

PART II – NEUROLOGICAL DISORDERS

CHAPTER 9
COMA AND TRANSIENT
LOSS OF CONSCIOUSNESS

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NEUROLOGY IN AFRICA

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CHAPTER 9

COMA AND TRANSIENT LOSS OF CONSCIOUSNESS

Loss or alteration in consciousness is a very common clinical disorder. This can be transient lasting seconds or minutes as occurs in syncope and seizures or more prolonged as occurs in coma. *Coma is by definition a state of impaired consciousness during which the patient is unrousable by external stimuli.* In states of coma the patient remains in a sleep like state with no purposeful movements or response to any external stimuli. These can be measured by the Glasgow Coma Scale which defines coma as a GCS $\leq 8/15$. Coma can be caused by disorders that affect either a part of the brain focally or the whole brain diffusely (Figs. 9.1-4). The causes of coma are generally classified as intracranial or extracranial and are outlined in Table 9.1. Episodes of transient loss of consciousness are by definition intermittent and usually sudden events from which the patient recovers fully. These arise either from the disorders of the cardiovascular system with an acute reduction of blood flow to the brain (syncope) or a disruption in brain electrical activity (seizure). The chapter outlines the main mechanisms, causes, investigations and management of coma and syncope. The student should aim to be familiar with these and be able to investigate and manage a patient presenting with loss of consciousness.

Pathophysiology

Consciousness is a person's awareness of themselves and their surroundings. Normal consciousness is maintained by an intact reticular activating system in the brain stem and its central connections to the thalamus and cerebral hemispheres. The reticular activating system keeps us awake and alert during the waking hours. Disorders that physically affect these areas can lead to disordered arousal, awareness and to altered states of consciousness. A focal brain lesion occurring below the tentorium (Figs.9.1 & 2) interfering with the reticular activating system can result in coma whereas a focal lesion occurring above the tentorium in one cerebral hemisphere results in coma only if the contralateral side of the brain is simultaneously involved or compressed (Fig.9.3) Diffuse lesions which affect the function of the brain as a whole including the reticular activating system can result in coma (Fig.9.4).

ASSESSMENT

Acute

Coma is an acute life threatening condition and evaluation needs to be quick, comprehensive and may involve starting emergency management even before the cause is established. Emergency management starts with an immediate assessment of airway, breathing and circulation (ABC) and involves the following steps. **Firstly** check that the airway is clear without secretions and that no cyanosis is present. This is achieved by rapid visual inspection and checking the vital signs. **Secondly** ensure that breathing rate is satisfactory (rate >10 -12/min), that there are

