COMA IN INFANCY AND CHILDHOOD

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BY

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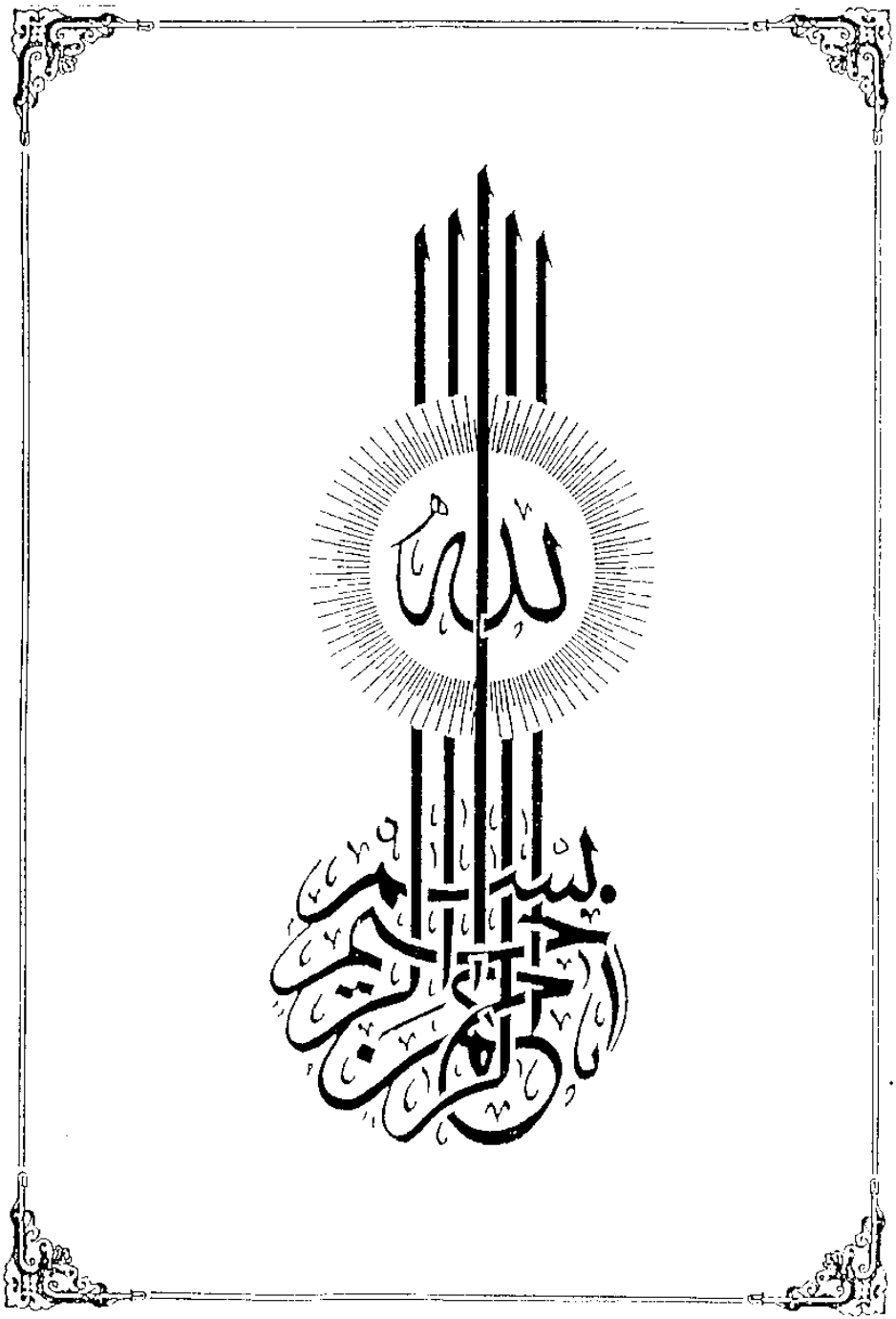

CUSTOMARY ABBREVIATIONS

IV	*Intravenous*
IM	*Intramuscular*
ICP	*Intracranial Pressure*
CSF	*Cerebrospinal Fluid*
CAT	*Computerized axial tomography*
PMN_s	*Polymorph nuclear cells*
ARAS	*Ascending reticular activating system*
SGOT	*Serum glutamic oxaloacetic transaminase*
SGPT	*Serum glutamic pyruvic transaminase*
CDC	*Center for disease control*
HVA	*Homovanillic acid*
5-HIAA	*5-hydroxyindole acetic acid*
AER	*Auditory evoked responses*
BAER	*Brain-stem auditory evoked responses*
CNS	*Central nervous system*
BUN	*Blood urea nitrogen*
Co_2	*Carbon dioxide*
0_2	*Oxygen*
P_aCo_2	*Carbon dioxide tension in arterial blood*
HCo_3	*Bicarbonate*
Na	*Sodium*
K	*Potassium*
ATP	*Adinosine triphosphate*

