AMMA JOURNAL VOL 6 ISSUE 3

DECEMBER 1997

The neurophysiologic aspects of G-induced loss of consciousness (G-LOC) [1]

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Introduction

The principal problem faced by the human cardiovascular system when exposed to a force environment greater than +1 Gz in magnitude is that the hydrostatic effects of the applied acceleration may overwhelm the system's ability to maintain the required level of cerebral perfusion. When this occurs, G-induced loss of consciousness (G-LOC) results. G-LOC has been formally defined 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. [1] G-LOC is perhaps the most significant consequence of exposure to accelerations greater than +1 Gz.

This paper briefly reviews the historical aspects and general physiology of G-LOC and then examines the neurophysiologic aspects of a typical episode of G-LOC. These neurophysiologic aspects are the result of a significant amount of research into the nature, duration and characteristics of the unconsciousness induced by exposure to high +Gz loads.

Historical Aspects of G-LOC

G-LOC is not a new problem. Indeed, it has been a challenge to pilots for many years, almost since man first took to the skies in powered aircraft. Episodes of "fainting in the air" were first described during and after the First World War by several authors. [2.3.4] In 1938 Livingston carried out significant research in England into this problem, using modified aircraft to test subjects.[2] He accurately reported the time of incapacitation, and also described the attendant event amnesia as well as some of the psychophysiological aspects of G-LOC.

In Germany in the 1930's, Dr Heinz von Diringshofen proposed the hydrostatic theory of tolerance to G, based on elegant experiments he carried out on pilots flying an instrumented aircraft for data collection.[4] Much of von Diringshofen's work remains valid today. He was able to identify the limits of human tolerance to acceleration. Indeed, he was arguably the first researcher to accurately describe the blood pressure changes occurring under acceleration stress and the cardiovascular compensations brought to bear as a result. [4]

By the Second World War, the problem of "aviator's vertigo" was receiving a lot of attention in many countries, including the United Kingdom, America and Canada. [1.3.4] This vertigo involved pilots with transient cognitive deficits or impairments not necessarily due to vestibular factors. As knowledge of this problem grew, it became apparent that the accelerations generated by the aircraft were compromising the cardiovascular systems of the aircrew. The body of knowledge concerning G-LOC was beginning to take shape. Man-carrying centrifuges became increasingly popular as research tools, as they were able to generate the same level of acceleration in a safe and reliable manner. Indeed, the first human centrifuge for aerospace medicine research was built in Berlin in 1934, by von Diringshofen and his brother, a mechanical engineer. [4] During and immediately after the Second World War, the Allied forces had 6 centrifuges which were conducting G-related research.

As aircraft became more powerful and manoeuvrable, G-LOC assumed a position of even greater significance. Modern materials technology has allowed fighter aircraft to be developed and produced that are remarkably strong and certainly capable of generating levels of acceleration beyond the tolerance of the human occupant. The introduction of the F-15 and F-16 fighter aircraft in the 1970's illustrates this point quite well. Experience with these new high-performance aircraft led to a resurgence of interest in the problem of G-LOC, which had not received much research attention since the mid-1950s. [1.5.6] These aircraft were involved in a significant number of accidents, many of which were fatal. A disturbing proportion of these accidents were attributed to pilot incapacitation as a result of G-LOC. It became apparent that the acceleration envelopes of these aircraft were greater than had previously been experienced, and existing countermeasures were not providing the necessary margin of safety for the pilots. Once again G-LOC became a serious problem in the high-performance aircraft community. Recent surveys of fighter pilots indicate that the problem of G-LOC is certainly still present and still responsible for significant losses of both expensive materiel and highly trained personnel. In one study among USAF fighter pilots, some 12% reported having had a G-LOC episode. [9] It thus remains a challenging problem for both researchers and pilots.

Physiology of G-LOC

The physiological problems inherent in exposure to accelerations beyond+1 Gz are simply accentuations of the same problems encountered by humans when they assume upright posture, which is essentially an applied acceleration of +1 G in the z-axis. The hydrostatic effects of this force have been well described. [10.11.12.13] These same hydrostatic forces are at work when the human is subjected to +2, +4 or +9 Gz, but are obviously several orders of magnitude greater. The physiological consequences are thus magnifications of what occurs at +1 Gz. Indeed, the cardiovascular system endeavours to cope with these effects in much the same way, albeit with differing results in many cases.

