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MANAGEMENT OF COMATOSE PATIENTS IN THE I.C.U.

Essay

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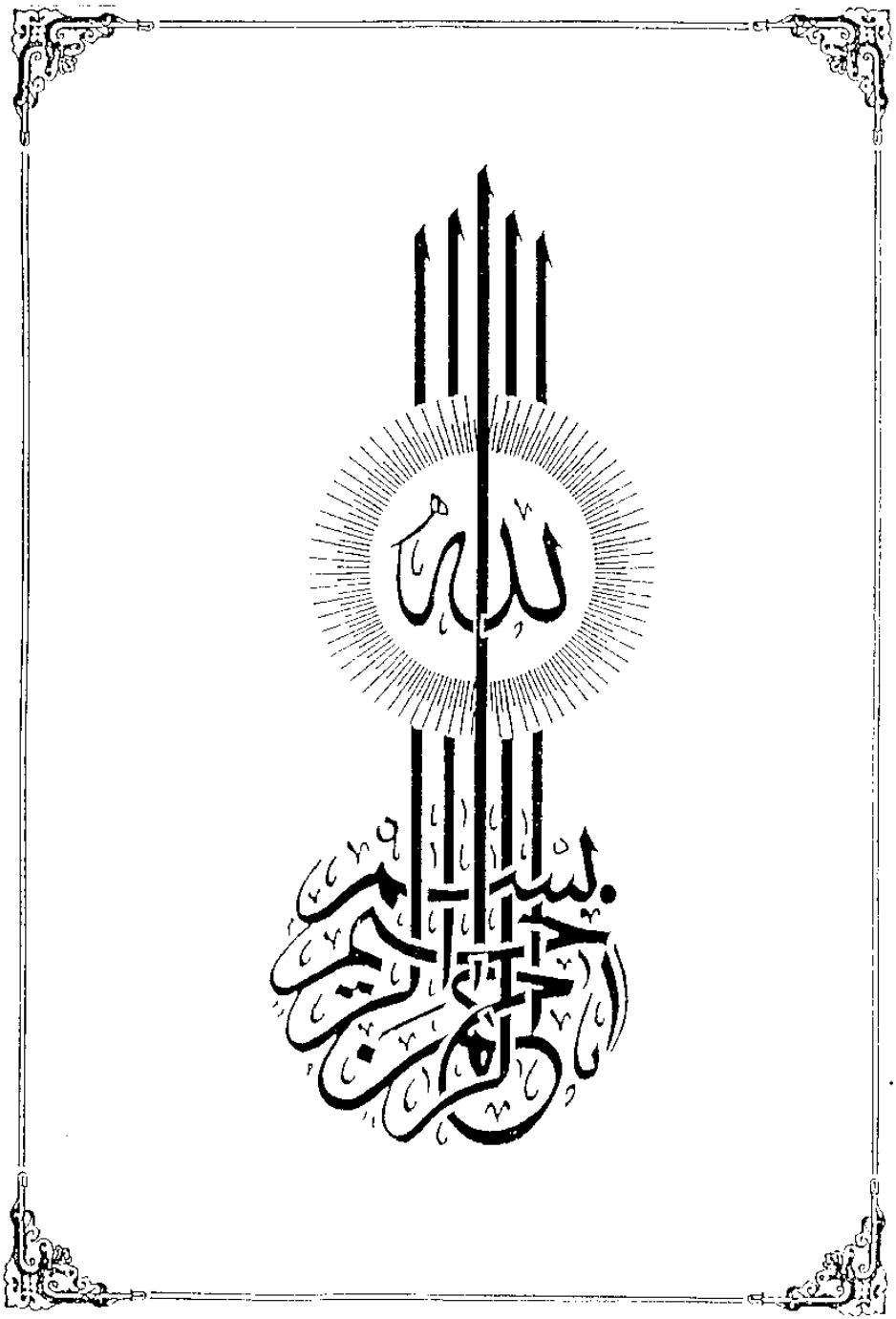
To Mom and Dad for their guidance
and support throughout the years.



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INTRODUCTION

Introduction.

