for initiating LOC preventive measures. Regardless, this technology is several years in the future and of no benefit to the immediate problems of LOC.

The Crew Technology Division, USAF School of Aerospace Medicine, has developed a research program, Aircrew Sudden Physiologic Incapacitation Research Efforts (ASPIRE), which addresses several of the problem areas described in this article. We should have some answers, including a better definition of the LOC problems, in the near future.

In the meantime we must remember that G-induced LOC can occur in aircraft at relatively low G levels and at moderate levels of G onset, usually as the result of a poorly executed straining maneuver. Such a deficiency in the straining maneuver can be the result of insufficient training, physical and/or mental difficulties, and improper preparation for the G maneuver. Because inadequate preparation cannot always be avoided, G-induced LOC in aircrew of high-performance aircraft will continue to be a problem until aircrew are supinated to a minimum of 60° to 65° seat back angle. This angle plus the angle of attack would approximate 75°. Providing this type of G protection would eliminate LOC as a serious problem for USAF high-performance aircraft operations and should be considered for aircrew stations for future USAF advanced high-performance aircraft.

EPILOGUE

Since this article was written and cleared for publication, five additional Class A mishaps have occurred that were caused by G-induced LOC—three F-16, one F-5, and one A-10.

REFERENCES

1. Bode FR. Recommended change to AFR 160-43, 10 Nov 1983. Letter from PACAF/SG to HQ AFMSG/SGPA, 8 June 1984.
2. Burns JW. Re-evaluation of a tilt-back seat as a means of increasing acceleration tolerance. *Aviat. Space Environ. Med.* 1975; 46:55–63.
3. Burns JW, Laughlin MH, Witt WM, Young JT, Ellis JP Jr. Pathophysiologic effects of acceleration stress in the miniature swine. *Aviat. Space Environ. Med.* 1983; 54:881–93.
4. Burns JW, Whinnery JE. Significance of headrest geometry in +Gz protective seats. *Aviat. Space Environ. Med.* 1984; 55:122–7.
5. Burns JW, Balldin UI. +Gz protection with assisted positive-pressure breathing (PPB). Preprints of the Annual Scientific Meeting Washington DC: Aerospace Med. Assoc. 1983:36–7.
6. Burton RR, MacKenzie WF. Heart pathology associated with exposure to high sustained +Gz. *Aviat. Space Environ. Med.* 1975; 46:1251–3.

