

An Overview of the Physiological Effects of Sustained High +Gz Forces on Human Being

K L Fong,*^{MBBS, Dip Av Med (UK), MS (Aerospace)}, S W Fan,**^{MBBS, Dip Av Med (UK)}

Abstract

In the search for the advantage in aerial combat, increasingly higher performance aircraft are being developed that impose ever greater acceleration force loads on the human pilot. The symptomatology associated with increasing +Gz acceleration forces, culminating in G-induced loss of consciousness caused by the cessation of cerebral blood circulation are described. The inability of the normal cardiovascular system mechanisms to provide adequate circulation to the brain and the eyes, and the resulting associated symptomatology and physiological changes are discussed.

The normal weight, hydrostatic pressure and physiological ventilation /perfusion gradients in the lungs are exaggerated under high +Gz forces resulting in increased pulmonary arterio-venous shunting. This causes impairment of circulatory oxygenation and may also result in acceleration inducedatelectasis. The effects of +Gz acceleration forces on the renal system and the limitations of the cervical musculature are also discussed.

This paper serves to describe the human physiological responses to increased +Gz acceleration forces in an attempt to provide a better understanding of the body's reaction to such demanding physical stressors.

Ann Acad Med Singapore 1997; 26:94-103

Key words: Acceleration, G, G-LOC, High-G, Incapacitation

Introduction

The evolution of high performance combat aircraft has resulted in ever increasing physiological stressors being imposed on the pilots of these vehicles. The performance capabilities of the present generation of fighter aircraft like the General Dynamics F-16 Fighting Falcon and the Dassault Mirage 2000 have reached and sometimes even exceeded the human physiological limits of unprotected tolerance to +Gz acceleration forces. This has led to occurrences of G-induced loss of consciousness (or G-LOC, as it is more commonly known as) in air forces throughout the world, and has given rise to the notion that the human pilot is now the limiting factor in the quest for higher flight performance in combat aircraft.¹ Future generations of high performance combat aircraft are expected to generate even greater acceleration stresses in flight.² This will necessitate the need for the enhancement of present G protection techniques and the development and use of new methods in order to maximize the potential of the pilot flying these new aircraft. Improvements in +Gz protection will also provide a greater margin of safety for the pilots flying in existing high performance aircraft.

This research aims to review the literature available regarding the physiological reactions of man in the high +Gz acceleration force environment and provides researchers with the necessary background information to enable them to assist in the development of better methods of protection against high sustained +Gz forces and to help aircrew to best prepare themselves for the rigours of exposure to these same physiological stressors.

Background

G-LOC has been described by Burton³ as “a state of altered perception wherein (one's) awareness of reality is absent as a result of sudden, critical reduction of cerebral blood circulation caused by increased G force”. G-LOC is not a phenomenon of recent origin, but was apparently first documented to have occurred in 1903 when Dr AP Thurston, Sir Hiram S Maxim's chief engineer, lapsed into unconsciousness at 6 +Gz while riding Maxim's first “captive flying machine” at the Crystal Palace park outside London, England.^{4,5}

In the 1914 book by Hamel and Turner,⁶ Alder described that “the use of abdominal belts is certainly a thing to be advised, because it keeps up the tone of the

* Head

Aviation Physiology and Training Branch

** Head

Environmental Physiology Section

Aeromedical Centre, Republic of Singapore Air Force

Address for Reprints: Dr Kenneth L Fong, 7 Padang Chancery, Singapore 307880

splanchnic areas and so guards against any sudden attack of fainting". Thus, G-LOC was already known to exist in the aviation environment of that period but it was Sir Henry Head who described the physiological experiences of increased +Gz stress of a Maj VH flying a Sopwith Triplane at 4.6 G in 1918.⁷

The first formal research into the problems of the physiological effects of acceleration forces experienced while flying was carried out by the French doctor, P Garsaux in 1918, using animal (dog) centrifuge studies.^{4,5} His improvised equipment did, however, preclude the conduct of comprehensive centrifuge-based research. Much of the research performed just after World War I was obtained by in-flight observations⁸ until the first human centrifuges were developed in the 1930s.

World War II led to much research into the problems of in-flight acceleration forces due to the great developments in aircraft performance, in the efforts to achieve air superiority among the warring nations. This resulted in much improved understanding of the physiology of high +Gz stress and led to the development of the Anti-G Suit and later, the M-1 Anti-G Straining Manoeuvre (or AGSM), as protective measures against G-LOC. The effectiveness of these measures coupled with the limited performance of the post-war fighters led to a reduced emphasis on +Gz research in the decades following World War II.^{9,10} It was only when new high performance fighters like the F-16 were developed in the 1970s did interest and concern about G-LOC resurface.

G-LOC epidemiological surveys have been carried out by many air forces in countries throughout the world. The United States Air Force (USAF) data compiled by Pluta¹¹ gave a 12% incidence rate of G-LOC occurring among the respondents. Similar results were obtained in surveys conducted by the United States Navy (USN) (12.23% to 13.24%),¹² the Royal Air Force (RAF) (19%),¹⁰ the Royal Netherlands Air Force (RNLAF) (15%),¹³ the Royal Canadian Air Force (RCAF), and the Royal Australian Air Force (RAAF).⁹

These statistics take on an even greater importance as persons who have experienced G-LOC often have no recollection of the episode.^{14,15} Glaister¹⁶ has reported that 50% of subjects experiencing G-LOC on the centrifuge do not remember having been unconscious. Based on this observation, an estimated 25% to 38% of pilots have experienced G-LOC at least once in their careers, illustrating the need to develop adequate methods to protect the pilot from high +Gz stress.

G-LOC has been reported to be a contributory factor in 18 Class A accidents (accidents causing more than US \$1 million worth of damage to the aircraft, or involving loss of life) in the USAF from the period between 1982 and

1990.¹⁷ These G-LOC accidents not only affected high performance aircraft but also occurred in older and relatively lower performing fighter types. USN and RAF surveys^{10,12} showed similar results. The development of new generations of even higher manoeuvrability combat aircraft will test the pilots' tolerance limits to +Gz forces to their absolute maximum.