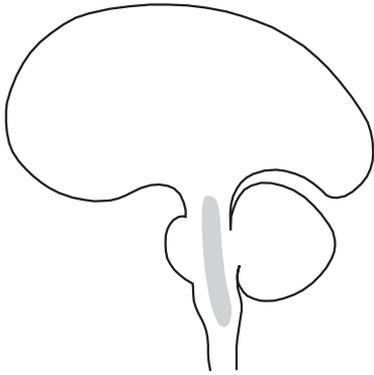


Figure 9.1 The reticular activating system

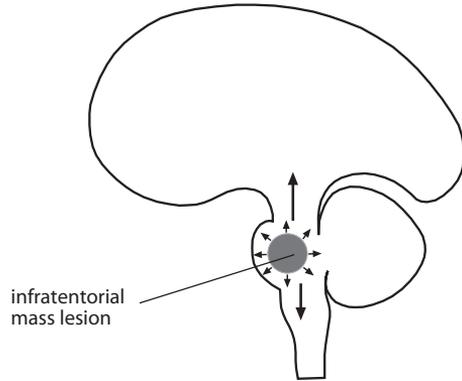


Figure 9.2 Sites that produce loss of consciousness

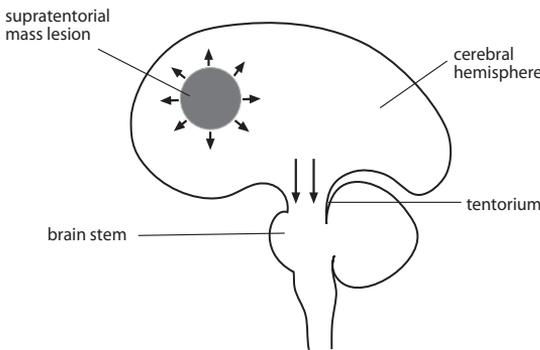


Figure 9.3 Sites that produce loss of consciousness

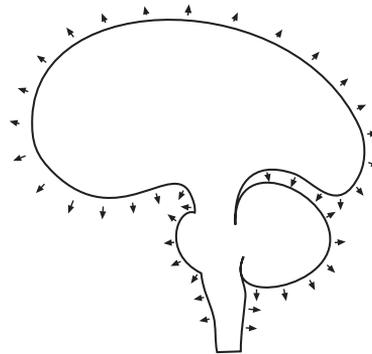


Figure 9.4 Encephalopathy diffuse

adequate breath sounds bilaterally on auscultation and that the oxygen saturation is >95%. If ventilation is inadequate or GCS is ≤ 8 then consider intubation and assisted ventilation. **Thirdly** check that the circulation is adequate by measuring pulse and BP. If the systolic blood pressure is <90 mm Hg then start immediate fluid resuscitation, and with inotropic support if BP remains persistently low despite adequate volume replacement. **Fourthly** insert an IV cannula and withdraw blood for laboratory studies. All comatose patients should have their blood glucose checked on arrival and treated immediately if hypoglycaemic (blood sugar <2.5 mmols/l) or hyperglycaemic. **Finally** treat any other immediately reversible cause without delay e.g. iv thiamine 100 mg in patients with history of alcoholism and naloxone 0.4-2 mg in patients with drug habituation (Table 9.4). If the patient is stable, then the clinical assessment can start.

Key points

- airway: make sure it is clear
- breathing: count RR & listen to the lungs, if respiration inadequate give O₂ & ventilate
- circulation: check pulse/BP and treat if systolic <90 mm Hg
- glucose: check urgently and treat any reversible cause immediately

Clinical assessment

This involves *history, general examination, level of consciousness and neurological examination.*

The history

The history is the most important part of the assessment as it frequently points to the underlying cause of coma. The diagnosis may already be obvious from the circumstances surrounding the coma e.g. head injury in a road traffic accident (RTA), stroke in hypertension or hyperglycaemia or hypoglycaemia in diabetes or a seizure in epilepsy. If the cause is not obvious then it is necessary to obtain a history from the patient's family members, friends or colleagues. The history should include information and details concerning the immediate circumstances and the possible cause of the coma. It should also include the patient's previous medical history, medications, allergies, possible toxins and details of social and family history including recent travel or anything relevant. In particular, ask if there was a recent preceding illness e.g. fever, headache, if the onset was sudden or gradual and check specifically for a history of trauma or fall, a history of epilepsy, alcohol or drugs. The main causes of coma are outlined below (Table 9.1).

Key points

- loss of consciousness is a medical emergency
- assessment needs to be brief and focused
- history is the most important part of the initial assessment
- cause may be obvious & reversible causes need to be considered
- main causes are head injuries, encephalopathies, infections & strokes

Table 9.1 Main disorders causing coma

Site/aetiology	Disorder
Intracranial	
Focal	
<i>stroke</i>	infarct, ICH, SAH
<i>infections</i>	brain abscess
<i>trauma</i>	haematoma (<i>ICH, EDH, SDH</i>)
<i>tumours</i>	primary or secondary
Diffuse	
<i>infections</i>	HIV, meningitis, malaria, encephalitis
<i>seizures</i>	post ictal/status epilepticus
<i>trauma</i>	traumatic brain injury
Extracranial	
<i>hypoxia</i>	cardiac, respiratory, renal, shock, anaemia
<i>metabolic/toxic</i>	hyper-hypoglycaemia, organ failure, hyponatraemia overdose, opiates, alcohol
<i>hypertension</i>	encephalopathy, eclampsia

