*Teaching File*

# Syncope in the air - A case report

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A 22 year-old ex-NDA flight cadet with a total of 105 hours of flying on HPT-32 and Iskra was undergoing stage II training on the Iskra. Mid way during his training, on 10 Feb 1995 at 1215 hours, he was strapped up for a dual general handling sortie with his instructor. He had already flown one solo sortie on that day at 1000 hours, which was uneventful. He was again scheduled for a second sortie for which he took off at 1250 hours (30 minutes waiting period on the tarmac) and climbed to 13000 feet (cabin altitude - 7000 feet, pressurization 2.5 PSI). All subsequent manoeuvres were carried out at this altitude.

During the sortie, after about 05-10 minutes, he first performed four steep turns with a +Gz component of 3.0-3.5, followed by stalls. His performance was observed to be below normal as described by his instructor. At this stage he was told by his instructor to perform a wing over manoeuvre and also to take sector reference.

The pupil first turned his head to the left to obtain sector reference and then to the right, by when he noticed that the nose of the aircraft had dropped sharply and that the aircraft had gone into a spin. He noticed that the aircraft speed had built up to 700 km/hr and he saw the ground rapidly coming towards him. He was alarmed and described the episode as 'I have never felt so afraid in my life'. He held his breath and pulled back the stick sharply as a result entered into a tight 5.2 +Gz turn. At this moment he felt his legs becoming heavy, he felt hot and noticed that he was sweating profusely, audio calls were becoming fainter and he had tunneling of vision following which he did not remember anything.

The instructor took over the aircraft at this juncture and leveled off at 10000 feet after a duration of one minute. Though not able to visualize his pupil (tandem seating), he noticed the pupil to be slumped to one side and not responsive to any verbal commands. He finally landed the aircraft back after a period of 08-10 minutes. A couple of minutes after landing, as the pupil was being unstrapped from the aircraft, the pupil regained consciousness. Return of consciousness was rapid and not accompanied by any confusion, drowsiness or disorientation, though he expressed surprise on finding himself back on ground. He was noticed to be drenched with sweat. He had no incontinence of urine, and gave no history of any headache, tongue bite or aura. Also no post-ictal phenomenon was observed. The instructor could not give any history of any observed convulsions. There was

no past history of any similar complaints, syncopal attacks, seizures, head injury or airsickness. Medical examination and investigation, done just after landing, which included a blood sugar random (80 mg%), failed to reveal any abnormality. There was no failure of pressurization or any malfunction of oxygen equipment.

On going through further details of antecedent history, the pilot had slept late the previous night (0100 hours) and had also woken up earlier (0500 hours). He had a light breakfast at around 0630 hours and had not taken any food or water thereafter. The day was unduly hot for that time of the year (Tdb-39°C at 1200 hours). There was no history of any alcohol consumption the day before.

The pilot with this history was referred to IAM IAF for further evaluation.

**Q. 1. What will be your approach in this patient?**

A case of episodic unconsciousness in the aviation environment requires thorough evaluation, as flight safety is the issue. The most important aid to the diagnosis of such cases is a reliable eyewitness account and history. Two studies exemplify the importance of a reliable history in these cases [1]. The first called the Boston study, revealed that initial diagnosis based on clinical assessment was correct in 172/196 cases. In the remaining 24 cases diagnosis could not be established even after a thorough investigation. The second study, called the Pittsburgh study, showed that initial diagnosis based on history and clinical assessment was made in 89/210 cases. In the remaining 121 cases the diagnosis after extensive investigation was possible in a further 13 cases. Further the yield after specialized investigations in a case of syncope of unknown etiology is not cost effective. However where aeromedical disposal is the issue, additional investigations, are necessary and may actually be cost effective.

A case of episodic unconsciousness needs to be approached systematically. The normal level of consciousness depends upon activation of the cerebral hemispheres by the reticular activating system (RAS) in the brain stem. The RAS and its connections from the cerebral hemispheres must be preserved for normal consciousness. Hence any disturbance in the normal level of consciousness could be due to two main reasons [2].

(a) Bilateral hemispherical damage.

(b) Brain stem lesion that damages the RAS.

