Initial Diagnosis and Management of Coma

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KEYWORDS

• Coma • Coma mimics • Pathophysiology

KEY POINTS

- Coma is a life-threatening process that requires immediate stabilization and a structured approach to diagnosis and management.
- The differential diagnosis for coma is long, but is often divided into structural vs. diffuse neuronal dysfunction; the latter is subdivided into toxic vs. metabolic.
- When available, historical information may be of great use in determining the etiology of coma; in all cases, a focused physical examination can help greatly refine the differential diagnosis.
- The definitive treatment of patients with coma is ultimately disease-specific.

INTRODUCTION

Many patients present to the emergency department with an alteration in mental status simply as a complication of many serious illnesses. A subset of these patients will present comatose, a clinical state that is a true medical emergency. Although coma is a relatively rare presenting condition in the emergency department, patients who present with coma are often in extremis and necessitate immediate evaluation and stabilization.

The approach to coma by the emergency physician is described, beginning with a discussion of pathophysiology and cause. Then, the practical clinical aspects of coma are addressed, including initial stabilization, obtaining the correct historical information, performing a thorough physical examination, ordering appropriate testing and imaging studies, and providing appropriate treatment.

PATHOPHYSIOLOGY

A neuronal network in the dorsal pons and midbrain give rise to the ascending reticular activing system (ARAS), which is responsible for arousal.[1] Neurons from these centers

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run together through the thalamus and then to the bilateral cerebral cortex; the cortex controls sensory processing and understanding, which generates awareness.[2,3] Coma results from an impairment of this axis by a process that affects the brain's arousal center, consciousness center, the tracts that connect them, or some combination thereof. Patients are, therefore, not aware and not awake. Importantly, coma from cortical impairment can only result from a bilateral insult; unilateral cortical deficits do not cause coma. Prolonged coma may result in awakening cycles (eyes open coma) without awareness. Because the comatose state is difficult to quantify, some patients diagnosed as comatose may be minimally aware (minimally conscious state) and others may be more aware than can be assumed or tested.

Although the final common physiologic pathway of coma is neuronal dysfunction in the ARAS-thalamic-cortical pathway, it is useful to subdivide the pathophysiology into structural versus diffuse neuronal dysfunction. Structural causes of coma are defined as those that precipitate cellular dysfunction through a mechanical force, such as pressure on key area or a blockade of delivery of critical cellular substrate. Diffuse neuronal dysfunction precipitates coma by abnormalities only at the cellular level and may be further divided into two general categories: toxic and metabolic. In a toxin-induced coma, an exogenous substance is responsible for the clinical findings; in a metabolic coma, a perturbation of an endogenous process, such as temperature or sodium regulation, has gone awry.

This classification, although useful, does have limitations. A metabolic process, such as hypoglycemia or hypoxia, may initially produce coma through diffuse neuronal dysfunction; however, if the process is uncorrected and cell death occurs, the cause of coma becomes structural. Similarly, a diffuse neuronal process, such as cerebral edema, may become a structural problem if the edema occludes vessels in the posterior circulation and produces brainstem ischemia.

CAUSES

A causal overview of coma is presented in **Table 1**, categorized based on this logic, and includes coma mimics, which are several disorders that may be easily mistaken

Table 1
Causal overview of coma and coma mimics

Coma			
	Diffuse Neuronal Dysfunction		
Structural	**Toxic**	**Metabolic**	**Coma Mimics**
Neoplasia	Sedative-hypnotics agents	Respiratory insufficiency	Locked-in syndrome
Hydrocephalus	Opioids	Dysthermia	Neuromuscular paralysis
Intracranial hemorrhage	Dissociative agents	Dysglycemia	Akinetic mutism
Vascular occlusion	Carbon monoxide	Electrolyte disorders	Psychogenic unresponsiveness
	Toxic alcohols	Infection	
	Antidepressants	Hypothyroidism	
	Antiepileptics	Thiamine deficiency	
	Agents of histotoxic hypoxia	Nonconvulsive status epilepticus	
	Simple asphyxiants		
	Serotonin syndrome		
	Neuroleptic malignant syndrome		
	Clonidine		