Easy way to remember the causes of coma

A = anoxia/apoplexy

E = epilepsy

I = injury/infection

O = opiates

U = uraemia

The general examination

This involves confirming the vital signs and checking for evidence of obvious injury or major underlying illness. Signs of head injury/basal skull fracture include lacerations or bruising to the head, around the eyes, behind the ear (Battle’s sign) or CSF leak from the nose or ears (Chapter 19). Palpation of the head/neck may show signs of a fracture and swelling. Signs pointing to an underlying illness include paresis, hypertension, tongue biting, ketoacidosis, jaundice and evidence of infection including fever, meningitis, and pneumonia or discharging ear.

The level of consciousness (LOC)

This is the most important part of the assessment of the unconscious patient. Altered states of consciousness range from *confusion* and *delirium* to *stupor* and *coma* (**appendix 1**). *Confusion* is characterized by the patient being fully conscious but with impaired attention, concentration and orientation. Confusion can be tested at the bedside by checking if the patient is fully orientated in time, person and place with a score of 10/10 being fully orientated (Table 9.2).

Table 9.2 Testing for orientation 10/10*

Time	Person	Place
time	name	hospital
day	age	town/district
month	year of birth	country
year		

*score one for each correct answer

In a state of *delirium*, the patient although fully conscious is confused and restless with hallucinations. In a state of *stupor*, the patient is in coma but is rousable after intense stimulation; this is in contrast to coma where the patient is unrousable. In general the use of these terms has been replaced by the **Glasgow Coma Scale (GCS)** (Table 9.3).

Glasgow coma scale

The depth of coma can be measured by the **GCS**. This measures eye opening, best motor and verbal response, and is a reliable method for measuring and monitoring level of consciousness. It should be carried out and if necessary repeated on every comatose patient. When using the **GCS**, look carefully at the patient’s face while assessing eye opening, and then check on the patient’s ability to follow simple motor commands and listen to the patient’s speech for content and orientation. If the patient is not responding to voice then test eye opening and limb movement response to deep pain by applying pressure to sternum or supra orbital ridge or nail beds. Record best eye opening, motor and verbal response as **E4**, **M6** and **V5**. Patients are considered comatose if the **GCS ≤ 8/15**.

Table 9.3 Glasgow Coma Scale

	Best response	Score
Eye opening (E)	spontaneously	4
	to speech	3
	to pain	2
	nil	1
Motor response (M)	obeys commands	6
	localizes stimulus	5
	flexes withdrawal	4
	flexes weak	3
	extension	2
	nil	1
Best verbal (V)	oriented fully	5
	confused	4
	inappropriate	3
	incomprehensible (sounds only)	2
	nil	1
Maximum score		15

The AVPU Method

A more simplified bedside assessment of the level of consciousness is the **AVPU method** (yes/no response). Its advantages are that it assesses the main levels of consciousness quickly and is easy and quick to use and communicate.

AVPU method

A: is the patient alert

V: is the patient responsive to voice

P: is the patient responsive to pain

U: is the patient unresponsive

Key points

- check the vital signs
- examine for signs of head injury or a systemic disorder
- assess the level of consciousness
- level of consciousness is the most important part of the assessment

THE NEUROLOGICAL ASSESSMENT

This frequently gives the clues towards establishing the aetiology of the coma. The neurological assessment in coma is necessarily shortened concentrating on the possible neurological causes of coma e.g. stroke, meningitis and the presence of any localizing signs. Note the level of consciousness and any obvious neurological abnormalities such as seizures, the pattern of breathing and the position of the eyes and posture of the trunk and limbs. In particular, record pupil size, equality, response to light and eye position or movements. Normal pupils are 3–4 mm in diameter and respond briskly to light. Abnormalities include fixed dilated pupil (s), >7 mm in size and non reactive to light. In states of coma the most common cause of a unilateral fixed pupil is herniation (Table 9.4.) and of bilateral fixed pupils is brain death. The presence or absence of the corneal reflexes should be noted and fundi checked for papilloedema. If there

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is no contra indication to moving the neck such as spinal or head injury neck stiffness should be tested although its absence is unreliable in coma.