Physics and Terminology of Acceleration Forces

Acceleration is a vector quantity and can be defined as the rate of change of velocity. This change can be in the form of the rate of straight-line movement (i.e. speed) or in the direction of movement, or a combination of both rate and direction. Accelerations can have positive (i.e. increasing) or negative (decreasing) values and are typically measured scientifically in meters per second squared (m/s^2). In the aviation industry, acceleration forces are more often expressed in terms of multiples of the acceleration force due to gravity (traditionally represented by the symbol "g" where $1g$ is 9.81 m/s^2). The value of an acceleration force, in comparison to the gravitational constant is thus a ratio and is dimensionless. It is expressed as the "G" value of the force and can be illustrated as follows:

$$G = \frac{\text{applied acceleration}}{g}$$

The magnitude of a 9 G acceleration force is thus: $9 \times 9.81 = 88.29 \text{ m/s}^2$

The present conventions in the terminology of describing the directions of G acceleration and inertial forces are illustrated in Table I. The reference to G forces without the use of any other descriptors refers to the positive Gz direction in common usage. Aircraft manoeuvres typically occur in this headwards (+Gz) direction during Air Combat Manoeuvres (or ACMs) and on performing evasive actions after dropping armaments on a ground target.

Acceleration forces can be described as linear, radial or angular. Linear acceleration occurs when there is a change in the speed of travel of an object, but not in the direction of movement. In the aviation environment, linear acceleration is commonly produced during aircraft take-offs and landings, and during afterburner

TABLE I: THREE-AXIS NOMENCLATURE FOR DESCRIBING ACCELERATION FORCE VECTORS ACTING ON MAN

Direction of acceleration travel	Common terminology
Forward	+Gx
Backward	-Gx
To the right	+Gy
To the left	-Gy
Headward	+Gz
Footward	-Gz

engagement during straight-line flight.

Radial acceleration occurs when there is a change in the direction of movement, without an associated change in the speed of the aircraft. These forces occur whenever the flight-path of an aircraft is changed and hence is present in almost all controlled aircraft manoeuvres (e.g. banked turns, pull-out from dives and loops). Modern aircraft like the F-16 are capable of sustaining 8 to 9 G of acceleration force during ACMs.

Angular acceleration occurs when there is change in both the speed and the direction of movement of an object (e.g. when the aircraft is speeding up or slowing down, while simultaneously in a turn).

The jolt is the rate of change of acceleration force and is most commonly described in terms of G change per second of time (or G/s). Combat aircraft can reach G-onset rates of up to 10 G/s transiently during in-flight manoeuvres.

Physiology of Increased +Gz Stress and G-induced Loss of Consciousness

Persons exposed to gradually increasing +Gz stress undergo subjective effects as illustrated in Table II. The sequence of symptoms listed is similar in most persons exposed to +Gz acceleration forces, however, the actual G levels at which the sensations are felt are extremely variable in different persons¹⁸ and may even change from day to day for a particular person.

The primary cause of G-LOC is due to the effects of the centrifugal +Gz forces acting on the cardiovascular system supplying blood to the brain. Cessation of blood

circulation to the brain results in loss of consciousness, due to hypoxia. Rossen et al¹⁹ showed that unconsciousness occurs between 6 to 6.5 seconds after the sudden occlusion of blood flow to the brain. The effects described in Table II result from increased weight together with the progressive reduction of blood circulation to the retina and the brain.^{14,16,20} These symptoms can be explained by the Hydrostatic Column Model of +Gz stress.

Hydrostatic Column Model

There are two main aspects to this model. The first component involves the actual physical difficulties of pumping blood to the brain against an increased gravitational force and the second with the problem of returning blood to the heart so that it may be recirculated to the central nervous system. These two main effects are illustrated in Figure 1.

The heart as a pump: The human heart functions as an organic pump, providing blood supply to all parts of the body. The heart's function is supervised by the various automatic feedback mechanisms found in the autonomic nervous system which are activated in times of vascular disequilibrium. The heart strives to maintain an average blood pressure of 120/80 mm of mercury (or a mean blood pressure of 100 mmHg) in order to maintain a relatively constant flow of blood throughout the circulatory system.

The eye level (and the base of the brain) is sited

TABLE II: SUBJECTIVE EFFECTS OF GRADUALLY INCREASING +Gz STRESS

Approximate +Gz level	Subjective effects
1	Ordinary sensations of the 1 g environment
2	Sensation of being pressed into the seat. Some heaviness of the limbs and head is felt. Movement is impaired.
3	Severely increased heaviness of body parts. Impossible to move or escape manually from aircraft.
3 to 4	Progressive dimming or greying of vision (grey-out).
3.5 to 4.5	Progressive loss of peripheral vision, dimming of central vision.
4.5 to 5.5	Complete loss of vision or "visual blackout". Hearing and mental function persists. Compensatory mechanisms may improve visual symptoms if G-level does not increase any further.
5 to 6 and higher levels	Loss of consciousness occurs. If rate of increase is quick enough, no visual warning symptoms may be felt.

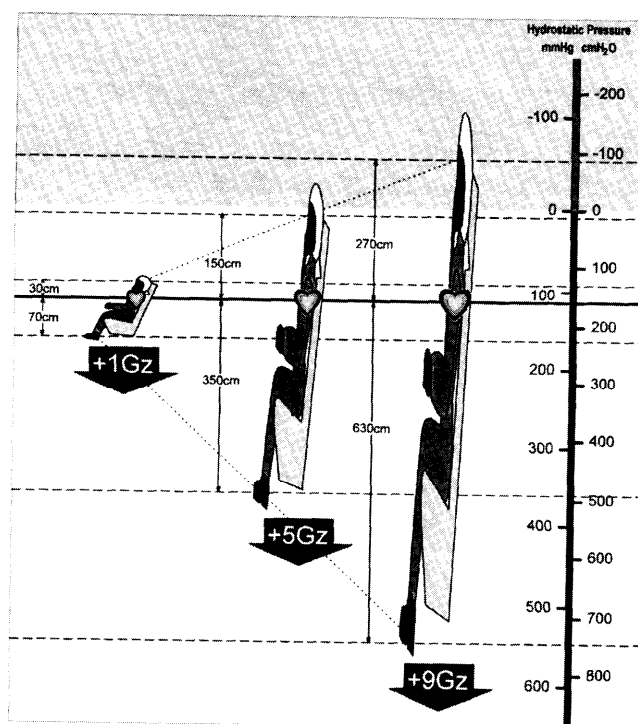


Fig. 1. Hydrostatic column effect on systolic arterial blood pressure at elevated +Gz levels.