These areas could be affected by mechanical damage resulting in the so called anatomical coma or due to a global disruption of the brains metabolic processes which is termed as metabolic coma. Coma due to a metabolic origin could be produced by interruption of energy substrate delivery (hypoxia, ischemia, hypoglycemia) or by alteration of neurophysiological responses of neuronal membranes (epilepsy, acute head injury, drug/alcohol intoxication).

The first step is to distinguish whether the loss of consciousness was due to a syncopal attack or due to a seizure. Seizure is a single episode of neurological dysfunction whereas syncope can be defined as a reversible, temporary loss of consciousness due to a qualitative or quantitative disturbance of cerebral blood flow. Syncope is of two types [3]:

(a) Simple syncope - no convulsive activity

(b) Convulsive syncope.

  (i) Syncope with convulsive accompaniments (brief twitching)

  (ii) Syncopal convulsion, due to prolonged global ischemia.

The task becomes more difficult if there is evidence of seizure like (myoclonic) activity during the episode. Convulsive activity during syncope occurs in 12% of cases [3]. The clinical history is the diagnostic cornerstone of convulsive syncope.

**Q. 2. How will you distinguish between syncope and a seizure ?**

No single point will distinguish the two but taken as a group and supplemented by an EEG the two may be distinguished. Table 1 given below shows certain differentiating characteristics between a convulsive syncope and a seizure (tonic-clonic) [3].

**Q. 3. What is the role of EEG in the diagnosis in such cases?**

EEG is the most sensitive tool for diagnosis of epilepsy. However, 20% of petit mal and 40% of grand mal epileptics have normal EEG's interictally despite multiple recordings. A standard EEG should specifically include hyperventilation, photic stimulation and sleep recording. Activation procedures increase the yield of EEG's interictally in epileptic individuals to 75-80%. A sleep deprived wake and sleep EEG is of particular importance when epilepsy is being strongly considered [4, 5].

**Table 1. Difference between epilepsy and syncope [3]**

| Function | Syncope | Seizure (Tonic-clonic) |
|---|---|---|
| Age | Any age, usually young | 75% cases-before 21 years |
| State of wakefulness | Usually awake | Awake/sleep |
| Position of patient | Usually erect | Any position |
| Emotional stresses | Important | Often unimportant |
| Physiological stresses | Could be important (heat) | Could be important (sleep deprivation) |
| Onset | Pre-syncopal symptoms | Usually sudden (aura ±) |
| General appearance | Pale, perspiring | Unchanged/cyanotic |
| Frothing at mouth/ tongue biting, automatism | No | Often |
| Respiration | Shallow | Noisy, blowing, rough |
| Injury on falling | Infrequent | Often |
| Length of unconsciousness | Short, depends on time taken to take recumbent posture | Relatively prolonged (2-5 min) |
| Urinary incontinence | Uncommon | Frequent |
| Pulse | Slow, weak | Bounding, rapid |
| Recovery time | Short (secs to a few mints) | Longer (min-hrs) |
| Post seizure sequel | Physical weakness, clear sesnsorium, perspiration | Coma like state (15-20 min) Mental confustion, headache, drowsiness, lethargy (1-2 hrs) |
| Amnesia | Minimal/absent | Prominent |
| EEG interictal | Usually normal | Often abnormal |
| EEG (during event) | Bilateral, synchronous beta/ theta waves, middle/high amplitude | Characteristic spikes and sharp waves |

**Q. 4. What is your diagnosis in this case?**

In this case the classical history of profuse sweating, tunneling of vision, audio calls becoming faint and absence of any signs and symptoms suggesting epilepsy enabled us to make a diagnosis of a vasovagal syncope. The diagnosis of vasovagal syncope was made based on the classical clinical history and by exclusion of other causes. An acute loss of consciousness with tunneling of vision strongly suggests a vascular cause with progressive centrally mediated ischemia affecting the brain centers subserving consciousness. Vascular causes in such an environment can be due to G-LOC, hypoxia, hypocapnia secondary to hyperventilation, primary neuro-circulatory collapse and vasovagal syncope in the air due to any provocative etiopathology. G-LOC for almost 8-10 minutes is not reported in the literature. Further the rate of build up of +Gz and the overall level of +Gz would not favour a G-LOC situation. Though it has been seen that G-LOC can occur in uninitiated pilots and 13 cases have been reported in Iskra aircraft [6]. Hypoxia also could not have been a cause at this operating altitude. Further there was no failure in pressurization.