In the comatose patient eye position and movements are observed and examined at rest and during head and neck movement. Eye movements may be checked by stimulating the vestibular system. The details concerning the methods of stimulation (Doll’s eyes and caloric tests), are outlined in **appendix 1**. Note the presence of any cranial nerve palsies, the position of the limbs and any signs of limb paralysis. Motor response to pain can be checked by noting limb movement in response to a painful stimulus e.g. deep Achilles tendon pressure or knuckling the sternum. A flexor response in the arms and concurrent extension of the legs indicates a cortical site of origin (**decorticate**) whereas an extensor response at the elbow coupled with internal arm rotation and extension of the legs (**decerebrate**) indicates an upper brain stem injury. Unilateral or asymmetrical posturing indicates a contralateral focal brain lesion/injury. Tone and reflexes in the limbs are then examined in the routine manner. Main localizing neurological features in coma are summarized below in Table 9.4.

Table 9.4 Main localizing neurological features & causes in coma

Neurology finding	Localization	Main causes
Respiration pattern Cheyne Stokes <i>(breathing progressively deeper and then shallower in cycles)</i>	brain stem herniation/ encephalopathy	↑ICP, stroke
Eye position <i>eyes looking to one side (conjugate deviation)</i>	unilateral lesion	stroke, head injury & any focal lesion
Pupils <i>fixed & dilated pupils</i> <i>a fixed & dilated pupil</i> <i>pin point pupils</i>	 brain herniation of medial temporal lobe through tentorium compressing 3rd CN pontine lesion	 anoxia, trauma, brain death unilateral mass lesion above tentorium due to any cause stroke, organophosphorous poisoning, opiate overdose
Meningism	meningeal inflammation	infections & haemorrhage
Limb position <i>hemiparetic/asymmetrical</i> <i>arms flexed, legs extended</i> <i>all 4 limbs hyper extended & extensor posturing to pain</i>	 unilateral lesion decorticate posturing decerebrate posturing	 stroke & any focal lesion cortical damage brain, brain stem damage
Limb examination <i>hypertonia & hyperreflexia & extensor plantars</i> <i>bilateral extensor plantars</i>	 brain/cord non localizing	 any diffuse or focal lesion coma any cause

Key points

- assessment of coma needs to be fast & focused
- history usually reveals the cause of coma
- general examination reveals the level of coma
- neurological examination may reveal the site
- don't forget fundoscopy

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for persistent coma involves all causes of unresponsiveness including psychiatric and neurological disorders. These include unresponsiveness of psychogenic origin locked-in syndrome, persistent vegetative state and brain death (**appendix 1**).

Psychogenic

This can be a manifestation of severe schizophrenia (catatonia), hysteria (conversion disorder) and malingering. These are all diagnoses of exclusion and should only be considered when other causes have been excluded and there is strong evidence in their favour. Neurological exam in these patients is invariably normal and most will exhibit resistance to eye opening and tensing or withdrawal to a painful stimulus. Investigations including caloric tests and EEGs if performed are normal. A return to full consciousness is usually the rule.

Locked-in syndrome

The term locked-in is a rare syndrome which describes patients who though fully conscious are quadriplegic and unable to speak. The main causes are stroke and trauma affecting the brain stem at the level of the pons/midbrain. The patients are usually able to see and hear but are unable to move or communicate apart from moving their eyes/eye lids. Only some of voluntary eye movements may be preserved e.g. looking upwards. This still allows for non verbal communication. The diagnosis can be confirmed by saying/indicating with your own eye movements to the patient to “open your eyes”, “look up”, “down” and “towards the tip of your nose”. Using this method a way can then be established to communicate with the patient. If in doubt ask a senior colleague to examine and check the patient. EEG is normal. Survival for months and years is the rule.