approximately 30 cm vertically above the level of the heart.^{9,16,20} The hydrostatic pressure exerted by a column of blood of this height is equivalent to 22 mm Hg downward pressure in a 1g environment. For a person with an average blood pressure of 100 mmHg, the mean blood pressure available at the level of the eye would typically be in the order of $100 - 22 = 78$ mmHg and with each additional increase of one +G force applied in the +Gz axis, the blood pressure at the level of the eye would be decreased by a further 22 mm Hg. Thus at +4.5 Gz, the mean blood pressure at the brain would be $100 - 99 = 1$ mm Hg with peak pulses of pressure of 21 mm Hg occurring to provide some blood flow to the brain, without including the compensatory effects of the autonomic nervous system. Blood supply to the brain would theoretically be completely cut off if the G stress is increased further, and loss of consciousness would follow thereafter. The compensatory reflexes of the autonomic nervous system would serve to increase the blood pressure as soon as the circulation to the brain is perceived to be significantly reduced and attempt to provide more blood flow to the vital organs. The beneficial effect of these automatic responses however can easily be overcome by additional increases in the +Gz stress applied. Five to six G is the approximate average maximal tolerance of relaxed subjects exposed to +Gz stress.

In view of this hydrostatic column model for the effects of +Gz stress, it is expected that a person with a shorter heart to head distance would have better G tolerance than a tall, thin and lanky person and this view has been supported by the available research data collected.²¹⁻²³

Resupplying the heart: The heart serves as a pump for the circulation of blood to all areas of the body and needs to be kept constantly resupplied with blood in order that the blood can be recirculated to areas of need. The hydrostatic pressure effects of +Gz stress unfortunately causes dilatation of blood vessels below the level of the heart, and causes the pooling of blood in the dependent regions. Blood is sequestered from the central circulation and accumulates in regions like the splanchnic tissue, skeletal muscle (especially in the highly distensible blood vessels of the legs), and skin, thus reducing venous return to the heart. This in turn causes a decrease in cardiac output and blood pressure.

Specific System Effects

Cardiovascular system: The hydrostatic pressure effects discussed cause a rapid fall in cerebral arterial blood pressure (Fig. 2) with the onset of +Gz force being applied. This in turn causes the activation of compensatory responses from the cardiovascular system, trying to maintain circulation to the vital organs, especially the brain. These responses are thought to be mediated by a combination of cardiopulmonary volume receptors to-

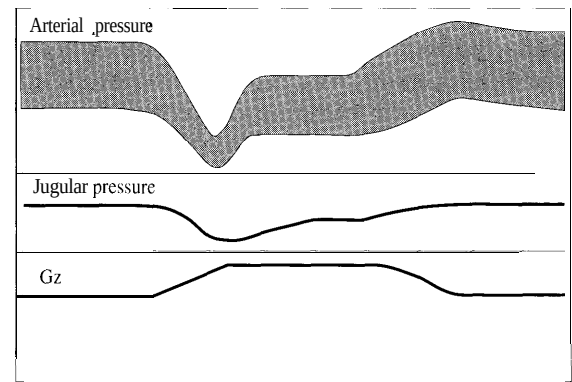


Fig. 2. Cerebral artery and jugular venous pressure changes during exposure to elevated +Gz forces.

gether with carotid and aortic baroreceptors²⁴⁻²⁶ and react within 5 to 10 seconds of exposure to +Gz forces.^{16,25} These result in reflex tachycardia, increased cardiac contractility, vasoconstriction and increased basal muscular tone. These benefits are extremely variable in individuals but have been observed to be able to enhance G tolerance by 0.5 to 3.5 G.²⁷ Rapid application of +Gz forces (more than 1G/s increase) do not allow the autonomic mechanisms enough time to react before maximal G tolerance is exceeded. As a result of this inability to respond with adequate speed, tolerance levels recorded during such rapid application of acceleration force can be decreased by up to 1.9 Gz.^{9,16,28}

From observations made of centrifuge subjects, progressive tachycardia is one of the most consistent cardiovascular clinical features of exposure to increasing +Gz stress.^{27,29} During gradual onset +Gz centrifuge runs (0.1G/s) the increase in heart rate matches that of the +Gz stress increase, but tends to lag behind the Gz level increase during rapid application of +Gz forces (1 G/s and 6 G/s onset rates).²⁷ Potentially, this "lag" is a possible physiologically limiting reaction in trying to adapt the human to aircraft capable of even higher G onset rates in the future.

Cardiac arrhythmias in otherwise asymptomatic centrifuge subjects^{20,29-31} are also known to commonly occur under conditions of high +Gz stress. These arrhythmias may be found acutely during exposure to high +Gz as well as after the acceleration load levels have been returned back to normal (1g condition). Atrial and ventricular ectopy are commonly found to occur during exposure to elevated levels of +Gz stress, while sinus arrhythmia, sinus bradycardia, sinus pauses, ectopic atrial rhythms, and atrio-ventricular dissociation are usually observed after returning to the 1g environment. Some form of atrial or ventricular ectopy or rhythm disturbance was observed in 88% of Whinnery's centrifuge subjects.³⁰ These arrhythmias were spontaneously self-limiting and were not associated with significant pathology or reduction in G tolerance in this study.