7. Burton RR, MacKenzie WF. Cardiac pathology associated with high sustained +Gz: I. Subendocardial hemorrhage. *Aviat. Space Environ. Med.* 1976; 47:711.
8. Burton RR, Storm WF, Johnson LW, Leverett SD Jr. Stress responses of pilots flying high-performance aircraft during aerial combat maneuvers. *Aviat. Space Environ. Med.* 1977; 48:301-7.
9. Burton RR. Human responses to repeated high-G simulated aerial combat maneuvers. *Aviat. Space Environ. Med.* 1980; 51:1185-92.
10. Burton RR, Shaffstall RM. Human tolerance to aerial combat maneuvers. *Aviat. Space Environ. Med.* 1980; 51:641-8.
11. Burton RR, Iampietro PF, Leverett SD Jr. Physiologic effects of seat back angles $<45^\circ$ (from the vertical) relative to G. *Aviat. Space Environ. Med.* 1975; 46:887-97.
12. Burton RR, Shaffstall RM, Jaggars JL. Development, test, and evaluation of an advanced anti-G valve for the F-15. *Aviat. Space Environ. Med.* 1980; 51:504-9.
13. Burton RR, Krutz RW Jr. G-tolerance and protection associated with anti-G suit concepts. *Aviat. Space Environ. Med.* 1975; 46:119-24.
14. Burton RR, Parkhurst MJ, Leverett SD Jr. +Gz protection afforded by standard and preacceleration inflations of the bladder and Capstan type G-suits. *Aerospace Med.* 1973; 44:488-94.
15. Burton RR, Leverett SD Jr, Michaelson ED. Man at high-sustained +Gz acceleration: a review. *Aerospace Med.* 1974; 45:1115-36.
16. DeHart RM. High "G" induced loss of consciousness (GLC). Letter from PACAF/SG to PACAF/OPS/SG, 29 May 1984.
17. Duane TD. Observations on the fundus oculi during blackout. *Arch. Ophthalmol.* 1954; 51:343-55.
18. Epperson WL, Burton RR, Bernauer EM. The influence of differential physical conditioning regimes on simulated aerial combat maneuvering tolerance. *Aviat. Space Environ. Med.* 1982; 53:1091-7.
19. Gillingham KK. Absence of high-G stress cardiopathy in a human centrifuge rider. USAF School of Aerospace Medicine, Brooks AFB, TX. 1978; SAM-TR-78-17.
20. Gillingham KK, Crump PP. Changes in clinical cardiologic measurements associated with high +Gz stress. *Aviat. Space Environ. Med.* 1976; 47:726-33.
21. Gillingham KK. Centrifuge training of USAF fighter pilots. *Aviat. Space Environ. Med.* 1984; 55:467.
22. Gillingham KK, Makalous DL, Tays MA. G stress on A-10 pilots during JAWS II exercises. *Aviat. Space Environ. Med.* 1982; 53:336-41.
23. Gillingham KK, Winter WR. Physiologic and anti-G suit performance data from YF-16 flight tests. *Aviat. Space Environ. Med.* 1976; 47:672-73.
24. Gillingham KK, McNaughton GB. Visual field contraction during G stress at 13° , 45° , and 65° seat back angles. *Aviat. Space Environ. Med.* 1977; 48:91-6.
25. Gillingham KK. G-tolerance standards for aircrew. Preprints of the Annual Scientific Meeting. Washington, DC: Aerospace Med. Assoc. 1978:77-8.
26. Glaister DH, Lisher BJ. Centrifuge assessment of a reclining seat. AGARD Proc. 1976; 189: A4-1 to A4-8.
27. Glaister DH, Lisher BJ. The effect of acceleration and seat back angle on psychomotor performance. Preprints of the Annual Scientific Meeting. Washington, DC: Aerospace Med. Assoc. 1978:58-9.
28. Ham GC. Effects of centrifugal acceleration on living organisms. *War Med.* 1943; 3:30-56.
29. Leverett SD Jr, Burton RR. The use of a fixed base simulator as a training device for high sustained or ACM (air combat maneuvering) +Gz stress. AGARD Proc. 1976; 189:A8-1 to A8-6.
30. Livingston PC. The problem of "blackout" in aviation (Amaurosis fugax). *Br. J. Surg.* 1938; 26:749-56.
31. MacKenzie WF, Burton RR, Butcher WI. Cardiac pathology associated with high sustained +Gz: II. Stress cardiomyopathy. *Aviat. Space Environ. Med.* 1976; 47:718-25.
32. McNaughton GB. Personal communication. HQ AFISC/SEL, 1984.
33. Newsom WA, Leverett SD Jr, Kirkland VE. Retinal circulation in man during centrifuge acceleration. *Trans. Am. Acad. Ophthalmol. Otolaryngol.* 1968; 72:39-49.
34. Parkhurst MJ, Leverett SD Jr, Shubrooks SJ Jr. Human tolerance to high sustained +Gz acceleration. *Aerospace Med.* 1972; 43:708-12.
35. Parnell MJ, Whinnery JE. The effects of long-term aerobic conditioning on tolerance to +Gz stress. Preprints of the Annual Scientific Meeting. Washington, DC: Aerospace Med. Assoc. 1982:22-3.
36. Pluta JC. LOC survey. *Flying Safety*, Jan. 1984; 25-8.
37. Shaffstall RM, Burton RR. Evaluation of assisted positive-pressure breathing on +Gz tolerance. *Aviat. Space Environ. Med.* 1979; 50:820-4.
38. Shubrooks SJ Jr. Positive-pressure breathing as a protective technique during +Gz acceleration. *J. Appl. Physiol.* 1973; 35:294-8.
39. Stoll AM. Human tolerance to positive G as determined by the physiological endpoints. *Aerospace Med.* 1956; 27:356-67.
40. Whinnery JE, Shaffstall RM. Incapacitation time for +Gz induced loss of consciousness. *Aviat. Space Environ. Med.* 1979; 50:83-5.
41. Whinnery JE, Shaffstall RM, Leverett SD Jr. Loss of consciousness during air combat maneuvering. *Aerospace Safety* 1978; 34:23-5.
42. Whinnery JE. G-tolerance enhancement: straining ability comparison of aircrewmen, nonaircrewmen, and trained centrifuge subjects. *Aviat. Space Environ. Med.* 1982; 53:232-4.
43. Whinnery JE. +z tolerance correlation with clinical parameter. *Aviat. Space Environ. Med.* 1979; 50:736-41.
44. Whinnery JE, Laughlin MH, Hickman JR. Concurrent loss of consciousness and sino-atrial block during +Gz stress. *Aviat. Space Environ. Med.* 1979; 50:635-9.
45. Whinnery JE, Laughlin MH. Coincident loss of consciousness and ventricular tachycardia during +Gz stress. *Aviat. Space Environ. Med.* 1980; 51:927-31.
46. Whinnery JE. Acceleration tolerance of asymptomatic aircrewmen with mitral valve prolapse. *Aviat. Space Environ. Med.* 1984; 55:467.
47. White WJ. Visual performance under gravitational stress; ch. 11. In: Gauer OH, Zuidema GD, ed. *Gravitational stress in aerospace medicine*. Boston: Little, Brown, 1961:72.
48. Wood EH, Lambert EH, Code CF. Do permanent effects result from repeated blackouts caused by positive acceleration? *Aerospace Med.* 1947; 47:471-82.
49. Von Beckh HJ. G-protective aircraft seats, with special consideration given to pelvis and legs elevating (PALE) seats. NADC-72262CS, 1972.
50. Von Beckh HJ, Voge VM, Bowman JS. Centrifuge evaluation of the G-protective PALE (pelvis and legs elevating) seat concept. Preprints of the Annual Scientific Meeting. Washington, DC: Aerospace Med. Assoc. 1975: 49-50.
51. Von Beckh HJ, Voge VM, Bowman JS. Dynamic testing of various 15-G rated acceleration protective seat assemblies using the PALE (pelvis and legs elevating) position at onset rates of 3.5 G/s. Preprints of the Annual Scientific Meeting. Washington, DC: Aerospace Med. Assoc. 1976: 31-2.

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