Persistent vegetative state

Patient with this disorder are awake but not aware. Their eyes open and close normally and they have a sleep-wake cycle because of an intact brain stem but they show no purposeful response to any external stimuli. The cause is usually cerebral hypoxia or ischaemia or occasionally a structural lesion. Recovery is uncommon.

Brain death

Brain death is the irreversible loss of all brain stem function. This is loss of consciousness, respiration, response to pain and loss of all brain stem reflexes (**appendix 2**). The patient should be unresponsive to any sensory input including pain and speech. The cause should be known, irreversible and sufficient to explain the death. Reversible causes of coma should be considered including CNS depressant drugs, metabolic disorders and hypothermia. If the patient is on assisted ventilation, the criteria for determining brain death are outlined in appendix 2.

INVESTIGATIONS

The main emergency investigations in patients presenting in coma are outlined below in Table 9.5. A blood glucose should be checked immediately and glucose given intravenously if <2.5 mmol/L. Screening for the presence of malaria parasites and checking of HIV status is routine practice in patients presenting in coma in Africa. When indicated bloods for other metabolic causes of coma should also be checked. A lumbar puncture is relatively contraindicated in states of coma. It is specifically contraindicated in states of undiagnosed deep coma (**GCS \leq 8**) until raised intracranial pressure and focal intracranial lesions have been excluded by fundoscopy and CT. Emergency X-rays are important in coma in particular in head injury. A skull X-ray may show a fracture and a CT a treatable cause of coma e.g. subdural haematoma.

Table 9.5 Emergency investigations

Bloods	blood sugar malaria parasite film FBC HIV biochemistry (renal & LFTs)
Urine	sugar & ketones
X-rays	
Skull/neck	may show fracture
Chest	may show pneumonia, PCP, TB
CT head	may show blood & fracture & structural lesion

MANAGEMENT

The emergency treatments of the main causes of coma are presented in Table 9.6. Medical and nursing care involves monitoring and regularly checking the vital signs, level of consciousness and pupils, usually every 15 minutes to four hourly depending on the clinical situation. It is important at this stage to avoid sedation and strong analgesics. General nursing care of eyes, mouth, bladder and bowels should be started early with special attention to avoiding pressure sores and prevention of deep vein thrombosis. The ongoing care of the comatose patient should ideally take place in an appropriate intensive care unit (ICU) setting, depending on the cause. The subsequent medical or surgical management will depend on the underlying cause. Consider intubation if GCS \leq 8.

Table 9.6 Emergency treatment of coma

Disorder	Acute management
Stroke (ischaemic)	aspirin 300 mg/po/stat
Infections	
<i>cerebral malaria</i>	artemether* 2.4 mg/kg iv/im stat <i>or</i> quinine 600 mg/iv over 4 hours
<i>acute bacterial meningitis</i>	ceftriaxone 2 gm/iv infusion stat
<i>cryptococcal meningitis</i>	fluconazole 1200 mg/po/ng tube
<i>toxoplasmosis</i>	co-trimoxazole 80/400mg/4 tablets stat/po/ng tube
<i>sepsis</i>	ceftriaxone 2 gm/iv infusion stat gentamycin 80 mg/im/stat
Hypoglycaemia	25-50 ml of 50% dextrose/iv/stat <i>or</i> glucagon 1mg/im stat
Ketoacidosis	soluble insulin 5 iu/iv & 5 iu/im stat & start hydration
Raised intracranial pressure	mannitol 20%: 100-200 ml iv stat dexamethasone 16 mg/iv stat
Poisoning e.g. organophosphates	atropine 1.0 mg/iv/stat & repeat every 15-30 mins
Respiratory depression (secondary to suspected opiate overdose)	naloxone 0.4-2 mg iv every 2-3 mins to max of 10 mg
Wernicke's encephalopathy	thiamine 100 mg iv
Anaphylaxis	adrenaline 0.5-1 ml of 1:1000/im/stat <i>or</i> adrenaline 0.5-1 ml of 1:10,000/iv/stat

* artemisinin drugs are the first line drugs recommended for use in cerebral malaria although quinine is mostly available