G-LOC is also not noted to be associated with any particular pattern of arrhythmia, however, it has been observed that persons with excessive vagal tone may be at particular risk of developing symptomatic arrhythmias or may suffer from more severe episodes of G-LOC.^{32,33} Haematological evaluations of centrifuge subjects have not shown any evidence of cardiac injury caused by or attributed to repeated Gz stress.³⁴

Increased hydrostatic pressure in the dependent areas of the body vasculature results in the formation of characteristic petechial haemorrhages in unsupported areas. These are commonly known as “G-measles” or “G-rash” and resolve spontaneously without requiring medical intervention. Less frequently, problems have been reported to occur from the rupture of larger vessels (e.g. causing a scrotal haematoma) or from acute venous engorgement (e.g. in varicocele) causing severe pain.²⁰ As such, it is important that potential pilots of high performance aircraft are adequately briefed and examined to look out for early signs of such potential problems and have them corrected before being further exposed to high +Gz forces.

Respiratory system: The application of +Gz acceleration force aggravates the existing ventilation and perfusion gradients that already exist in the lungs in a 1g environment. Blood circulation in the lung is affected by +Gz acceleration forces in a similar manner as that described by the hydrostatic column model for the circulatory system. In a 1g situation, pulmonary arterial and venous pressures are typically 15 mmHg and 0 mmHg respectively at the junction of the middle and lower thirds of the lung. Due to the physics of hydrostatic pressure, pulmonary arterial pressure is further reduced as blood travels towards the lung apex, such that even at 1g, the mean arterial pressure is reduced to zero at the apex of the lung.³⁵ Exposure to elevated +Gz forces increases this hydrostatic pressure gradient and causes a further aggravation of the ventilation / perfusion imbalance in the lung and results in an increasing proportion of the lung (from apex downwards) being unperfused as +Gz forces increase. This leads to an increase in the respiratory dead space in the lung.

The weight of the lung creates a pleural pressure gradient that increases progressively from the apex to the base of the lung.^{9,16,20,36} This pressure gradient results in the gradual size variation of the lung alveoli such that the air sacs at the apex of the lung are more distended than those at the base of the lung.⁹ Consequently, there is less ventilation of the apical air sacs per incremental increase in lung pressure as compared to the basal alveoli. Increased +Gz forces accentuate this situation as the pressure gradient down the lung rises proportionally with the increased acceleration forces. There is thus increased alveolar expansion at the lung apex while at the base of the lungs the alveoli approach their mini-

mum volumes, resulting in the closure of their associated terminal bronchioles and cessation of ventilation to the region. Blood flow to the lower lung regions will hence, not be involved in respiratory gaseous exchange and will constitute a right-to-left circulatory shunt and causes an overall lowering of arterial oxygen saturation within a few seconds of the onset of high +Gz forces.” This is first apparent in humans at approximately +3 Gz. At +5 Gz, up to 50% of the cardiac output may be shunted through the lungs in this manner³⁶ and can cause the saturation of oxyhaemoglobin to fall to about 85%. Use of 100% oxygen reduces the magnitude of the fall in saturation and delays the onset of desaturation but is associated with problems of basal atelectasis.

Reopening of the terminal bronchioles occurs with the return to a lower +Gz force environment, reducing the extent of the right-to-left shunt and results in the re-establishment of a more beneficial ventilation and gaseous exchange. However, repeated exposures to +Gz stress³⁷ cause arterial desaturation to recur at a faster rate and to a greater magnitude. This effect will be of significance in prolonged high-G air combat where repeated (and possibly protracted) exposure to elevated +Gz levels may occur.

Alveolar collapse also depends on the relative solubility of the gas inside the alveoli. Atmospheric air contains 80% nitrogen, which is not as easily absorbed by the lungs as is oxygen. When terminal bronchioles close as a result of increased +Gz stress, the oxygen trapped in the alveoli is absorbed rapidly, leaving behind the nitrogen to prevent the collapse of the alveolar sacs. When 100% oxygen is breathed however, the trapped oxygen is rapidly and completely absorbed, causing the total collapse of the air sacs. This results in the formation of acceleration induced atelectasis. Symptomatically, the atelectasis causes retrosternal discomfort, difficulty in taking deep breaths and results in an increased tendency to cough. Reduction of +Gz forces after atelectasis has occurred does not result in the spontaneous reinflation of the collapsed areas but relief can be obtained by making maximal inspirations or by coughing. Acceleration atelectasis increases with increased +Gz stress³⁸ and by the upward displacement of the diaphragm against the lung bases (e.g. due to the compression of the abdomen by the inflation of the abdominal bladder of an anti-G suit). Acceleration atelectasis can be minimised by ensuring that the breathing air contains at least 40% inert gas⁹ and by refraining from smoking as the condition has been found to be three and a half times worse in persons who smoke as compared to non-smokers.³⁹

In view of all these problems, Wood and Hoffman⁴⁰ have deemed that the air-filled lungs are a major limitation in the strive for ever increasing tolerance to high +Gz force environments. Replacement of the breathing

gas by a high oxygen content liquid⁴¹ appears to resolve the problems of right-to-left shunting and oxygen desaturation in experimental animals. Unfortunately, the multitude of problems of utilising liquid breathing devices preclude its use for protection enhancement against increasing +Gz stress at this time.⁴²

Vision: Gradually increasing exposure to +Gz forces decreases the blood supply pressure to the eyes and results in the “greying” of vision and constriction of the visual field. The eye has an intraocular pressure of approximately 20 mmHg for the maintenance of the spherical shape of the eyeball. Incoming blood to the eye must exceed this opposing back-pressure in order to supply blood to the interior of the eye. Increases in +Gz acceleration forces reduce the arterial blood pressure to the head and neck regions, depriving the eye of vital blood supplies resulting in the progressive “greying out” of the visual field as colour perception is lost when the cone cells lose their proper function due to the ischaemia. When circulating blood pressures fall to below 20 mmHg, closure of the intraocular vessels occurs, resulting symptomatically in loss of vision or “black-out”. At this level of +Gz stress, the brain is still perfused and is fully functional. The pilot is still awake and is able to think, hear and act consciously, but is unable to see. Thus, visual “blackout” occurs when vision is lost but cerebral function is not yet significantly impaired. In contrast, loss of consciousness (LOC) occurs when cerebral function is lost and the pilot is no longer in conscious control of himself. Due to the 20 mmHg of intraocular back pressure, the eyes can be considered to have a G-tolerance level that is approximately 1g less than that of the brain, although they are both sited at approximately the same vertical distance from the heart. A reduction in +Gz forces re-establishes blood flow to the intraocular vessels and results in the return of vision to the pilot. In order for cerebral function to be lost, it would be necessary for the acceleration force levels to be further increased (by approximately 1g) and this will result in loss of blood supply to the brain, causing G-LOC to occur. This 1g margin is very narrow and can be easily exceeded especially when high performance aircraft are able to fly at G onset rates of up to 6 to 10 G/s.