Heinz von Diringshofen's haemostatic theory has contributed much to acceleration research. His theory has undergone little change since it was first put forward. Much of what we know about the effects of acceleration on the human cardiovascular system is based on von Diringshofen's theory. The haemostatic theory generally explains the effects of applied acceleration in a satisfactory way.

In the upright posture, the arterial pressure of the human varies in accordance with the magnitude of the hydrostatic force. Assuming a mean arterial pressure of 100 mmHg, the arterial pressure above the heart at the base of the brain is about 78 mm Hg. His represents a heart-brain pressure differential of 22 mmHg.

This is an important point. It allows us to state that the pressure differential between heart and brain at an applied acceleration of +1 Gz is 22 mmHg. This pressure differential is increased by a factor proportional to the magnitude of the applied acceleration. Thus, at +2 Gz, the pressure differential will be 44 mmHg, and at +4 Gz it will be 88 mmHg. From this analysis, it can be readily deduced that if the mean arterial pressure remains at a level of 100 mmHg, blood flow to the brain will cease at an applied acceleration of +4.5 Gz. This would then constitute G -LOC. Indeed, it was von Diringshofen that originally described tolerance to G as being within the range of +4.5 to +5.5 Gz. [4]

In practical terms, blood flow does not actually cease at the hydrostatically-predicted +Gz level. The hydrostatic forces involved in +Gz exposure are applied equally to the venous system and the cerebrospinal fluid, as well as the arterial system. The consequent development of a highly negative intracranial pressure sustains cerebral blood flow under gravitational stress. This theory was first postulated by Ranke in 1937, [14] and has subsequently been examined by several researchers. [15] This "perfusion without pressure" concept permits cerebral blood flow beyond the level of gravitational force that hydrostatic considerations would predict. However, this only occurs up to a point, beyond which it does effectively cease and unconsciousness results. Typically, this level is approximately +5 Gz for a relaxed, healthy subject. [10,11,16,17,18,19]

As the human cardiovascular system is exposed to acceleration, the increasing hydrostatic force is well tolerated up to a certain point. The most significant of these affects the visual system, particularly the retina. The

human eye has an internal pressure of 20 mmHg. As the pressure differential continues to increase with acceleration, there comes a point when the perfusing arterial pressure is 20 mmHg or less. At this point, the level of function of the eye becomes impaired due to relative retinal ischaemia arising through lack of the appropriate level of blood flow. The changes that occur in the vision of the human subject undergoing the applied acceleration are well documented. [10,11,16,20]

At a level of +3 to +4 G, retinal blood flow becomes degraded as arterial pressure approaches the level of internal ocular pressure. The retinal periphery is first affected, essentially as a function of distance from the blood supply entering the retina. [20] Peripheral vision, therefore, becomes impaired, and this phenomenon is known as "grey-out." This loss of peripheral vision manifests itself as a grey vision or tunnel vision. It may also occur in an asymmetric fashion, especially if the head is tilted under high +Gz. It is the first sign to a pilot that he is approaching the threshold of cardiovascular compromise. At a slightly higher level of acceleration, in the order of +4 to +4.5 Gz, blood flow into the retina is prevented by the higher internal ocular pressure relative to the driving arterial pressure. Complete loss of vision occurs. This is termed "blackout." This term should not be confused with syncope, with which it is popularly associated. This complete loss of vision typically occurs with the pilot still fully conscious. He is able to still manoeuvre his aircraft, receive radio transmissions, etc. His higher cognitive functions are all still intact. It is only his visual input that has been lost. There is an interval of approximately 4-6 seconds between arterial pressure falling below the critical level of 20 mmHg and complete loss of vision. This is due to the existence of a reserve oxygen store within the eye itself. [10,11,16]

When the applied acceleration is greater than +4.5 to +5.5 Gz, the driving pressure generated by the heart is insufficient to overcome the magnitude of the hydrostatic force. Cerebral blood flow ceases and unconsciousness results. This is G-LOC. It can be seen that G-LOC represents the failure of the cardiovascular system to tolerate a high applied acceleration.

