MANAGEMENT OF EPISODIC UNCONSCIOUSNESS

A REVIEW

By

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Transient loss of consciousness is a vexatious symptom especially in flying personnel. The exact definition of unconsciousness is difficult, but it may generally be taken, for our purpose, as a lack of or reduced awareness of the environment for a short duration. This ranges from (a) light headedness, reduced alertness, faints, classical syncopes on one hand, to (b) various ictal phenomena on the other.

The significance of the correct diagnosis, management and disposal need not be stressed, especially in the case of aircrew. The first group may be regarded as symptoms of a transient disorder of cerebral function mostly of benign origin, mediated by impaired cerebral circulation or by purely neurogenic factors. Organic disorders of the heart and metabolism may also be responsible in a minority of cases, thus warranting a careful assessment of each case. The second group is nearly always due to an organic lesion of the C. N. S. and the prognosis, management and disposal is entirely different. Classification in the above two groups is not easy. If the precipitating factor is prolonged, convulsions may result in the benign group. The problem is further complicated by the fact that many epileptics give a history of faints in the past, and epilepsy may be induced by factors normally producing syncope. This paper is an attempt to discuss the practical management of these cases to assist in the correct and balanced assessment. A brief discussion of pathophysiology is essential for a proper understanding.

Conditions causing transient loss of consciousness are multiple and confusing if discussed individually. For clarity, the following grouping is useful :

(*1*) *Circulatory* — (reduced blood supply to the brain),

a) Peripheral circulatory failure.
Vaso vagal (psychogenic syncope).
Postural hypotension.

Tussive syncope — due to interference with the venous return caused by increased intrathoracic pressure, during bouts of coughing and pressure breathing.

Shock — following injuries, burns, instrumentation etc.
Vaso-motor weakness following prolonged illness—"parade faints".
Drugs — Vasodilator group or hypotensive drugs.
Vasomotor weakness induced by syphilitic and diabetic myelopathy.

b) Reduced blood volume.
Internal & external hæmorrhage.
Effects of heat.
Loss of tissue fluids e. g. diarrhoea.

c) Cardiac causes :—

(i) Alteration in rate or rhythm,

(aa) Bradycardia.
Neurogenic e. g. carotid sinus syndrome.
Reflex bradycardia.
Conduction defects e. g. heart block and cardiac stand-still.

(ab) Ectopic beats and tachycardia of ectopic origin — the cardiac filling or output falls in this group.

(ac) Congenital heart disease (cyanotic group) — syncope is associated with exertion as a result of further lowering of oxygen saturation.

(ii) Acute myocardial injury e. g. infarction.

(iii) Mechanical hindrance to the flow of blood, aortic valvular disease, pulmonary embolism.

(2) Metabolic causes

Anoxia and hypoxia.
Hyperventilation.
Hypoglycæmia.
Exposure to noxious gases and extreme cold.
Depression of C. N. S. by drugs and alcohol.

(3) Cerebral lesions

Epilepsy.
Cerebral vascular disturbance.
Hypertensive encephalopathy.

(4) Psychosomatic conditions

Hysteria.
Anxiety state with or without hyperventilation .

Circulatory Disorders

Circulatory syncope : The basic pathology is pooling of blood in muscles and tissue spaces with ischæmia of skin. The cardiac output may or may not be affected. Cerebral ischæmia ensues only in the erect posture, with restoration to normal in the supine position. If ischæmia is prolonged, clonic movements may be noted. A similar mechanism operates in positive 'G' blackouts.

Vasovagal Syncope

Syncope only occurs in the erect posture and is relieved by lying down. The period of unconsciousness is short, but may recur for hours on standing up. The pulse may be slow or rapid but the systolic pressure falls to 70 mm Hg. or so. Syncope is precipitated by strong emotional stimuli, pain, sight of blood, injections etc. It is facilitated by preceeding illness, overcrowding, hunger, fatigue etc. Unconsciousness is always preceeded and accompanied by weakness, light headedness, nausea and cold sweat. It is never sudden. The patient gradually slumps down, unlike sudden falling like a log in epilepsy.