Vasovagal syncope could have been triggered off due to two main reasons, which were both present during this particular sortie.

(a) Loss of control of the aircraft caused an intense emotional stress on the individual. Intense emotional stress in a warm environment, as in this case, is one of the most important triggering causes of syncope.

(b) Frequent exposures to low levels of +Gz exposure (near the black out threshold), especially in a warm environment makes an aviator prone to syncope [7].

**Q. 5. What is the incidence and causes of syncope in military aviation?**

The incidence of syncope in USAF presence is 12-48% i.e. they have had at least one syncopal episode during their service tenure. The life time incidence in general population is around 3 to 48%. Syncope accounts for 1-3% of emergency room visits [3]. Even though we can determine the cause of syncope in many episodes the etiology remains elusive in 11-52% cases. The causes of syncope, relevant to the aeromedical physician are enumerated in Table 2.

**Q. 6. Discuss the role of acceleration in this case?**

Majority of cases of unconsciousness in the air/centrifuge are attributed to acute cerebral ischemia induced by high +Gz forces (G-LOC). While G-LOC is known to occur during sudden high G levels it can also occur in an unprepared pilot or in relatively inexperienced pilots at rather low G levels or due to improperly done anti G straining manouevres. Sometimes the loss of consciousness is delayed. This occurs in centrifuge experiments, which last for a long time at acceleration magnitudes near the black out threshold. The rates of onset of +Gz forces are usually slow enough for full cardiovascular compensation to take place and suddenly, after a long period during which no untoward incident has taken place, collapse occurs. Physiological recordings made during such episodes indicate that it is due to peripheral vascular failure. When consciousness is regained, the subject is pale and sweating, in sharp contrast to the flushed appearance seen after the more usual period of LOC on the centrifuge. This form is due to vasovagal syncope. The loss of effective blood volume caused by pooling of blood in dependent parts of the body is aggravated by the filtration of fluid through the walls of the vessels, and when a critical quantity is lost, syncope will occur [7]. However the maximum G-LOC reported in the literature is for 110 seconds [8]. In studies on G-LOC during centrifuge training at IAM IAF where the G-LOC duration (absolute and relative) reported is 130 seconds [9]. Absolute incapaci-

**Table 2. Causes of syncope relevant to the aviation environment**

| | | |
|---|---|---|
| (a) Circulatory causes (decrease in blood flow to the brain) | | |
| (i) | Inadequate vasoconstrictor mechanisms: | Vaso-Vagal syncope, Postural hypotension, Primary autonomic insufficiency, Carotid sinus syncope, Hyperbradykinemia. |
| (ii) | Hypovolemia | |
| (iii) | Mechanical reduction of venous return: | Valsalva, cough, micturition, atrial myxoma. |
| (iv) | Reduced Cardiac Output: | Obstruction to LV outflow (Aortic stenosis, hypertrophic sub aortic stenosis) |
| | | Obstruction to pulmonary flow (Primary pulmonary hypertension, pulmonary embolism, pulmonic stenosis) |
| | | Myocardial causes (Myocardial infarction with pump failure) |
| | | Pericardial causes (tamponade) |
| (v) | Arrhythmias: | Bradyarrhythmias- Ventricular asystole, Sinus bradycardia, Sinus arrest, SA block, Carotid Sinus Syncope, Atrio ventricular block (2/3 degree with Stokes Adam syndrome) |
| | | Tachyarrhythmias- Ventricular tachycardia, episodic ventricular fibrillation with or without bradyarrhythmias, supraventricular tachycardia without AV block. |
| (b) Other causes | | |
| (i) | Altered state of blood to the brain: Hypoxia, Anemia, Hyperventilation, Hypoglycemia. | |
| (ii) | Cerebral causes: Cerebrovascular disturbances, Emotional disturbances, Anxiety attack. | |
| (c) Aviation related causes | | |
| (i) | Related to high altitude - | Hypoxia, PPB, Hyperventilation, DCS, Pressurization failure. |
| (ii) | Related to acceleration - | Low G tolerance, HSG, Faulty AGSM, failure of G suit pressurization, unprepared pilot. |
| (iii) | Miscellaneous causes - | Airsickness |

tation period is reported to be 26 seconds [10] though it may vary depending upon the rates of onset. Recovery after G-LOC is usually slow and associated with confusion and disorientation (relative incapacitation period) and may take up to another 15 seconds.

