Symposium: HIGH SUSTAINED G FORCES

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HIGH SUSTAINED G - AEROMEDICAL ASPECTS

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THE potentially disabling effects of G forces on aircrew, viz., grey out, black out and loss of consciousness (LOC), are well known. Aircrew have been exposed to +Gz forces for decades. However, G-induced loss of consciousness (G-LOC) has emerged as a premier human factor challenge to those who fly modern generation aircraft. Besides generating and sustaining G forces far in excess of what the human body can tolerate, new electronic flight control systems allow high Gs to build up so rapidly that aircrew may lapse into unconsciousness without passing through the stages of grey out or black out.

CASE REPORT

A case of G-LOC in a Vajra pilot of Indian Air Force (IAF) came to light during a recent anonymous questionnaire study.

The exercise was medium level practice interception culminating in a 1-Vs-1 situation on visual pick up. The target was a trainer and the attacker a fighter. The trainer was flown by a student pilot from the front seat with his instructor in the rear. The first two engagements were uneventful. For the third engagement, the instructor was on the controls. He initiated a hard turn in which the G quickly peaked at 7 +Gz. The student pilot crouched forward and performed the straining manoeuvre but nevertheless experienced a grey out and narrowing of vision. The instructor relaxed the G almost completely and turned to the other side. The pupil then lay back in the seat with his head against the head-rest and looked back in an attempt to locate the fighter. At this stage, the instructor once again initiated a hard turn to the right and upwards. The pupil was unable to crouch forward. He attempted the straining manoeuvre but could not perform it correctly due to lack of anticipation. He felt the onset of grey out and soon lost consciousness. The extent of LOC was probably about 10-15 sec. It took him another 4-5 sec to come out of the state of almost total disorientation. After recovery, he found his head slumped forward and felt twitching in his arms. Shortly thereafter, he was given the control and carried out a normal recovery to base.

No post-incident aeromedical evaluation could be undertaken as it came to light much later during the anonymous questionnaire study after frequent discussions/indoctrination. It is obvious that similar cases are not reported immediately after the incident due to the fear of grounding. The aeromedical response to this problem thus should be education rather than medical disqualification.

The most probable cause leading to G-LOC in the above case was the abrupt onset of +Gz when the pupil pilot least expected it. He was thus not able to perform effective straining manoeuvre which is mandatory to sustain high +Gz. A contributory factor may be physical exhaustion due to the high +Gz during the first two engagements which fatigued him and hampered his ability to perform effective and timely anti-G straining manoeuvre during the third engagement.

HUMAN TOLERANCE

Tolerance to high sustained G (HSG) varies from individual to individual and in the same individual, to some extent, from time to time. High haemodynamic tolerance has been reported among motivated persons utilising different means of protection, e.g., +8 Gz for 60 sec, +9 Gz for 45 sec and +10 Gz for 40 sec (6).

Tolerance to HSG is measured as the ability to endure specified levels and durations of exposure to HSG , (a) with maximum of 100% peripheral light loss and 50% central light loss / dimness, (b) without occurrence of serious cardiac rhythm disturbances or heart rate exceeding 200 bpm and, (c) without complete subjective fatigue.

SUBJECTIVE EFFECTS

G-induced loss of consciousness is one of the serious challenges faced by any modern Air Force. The victim of G-LOC usually does not recollect the event.

Petechial haemorrhages are noted in parts of the lower extremities not covered by anti-G suit. These are painless and disappear within a few days. Whether haemorrhages of any significance occur in other organs is not known.

Mild to moderate pain in the left forearm is reported after sorties involving HSG. There may be associated petechial haemorrhages in the left forearm which are painless and last for a few hours. These have been observed in a Vajra pilot on two occasions when he pulled 8.5 to 9 G.

Minimal oedema of feet may occur due to prolonged high hydrostatic pressure.

Incidence of neck pain in pilots of aircraft with HSG capability has been increasing. Persistent discomfort may cause preoccupation and distraction and may interfere with performance. The pain may appear in both acute and chronic forms. It may occur when a pilot while pulling a high G load is also turning his head. Vanden-bosch reported a case of subluxation of C4 vertebra with probable discal hernia in a Belgian Air Force pilot whose head had turned under acceleration during flight (8). Weight of the flying helmet is

the principal cause for cervical pain. Approximate weight of the head and neck upto C5-C6 can be taken as 4 Kg. With the equipment on head weighing 1.7 Kg, the load at C5-C6 will be 28.5 Kg at 5 G and 51.3 Kg at 9 G!

When the neck is bolted during wait for a HSG, the cervical spine perfectly resists an acceleration of 8 to 9 G. From 5 G onwards the head, surprised by an attitude other than the position of bolting, will have an apparent weight of 30 Kg or more and the head movement is going to be pursued till braked by anatomical formations. One can envisage the sudden occurrence, at 8 to 9 G, of lesions like cervical sprains, especially in a trainer aircraft, when one of the pilots is surprised by the manoeuvre. Similar aetiology can cause backache in any region of the spine. Thus, it is indispensable that the pilot maintains his resistance and develops his spinal muscles. Moreover, the ideal solution for cervicalgia during HSG is the development of an extremely light weight helmet with a low resultant centre of gravity.

Sensory illusions during build up and decay of acceleration are known. Disorientation problems which persist for several hours and upto a day after HSG have been described (9). These are due to disadaptation of organs of equilibrium at cessation of stimulus. However, the hypothesis of minor lesions of otolithic organs cannot be ruled out. It has been described that under HSG, it is difficult to perceive and maintain the neutral position of the stick. Thus, when the pilot looks outside, he has a tendency to tilt the stick towards the side of his gaze or to push it if he is looking downwards.

