Emergency management of the unconscious patient

Introduction

Acute presentation of an unconscious patient is a common medical emergency and presents a challenge in the emergency department. There is usually some history available and the list of potential causes and hence treatments is considerable. This review looks at the definition, pathophysiology, causes, history, examination and management of the condition.

Definitions

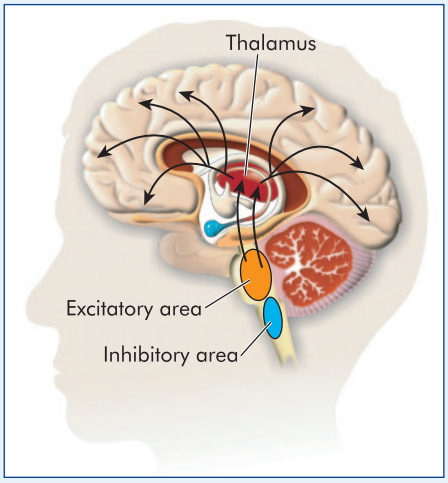
Consciousness is the state of awareness of self and environment after adequate stimuli (Bates, 2003). Normal consciousness is a product of interconnections between the ascending reticular activating substance (ARAS) and neuronal connections between areas of the cerebral cortex (*Figure 1*).

Coma is the state of unarousable unconsciousness without any psychologically understandable response to external stimuli or inner need (Plum and Posner, 1982). It is usually defined using the Glasgow Coma Scale (GCS) (Teasdale and Jennett, 1974) as ≤8. Impaired consciousness is a GCS between 9 and 14. The maximum value is 15 and the minimum 3.

Pathophysiology

Consciousness is composed of content and arousal (Oschatz and Laggner, 1999).

Figure 1. Reticular activating system.



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Content is mediated by the cerebral hemispheres and marked by use of language and purposeful motor function. Diffuse bilateral cortical dysfunction is needed to produce coma. Metabolic disturbances usually cause coma by damaging this area.

The ARAS of the brainstem determines arousal, an on-off switch for consciousness (*Figure 1*). Arousal can still occur if the ARAS is intact despite bilateral cortical destruction. Structural lesions usually cause coma by damaging this area (Plum and Posner, 1982).

History

History is pivotal and, in this scenario especially, needs to incorporate additional information from witnesses, relatives, police and paramedics, whenever possible. Priority is given to airway, breathing and circulation (ABC) and resuscitation should not be interrupted, so history taking, resuscitation and examination often occur together.

History can be particularly helpful in determining the speed with which coma occurred, thereby acting as pointer to the underlying diagnosis (*Tables 1* and *2*).

A previous history of head injury, trauma, epilepsy, hepatic, renal, cardiac and endocrine problems as well as one of drug misuse will help reach a diagnosis.

Examination

General

Signs suggesting the underlying cause of coma may lie in any system and a full

Table 1. Causes of coma

Bilateral cortical disease process	Trauma	Head injury
	Hypoxia/anoxia	Cardiorespiratory arrest, global cerebral ischaemia, sinus thrombosis
	Infection	Meningitis, encephalitis, cerebral malaria, abscess
	Sub-	Subarachnoid haemorrhage, subdural haematoma, extradural haematoma, cerebral haemorrhage, cerebral infarction
	Metabolic	Diabetic ketoacidosis, hyperosmolar diabetic coma, hypoglycaemia, hyper-/hyponatraemia, hypo-/hyperthermia, hypercalcaemia
	Organ failure	Hepatic encephalopathy, renal failure
	Post-ictal	Epilepsy or seizures
	Endocrine	Thyroid storm, myxoedema coma, adrenal insufficiency
	Drugs	Alcohols, opiates, cocaine, ecstasy, carbon monoxide, benzodiazepines, antidepressants, barbiturates, methaemoglobinaemia
Brainstem	Supratentorial/ infratentorial/ intrinsic lesion	Subdural haemorrhage, extradural haemorrhage, intracranial haemorrhage, ischaemia with oedema, infection with oedema, tumour with oedema, trauma with oedema

Table 2. Onset of coma

Speed of onset	Cause	
Acute (seconds to minutes)	Cerebral vascular disease	Haemorrhagic
		Ischaemic
	Cardiac arrest	
	Subdural or extradural haemorrhage	History of head injury
Subacute (minutes to hours)	Sepsis or infection	
	Drugs, hypoglycaemia	
Protracted acute (hours to days)	Mitotic lesion	
	Metabolic	

examination is vital. The fingertips should be examined for evidence of capillary blood glucose monitoring (hypoglycaemia in a diabetic). The skin should be inspected for a rash, jaundice, bruising and puncture wounds (drug misuse). The head and scalp should be carefully examined for signs of head injury, including those of a basal skull fracture (CSF rhinorrhoea, CSF otorrhoea and raccoon eyes). Neurological examination may further help the clinician make a broad distinction between a toxic-metabolic cause or a structural cause of coma. Approximately one third of comas of unknown aetiology are attributed to mass lesions, one third to drug overdoses and the remaining third to diffuse metabolic causes (Plum and Posner, 1982).