Prognosis

The underlying cause of the coma is the most important factor in determining outcome. Acute reversible causes have the best prognosis; these include acute infections, seizure disorders, drug overdose and treatable head injury. Patients with reversible causes may remain unconscious for long periods. However significant recovery is very unlikely in those comatose patients without a reversible cause who remain unconscious for more than 24 hours after admission. The outcome is poorest in those patients with anoxic, ischaemic and structural lesions. In these patients the persistent absence of corneal and oculovestibular reflexes and absence of movement to pain are bad prognostic signs with the chances of meaningful recovery set at <3%.

Key points

- emergency treatment of the cause of coma should be started without delay
- patients with reversible causes can remain unconscious for long periods
- patients comatose for >24 hours without a reversible cause have a poor prognosis

TRANSIENT LOSS OF CONSCIOUSNESS

Transient loss of consciousness is a very common clinical disorder. The main causes are outlined below in Table 9.7. The most common type is vasovagal or simple faints which occur in all age groups. The main differential diagnosis for vasovagal syncope is an epileptic seizure (Table 9.8).

Table 9.7 Common causes of transient loss of consciousness

vasovagal syncope	pseudoseizure
cardiac syncope	hypoglycaemia
postural syncope	transient ischaemic attack (TIA)
epileptic seizure	

Vasovagal Syncope (Faint)

Syncope is a sudden episodic loss of consciousness and postural tone resulting from an acute reduction of blood flow to the brain. Syncope of vasovagal origin is the most common form of transient loss of consciousness and can affect all age groups especially teenagers, young adults and also older persons. It is of cardiovascular origin. There are many contributory causes and syncope is often triggered by prolonged standing in crowded warm, indoor surroundings e.g. schools, churches, hospitals. Psychogenic factors, emotion, tiredness, fear, pain and sometimes even seeing blood, needles or medical procedures may precipitate the syncope or fainting. Rarely, it is provoked by micturition or coughing.

During a typical faint, the person first describes feeling dizzy, hot and cold, sweaty; their vision becomes blurred and darkened and nearby voices become distant. This comes on relatively quickly over 10-15 seconds or at most a few minutes. The attack may be prevented at this stage, by lying flat or alternatively made worse by staying upright in either the standing or sitting position. If the attack is not prevented, there is a sudden loss of consciousness with a falling to the ground (*syncope*). The person then remains unconscious lying motionless, pale and sweaty for a short period, usually for less than 30 seconds but not more than a minute. There may be transient, brief ictal-like limb jerking and very rarely a seizure if there is prolonged anoxia. Bradycardia and dilated pupils are usually present. Persons then regain full consciousness remembering the events leading up to and immediately after the blackout. They may continue to feel sweaty and nauseated but are usually back to normal within 10-15 minutes or less. The attack of syncope may recur on attempting to stand up too soon. The differential diagnosis includes the other causes of transient loss of consciousness and in particular a seizure disorder. Management is one of reassurance and advice to avoid the precipitating factors.

Cardiac syncope

A cardiac cause is suspected when syncope occurs either in relation to exercise or alternatively occurs in the sitting or lying position or in a patient with known cardiac disease. *The syncope is characterized by its sudden onset usually without any warning* and then by an equally rapid recovery of consciousness on falling to the ground, the entire episode usually lasting a matter of seconds. The main causes are arrhythmias e.g. complete heart block and supra ventricular tachycardia and cardiac outflow obstruction e.g. aortic stenosis and hypertrophic cardiomyopathy. Investigations include ECG, monitoring for arrhythmias and cardiac Echo. Management is the treatment of the underlying cardiac disorder.

Cerebrovascular syncope

This is an uncommon cause of syncope. The main causes are basilar artery insufficiency, large vessel occlusive disease and carotid sinus compression or hypersensitivity. *Basilar artery insufficiency is characterized by sudden brief attacks of dizziness and vertigo, and sometimes transient symptoms of lower cranial nerve dysfunction.* These attacks last seconds to minutes and may be coupled with a loss of consciousness which can occasionally be more prolonged. Management is similar to that outlined in cerebrovascular disease (Chapter 5). Carotid syncope is caused by a tight collar or mass in the neck pressing on the carotid sinus or by an abnormally sensitive carotid sinus. It is more common in older males and in those taking antihypertensive medications, in particular beta blockers.

