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Coma etiology

Toxic/metabolic causes of coma

- 1. electrolyte imbalance: especially hypo- or hypernatremia, hypercalcemia, renal failure with elevated BUN & creatinine, liver failure with elevated ammonia
- 2. endocrine: hypoglycemia, nonketotic hyperosmolar state, DKA (diabetic ketoacidosis, AKA diabetic coma), myxedema coma, Addisonian crisis (hypoadrenalism)
- 3. vascular: vasculitis, DIC, hypertensive encephalopathy
- 4. toxic: EtOH, drug overdose (including narcotics, iatrogenic polypharmacy, barbiturates), lead intoxication, carbon monoxide (CO) poisoning, cyclosporine (causes an encephalopathy that shows white-matter changes on MRI that is often reversible with discontinuation of the drug)
- 5. infectious/inflammatory: meningitis, encephalitis, sepsis, lupus cerebritis, neurosarcoidosis, toxic-shock syndrome
- 6. neoplastic: leptomeningeal carcinomatosis, rupture of neoplastic cyst
- 7. nutritional: Wernicke's encephalopathy, vitamin B12 deficiency
- 8. inherited metabolic disorders: porphyria, lactic acidosis
- 9. organ failure: uremia, hypoxemia, hepatic encephalopathy, Reye's syndrome, anoxic encephalopathy (e.g. post-resuscitation from cardiac arrest), CO2 narcosis
- 10. epileptic: status epilepticus (including non-convulsive status), post-ictal state (especially with unobserved seizure).

Structural causes of coma

- 1. vascular:
- a) bilateral cortical or subcortical infarcts (e.g. with cardioembolism due to SBE, mitral stenosis, A-fib, mural thrombus...)
- b) occlusion of vessel supplying both cerebral hemispheres (e.g. severe bilateral carotid stenosis)
- c) bilateral diencephalic infarcts: well described syndrome. May be due to occlusion of a thalamoperforator supplying both medial thalamic areas or with "top-of-the-basilar" occlusion.

Initially resembles metabolic coma (including diffuse slowing on EEG), patient eventually arouses with apathy, memory loss, vertical gaze paresis

2. infectious: abscess with significant mass effect, subdural empyema, herpes simplex encephalitis

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- 3. trauma: hemorrhagic contusions, edema, hematoma
- 4. neoplastic: primary or metastatic
- 5. herniation from mass effect: presumably brainstem compression causes dysfunction of reticular activating system or mass in one hemisphere, causing compression of the other, and resulting in bilateral hemisphere dysfunction
- 6. increased intracranial pressure: reduces CBF
- 7. acute lateral shift (midline shift) of the brain: e.g. due to hematoma (subdural or epidural)

Coma results from one or more of the following:

- dysfunction of the high brainstem (central upper pons) or midbrain
- bilateral diencephalic dysfunction
- diffuse lesions in both cerebral hemispheres (cortical or subcortical white matter)

State of unconsciousness lasting more than six hours in which a person: cannot be awakened; fails to respond normally to painful stimuli, light, or sound; lacks a normal sleep-wake cycle; and, does not initiate voluntary actions.

A person in a state of coma is described as being comatose.

Coma from supratentorial mass

see Coma from supratentorial mass

Medically induced coma see General anesthesia.

Structural causes of coma

Midline shift of the brain due hematoma (subdural hematoma, epidural hematoma).

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