Level of consciousness

The GCS (*Table 3*) is the most widely used tool for assessment of level of coma. Documentation of the GCS both initially and serially provides a valuable indicator for further management. A GCS <9 is consistent with the definition of coma.

Patients with a metabolic problem often have milder alterations in consciousness and fluctuate, whereas structural lesions are associated with a stable or progressively deteriorating GCS.

Table 3. Glasgow Coma Scale

Glasgow Coma Scale		Score (total 15)
Eye opening	Spontaneous	4
	To speech	3
	To pain	2
	Nil	1
Verbal response	Orientated	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	Nil	1
Best motor response	Obeys command	6
	Localizes to pain	5
	Withdraws	4
	Abnormal flexion	3
	Extension	2
	Nil	1

From Teasdale and Jennett (1974)

Respiration

Abnormal breathing patterns can help localize level of involvement.

- Kussmaul breathing – deep regular respiration seen in metabolic acidosis
- Irregular respiration with random deep and shallow breaths, associated with medullary lesions
- Prolonged inspiratory gasp with a pause at full inspiration suggestive of a brainstem lesion
- Cheyne–Stokes respiration – a relatively non-specific pattern of alternating apnoea and tachypnoea.

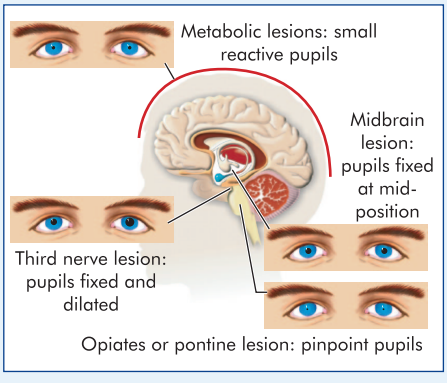
Pupils

Pupil size (*Figure 2*) and reactivity (*Table 4*) can help delineate structural from metabolic causes of coma and provide a warning of impending brainstem herniation.

As a general rule, structural lesions tend to be associated with pupil asymmetry and loss of the light reflex, whereas metabolic disturbances are characterized by small pupils with preserved light reflex.

It is important to remember that a sluggishly reactive pupil may be one of the first signs of herniation, before unilateral dilatation becomes apparent. Misleading causes of a unilaterally dilated pupil must not be forgotten, including prior administration of mydriatics, previous eye trauma or surgery, and carotid insufficiency.

Figure 2. Pupil signs.



Eye movements

The resting position of the eyes and both spontaneous and reflex movements should be assessed.

- Conjugate deviation from the midline suggests an ipsilateral cerebral hemisphere lesion or a contralateral pontine lesion
- 'Sunsetting' deviation – midbrain compression from above (herniation) and metabolic disturbances
- Horizontal slow eye roving – mild metabolic.

Reflex eye movements usually remain intact in toxic-metabolic coma compared with structural causes (unless deep coma or rarely with phenytoin). The oculocephalic (doll's head) reflex (caution suspected cervical injury) is tested by rotating the head side to side; the response is normal if the eyes move conjugately in the opposite direction to the head and indicates an intact pons.

If cervical trauma is suspected the caloric oculovestibular reflex can be tested by putting ice-cold water into the external ear canal (after excluding tympanic membrane rupture) – a tonic (slow) movement towards the stimulated side is a normal response in the unconscious patient, indicating an intact brainstem. The corneal reflex is usually preserved until the late stages.

Fundoscopy should be undertaken to look for signs of raised intracranial pressure (ICP) with loss of retinal vein pulsation being an early sign, as papilloedema can be a late manifestation.

Table 4. Pupil reactivity

Pupil size and symmetry	Response to light	Causes
Dilated, unilateral	Non-reactive	Cranial nerve III damage (herniation or other trauma) Posterior communicating artery aneurysm
Dilated, equal	Non-reactive	Significant brainstem damage
Mid-size, equal	Non-reactive, may demonstrate hippus	Midbrain lesion
Small, equal	Reactive, may be unduly brisk	Metabolic disturbance, thalamic lesion
Constricted, unilateral	Reactive	Horner's syndrome (lateral medullary syndrome or carotid artery damage)
Pinpoint, equal	Reactive	Opiates, pontine lesion

Motor function

This is an important part of the assessment and occurs as part of the GCS. Spontaneous movements should be looked for initially. If there is no response then a stimulus should be applied. The response will give vital clues as to the site of pathology. Asymmetry implies a contralateral focal cerebral lesion.

