Clinical approach to comatose patients

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Abstract

The nature of consciousness itself belongs within a group of 'underdetermined questions' to which we might not be able to find an answer. Similarly, we have a limited understanding of disorders of consciousness. In this brief article, we discuss a practical approach to the comatose patient and the importance of promptly identifying the cause to prevent permanent neurologic damage.

Keywords Coma; GCS; FOUR score; consciousness; neurological assessment; prognosis

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Learning objectives

After reading this article, you should be able to:

- understand the definition of consciousness and coma
- use an organized approach to identify the cause of coma
- initiate effective and comprehensive early management
- identify conditions that can mimic coma
- identify relevant prognostic factors in acute coma

Introduction

Consciousness remains one of the major unsolved questions, despite being the subject of intense study for many centuries. In the 19th century, autopsy studies of patients with impaired conscious states (i.e. encephalitis lethargica and Wernicke's encephalopathy) identified lesions in the upper brainstem and diencephalon. The role of these structures in maintaining wakefulness was later confirmed by a series of elegant experimental studies by Moruzzi and Mogun. However, it was only with the advent of mechanical ventilation and the consequent possibility of closely observing comatose patients that two fundamental components of consciousness were described: arousal and awareness. Arousal is mediated by the reticular system and projections to the thalamus, whereas awareness is mediated by the thalamus and the cerebral cortex.

Coma (from the Greek 'κομα' to fall asleep) is a deep state of unconsciousness characterized by the absence of arousal and awareness. Neuroimaging studies have shown that regardless of the aetiology, coma is associated with a marked and diffuse reduction in metabolism, suggesting that coma is a state of transient 'cerebral energy failure'. Coma is typically a transitional state evolving towards recovery of consciousness, minimal conscious state (fluctuating minimal level of consciousness), unresponsive wakefulness syndrome (preservation of arousal, but the absence of awareness) or brain death.

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Clinical assessment of conscious state

A number of different coma scales have been devised for the clinical assessment of consciousness (e.g. Innsbruck coma scale, Edinburgh-2 coma scale and Reaction level scale). However, the Glasgow Coma Scale (GCS) remains the most widely used. It comprises three subscales: eye opening, motor function and verbal abilities. Coma is defined as a post-resuscitation GCS score of 8 or less lasting for more than an hour. The GCS score should be determined, focussing on individual component scoring rather than the overall score. The examiner determines the level of responsiveness with stimuli of increasing intensity, starting with verbal cues. If no response is elicited, noxious stimuli can be delivered to the sternum, trapezius, supraorbital nerve, temporomandibular joints or to the peripheries. This enables the examiner to test for localization of pain and abnormal motor responses. Despite its widespread use, the GCS has limitations in the assessment of patients who are intubated or have craniofacial trauma. An alternative to the GCS is the *Full Outline of UnResponsiveness* (FOUR) *scale* which is comprised of four subscales assessing: motor, ocular responses, brainstem reflexes and breathing (Table 1). Compared to the GCS score, the FOUR score has a lower inter-observer variability and provides a tool to identify patients with locked-in syndrome.

Management of coma in the first hour

The initial step should be directed to ensuring adequacy of resuscitation measures, and to rapidly treat reversible causes of coma. Urgent and empiric therapy must be given to avoid additional cerebral insult. Oxygenation must be assured by establishment of an airway and ventilation of the lungs. Maintenance of a $PaCO_2$ in the low–normal range (35–40 mmHg) may have a therapeutic effect on decreasing intracranial pressure, while not being so low as to compromise cerebral oxygenation. High-concentration supplemental oxygen therapy is to be given to prevent cerebral hypoxia. Circulatory hypotension requires intravenous resuscitation with fluid volume infusion. Appropriate fluid resuscitation, aiming for a mean arterial pressure at about 100 mmHg, is an adequate target for most patients. As hypoglycaemia is a frequent cause of altered consciousness, give glucose 25 g as a 50% solution intravenous accompanied with thiamine 100 mg to prevent precipitation of Wernicke's encephalopathy. If opiate toxicity is suspected then administer naloxone 0.4–2 mg intravenously repeated as needed up to 4 mg. Benzodiazepine intoxication can be reversed with flumazenil, with caution, so as to not induce a withdrawal seizure. A rapid initial survey should follow, looking for injuries or other notable physical signs (ecchymoses, jaundice, rashes, and needle track marks). If trauma is suspected the cervical spine should be immobilized. Once the patient is stabilized a search for the causes

