Guidelines

For The Management Of

Coma

By

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Setting Clinical and Professional Excellence
Coma is a symptom, not a diagnosis. It may be a transient phenomenon during acute illness or persist in the long-term. It is a loss of consciousness involving a disturbance of awakness (a function of the reticular formation in the brainstem) and awareness (a function of cerebral cortex). Coma can result from mechanical destruction of crucial areas of the brain stem or cerebral cortex (anatomic coma) or from widespread (global) disruption of brain metabolic processes (metabolic coma).

Head trauma, drug use, epilepsy, and brain infections are the most common causes of coma in individuals less than 40 years of age. Cardiovascular disease (especially stroke) and metabolic disorders (e.g., diabetes mellitus, hypoglycemia, coma from liver failure, electrolyte disorders, and uremia) are common causes in those over 40.

The aim of immediate management is to minimize any ongoing neurological damage whilst making a definitive diagnosis through a comprehensive assessment starts with detailed history and neurological and systemic examination.

**History and examination**

*Details of the individual's medical history and circumstances surrounding the onset of coma and subsequent events may be gathered from the individual's family members, bystanders, paramedics or even the police like past history of hypertension, seizures, diabetes, infection, cardiac, previous similar episodes or drug history.*

If the patient is unaccompanied; search for personal I.D, disease or drug label.

*Baseline observations - BP, PR, temperature (rectal ideally), RR, oxygen saturation, serum glucose and the Glasgow Coma Scale (GCS).*

*Breathing patterns abnormality (cheyne–Stokes, cluster breathing and central neurogenic hyperventilation) may point to damage at different levels of the brain or brain stem.*

*Examine the skull and spine and test for neck stiffness and Kernig's sign (if there is no cervical spine trauma).*

*Check for presence of sugar in any secretion from the nose or ear to exclude CSF leak*

*Smell: Classic foul breaths are dirty toilet (uraemia), fruity sweat (ketoacidosis), fishy (acute hepatic failure), onion (paraldehyde), garlic (organophosphates) and alcohol.*

*Posture & response to painful stimuli, as withdrawing a limb is a likely indication that the sensory and motor nervous pathways are intact. Abnormal body posturing (decorticate or decerebrate) or no movement in response to painful stimuli suggests serious damage at the level of the brain stem.*
Asymmetry in movement or reflexes may indicate structural damage to one side of the brain.

* Corneal & Gag reflex
* Fundoscopy & pupil examination for abnormal movements, size, response to light stimulus. The presence of a single, dilated pupil (anisocoria) is a serious sign and may indicate increased intracranial pressure or extradural haematoma.

Tiny pupils that respond to light tend to occur in metabolic derangements. Structural brain lesions or non-convulsive status epilepticus can cause the eyes to be positioned to one side or in a downward gaze.

* Primary survey of skin and mucous membranes - scalp bruising or haematoma any evidence of hyperpigmentation, sepsis, myxoedema, intravenous or subcutaneous drug marks, anaemia, jaundice, purpura

**Investigations**

- Glucose
- Urea and electrolytes
- Liver function test
- Full blood examination
- Arterial blood gas
- Thyroid function tests
- Blood smear for schistocytes
- ECG & cardiac enzymes
- Culture of blood and urine
- Blood and urine toxicology screen
- Coagulation screen
- CT scan or MRI
- CT-Angiogram (or MRA) in suspected acute basilar artery occlusion or SAH.
- Lumbar puncture may be done if a CT scan or MRI shows no lesions or tumors to help diagnose an infection or hemorrhage in the brain. Check opening pressure and send CSF for protein, cells, glucose, G-stain, culture, India ink stain, cryptococcal antigen, viral PCRs.
- EEG in selected patients e.g. suspected non-convulsive status

**The Glasgow coma scale GCS** is a practical and standardized system for assessing the degree of coma by scoring a patient's conscious level. It is also useful in monitoring progress of the patient and predicting the duration and ultimate outcome, especially in
individuals with head trauma.
The GCS is determined by assessing three aspects: eye opening (4 levels), verbal response (5 levels) and motor response (6 levels).
If the patient gets a score of:
13 to 15 correlates with a minimal impairment such as a concussion
9 to 12 correlates with a medium level of impairment
8 & lower indicate severe reduction in consciousness and the patient may be unable to maintain their airway spontaneously, thus should be intubated and ventilated.