Heat stress also results in a decreased tolerance to acceleration due to vasodilatation, which results in opening up of large areas of cutaneous vascular bed resulting in decrease in circulatory reserve. In conjunction with accelerative forces the compensation is greater than what the circulation can provide. At higher heat loads the decrease in +Gz tolerance has been estimated to be +1 Gz.

In this case syncope occurred just after he entered into a tight +5.2 Gz turn, it was associated with features of gray out like tunneling of vision followed by black out and loss of consciousness. However the duration of loss of consciousness has been unusually long, about 15 minutes, and there was no spontaneous recovery once the acceleration stress was removed. Also the instructor did not experience any symptoms of acceleration stress.

**Q. 7. Could the syncope be of cardiac origin?**

Syncope of cardiac origin could be related to bradyarrhythmias, which are poorly picked up by electrophysiological studies. Tachyarrhythmias of significance are usually seen in cases where there is underlying evidence of structural heart disease. When syncope is related to a disorder of cardiac function there is a combination of pallor and cyanosis, with pronounced dyspnoea, and often the jugular veins are distended [5]. This is because the ischemia in these cases is more global and prolonged. Persistent asystole may lead to cerebral injury and focal neurological signs may appear. Normally syncope associated with arrythmias lasting for more than 15 seconds will also result in generalized tonic spasms and incontinence. The causal relation between arrythmias and syncope is uncertain and only 4% of patients may have symptomatic correlation with arrythmia on holter monitoring. Extending the duration of monitoring to longer than 24 hours does not improve the yield of symptom producing arrhythmias [11].

**Q. 8. What were the investigations carried out in this case?**

For reaching the diagnosis and to rule out other causes of syncope the following investigations were carried out on this aircrew:

* *GTT (2 hour and 5 hour)* - to rule out reactive hypoglycemia.
* *ECG* - Normal and Treadmill Stress Test, 24 hour ambulatory Holter monitoring - to exclude cardiogenic causes of syncope. The diagnostic yield for Holter monitoring in this setting is approximately 10%. Event recording using loop recorders is a relatively new technique, which has a better diagnostic yield of 36% [12].
* *Echocardiography* - to exclude structural heart disease.
* *Tilt Table studies (70 degree HUT)* - this test is 65 to 75% sensitive in classical syncope [5] and 90% in the acute situation [13]. Passive tilt testing should be done for not less than 30 minutes and preferably 45 minutes. Drug challenge with isoproterenol (1 to 3 µg) and recently glyceryl tri nitrate IV or sublingually has also gained favour [12]. The use of digital plethysmography to monitor arterial blood pressure has made it possible to demonstrate different types of heamodynamic reactions to tilt testing.
* *Hypoxia studies* - Exposure to simulated altitude of up to 18000 feet with and without hyperventilation.

- *Acceleration Studies* - relaxed and straining +Gz tolerance with GOR and ROR profiles. ROR tolerance of this cadet was 4.1 G and GOR tolerance was 6.68 G.
- *EEG* - including with activation procedures and a sleep deprived and a sleep EEG. (In this case it was done at three different centres and seen by three different neurophysicians)
- *Heat stress studies* - to exclude undue heat intolerance.
- *ENT evaluation* - to rule out any vestibular pathology which could precipitate syncope.
- *Psychological and Psychiatric evaluation*- Psychiatric syncope is a known entity but is associated with panic disorders, major depression and conversion reactions (somatoform disorders) [14].
- *X-Ray Cervical spine and Chest* - the former has been done to ascertain if there were any craniovertebral anomalies and the latter to assist the cardio-respiratory assessment.
- *CT Scan Head/MRI:* CT Scan (with and without contrast) was done to exclude any organic lesion. MRI was not done in this case.

All the above investigations were found to be within normal limits.