Abnormal movements such as decorticate (flexion of elbows and wrists with extension of legs) and decerebrate (extension of arms and legs) indicate lesions above the midbrain in the cerebral white matter and brainstem respectively (Bleck and Webb, 1999). Prognostically, decerebrate movement is worse than decorticate.

Immediate management

Once unresponsiveness is established, the airway must be protected while maintaining stabilization of cervical spine if there is any suspicion of trauma. Supplemental oxygen should always be given and breathing may need to be assisted.

Then the circulation should be assessed, intravenous access established, blood taken for estimation of blood glucose levels and samples sent for haematological, biochemical, microbiological and toxicological analysis, including full blood count, urea and electrolytes, paracetamol and salicylate levels, clotting, liver function tests and blood gases. An electrocardiogram and a chest X-ray are also necessary. Serum insulin and C-peptide levels will help with the diagnosis of an insulinoma in spontaneous hypoglycaemia. If hypoglycaemic, dextrose (50 ml 50%) is given. If alcohol abuse or malnutrition is suspected, thiamine should precede glucose to prevent precipitation of Wernicke's encephalopathy. If tissue perfusion is inadequate fluid boluses are given.

Small pupils and hypoventilation suggests opiate intoxication and naloxone (0.4–2 mg repeated at 2–3-minute intervals to maximum of 10 mg if respiratory function does not improve) should be given.

Flumazenil (a pure competitive benzodiazepine antagonist) may reverse benzodiazepine-induced sedation but risks precipitation of arrhythmias and seizures in acute benzodiazepine withdrawal, especially in patients who have concomitantly taken proconvulsant or proarrhythmic drugs, e.g. tricyclic antidepressants, chloral hydrate or carbamazepine. After a risk–benefit analysis and careful consideration flumazenil can be given slowly and in small doses (initially 0.2 mg, up to 1 mg). This ensures only 50% of benzodiazepine receptors will be occupied (Hoffman and Goldfrank, 1995).

Seizures

Prolonged seizures should be treated with intravenous lorazepam 4 mg or diazepam 10–20 mg (2.5 mg per 30 seconds).

Further management

Depending on the history and examination findings, thyroid function tests, carboxyhaemoglobin levels, malaria film and plasma osmolarity (increased in methanol, ethylene glycol and isopropyl alcohol) may be required.

Head injury and raised ICP

A computed tomography (CT) scan should be done if there is a deterioration in GCS, focal neurology, meningism, raised ICP or if a significant head injury is suspected. Brain injury severe enough to cause coma can cause a 40–50% reduction in cerebral blood flow during the first 6–12 hours after injury. Reduction in blood flow to the injured brain leading to hypoxia will exacerbate the underlying pathology, as will hypercarbia and hypotension resulting in secondary brain injury. Therefore give supplementary oxygen, aim for a mean arterial pressure of 80–95 mmHg, and a partial pressure of carbon dioxide (CO_2) between 4 and 4.5 Kpa.

In a patient with a GCS of 8 or less, it is best that the airway is secured with an endotracheal tube (prevents aspiration, ensures adequate oxygenation and helps control CO_2 levels, thereby reducing ICP).

Intravenous fluids should be only used if needed and to maintain normovolaemia. It is best to use normal saline as hyperglycaemia from dextrose-containing fluids is harmful to the injured brain.

Coma with meningism or rash

If clinical features suggest bacterial meningitis, antibiotics should be given immediately and blood cultures sent thereafter.

Meningeal irritation may also be caused by a subarachnoid haemorrhage in which case a CT scan should be requested and depending on the timing and result, a lumbar puncture may be needed. Abdominal examination may reveal enlarged polycystic kidneys.

If there is a high temperature (usually above 39°C) or a seizure (especially focal) then it is prudent to administer intravenous acyclovir.

Malaria should be excluded on blood films in patients who have travelled through endemic areas.

Conclusions

The management of the unconscious patient should be done in an expedient yet systematic manner, with priority given to ABC. Airway compromise is a grave danger and anaesthetic involvement should occur early. History, examination and resuscitation are often done simultaneously, looking to treat easily reversible causes such as hypoglycaemia. BJHM

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KEY POINTS

- The unconscious patient is a common medical emergency.
- Priority should always be given to airway, breathing and circulation.
- Always check glucose as this is an easily reversible cause and delay in giving dextrose can be extremely detrimental to the patient.
- In a coma patient with a history of alcohol abuse always give thiamine before the glucose to prevent Wernicke's encephalopathy.
- Be cognisant of the causes of coma – it may help to remember the mnemonic THIS MOPED.