**Immediate management**

- Attention to airway, breathing and circulation (ABC).
  Assess and monitor pulse, respiratory rate, BP, temperature, oximetry, ECG monitoring and conscious state using GCS. Proper oxygenation aiming at a pulse oximeter saturation of >95%
- If traumatic cause is possible; immobilize cervical spine and arrange urgent neurosurgery involvement.
- Intubate if patient cannot protect the airway e.g. GCS 8 or less
- Correct hypotension with IV crystalloids and extreme hypertension (systolic above 250 mm Hg or mean arterial pressure above 130 mm Hg) with:
  labetalol (10 mg IV), hydralazine (10 mg IV) or nicardipine (5 mg/h).
  \[ MAP = DP + \frac{1}{3} (SP-DP) \quad \text{OR} \quad \frac{2xDP + SP}{3} \]
- Perform blood glucose; if glucometer <45mg/ml (2.5 mmol/l) in a non-diabetic, send specific bloods tests, administer dextrose with IV thiamine (see below)
- Control seizure: lorazepam 4mg I.V slowly, medazolam 5mg I.V slowly or diazepam 10-20mg I.V slowly. Follow status epilepticus pathway.
  If no response: call anesthetist for help and administer phenytoin 15-20mg/kg at a rate of 50mg/min, phenobarb 15-20mg/kg at a rate of 50mg/min or sodium valproate 200-400 mg I.V.
- Thiamine hydrochloride 100 mg I.M or I.V with dextrose solutions in alcoholics or others in a malnourished state or in case of vomiting preceding coma. Give intravenous thiamine and glucose to all in whom diagnosis is unclear. Remember to give thiamine first, as glucose may cause Wernicke's encephalopathy in those who are malnourished
- Consider naloxone 0.1 mg/kg (max. 2 mg) I.V. (repeat every 3 min intravenously if opioid intoxication is suspected).
• Treat severe hyponatraemia with hypertonic saline and furosemide (3% hypertonic saline, 0.5 mg/kg hourly) after placing central venous catheter.
• Reduce suspected increased intracranial pressure with osmotic agents; mannitol, initial dose 1–2 g/kg IV followed by 0.25-0.5g/kg repeated every 2-6 hrs as required. Dexamethason phosphate: IV 10-50 mg (0.5-1.5 mg/kg) then IM/IV 4-20 mg (0.2-0.5 mg/kg) every 6 hrs. (Seek neurologist and anesthetist advice). Reducing carbon dioxide by raising the respiratory rate (hyperventilation) helps decrease intracranial pressure. In some cases, surgical insertion of an intracranial pressure monitor may be needed.
• If CNS infection is even a remote possibility, start full coverage with cephotaxime (8–12 g daily, divided doses every 6 h I.V), vancomyocin (2 g daily, divided dose every12 h I.V) and ampicillin (12 g daily, divided dose every 4 h I.V) in combination with intravenous aciclovir (10 mg/kg every 8 h).
• Further management depends on the cause & progress of coma.

Rehabilitation
Rehabilitation should begin almost immediately in acute coma, with passive range of motion and frequent turning to avoid limb contractures and bedsores.

Failure to Recover: Points of consideration
• Was there any extended delay between the onset of symptoms and treatment?
• Was necessary life-saving support (airway, breathing, cardiac compressions) provided as appropriate?
• Was treatment appropriate for the underlying condition?
• Was surgery required?
• Would individual benefit from consultation with a physiatrist or specialist in neurological rehabilitation?

AVPU Scale
A scale used to measure a patient's conscious level. It is simpler than the GCS and can be used by doctors, nurses, first aiders and ambulance crews. Four elements are tested:
• A lert - meaning spontaneous eye opening, speaking and intact motor functions,
• V oice - responds when spoken to, eg grunt or actual speech.
• P ain - responds to pain, eg sternal rub.
• U nresponsive - if no response to pain, ie no eye, voice or motor movement.
Ambulance crews usually use AVPU initially and, if the patient scores anything other than an 'A', they record a formal GCS.

References
   www.patient.co.uk/doctor/Coma.htm
   www.tandurust.com/neurological-disorders/coma-causes-diagnosis-treatment-management.html
   www.aan.com/globals/axon/assets/4458.pdf
8. www.mdguidelines.com/coma