Evaluation of performance is important for flight safety and conception of pilot aircraft interface. Centrifuge studies by US Navy show that there is a decrement of mental performance (choice reaction time and simple computation) lasting up to two minutes after LOC. It has also been demonstrated that there is a rapid fall of performance in tracking tasks after +6 Gz.

OBJECTIVE EFFECTS

Cardiovascular Effects

Sustained accelerations of high intensity have given rise to a new risk of cardiac rhythm abnormalities. Up to +5 Gz, the rhythm anomalies observed pose very little problem. However, from +7 Gz upwards the extent of ventricular excitability makes it necessary to recognise the rhythmogenic nature of HSG. Dysrhythmias observed under HSG are as follows:

a. Sinusoidal Rhythms

i. Sinusoidal tachycardia : At low G levels, heart rate can be directly correlated with level and duration of +Gz exposure. At HSG, the G - heart rate relationship is less discernible as a maximal heart rate of about 200 bpm is reached at relatively low G levels, viz., +7 Gz.

ii. Sinusoidal bradycardia and bradyarhythmias (occasionally).

iii. Sino-auricular blocks and junctional rhythms.

b. Disturbances of auriculo-ventricular (AV) conduction. AV Block (I, II or III degree)

c. Disturbances of auricular excitability - Auricular fibrillation

d. Disturbances of ventricular excitability - Ventricular extrasystoles

The mechanisms which induce rhythm disturbances during acceleration have been widely debated. Some of the possible mechanisms postulated are :

- mechanical role of acceleration on position of heart,
- diminution of coronary supply,
- reflex slowing of sinus pacemaker due to straining manoeuvres,
- right auricular distension,
- increase in sympathetic adrenergic activity, and
- neuro-vegetative disequilibrium with increase in vagal tone.

The role of dysrhythmias in limiting tolerance to HSG is not definitely known. Thus, when dysrhythmia is accompanied by G-LOC or a bad tolerance to acceleration, it is not proved that the former is responsible. It appears that there is a common cause to both these phenomena. However, the risk of dysrhythmias especially VPBs cannot be underestimated. Haemodynamically, when frequent VPBs appear suddenly in a pilot, who at 7 or 8 G has already a limited central circulatory supply, he can black out or even lose consciousness.

Respiratory Effects

Exposure to levels of +7 to +9 Gz for 45 sec combined with anti-G suit inflation, especially in smokers, is sufficient to produce regional compression atelectasis and/or airway closure without having to invoke rapid absorption of oxygen distal to occluded airways which is necessary at lower acceleration levels. Thus there are more chances of acceleration atelectasis under HSG.

Pathological Effects on CVS

The occurrence of pathology in a human analog (an experimental animal with HSG responses similar to man) exposed to HSG, tolerable to both man and the experimental animal, has been investigated using adult miniature swine (2). Conscious pigs serve as adequate human acceleration pathophysiologic model because they have similar vascular hydrostatic column length as man, perform M-1 manoeuvre during +Gz resulting in similar arterial pressure responses at eye level, and have similar acceleration tolerance levels with and without anti-G suit inflations.

Burton and Mckenzie (3) reported subendocardial haemorrhage and minor cardiomyopathy (myofibrillar degeneration and myocardial necrosis) in miniature swine exposed to +Gz stress. These observations stimulated concern that exposure to sustained high G stress

might cause cardiac damage in aircrew and human experimental subjects. Laughlin (5), in a review of the available literature, concluded that +Gz exposure poses no significant risk for cardiac damage in humans. Three primary considerations led to this conclusion :

(a) The lesions in swine probably result from very high levels of both sympathetic adrenergic tone to the heart and circulating plasma catecholamines (CA) acting on the cardiac cells. Most of these CA appears to be released as a result of the overall stress involved in exposing conscious miniature swine to +Gz on the centrifuge and not directly as a result of the +Gz per se.

(b) +Gz exposure is not as psychologically stressful for humans. Thus CA levels (cardiac or systemic) in men exposed to +Gz were found to be much less as compared to miniature swine.

(c) A large number of clinical cardiologic data (ECG, thoracic radiography, phonocardiography, systolic time intervals and VCG) available from the humans who have been exposed to +Gz stress does not indicate any degree of cardiac damage. However, all the techniques mentioned above would not be able to detect limited histological lesions observed in experimental animals.

There remained the possibility that cardiac damage was occurring but at a level undetectable with available techniques. The strongest argument against this possibility was the report (4) concerning a volunteer for a study programme of HSG in centrifuge, who was fatally injured in a traffic accident. For the preceding two years he had been exposed 142 times to +7 Gz, 18 times to +8 Gz, 6 times to +9 Gz and 18 times to +10 Gz. He had been exposed at least 15 times to +7 Gz in the month prior to his death. Histological study of myocardium of this individual did not show any evidence of high G stress cardiomyopathy. This one case combined with all existing clinical and experimental data, indicate that operational levels of the stress do not provide an environment which will cause cardiac damage in normal humans.

A recent echocardiographic study in France (personal communication), however, revealed the presence of significantly more frequent right ventricle anomalies (right ventricular dilatation and mitral valve prolapse) among fighter pilots as compared to transport pilots. This was probably due to long term effects of combat aircraft accelerations. The hypotheses put forward were : (a) simple adaptation of right ventricle to acceleration; (b) early myocardium disorder.

It is concluded that strict medical follow up studies and a comparison of the results with other studies will perhaps bring forth an explanation for these findings.

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