Scales used for the assessment of conscious state

Full Outline of UnResponsiveness (FOUR) scale

Eye response	
Eyelids open or opened, tracking, or blinking to command	4
Eyelids open but not tracking	3
Eyelids closed but open to loud voice	2
Eyelids closed but open to pain	1
Eyelids remain closed with pain	0
Motor response	
Thumbs-up, fist or peace sign	4
Localizing to pain	3
Flexion response to pain	2
Extension response to pain	1
No response to pain or generalized myoclonic status	0
Brainstem reflexes	
Pupil and corneal reflexes present	4
One pupil wide and fixed	3
Pupil OR corneal reflexes absent	2
Pupil AND corneal reflexes absent	1
Absent pupil, corneal and cough reflex	0
Respiration	
Own airways, regular breathing pattern	4
Own airways, Cheyne—Stokes breathing pattern	3
Own airways, irregular breathing	2
Breathes above ventilator rate	1
Breathes at ventilator rate or apnoea	0

Glasgow Coma Scale

Eye opening	
Spontaneous	4
To voice	3
To pain	2
No response	1
Verbal	
Oriented	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
No response	1
Motor	
Obeys commands	6
Localizes to pain	5
Withdrawal to pain	4
Flexion posturing	3
Extension posturing	2
No response	1

Table 1

of the coma begins with serologic investigations and arterial blood gases to identify potentially reversible metabolic or toxicological causes.

History

Collateral history should focus on: (a) the time course of the alteration in consciousness (abrupt onset suggests a stroke, seizure, drug poisoning or trauma, whereas a gradual onset is more suggestive of a metabolic process); (b) the presence of premonitory signs (headaches or focal signs prior to coma); (c) the setting (objects in the vicinity of the patient such as empty medicine bottles); and (d) active and past medical and surgical relevant conditions.

Neurological assessment

The initial assessment should focus on the detection of lateralizing signs that suggest a focal lesion and require rapid imaging evaluation. In examining a comatose patient, confounding factors such as sedation, muscle paralysis, hypothermia, shock and drug intoxication should be taken into consideration (Table 2). For instance, sedation can not only suppress brainstem reflexes (opioids and midazolam are more suppressive than propofol and dexmedetomidine), but also can generate false localizing signs (i.e. phenytoin suppression of oculocephalic and vestibulo-ocular reflexes).

Fundoscopy and pupillary responses

Fundoscopy may reveal important signs such as papilloedema (i.e. raised intracranial pressure or acute asphyxia) and subhyaloid haemorrhage (subarachnoid haemorrhage and shaken baby syndrome). Visual fields and neglect can be grossly assessed with the blink threat response. The size, symmetry, shape and reactivity of the pupils should be noted. A unilateral dilated pupil suggests pressure on the oculomotor nerve (i.e. uncal herniation or a posterior communicating artery aneurysm). Unilateral miosis is suggestive of Horner's syndrome. Bilateral fixed midposition pupils are a sign of extensive midbrain injury. Bilateral pinpoint pupils are often due to narcotic overdose, a ponto-tegmental lesion, or cholinergic toxicity. Pupillary reflexes and ciliospinal reflexes are preserved in metabolic coma.

Ocular motor function

The primary position of the eyes should be documented. Eyes found to be heterotropic (non-parallel) must be classified according to their relative positional defect: esotropia (one eye deviated towards nose), exotropia (one eye deviated towards temple) and hypertropia or skew deviation (one eye deviated upwards). Two patterns are commonly encountered: oculomotor

Half-lives of medications that affect neurological assessment of coma

Medication	Half-life (hours)
Phenobarbital	100
Diazepam	40
Amitriptyline	24
Lorazepam	15
Thiopental	10
Midazolam	6
Fentanyl	6
Morphine	3
Rocuronium	1
Atracurium	0.5

Table 2