Postural or Orthostatic Hypotension

This condition occurs in some normal individuals, but is more commonly seen after prolonged illness in old people. The condition is occasionally seen in association with diabetic myelopathy and tabes dorsalis. In such individuals there is a sudden fall of systolic and diastolic blood pressure leading to syncope, whenever they quickly stand up. Recovery follows on lying down. There are no associated symptoms. The pooling of blood peripherally does not excite compensatory vaso-constriction. A similar mechanism operates after prolonged standing still in "parade faints."

Syncope may occur after bouts of coughing and during performance of the Valsalva manoeuvre. Intrathoracic pressure increases to the extent of impeding venous return resulting in a fall of cardiac output and syncope.

Reduced blood volume — syncope is caused by reduced blood pressure due to low circulating fluid volume and low cardiac output.

Cardiac Abnormalities

These result in poor cardiac output. Adam Stokes syndrome is caused by disease of the conduction system — the pulse is usually below 40 per minute. On resumption of the heart beat, there is flushing of head and neck producing a sense of heat. The electro-cardiogram reveals heart block with or without evidence of myocardial damage. Heart block may also be caused reflexly by irritation of the vagus nerve. Thus syncope may be associated with oesophageal, pleural, pericardial or severe abdominal irritation. Aortic stenosis is often a cause of giddiness and fainting following exertion. Another important member of this group is carotid sinus syncope. Irritation

of the carotid sinus in sensitive individuals leads to bradycardia and hypotension. A tight collar, sudden turning of head etc., may lead to a short lived syncope in the erect posture only.

Metabolic Disorders

The C. N. S. is highly sensitive to hypoxia, CO_2 levels and hypoglycæmia. Any alteration in these may lead to syncopal attacks as a result of interference with cerebral cellular metabolism. Syncope, following hypoglycæmia and hyperventilations deserve special mention. Effects of hypoxia are too well known to be discussed.

Hypoglycæmia : Syncope may be induced by injudicious use of hypoglycæmic agents, tumours of islets cells or spontaneous (reactive) hypoglycæmia. It may also be associated with severe hepatic, adrenal or pituitary deficiency. Attacks are associated with sweating, giddiness, hunger, lassitude, unsteadiness, choking and are masked by confusion, stupor or coma. The history or marks of insulin administration, if present, are helpful. Primary hyperinsulinism is associated with low fasting blood sugar. A more interesting condition is reactive hypoglycæmia. Symptoms usually occur 2 to 4 hours after a heavy carbohydrate meal. Fasting blood sugar levels are normal. The condition is usually missed, unless suspected. G. T. T. prolonged upto 4 hours is diagnostic.

Hyperventilation is usually associated with anxiety and results in washing out of plasma CO_2 leading to cerebral vaso-constriction and syncope. Such a fall in plasma CO_2 alters the *pH* of blood which becomes more alkaline. The oxygen dissociation curve shifts to the left — which shows that Hb does not part with O_2 so easily. This factor in continuation with cerebral vascular constriction may cause cerebral hypoxia resulting in unconsciousness. The patient should be closely questioned to exclude the possibility of hyperventilation especially under stressful situations.

Cerebral Disorders

EPILEPSY — This is a common cause of short lived loss of consciousness. It is caused by an excessive and disorderly discharge from cerebral neurones resulting in instantaneous disturbances of sensation and consciousness, convulsive movements or some combination thereof. The symptomatology of the classical epileptic fit is well known. However, it should be noted that the motor element may be absent or insignificant. Attacks may be purely psychic or sensory. Petit mal and akinetic episodes are examples. But practically all varieties have recurrent episodes with an aura and associated with a varying degree of unconsciousness. The classical fit includes aura, a sudden "falling like a log", rigidity, convulsions, outcry, frothing, changes of reflexes, loss of sphincter control. Injuries are often sustained during the attack.