Coma is a common problem in general medicine , it is estimated that up to 3 percent of admissions to the emergency ward of large municipal hospitals are due to diseases that cause a disorder of consciousness . The importance of this class of neurologic disorders points to the necessity of acquiring a systematic approach to their diagnosis and management . The increased availability of computerized tomography (CT) has resulted in an artificial orientation to the diagnosis of coma by focusing attention on lesions that are detected by CT . This approach, although at times expedient, is often imprudent because most Coma is metabolic or toxic in origin. The physician confronted with an unresponsive patient should formulate a differential diagnosis based on the history and clinical examination before leaving the bedside . Certain signs observed during general neurologic examination allow the physician to decide which of several generic diseases is responsible for Coma, thus limiting the diagnostic possibilities . A rational approach to the precise diagnosis and subsequent management can then be planned and clinical changes anticipated . The clinical approach must be coupled to knowledge of the pathologic entities that cause Coma .

•Chapter 1

Pathophysiology of coma

Some physiological aspects and anatomic correlates of consciousness .

Wakefulness is not anatomically represented in any region of the cerebral hemispheres, howeve, it is related in a semiquantitative way to the total mass of functioning cortex. A normal level of consciousness depends upon the activation of the two cerebral hemispheres by the brainstem reticular activating system (RAS). Both cerebral hemispheres, RAS, and the connections between them must be preserved for normal consciousness. The principal causes of coma are, therefor either, bilateral hemispheral injury or injury to the diencephalic and brainstem RAS *(Ropper and Martin, 1991)* . The type of injury may be structural (anatomic) or in the form of metabolic derangement and suppression . Hemispherical lesion may produce coma in one of three ways . First, if a wide area of the two cerebral cortices are damaged or suppressed e.g., bilateral extensive contusions, encephalitis, ischemic encephalopathy, and drugs . Second, a unilateral mass, or secondary brain swelling (edema) initially confined to one side of the brain may compress the

contralateral hemisphere effectively creating bilateral hemispheral lesion. Third, a large lesion in one or both hemispheres (supratentorial mass) may compress the diencephalic and brainstem RAS downwards (Transtentorial herniation). *(Ivan and Bruce, 1982)*

The pathophysiology of coma can be divided into four basic mechanisms according to a classification modified from Plum and Posner(1980).They are : supratentorial structural lesions ; infratentorial structural lesions ; metabolic or diffuse encephalopathy; and hysterical coma . *(Plum and Posner, 1980)* .

Supratentorial mass impairs consciousness by compressing the other cerebral hemisphere leading to bilateral hemispherical damage, or by compressing the diencephalic RAS from rostral to caudal direction and the brainstem herniates downward through the tentorial notch (Transtentorial herniation) . In centrally placed masses (e.g., generalized brain edema), the brainstem is uniformly compressed in a rostrocaudal direction (central transtentorial herniation) . Results on neurologic examination are consistent with the anatomic level of brain involvement . Uncal (lateral) hernia-

tion, on the other hand, occurs in lateral hemispheral lesion (e.g.,subdural hematoma). The uncus is squeezed over the notch of the tentorium compressing the ipsilateral occulomotor nerve and the cerebral peduncle, resulting in ipsilateral pupillary dilatation and contralateral hemiparesis *(Posner,1975)*

Supratentorial space occupying lesions (SOL) :

A - Hemorrhagic . (as in truma, hypertension, coagulopathies)

1- Epidural .

2- Subdural .

3- Intracranial .

B- Infarction plus edema .

C- Neoplastic .

D- Inflammatory (abscess)

(Plum and Posner, 1980)

Table 1

Infratentorial structural lesions produce coma by compressing or directly destroying the brainstem RAS . Such lesions may also cause brain herniation, either transtentorially

upwards (with mid brain compression), or downwards through the foramen magnum (with distortion of the medulla by the cerebellar tonsils). Abrupt tonsillar herniation causes apnea and circulatory collapse. Coma is then secondary, for the medullary RAS probably has little direct role in arousal .

Subtentorial structural lesions
A- Hemorrhagic
1- Pontine
2- Cerebellar
3- Subtentorial hematoma (epidural or subdural)
B- Infarction
1- Cerebellar infarction plus edema
2- Brainstem infarction (basilar artery thrombosis or embolic occlusion)
C- Neoplastic
D- Abscess
(Plum and Posner, 1980)

Table 2

Metabolic, diffuse, or multifocal encephalopathy is a diffuse disorder of the cerebral function due to impaired neuronal metabolism due to ; oxygen or substrate deprivation ; disturbance of neuronal metabolism by disease of organ rather than the brain ;