EC NEUROLOGY EDITOR'S COLUMN - 2019

Coma, Overview of Anatomic Localization

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COLUMN ARTICLE

Introduction

Coma is a medical emergency and its management is an important part of all doctor's clinical practice.

Neurologists are specially involved in evaluation of such patients.

Clinical evaluation and examination of comatose patient must follow a standard protocol.

History and examination is important in correct diagnosis and management of the underlying condition.

In the following sentences, we will discuss some points and pearls of coma management.

We need to review some definitions, first:

- Consciousness: State of awareness of self and environment.
- Alertness: An alert patient is fully conscious and aware of their environment.
- Confusion: Inability to think with customary speed, clarity, and coherence.
- Lethargy: Difficult to maintain the arousal state.
- Obtundation: Responsive to stimulation other than pain.
- Stupor: Responsive only to pain.
- Coma: The patient is unconscious, unaware, and unresponsive to external stimuli.
- Delirium: Clouding of consciousness with reduced ability to sustain attention to environmental stimuli.
- Encephalopathy: A broad term used to denominate a syndrome of diffuse cerebral dysfunction.

Neuroanatomy of consciousness

Maintaining alertness is primarily a function of the ascending reticular activating system (ARAS), which projects from the brainstem tegmentum through synaptic relays in the rostral intralaminar and thalamic nuclei to the cerebral cortex.

Coma, overall approach to the patient

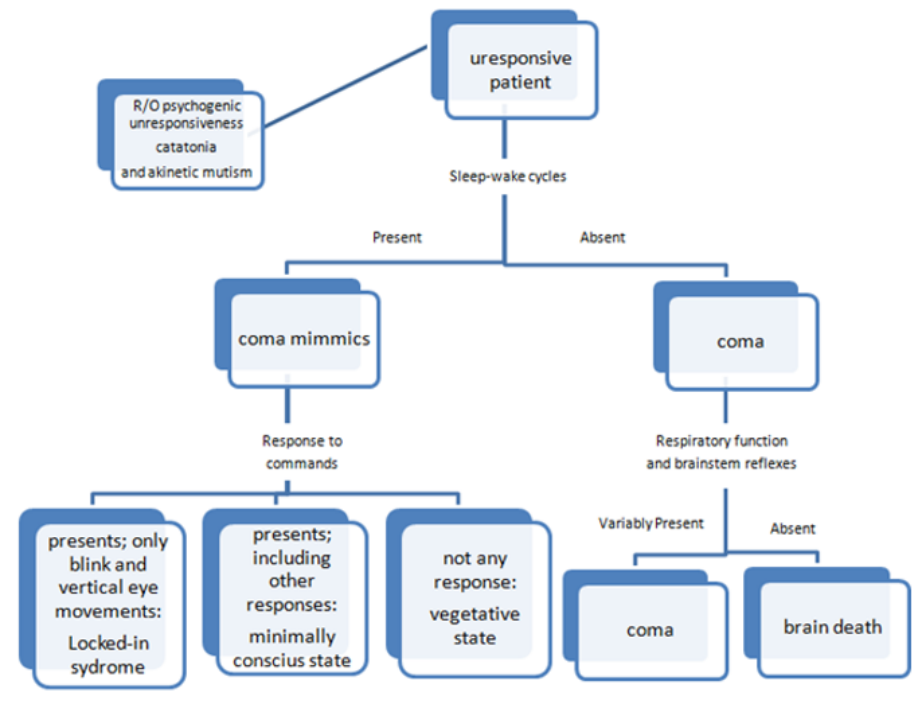
Several different states of impaired cognition or consciousness may appear similar to coma or be confused with it.

Differentiation of these states from true coma is important to allow administration of appropriate therapy and help determine prognosis.

Algorithm 1 summarizes useful clinical clues to differentiate coma from its mimics.

When coma is confirmed, the patient should be classified into one of the following groups:

Citation: Hadi Gharebaghian Azar. "Coma, Overview of Anatomic Localization". EC Neurology ECO.02 (2019): 21-24.



Algorithm 1: Approach to unresponsive patient.