Blood supply to the retina is by means of an end-artery system formed by the central retinal artery which enters the eye at the optic disc and branches repeatedly to give rise to smaller size vessels as it passes forward and move peripherally. As subdivision of the artery occurs, the blood pressure within the branch vessels falls resulting in their earlier obliteration with increasing +Gz force. This results in the occurrence of “tunneling” of vision as the blood supply to the periphery of the retina is compromised more quickly than that around the macular area.

When +Gz increase occurs rapidly (e.g. during high-

G onset rates), collapse of the central artery occurs suddenly resulting in the simultaneous cessation of blood flow to all parts of the retina. With this sudden occlusion, the progressive symptoms of “greying out”, “tunneling of vision” followed by “blacking out” occur almost simultaneously resulting in only the symptom of “black out” to be experienced. The effects of increasing +Gz exposure on visual and neurological symptoms, and how they are modified by the baroreceptor reflexes are illustrated in Figure 3.

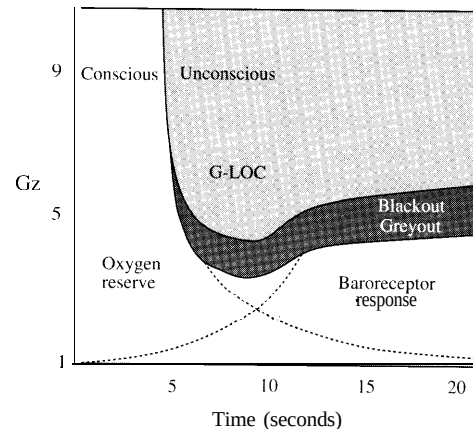


Fig. 3. The G-time tolerance curve.

Gradual and moderate rates of G onset result in progressive visual symptoms before LOC occurs. Transient (generally considered to be less than three to seven seconds total duration) exposure to rapid onset, high +Gz forces do not cause symptoms due to the availability of dissolved oxygen in the eyes and the central nervous system. Prolonged rapid onset +Gz forces can however result in loss of consciousness without prior visual warning symptomatology. These differing effects of onset rate on G tolerance are illustrated in Figure 4.

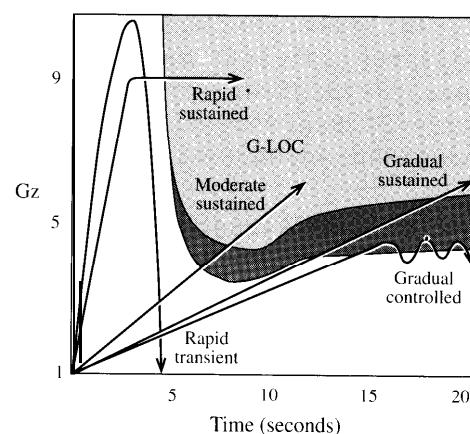


Fig. 4. Effect of G onset rate on G tolerance.

The recognition of these features of visual symptoms is important as pilots of combat aircraft make use of these symptoms to assess their G tolerance limits and manoeuvre their aircraft accordingly, so as to fly at their physiological maxima whenever necessary. Aircraft that are capable of flying sustained high G, rapid onset rate manoeuvres however do not give their pilots the opportunity to use these visual symptoms as a measure of their tolerance limits and these pilots may easily exceed their tolerance capabilities and lapse into G-LOC. Education regarding the differences in the human physiological response to high and low-G onset rates is thus essential to reduce the risks of G-LOC occurring.

Central nervous system: It has already been discussed that the brain can be considered to be at the same vertical height above the heart as the eyes and that the intraocular back-pressure accounts for a part of the reason as to why vision is affected by increasing +Gz forces before the brain is similarly afflicted. Other factors also contribute to the maintenance of blood circulation to the brain.

The brain and the cerebral vessels are enveloped by cerebrospinal fluid (CSF) and contained in the rigid bony skull. In response to increased +Gz forces, the effects of the hydrostatic column model have been postulated to affect the CSF to the same extent as the blood in the cerebral circulation. As a result of this effect, the CSF pressure falls in parallel to that experienced by the cerebral arterial blood pressure^{9,16} and helps to maintain the patency of the cerebral vessels, thus increasing cerebral circulation.

The venous blood column in the neck causes a siphon effect as it flows away from the head and this helps to maintain an arterio-venous pressure differential and hence supports blood circulation to the brain.⁴³ This effect is not large enough to fully compensate for the fall in arterial pressure as +Gz levels increase but it does help to provide up to -50 mmHg suction pressure at 4 to 5 +Gz. Further increases of +Gz force eventually cause so much lowering of the pressure in the veins that disruption of the siphon effect will ultimately occur, halting blood supply to the brain. Vasodilatation of the cerebral vasculature also occurs in response to the local hypoxia that would occur with a fall in the circulating blood pressure,⁴⁴ thus helping to maintain blood supply to the brain.

All of these mechanisms cannot ultimately compensate for large increases in +Gz force, and if no other techniques are applied, blood flow to the brain will be compromised and G-LOC will occur after a short latent period. This latent period (or central nervous system functional buffer period) has been quoted to last from 3 to 7 seconds^{16,45} and represents the time taken by the cerebral tissue to completely utilise the dissolved oxygen in the CSF bathing the brain.