Neurophysiologic Aspects

G-LOC should not be considered as an abnormal event. Rather, it is a normal physiological response to an abnormal stimulus, ie high +Gz acceleration. It is also not a form of seizure or syncope. It is best defined as a sudden, orderly and progressive shutdown of the brain, largely thought to occur as a self-protective mechanism. It reflects a continuum of +Gz effects from consciousness to unconsciousness. There is an important period, from the onset of high +Gz, during which the brain is able to still function despite the absence of any effective cerebral blood flow. This has been described as the functional buffer period, or metabolic energy reserve, and has in several studies been shown to have a duration of approximately 6 seconds. [21.22] The mechanism underlying this buffer period is unknown, but it is generally considered to have a protective role, in that it allows for large scale accelerations to be tolerated as long as they are not sustained beyond the critical 6 second mark. [21] At the end of this buffer period, consciousness is said to terminate abruptly with complete brain shutdown.

An episode of G-LOC has several features that have been well described in the aerospace medicine literature. [11,21,23.32]In simple terms, a G-LOC episode will result in a period of unconsciousness followed by a period of disorientation and confusion. The time of overall pilot incapacitation is defined as the total incapacitation period. This represents in operational terms the total amount of time that the pilot is not in control of his aircraft. The total incapacitation period consists of an absolute incapacitation period and a relative incapacitation period. [21.23.32] The absolute incapacitation period represents complete incapacitation or true unconsciousness, which usually lasts for about 15 seconds on average. In experimental terms, it is defined as the time from the subject's head dropping at the moment of unconsciousness to the raising of the subject's head as consciousness is restored. [23]

This absolute incapacitation period is then followed by a period of relative incapacitation which has a duration of approximately 10-15 seconds. This period is defined as the time interval from head raising to the first voluntary purposeful limb movement. [23] During the relative incapacitation period, the pilot is once again conscious, but only in a technical sense. That is, while blood flow to the brain has been restored, the pilot is somewhat dissociated from his situation and unable to function appropriately. He is disoriented and confused, with significant cognitive slowing, and his higher cortical centres are largely dysfunctional. [21.21.23.32] As a result, he is incapable of

appropriately assessing his situation and thus perceiving danger. Fine motor control is absent, and often only gross motor acts are carried out. He is thus still incapacitated inasmuch as he is unable to save himself or correct his situation. He is still recovering from the major ischaemic insult his cerebral cortex has sustained. It has been reported that some pilots in such situations have watched in fascination as their altimeters have registered their rapid descent to subsequent ground impact, the pilot not appreciating the significance of what he is seeing and therefore not reacting to it appropriately. Obviously, in a supersonic jet fighter pulling high +Gz levels at low altitudes, the potential for disaster in such a situation is enormous.

There is also a characteristic recovery process from an episode of G-LOC. Tingling in the extremities and perioral numbness have been reported in several studies. [25] Myoclonic convulsions and flailing of the arms commonly occur, generally coinciding with the reestablishment of effective cerebral blood flow. Typically these convulsions occur in the latter third of the absolute incapacitation period, generally in the last 4 seconds of this so-called convulsion prone period, and are not associated with any electroencephalographic abnormalities. [26] Whinnery has proposed that these convulsions occur as a result of a functional caudal reticular formation becoming disinhibited by a non-functional cerebral cortex. [26]

In addition, cognitive distortions of a dream-like nature are said to occur towards the very end of the absolute incapacitation period. [23.33] These dreams can often incorporate the myoclonic convulsions which typically occur in the same phase of the G-LOC event.[26] No link has been established between these dreams and REM sleep, but they are akin to the hypnopompic dreams which can occur at the end of a normal sleep cycle, in that they tend to be associated with the end stages of unconsciousness. [26]