1. Structural coma
2. Metabolic coma
3. Pseudocoma.

History and systemic examination

1. After the patient becomes stable, take history from accompanies
   a. Determine the course
   b. Similar events that happened before
   c. Medical and social history
   d. Drug history and substance abuse
   e. Symptoms before onset of coma
2. Important systemic examination: vital signs, meningeal irritation, general appearance, evidences of trauma, examination of skin, nails, mucosal membranes, respiration (including odor), heart and lung auscultation.

Neurologic examination

The purpose is to differentiate structural from non-structural causes and then to localize structural causes within neuraxis and consists of four major parts:

a. Respiratory pattern
b. Pupils
c. Ocular movements
d. Motor response.

Respiratory pattern with associated localizing value

- **Cheyne-Stokes respiration:** Diffuse forebrain damage, upper brainstem damage, metabolic encephalopathy, impending transtentorial herniation.
- **Central neurogenic hyperventilation:** Lesions of rostral brainstem (hyperventilation is more common in metabolic acidosis, pulmonary congestion, hepatic encephalopathy and analgesic overdosage).

- **Apneusis:** Pontine lesions specially infarct, rarely with metabolic coma or transtentorial herniation.
- **Cluster breathing:** Lower pontine tegmental lesion.
- **Ataxic breathing:** Lesion of the reticular formation of the dorsomedial part of the medulla.

Pupils

Anisocoria is always considered pathologic in comatose patient and represents an abnormality in autonomic innervation.

Various patterns of pupillary abnormality with their localizing value

- 3rd nerve compression: Dilated fixed.
- Sympathetic system damage (hypothalamus, medulla, cervical ganglion…): Horner's syndrome.
- Thalamus: Small reactive
- Midbrain: Midposition, fixed
- Pons: Pinpoint reactive.

Metabolic diseases

Usually small reactive, (dilated unreactive pupils after anoxic coma suggests a bad prognosis).

Metabolic diseases do not affect pupil reactivity (except for anticholinergic and barbiturate poisoning and hypothermia).

Opiate poisoning causes small reactive pupils.

Unilateral or bilateral mydriasis can be seen during or soon after epileptic seizures.

Ocular movements

Conjugate deviation:

a. To the side of hemiparesis: destructive cerebral lesion.
b. To the opposite side of hemiparesis: pontine lesion, adversive seizure, thalamic hemorrhage.
c. Downward: thalamic or midbrain lesion, metabolic (barbiturate poisoning).
d. Ping-pong gaze: bilateral cerebral infarct or cerebellar ICH.
e. Bobbing: pontine, cerebellar, metabolic.

1. Disconjugacy: paresis of specific EOMs, INO, unmasked preexisting phoria or tropia, skew deviation (cerebellar or pontine lesion).
2. Nystagmus.
3. Oculocephalic and oculovestibular testing.

Motor response

1. Spontaneous movements: myoclonus, clonic jerking, automatism.
2. Muscles tone (hypotonia, hypertonia, paratonia).
3. Simple motor commands.
4. Response to noxious stimuli.
5. Posturing: Decorticate and decerebrate (often with hemispheric lesions or metabolic coma).

Flexor response implies more rostral lesion and better prognosis.

CONCLUSION

In conclusion, we can classify the comatose patient and localize the possible underlying lesion based on the mentioned clinical features that are summarized in table 1.

Further evaluation, treatment and determination of prognosis is completely related to these results.

Dysfunction	Motor response to noxious stimulus	Pupils	Eye movements	Breathing
Both cortices	Withdrawal	Small, reactive	Conjugate gaze deviation, roving movements, normal response to OCR and caloric	Cheyne-Stokes respiration
Thalamus	Decorticate	Small reactive	Same as above	Same as above
Midbrain	Deco': cate or decerebrate	Midposition, fixed	Adduction deficit (CNIII damaged)	Usually same as above, rarely CNH

Pons	Decerebrate	Pinpoint, reactive or Homer's syndrome	Loss of conjugate horizontal movements, conjugate contralateral or inward deviation	CNH, cluster, and apneustic breathing
Medulla	Weak leg flexion or none	Usually small, Homer's syndrome	No effect on spontaneous eye movements; may interfere with OCR and caloric	Ataxic respiration
Metabolic	Symmetric spontaneous or pain induced movements	Small, reactive	Roving eye movements, normal OCR and caloric	Normal or rapid due to metabolic acidosis

Table 1: Coma, overview of lesion localization.