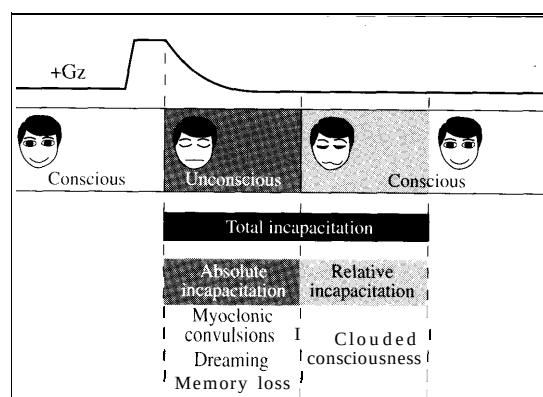
Features of G-LOC: G-LOC results in a period of incapacitation during which a person is not able to perform intelligent, purposeful actions. This period can vary considerably from 10 to 64 seconds (mean value of approximately 31 seconds)⁴⁶ and can be further subdivided into a period of absolute incapacitation (where the subject is unconscious) followed by a period of relative incapacitation (where the subject is awake but is confused and is not in full control of himself nor is he fully aware of his surrounding situation).

The period of absolute incapacitation has been found to last for an average of 15 seconds in human centrifuge studies and can also be subclassified into two types depending on the presence or absence of specific symptoms during the LOC episode. Type I LOC episodes are shorter and are uneventful while type II LOC episodes can be considered to be “deeper” and typically last longer⁴⁶ and have been associated with convulsive movements and sometimes vivid dream states. Myoclonic contractions have been noted to occur in 70% of episodes of LOC⁴⁷ and tend to occur in the last four seconds of the absolute incapacitation period. They have also been known to reflect the events occurring in a subject’s dream-state.⁴⁵ The exact causative mechanisms of these events have yet to be established.⁴⁵ Further research in this area may have potential important applications in enhancing G-LOC recovery.

The period of relative incapacitation follows that of the period of absolute incapacitation and also lasts for approximately 15 seconds. The duration of this period is more variable and is dependent on the G-LOC experience level of the person.^{48,49} G-LOC experienced subjects have been shown to be able to reduce their period of relative incapacitation by up to 8.5 seconds when they are re-exposed to G-LOC.⁴⁸ The recovery to consciousness may also be reduced by subjecting the unconscious subject to familiar warning signals (e.g. master caution warning buzzer). Auditory stimuli appear to be more effective than visual devices for this function, however further research is required to determine the optimum warning mechanism that would best rouse a pilot from G-LOC^{46,49} and in so doing, may provide a greater margin of safety for the pilot. The periods of G-LOC are illustrated in Figure 5.

G-onset and offset rates are also known to affect the period of incapacitation of a person experiencing G-LOC. Faster recovery has been noted in persons who are made unconscious by rapidly applied +Gz forces (in contrast to gradually increasing +Gz forces).^{9,45,47,49} G-LOCed subjects also recover faster if they are subjected to faster centrifuge deceleration rates.⁵⁰

Temporary electroencephalographic changes have been noted when a person experiences G-LOC. Commonly this takes the form of a shift from a normal higher frequency range of between 8 to 20 Hz (alpha-beta



Time periods associated with G-induced loss of consciousness.

range) to a lower 3 to 7 Hz (delta-theta range)“ during unconsciousness. These patterns persist until shortly before the return of consciousness.⁹ Anecdotally, repeated exposure to G-LOC has not resulted in any known permanent sequelae, however no definitive controlled studies have been performed using modern neurological techniques as yet.⁵²

Other effects: Exposure to increased +Gz forces is known to have little effect on the endocrine system, except for causing the elevation of serum cortisol levels. These levels have been found to peak approximately 20 minutes after acceleration stress.⁵³

Proteinuria and the passage of hyaline casts in the urine⁵⁴ have been demonstrated to occur after prolonged exposure to high-G simulated air combat manoeuvre types of centrifuge profiles. Blood flow to the renal cortex and medulla has been shown to decrease to zero between 5 to 7 +Gz, and remain depressed for up to 10 minutes after the +Gz exposure.““ Oliguria has been known to occur in human subjects exposed to +Gz stress lasting for durations of up to 1 hour after centrifuge experience? The mechanism of this renal dysfunction has not yet been elucidated, however, blood supply to the kidneys decreases in response to man assuming an upright posture and during physical exercise. High +Gz stress can be likened to an exaggerated orthostatic response and combined with the severe physical exertion of the Anti-G Straining Manoeuvre, these may possibly cause the decreased renal blood flow. This mechanism is a cause for concern as the possibility of the future use of vasoconstrictors in increasing pilots' +Gz tolerance may cause further reduction in renal flow to an extent that permanent renal damage may occur. Further analysis in this area is therefore required.

The effects of increased +Gz acceleration forces on the musculoskeletal system have been described in Table II previously. Neck injuries have been a source of particular concern of pilots of high performance aircraft, prompting epidemiological research into this area.⁵⁷⁻⁶¹ Incidence rates of neck injuries have been found to vary between

26.8% to 74% for US Marine and Navy aviators and 50.6% in USAF aircrew. Neck injuries were more common in higher performance aircraft [F/A-18>A-4>A-7⁵⁹ and F-16>F-15>F-5⁶¹] and commonly occurred in air-to-air combat, especially when the pilot's head is in a less stable position (e.g. looking backwards in a “check six” position). Some neck injuries have occurred when the aircrew were unprepared for sudden high G manoeuvres. The neck injuries seen have varied from muscle strains to compression fractures of the cervical vertebrae and cervical disc prolapse, and these have resulted in the rechanneling^{58,60} of several pilots to the flying of less demanding aircraft.

Conclusion

This compilation of information illustrates the physiological responses caused by exposure to elevated levels of +Gz acceleration force. Many of the physical responses caused by the elevated weight and hydrostatic forces are now easily conceptualised and understood. With such information, it is possible to provide counter-measures so as to reduce the risk of G-LOC to the pilots of high performance aircraft. Such methods would include techniques of elevating blood pressure levels when required, by physiological means, and possibly by pharmaceutical methods in the future.