Postural syncope

The main causes are hypovolaemia, antihypertensive medications and polyneuropathies e.g. in DM. These result in either reduced blood volume or autonomic dysfunction. This may occur in any age group but is more common in the elderly on medications for hypertension. *Syncope secondary to posture should be suspected if the patient with a known risk factor complains of light-headedness or loss of consciousness when standing up rapidly which is relieved when either sitting or lying down.* The diagnosis is confirmed by a symptomatic drop in systolic blood pressure of >30 mm Hg on standing as compared to lying. Management includes changing or discontinuing any likely causal medications and giving clear instructions to the patient, concerning standing up gradually and the wearing of support stockings.

Hyperventilation syncope

Hyperventilation is a common cause of dizziness and faintness but rarely causes syncope. It occurs most commonly but not exclusively in teenage or young women, usually in response to stress and anxiety. *It is a benign disorder which is caused by over breathing in response to anxiety.* This results in a respiratory alkalosis and cerebral vasoconstriction which can cause light-headedness, dyspnoea, and a feeling of unreality, and may proceed to blackouts which mimic seizures. The diagnosis should be suspected if the patient is anxious and describes an episode of difficulty of getting their breath before and just as the attack develops. There is typically associated tingling on the lips, the tip of the nose and finger tips and rarely muscle twitches of the fingers, followed by a short episode of loss of consciousness usually lasting seconds. The symptoms can be reproduced by getting the patient to hyperventilate voluntarily and then stopped promptly by rebreathing from a paper bag.

Syncope and seizures

The main differential diagnosis of syncope is with epileptic seizures. Distinguishing these can be difficult but there are some differences. A clear description of the episode from both the patient and an observer is essential. Syncope of vasovagal origin is largely non recurring and tends to happen in stereotyped situations e.g. triggered by standing, heat, pain, blood, straining. Syncope arising from cardiac, posture and stress disorders is recurring but the underlying cause is usually apparent. Seizures are by their nature recurring and happen anywhere. The immediate preceding warning symptoms are different in both types of LOC. Patients with syncope have a characteristic set of vasovagal warning symptoms including light-headedness, nausea, blurred vision sweating and pallor lasting seconds or minutes. This is in contrast to the aura in seizures which if present is usually more stereotyped and of shorter duration lasting a second or less. The duration of LOC is also shorter in syncope lasting seconds to 1-2 minutes

whereas in epilepsy it usually lasts 4-5 minutes or longer. During a syncopal attack the patient lies motionless with only occasional muscle twitching of the limbs and infrequently urinary incontinence and only very rarely if brain hypoxia occurs are there associated tonic clonic like limb movements and tongue biting. In contrast these are the characteristics of a seizure. In syncope recovery of full consciousness occurs within seconds of awakening whereas in seizures there is typically a period of postictal confusion which lasts for as long as 10-15 mins. These differences are summarized in Table 9.8.

Table 9.8 Clinical differences between a fit and a faint

Clinical features	Vasovagal attack	Epileptic seizure
posture	upright	any posture
onset	gradual	sudden
incontinence/tongue biting	rare	common
period/unconsciousness	seconds	minutes
recovery	rapid within seconds	slow
precipitating factor	stereotyped	non stereotyped
recurring pattern	no	yes

Pseudoseizures

Some patients have unexplained seizure-like episodes of loss of consciousness. These episodes resemble seizures and are considered to be psychogenic or non epileptic in origin. The diagnosis should be suspected if there are *atypical episodes of loss of consciousness occurring mostly in teenagers and young adults, often females, usually lasting longer than 5 minutes*. The attacks can mimic an epileptic seizure and may occur in association with known epilepsy. The main differences are already outlined in chapter 4.