CEREBRO—VASCULAR DISTURBANCES

Patients suffering from atherosclerosis of cerebral arteries or internal carotid thrombosis may have repeated attacks of focal symptoms, depending on the ischaemic

area — dimness of vision, slurred speech, sensory or motor paresis, dizziness and short lived impairment of consciousness may be repeatedly complained of. Symptoms are usually unilateral and associated with signs during the attacks, usually lasting 5 to 10 minutes. In these individuals, the cerebral circulation is critically balanced and any alteration in the haemodynamics results in an ischaemic episode. Attacks are usually repeated and have more or less a set pattern. They are common in the older age groups (in the fifties.)

Similarly hypertensive encephalopathy produces intermittent signs and symptoms associated with a sudden increase of blood pressure in a known hypertensive individual.

Psychosomatic Conditions

Hysteria — Occurs in young individuals of poor personality make up under adverse circumstances. Fainting is dramatic in the presence of a sympathetic audience. During the attacks, there may be no movement at all, but more commonly, struggling, mumbling and well directed purposeful movements are present. There are no signs. There is no change in the pulse, B. P. or reflexes. Sphincter control is not lost, injuries are absent, attempts at examination are resisted.

Hyperventilation associated with anxiety has already been discussed.

Management

During syncope: The patient should be laid flat and the head turned to one side. The mouth should be cleared of any foreign matter. A mouth gag of suitable material should be used if epilepsy is suspected. Tight clothing should be loosened. A brief history should be obtained from any witnesses available, and a quick examination of pulse, pupils, reflexes and blood pressure should be made. This examination will be of great help in the final diagnosis. Note should also be made of any injuries or bleeding especially from nose, throat and ears. Sprinkling of cold water and inhalation of spirit ammonia aromaticus may be useful. The patient should not be permitted to get up till the sense of weakness has passed off. He should be observed for some time afterwards.

After syncope: As soon as is practicable, the patient should be closely questioned regarding the nature of the onset, aura, peculiar sensations if any, origin of the faint and associated symptoms. He should be questioned as to (a) what he had eaten, (b) any unusual exertion preceeding the fainting (c) associated illness, fatigue etc., (d) if he had any previous attacks of similar nature. Emotional factors should be elicited. Hyperventilation should be excluded. The history should be verified from witnesses, who often are in a position to add something or to clear some doubtful points not noted by a confused patient. A detailed physical examination, including funduscopy, should be made and urine tested for albumin and sugar. If possible, an attempt should be made to re-

produce symptoms, if there is any doubt regarding their nature. This is controversial. However, only conditions, that can be reproduced, are benign in origin. Such a demonstration clears the diagnosis, gives the patient an insight into his condition and helps in the prevention of subsequent attacks. Methods commonly used are forced hyperventilation, prolonged standing still, sudden standing after rest on bed, massage of carotid sinus etc. In all such techniques, it must be remembered that the crucial point is not whether symptoms are produced but whether the exact pattern of symptoms during the attack is reproduced.

Investigations: In addition, special investigations may be necessary, Fasting blood sugar 3 -4 hours after meals, blood urea, haemoglobin percentage, red cell count, W.R.& Khan, X-ray of heart and skull, E. C. G. and finally E. E. G. may be required to arrive at a definite diagnosis. The fundus should be examined. If an intracranial lesion is suspected, special tests like angiography will be necessary. A fairly good diagnosis can be made in most cases without these tests, only on the basis of the history and the evaluation of physical signs and symptoms. Therefore, the above tests should not be carried out as a routine but only where definitely indicated and then too, only appropriate tests, which are likely to prove valuable, should be done. Routine battery of investigations is no substitute for clinical diagnosis.

If hypoxia, effects of G and hyperventilation are suspected, the patient must be closely questioned regarding altitude, use and servicability of oxygen equipment, fitting of the oxygen mask, manouveres undertaken, presence of fumes or smell, possibility of hyperventilation etc. These statements should be verified by examination of aircraft and equipment. A knowledge of aviation medicine, and aircraft and equipment is essential to deal with such cases.