**Q. 9. How did the pilot recover while still in the sitting posture and why were there no residual effects even after such a prolonged period of unconsciousness?**

Vasovagal syncope can also occur in a sitting posture. Attacks due to hypoglycemia, hyperventilation or heart block are not dependent upon posture. Syncope associated with a decline in blood pressure or ectopic tachycardia usually occurs in the sitting or standing posture [7]. An episode of vasovagal syncope ends when the patient is put in supine posture. The duration of unconsciousness may persists for a longer time which may also result in a syncopal convulsion if the head does not become dependent when the prolonged cerebral ischemia does not get corrected [3]. This status may continue till the impulses which are causing vasodilatation (decreased peripheral resistance) and cardio-inhibition (decrease in heart rate, cardiac output and cerebral blood supply) are removed. Thereafter the patient would regain consciousness though not as rapidly as seen when put in a supine position.

No brain damage occurred in this case though the patient was unconsciousness for a period of almost 10 minutes. This is because of the fact that brain is markedly dependent on continuous blood flow for the delivery of oxygen and glucose for its metabolic needs. These are consumed at the rates of 3.5 mg/100/gm/min and 5mg/100gm/min respectively. If blood flow to the brain is stopped abruptly and completely, unconsciousness occurs in 08-10 seconds though the brain store of glucose provides energy for a period of 02-03 minutes. Damage would occur only after this period. Normal resting cerebral blood flow is 75 ml/100gm/min in white matter with a mean of 55ml/100gm/min. The EEG becomes slowed below this level of flow and at 15mg/100g/min, the brain electrical activity ceases. It is only when flow falls below 10ml/100gm/min, does irrevocable brain damage occurs [2]. In this case blood flow to brain was reduced sufficiently enough to cause loss of consciousness but insufficient to cause any brain damage.

**Q. 10. Why could hypoglycemia not be a cause of unconsciousness in this case?**

The brain depends primarily on glucose for its energy supply. In a normal person the body maintains plasma glucose in a safe range for CNS

metabolism and also to provide energy for other tissues in the body. This is done by various processes like glycogenolysis, gluconeogenesis, lipolysis, proteolysis and ketogenesis, all these processes increase blood sugar levels and are initiated by a fall in plasma insulin. Glycogen plays the primary role in these processes followed by epinephrine. There is also a rise in cortisol and growth hormone, which antagonize insulin action. These protective mechanisms are activated much before symptomatic threshold is reached. Therefore in normal persons, hypoglycemia is not possible.

**Q. 11. What was your disposal in this case?**

This motivated student pilot with no antecedent illness, a good flying record and a normal psychoanalytical profile experienced a fainting episode in the air lasting about 15 minutes. The pilot had a late night, early breakfast and nothing to eat or drink till the episode. It was an unusually hot summer day. He had flown a 45 minute solo sortie two hours prior to this particular sortie. During the second afternoon sortie he completed three +Gz manouevres, as directed by his instructor, and when performing the fourth manouevre he lost control of the aircraft due to momentary attention diversion to check his bearings. As a consequence he entered into an unexpected downward tight turn and experienced an extreme degree of fright as the ground was rushing at him. He pulled on the joystick when he noticed that he was bathed in sweat, and his vision and other sensory faculties were dimming. He, thereafter, does not recall anything further. His instructor recovered to base after failing to verbally rouse the slumped student. On return to ground the pilot spontaneously recovered and got out of the aircraft on his own. Medical examination subsequently did not reveal any abnormality. Independent assessment by two neurophysicians and a psychiatrist ruled out any organic illness in the individual. The combination of repeated G stress situations with another sudden onset G manouevre in a stressed, possibly fatigued, unprepared student pilot who was exposed to sudden emotional disturbance in a rather hot environment, has led to syncope in the air preciptated by a G manouevre. Considering the benign nature of his illness he was returned back to flying in a restricted flying category. After an adequate period of observation he would have returned back to full flying status provided there were no further similar episodes. In a USAF study 64.1% such cases were returned back to flying [16].

**Q. 12. What are the causes of tunneling of vision in aviation?**

In aviation, tunneling of vision could be due to 5 main causes:-

(a) Acceleration induced
  (i) G-LOC
  (ii) Vasovagal Syncope
(b) Hypoxia
(c) Hyperventilation
(d) Positive pressure breathing

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