Knowledge of the physical basis of cerebral blood flow reduction under high-G loading also enables the development of mechanical devices to improve the reduced blood pressures to the brain under high +Gz loading and design systems to minimise the vertical height between the heart and the head. Examples of such devices include the anti-G suit and improved G-valve systems, positive pressure breathing systems, and supinating seat systems.

It is also hoped that limited knowledge in the neurophysiological aspects of G-LOC will stimulate research in these areas, leading to a better understanding of the workings of this area. This may, in turn, lead to the development of new techniques to minimise the duration of unconsciousness and assist the pilot in recovery to normal function.

REFERENCES

1. DeHart R. Human factors problems in the Tactical Air Command. *Aviat Space Environ Med* 1986; 57:725-6.
2. Thompson A, Henneman T. Technology impacts of advanced technology crew protection (ATCP). Dayton, Ohio: Air Force Wright Aeronautical Laboratories; 1985. Report Number: AFWAL-TR-85-3074.
3. Burton R R. G-induced loss of consciousness: Definition, history, current status. *Aviat Space Environ Med* 1988; 59:2-5.
4. Clark C C, Hardy J D, Crosbie R J. A proposed physiological acceleration terminology with a historical review. Johnsville, Philadelphia: Aviation Medical Acceleration Laboratory, US Naval Air Development Center; 1961. Report Number: NADC-MA-6118.
5. White W J. A history of the centrifuge in Aerospace Medicine. California:

- Douglas Aircraft Company Inc., 1964.
- 6 Alder. Some notes on the medical aspects of aviation. In: Hamel G, Turner CC. Flying: Some Practical Experiences. London: Longmans, Green and Co, 1914.
 - 7 Head H. The sense of stability and balance in the air. Medical Research Council reports of the Av. Medical Investigation Committee, Special Report Series No. 28, London, 1919. Reprinted In: Milford H, editor. The Medical Problems of Flying. London: Oxford Univ Press, 1920.
 - 8 Poppen J R, Drinker C K. Physiological effects and possible methods of reducing the symptoms produced by rapid changes in the speed and direction of airplanes as measured in actual flight. *J Appl Physiol* 1950; 3:204-15.
 - 9 Aerospace Medical Panel (AMP) working Group 14, editors. High G physiological protection training. Neuilly-Sur-Seine, France: North Atlantic Treaty Organisation; 1990. AGARDograph AGARD-AG-322.
 - 10 Prior A R J. RAF experience of G induced loss of consciousness. Proceedings of High G and High G Protection: Aeromedical and Operational Aspects. London: The Royal Aeronautical Society, 1987.
 - 11 Pluta J C. LOC survey. Flying Safety 1984 Jan;25-8.
 - 12 Johanson D C, Pheeny H T. A new look at the loss of consciousness experience within the US Naval Forces. *Aviat Space Environ Med* 1988; 59:6-8.
 - 13 Van den Biggelaar H, Hoekstra G. Centrifuge operations and training in the Royal Netherlands Air Force. AGARD Proceedings 377, Results of Space Experiments in Physiology and Medicine and Informal Briefings by the F-16 Medical Working Group. Neuilly-Sur-Seine, France: North Atlantic Treaty Organisation, 1984.
 - 14 Whinnery J E, Marshall R M. Incapacitation time for +Gz-induced loss of consciousness. *Aviat Space Environ Med* 1979; 50:83-5.
 - 15 Whinnery J E, Jones D R. Recurrent +Gz-induced loss of consciousness. *Aviat Space Environ Med* 1987; 58:943-7.
 - 16 Glaister D H. The effects of long duration acceleration. In: Ernsting J E, King P, editors. 2nd ed. Aviation Medicine. London: Butterworths, 1988:139-58.
 - 17 Lyons T J, Harding R, Freeman J, Oakley C. G-induced loss of consciousness accidents: USAF experience 1982-1990. *Aviat Space Environ Med* 1992; 63:60-6.
 - 18 Cochran LB, Gard P W, Norseworthy M E. Variations in human G tolerance to positive acceleration. Pensacola, Florida: US Naval School of Aviation Medicine; 1954. Research Report Number: NM 001 059.02.10.
 - 19 Rossen R, Kabat H, Anderson J F. Acute arrest of cerebral circulation in man. *Arch Neuro Psychiatry* 1943; 50:510-28.
 - 20 Leverett S D Jr, Whinnery J E. Biodynamics: sustained acceleration. In: DeHart R L, editor. Fundamentals of Aerospace Medicine. Philadelphia: Lea and Febiger, 1985:202-49.
 - 21 Gillingham K K, Schade C M, Jackson W G, Gilstrap L C. Women's G tolerance. *Aviat Space Environ Med* 1986; 57:745-53.
 - 22 Webb J T, Oakley C J, Meeker L J. Unpredictability of fighter pilot G tolerance using anthropometric and physiologic variables. *Aviat Space Environ Med* 1991; 62:128-35.
 - 23 Whinnery J E. +Gz tolerance correlation with clinical parameters. *Aviat Space Environ Med* 1979; 50:736-41.
 - 24 Abboud F M, Eckberg D L, Johannsen V J, Mark A L. Carotid and cardiopulmonary baroreceptor control of splanchnic and forearm vascular resistance during venous pooling in man. *J Physiol (Lond)* 1979; 286:173-84.
 - 25 Laughlin M H. The effects of +Gz on the coronary circulation: A review. *Aviat Space Environ Med* 1986; 57:5-16.
 - 26 Shepherd J T. Circulation to skeletal muscle. In: Shepherd J T, editor. Handbook of Physiology. The Cardiovascular System. Washington D C: American Physiological Society, 1983.
 - 27 Forster E M, Whinnery J E. Reflex heart rate response to variable onset +Gz. *Aviat Space Environ Med* 1988; 59:249-54.
 - 28 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* 1956; 27:482-9.
 - 29 Shubrooks S J Jr. Changes in cardiac rhythm during sustained high levels of positive (+Gz) acceleration. *Aerospace Med* 1972; 43:1200-6.
 - 30 Whinnery A M, Whinnery J E, Hickman J R. High +Gz centrifuge training: The electrocardiographic response to +Gz-induced loss of consciousness. *Aviat Space Environ Med* 1990; 61:609-14.
 - 31 Whinnery C C, Whinnery J E. Acceleration induced electrographic interval changes. *Aviat Space Environ Med* 1988; 59:102-6.
 - 32 Whinnery J E, Laughlin M H, Hickman J R Jr. Concurrent loss of consciousness and Sino-atrial block during +Gz stress. *Aviat Space Environ Med* 1979; 50:635-8.
 - 33 Whinnery J E, Laughlin M H, Uhl G S. Coincident loss of consciousness and ventricular tachycardia during +Gz stress. *Aviat Space Environ Med* 1980; 51:927-31.
 - 34 Gillingham K K, Crump P I. Changes in clinical cardiologic measurements associated with high +Gz stress. *Aviat Space Environ Med* 1976; 47:726-33.
 - 35 West J B. Regional differences in gas exchange in the lung of erect man. *J Appl Physiol* 1962; 17:893-8.
 - 36 Glaister D H. The effects of gravity and acceleration on the lung. Neuilly-Sur-Seine, France: North Atlantic Treaty Organisation; 1970. AGARDograph Number 133.
 - 37 Barr P O. Hypoxemia in man induced by prolonged acceleration. *Acta Physiol Scand* 1962; 54:128-37.
 - 38 Jones J G, Clarke S W, Glaister D H. Effect of acceleration on regional lung emptying. *J Appl Physiol* 1969; 26:827-32.
 - 39 Browning W H. Deleterious effects of cigarette smoking and 100% oxygen on aircrew members in high performance aircraft. *Aerospace Med* 1970; 41:39-42.
 - 40 Wood E H, Hoffman E A. Lungs, the "Achilles Heel" of air breathers in high force environments. *Physiologist* 1984; 27:47-8.
 - 41 Sass D J, Ritman E L, Caskey P E, Banchemo N, Wood E H. Liquid breathing: prevention of pulmonary arterial-venous shunting during acceleration. *J Appl Physiol* 1972; 32:451-5.
 - 42 Clark L C Jr, Gollan F. Survival of mammals breathing organic liquids equilibrated with oxygen at atmospheric pressure. *Science* 1966; 152:1755-6.
 - 43 Henry J P, Gauer O H, Kety S S, Kramer K. Factors maintaining cerebral circulation during gravitational stress. *J Clin Invest* 1950; 30:292-300.
 - 44 Ernsting J E, Sharp G R, revised by Harding R M. Hypoxia and hyperventilation. In: Ernsting J E, King P, editors. 2nd ed. Aviation Medicine. London: Butterworths, 1988:45-59.
 - 45 Whinnery J E. Observations on the neurophysiologic theory of acceleration (+Gz) induced loss of consciousness. *Aviat Space Environ Med* 1989; 60:589-93.
 - 46 Whinnery J E, Burton R R, Boll P A, Eddy D R. Characterization of the resulting incapacitation following unexpected +Gz-induced loss of consciousness. *Aviat Space Environ Med* 1987; 58:631-6.
 - 47 Whinnery J E. Recognizing +Gz-induced loss of consciousness and subject recovery from unconsciousness on a human centrifuge. *Aviat Space Environ Med* 1990; 61:406-11.
 - 48 Whinnery J E, Burton R R. +Gz-induced loss of consciousness: A case for training exposure to unconsciousness. *Aviat Space Environ Med* 1987; 58:468-72.
 - 49 Whinnery J E. Converging research on +Gz-induced loss of consciousness. *Aviat Space Environ Med* 1988; 59:9-11.
 - 50 Whinnery C C M, Whinnery J E. The effect of +Gz offset rate on recovery from acceleration-induced loss of consciousness. *Aviat Space Environ Med* 1990; 61:929-34.
 - 51 Squires R D, Jensen R E, Sipple W C, Gordon J J. Electroencephalographic changes in human subjects during blackout produced by positive acceleration. Johnsville, Philadelphia: Aviation Medical Acceleration Laboratory, US Naval Air Development Center; 1964. Report Number: NADC-MA-6402.
 - 52 Jones D R. A review of central nervous system effects of G-induced loss of consciousness on volunteer subjects. *Aviat Space Environ Med* 1991; 62:624-7.
 - 53 Mills J F, Marks V. Human endocrine responses to acceleration stress. *Aviat Space Environ Med* 1982; 53:537-40.

54. Noddeland H, Myhre K, Balldin U I, Andersen H T. Proteinuria in fighter pilots after high +Gz exposure. *Aviat Space Environ Med* 1986; 57:122-5.
 55. Laughlin M H, Witt W M, Whittaker R N Jr, Renal blood flow in miniature swine during +Gz stress and anti-G suit inflation. *J Appl Physiol*, 1980; 49:471-5.
 56. Stauffer F R, Errebo-Knudsen E O. Positive acceleration and urine output. *J Aviat Med* 1950; 21:500-6.
 57. Andersen H T. Neck injury sustained during exposure to high-G forces in the F-16B. *Aviat Space Environ Med* 1988; 59:356-8.
 58. Clark J B. Cervical dystonia following exposure to high-G forces. *Aviat Space Environ Med* 1990; 61:935-7.
 59. Knudson R, McMillan D, Doucette D, Seidel M. A comparative study of G-induced neck injury in pilots of the F/A-18, A-7, and A-4. *Aviat Space Environ Med* 1988; 59:758-60.
 60. Schall D G. Non-ejection cervical spine injuries due to +Gz in high performance aircraft. *Aviat Space Environ Med* 1989; 60:445-56.
 61. Vanderbeek R D. Period prevalence of acute neck injury in US Air Force pilots exposed to high G forces. *Aviat Space Environ Med* 1988; 59:1176-80.
-