The rate of onset and offset of high +Gz has also been shown to affect the nature of the subsequent G-LOC episode. Gradual onset of +Gz has been shown to result in longer absolute and relative incapacitation periods. [23.26.28] This is due to the longer period of absent or reduced cerebral perfusion during gradual onset exposure. Gradual onset thus produces a greater perfusion deficit and hence greater embarrassment to the central nervous system than rapid onset. Several centrifuge studies have shown that gradual onset runs (GORs) consistently produce prolonged incapacitation times. Rapid onset runs (RORs), on the other hand, typically produce shorter incapacitation periods. In one centrifuge study, for example, incapacitation following rapid onset was 23.7 seconds, compared with 32.3 seconds for gradual onset. [23]

Gradual offset of +Gz tends to prolong the time of incapacitation in much the same way as gradual onset, as it prolongs the period of overall cerebral hypoperfusion and delays reestablishment of full cerebral blood flow. Centrifuge studies involve offset rates in the region of 0.5 to 0.9 Gs [1], while inflight G offset rates have been found to be approximately 0.5 Gs 128. A centrifuge ROR usually takes 9 seconds to return to +1 Gz from a peak of +9 Gz. [34] Obviously the quicker the G offset, the quicker cerebral blood flow will be returned to normal, and the shorter the resultant incapacitation time. In terms of the rate of recovery from an episode of G-LOC, studies have shown that there is considerable individual variation. Indeed, it appears that a particular subject's G tolerance and their rate of recovery from an episode of G-LOC are not related. They are, in fact, independent variables. [23]

Rapid onset of +Gz is often not associated with myoclonic convulsions or dreams. This is thought to be due to the fact that rapid onset of high +Gz may disable the reticular formation just as quickly as the cerebral cortex. [26] This would prevent myoclonic convulsions from occurring.

The unconsciousness resulting from G- LOC has been compared with that resulting from acute arrest of the cerebral circulation. [28] In an experimental setting, the latter unconsciousness is generally produced via the use of a cervical occlusion cuff. Important facts emerge as a result of this comparison. Firstly, the times of incapacitation are different between the two types of unconsciousness. G-LOC results in longer incapacitation times than acute arrest of the cerebral circulation. This is related to onset and offset rates. As we have seen, onset and offset tend to take at least several seconds during +Gz stress. On the other hand, acute arrest of the cerebral circulation can be achieved almost instantaneously and can be reversed just as quickly. Higher onset and offset rates produce less overall incapacitation. However, the two types of unconsciousness are strikingly similar in one

important aspect. The time from application of the stress (high +Gz or acute arrest) to onset of unconsciousness is virtually the same in both cases, being 6 to 7 seconds. This represents the functional buffer period, which as we have already seen is the period of time that the central nervous system can function after the blood supply has become inadequate. [2s] This buffer period is due to the stored oxygen and metabolic substrate supplies within the brain, which are exhausted after this critical time.

The neuropsychological effects of G-LOC have also been documented, and are accentuated by repeated G-LOC episodes. These effects include denial, euphoria, irritation, embarrassment, confusion, dissociation and anxiety, among others. [2B. 30] It has been reported that G- LOC has the potential to "exert a temporary psychologically crippling effect" on the combat effectiveness of tactical aircrew, who may have altered judgement, and a loss of aggressiveness and motivation to carry out their mission. [2a.so] In the post-G -LOC period, psychological mechanisms often result in suppression and denial of the actual G-LOC event. Indeed, recovery from G-LOC is typically associated with event amnesia, with the pilot not recollecting having had a period of unconsciousness at all. These post-G- LOC psychological reactions can have a negative impact on flight safety. Full psychophysiological recovery from an episode of G-LOC is generally believed to be reached only after a complete sleep cycle.

Conclusion

G-induced loss of consciousness continues to be a challenge for the fighter pilot. The unconsciousness produced by overwhelming exposure to applied +Gz loads can produce a myriad of neurophysiological effects. Convulsions, dreams and various psychological reactions can all occur. All of these features of the post-G -LOC environment can have significant implications for flight safety.

With the continued advances in fighter aircraft technology, the elimination of G-LOC as a cause of aircraft loss seems a highly unlikely proposition. What is required is a greater level of understanding of the nature of G-LOC and its neurophysiologic consequences. In this way more effective anti-G countermeasures can be developed for the protection of the aircrew who operate in the high G environment. Only by understanding the problem more thoroughly can we hope to produce an effective solution.