HYPOGLYCAEMIA

The diagnosis should be suspected if there are *feelings of hunger, sweating, nervousness and palpitations* coupled with episodes of *confusion, abnormal speech* or *unusual* behaviour in a patient not at risk for seizures or syncope. A blood glucose <2.5 mmols/L is considered to be hypoglycaemia. These attacks occur most frequently in diabetics taking oral hypoglycaemic agents or insulin. Other less common causes include during or after a period of prolonged exercise, fasting, liver disease and malignancies including hepatoma and insulinoma. The loss of consciousness in hypoglycaemia can be prolonged (>30 mins) and seizures may occur. If untreated this may proceed to coma and brain damage. The diagnosis is confirmed by measuring blood glucose during the episode and by its response to treatment. While a blood sugar level should always be checked in the unconscious patient in whom hypoglycaemia is suspected, intravenous glucose should be given without waiting for the results of a confirmatory blood sugar.

TRANSIENT ISCHAEMIC ATTACK (TIA)

A TIA is a sudden ischaemic focal neurological deficit that completely resolves in <24 hours (Chapter 5). The neurological findings in TIA typically last for minutes and occasionally, hours and the presentations are the same as for stroke and include a loss of consciousness. They are mostly caused by thromboemboli arising from the internal carotid arteries or from the heart in atrial fibrillation or mitral valve disease. All TIAs should be treated with the same sense of urgency

as stroke. The aim is to identify risk factors and prevent a stroke occurring as the risk over the following days and weeks is high (>10%). Antiplatelet drugs and anticoagulants are used as in stroke.

Key points

- transient loss of consciousness is a common outpatient problem
- main differential diagnosis is an epileptic seizure
- causes tend to be stereotyped
- history is the key to the correct diagnosis

APPENDIX 1 DESCRIPTIONS OF STATES OF ALTERED CONSCIOUSNESS

Confusion	disturbed consciousness and impairment of higher cerebral function
Delirium	confusion with motor restlessness, and transient hallucinations and delusions
Stupor	conscious but rousable only with intense stimulation
Coma	unrousable unresponsiveness (GCS ≤ 8/15)
Vegetative state	loss of consciousness with preservation of brain stem function
Death	loss of consciousness and capacity to breathe spontaneously: irreversible

APPENDIX 2 EXAMINING THE COMATOSE PATIENT FOR SIGNS OF BRAIN DEATH

Brain death is the irreversible loss of all brain stem function. This is loss of consciousness, respiration, response to pain and loss of all brain stem reflexes. In brain death the following brain stem reflexes must be absent:

- light reflex
- corneal reflex
- gag reflex
- cough to deep tracheal suction
- eye movements to testing (Doll’s eyes)
- oculovestibular reflex (caloric test)

Doll’s eyes or oculocephalic reflex

Check for the normal oculocephalic reflexes by rotating the head 180 degrees to the left or right (or up down) holding it there for 3-4 secs and observing the eyes. In the normal intact brain stem the eyes deviate to the side opposite to that of the head movement. In brain stem disease or deep coma the eyes remain fixed and move in the same direction as the head which is abnormal. The oculocephalic reflex is suppressed in the fully conscious patient so it can only be tested in the unconscious patient.

Caloric testing or oculovestibular reflex

First check that the external canal is clear and the tympanic membranes are intact. Then irrigate the tympanic membrane in each ear with 50-100 mls of iced cold water. The normal response is that the eyes deviate slowly towards the stimulated ear. In brain death and overdose there may be no response or if the brain stem is damaged the eyes may diverge.

Certifying brain death in a patient on a ventilator

In order to declare a patient brain dead whilst on a ventilator ideally two doctors must be certain that all brain stem reflexes are absent and that there is no reversible cause. The ventilation should have continued with the patient in this state for 24 hours. If a patient is on a ventilator there should be no spontaneous ventilation 10 minutes after disconnecting from the ventilator having preoxygenated the patient with 100% O₂ for at least 10 mins.

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