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Disorders of Consciousness

Mohamad Chmayssani and Paul M. Vespa

INTRODUCTION

Coma is a sleeplike state in that the patient is unresponsive and the eyes remain closed even with vigorous stimulation. A poorly responsive state in which the eyes are open, or an agitated and confused state, or delirium is not coma but may represent early stages of the same disease processes and should be investigated in the same manner.

Consciousness requires that the brainstem reticular activating system and its cortical projections be intact and functioning. The reticular formation begins in the midpons and ascends through the dorsal midbrain to synapse in the thalamus; it then innervates higher centers through thalamocortical connections. Knowledge of this anatomic substrate provides the short list of regions to be investigated in the search for a structural cause of coma: Brainstem or bihemispheric dysfunction satisfies these anatomic requirements, whereas structural lesions elsewhere are not the cause of the patient's unconsciousness. In addition to structural lesions, meningeal inflammation, metabolic encephalopathy, and seizures diffusely affect the brain and complete the differential diagnosis for the patient in coma.

PATHOPHYSIOLOGIC FACTORS

Meningeal irritation caused by infection or blood in the subarachnoid space is an essential early consideration in coma evaluation because its cause requires immediate attention (especially with purulent meningitis) and may not be diagnosed by computed tomography (CT).

Hemispheric mass lesions result in coma either by expanding across the midline laterally to compromise both cerebral hemispheres or by impinging on the brainstem to compress the rostral reticular formation. These processes—*lateral herniation* (lateral movement of the brain) and *transtentorial herniation* (vertical movement of the brain)—most commonly occur together. At the bedside, clinical signs of an expanding hemispheric mass evolve in a level-by-level, rostral- caudal manner (Fig. 105-1). Hemispheric lesions of adequate size to produce coma are readily seen on CT.

Brainstem mass lesions produce coma by directly affecting the reticular formation. Because the pathways for lateral eye movements—the pontine gaze center, medial longitudinal fasciculus, and oculomotor (third nerve) nucleus)—traverse the

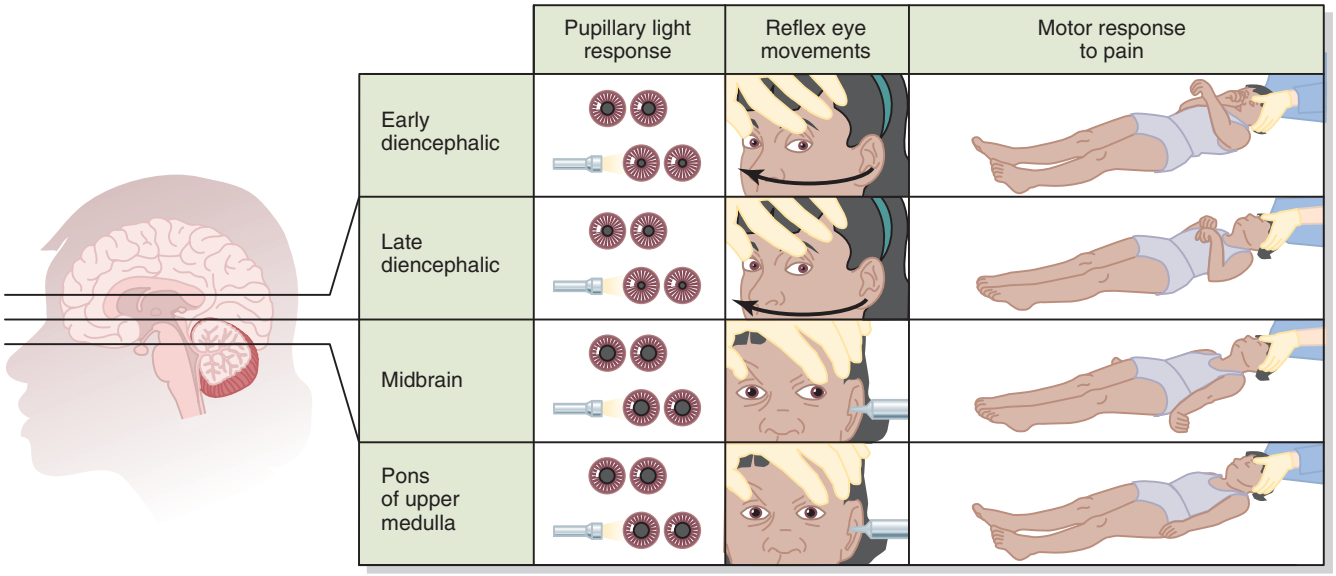

FIGURE 105-1 The evolution of neurologic signs in coma from a hemispheric mass lesion as the brain becomes functionally impaired in a rostral-caudal manner. The terms *early diencephalic* and *late diencephalic* refer to levels of dysfunction just above and just below the thalamus, respectively. (From Aminoff MJ, Greenberg DA, Simon RP: Clinical neurology, Stamford, Conn., 1996, Appleton and Lange.)