Differential Diagnosis

The most important factor in the differential diagnosis is the exclusion of epilepsy. While a classical attack is not likely to be missed, minor varieties pose difficult problems. Even these differ from syncope in important aspects. Epilepsy is sudden in onset. The aura lasts for a few seconds only. Attacks occur at any time and in any posture. There is no pallor. Injuries and loss of sphincter control are frequently noted. Tonic contractions are common and the period of unconsciousness is longer in epilepsy and is followed by confusion, sleep, drowziness headache etc. Physical weakness with a clear mind seen in syncope is not present in epilepsy. Epileptic attacks are repeated. In contrast, syncope is preceeded by feeling unwell, nausea, weakness, gradual slumping down to ground followed by quick recovery to full consciousness in a relatively short time. Tonic convulsions, loss of sphincter control, and drowziness are uncommon. Pallor, pulse and blood pressure changes are noticed.

The differential diagnosis of commoner conditions can be summarized in the Table I based on Harrison's "Principles and Internal Medicine."

TABLE I

DIFFERENTIAL DIAGNOSIS OF SOME OF THE MORE COMMON CAUSES OF RECURRENT ATTACKS OF WEAKNESS OR UNCONSCIOUSNESS

Group		Disorder	Type of onset	Position at onset	Duration of attack	Factors affecting attack		Important findings during attacks.	Remarks
						Precipitating.	Alleviating.		
1. Intermittent bradycardia.	(a)	Heart block.	Sudden.	Any.	Seconds to hours.	Unknown.	Ephedrine*	Heart rate less than 40.	Enlarged heart, Electro-Cardiogram.
	(b)	Hypersensitive carotid sinus.	Sudden.	Sitting or standing.	Seconds.	Turning head, tight collar.	Recumbent posture.	Bradycardia, Hypotension.	Attack reproduced by pressure on carotid sinus.
2. Intermittent tachycardia.	(a)	Orthostatic.	Sudden.	Standing.	Minutes.	Standing.	Recumbency.	Tachycardia on standing	
	(b)	Paroxymal ectopic @ tachycardia.	Instantaneous.	Any.	Minutes to days.	Indefinite.	Pressure on eye ball.	Heart rate 150 +	Electro-cardiogram, palpitation.
3. Temporary hypotension.	(a)	Orthostatic.	Sudden.	Standing.	Minutes.	Standing.	Recumbency.	Marked decline in blood pressure on standing.	
	(b)	Vasodepressor syncope.	Sudden.	Standing.	Seconds to minutes.	Anxiety, fright, pain.	Recumbency.	Hypotension, bradycardia.	Subjects usually healthy.
4. Chemical disorders.	(a)	Hypoglycaemia.	Gradual.	Any.	Minutes.	Excessive starch intake.	Eating.	Palpitation, anxiety, sweating.	Usually begins 2-4 hrs after meals.
	(b)	Hyperventilation.	Gradual.	Any.	Minutes to hours.	fatigue, emotional upset, anxiety.	Rebreathing.	Tremor, sighing, panting, numbness.	No evidence of disease of heart or lungs.
5. Etiology unknown.		Idiopathic epilepsy.	Sudden.	Any.	Minutes to hours.	Unknown.	Sedatives.	Deep coma, convulsions.	Family history, electro-encephalograms.

* Only when attacks are related to cardiac arrest. Diagnosis in the cases due to ventricular fibrillation requires an ECG during attack.

@ Including auricular fibrillation, auricular flutter, auricular tachycardia, and ventricular tachycardia.

Conclusion

In conclusion, it should be emphasized that the majority of conditions which produce a transient loss of consciousness in a relatively young population as seen in Services, are not serious. Most cases of serious conditions, associated with abnormalities of cardio-vascular and central nervous system, can be easily excluded on the basis of the history and the physical examination. Hypo-glycaemia and hypoxia should always be considered and excluded. After exclusion of the serious conditions, the problem is one of reassurance and education as to how to prevent further attacks and improvement of general health of the individual. Indoctrination of aircrew is essential in prevention of syncope in air.