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EEG	*Electroencephalogram*
P_aO_2	*Oxygen tension in arterial blood*
BAL	*Dimercaprol*
EDTA	*Ethylene diamine tetra-acetic acid*

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CONTENTS

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AIM OF THE WORK

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Coma in infancy and Childhood is a frequent and formidable problem in management and diagnosis facing the paediatrician. So our intention here is to review the literature on coma in infancy and childhood, to point out new and basic informations on the pathogenesis, diagnosis and treatment of coma, and to provide practical protocols of diagnosis and therapy.

INTRODUCTION

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Coma has been the target of many and diverse investigations and opinions. Strictly speaking, coma indicates a disturbance in reticular-hypothalamic function. It carries a bad prognosis for two reasons; the patient can not guard himself against common physical dangers(fire, water, asphyxiation and exposure) and the underlying causes are often life threatening.

Coma is not an independent disease entity but is always a symptomatic expression of the disease. A wide range of conditions may be associated with coma or impaired consciousness. Coma may result from the presence of an expanding intracranial mass lesion, or may follow a variety of vascular, metabolic, or toxic disturbances. Sometimes coma is multifactorial-a combination of both occult head injury and a metabolic disturbance such as diabetic ketoacidosis.

All too often, the patient is brought to the hospital in a state of coma, and little or no information is immediately available. The diagnostic and therapeutic attempts of the first few minutes often determine whether the patient survive and whether he will suffer irreversible central nervous system damage. Therefore it is essential that the paediatrician have a well planned immediate approach.

The paediatrician must then subject the clinical problem to careful analysis from many directions. To do this efficiently he reaquires a

broad knowledge of the disease and systematic approach that leaves none of the common and treatable causes of coma unexplained.

Within the past decade, new understanding of neurological pathophysiology and major advances in radiological diagnosis have given us the technical means for evaluating coma, while published comparative experience in a variety of treatment modalities has offered new insights for its management.

DEFINITION OF COMA

Coma is a state of depressed neurological function in which the patient can not be aroused [Fred, B., 1978]. A comatosed patient is out of touch with his surroundings, cannot communicate sensibly, responds imperfectly or not at all to external stimuli, and can not be aroused to normal waking behaviour. [Hall, G.H.1981]. So, the patient who appears to be asleep and is at the same time incapable of sensing (unreceptive) and responding adequately to either external stimuli or inner needs (unresponsive) is in a state of coma. [Adams, R.D., 1980].

STAGES OF COMA

Stage I = Stupor:

It is a state in which the patient may appear awake or in light sleep. Patient may be aroused by mild external stimuli, such as speech or light touch and will respond to questions and commands, however slowly. When the external stimuli are removed he lapses again into an immobile or sleep-like state.

The term "obtundation" has similar meaning and does not clearly differentiate the state of unconsciousness. [Lockman, L.A. 1975].

Stage II = light Coma:-

The patient can not be aroused; even with painful stimuli, he may moan and make semipurposful avoidance movements. [Huttenlocher, P.R. 1979]

Stage III = Deep Coma:

This term may either be used to indicate coma in which the patient does not respond to painful stimuli (painful stimuli may lead to extension and pronation of arms = decerebrate posturing) or loss of consciousness accompanied by deterioration of respiratory and cardiovascular function.

Numerical gradations, modified from classifications of the stages of anesthesia, have been applied to states of altered consciousness. This numerical grading is not uniformly used and is therefore of limited value. [Lockman,L.A., 1975]

Ambiguity may be prevented by the use of the terms "loss of consciousness" or "decreased consciousness" which indicate quantitative decrease in the patient's awarness of, and reactivity to, the environment. These terms must be modified by a description of the vegetative state and reaction to environmental stimuli. [Lockman, L.A., 1975].

"Vegetative state" describes the behavior of a group of patients sharing the feature of wakefulness without evidence of awareness of self and environment. [Dougherty,J.H. et al. 1981]

Table [1] : Clinical classification of Coma:

Symptoms	Class
* Asleep, but can not be aroused and can answer questions.	0
* Comatosed, withdraw from painful stimuli, reflexes intact.	1
* Comatosed, does not withdraw from painful stimuli, no respiratory or circulatory depression, most reflexes intact.	2
* Comatosed, most or all reflexes absent but without depression of respiration or circulation.	3
* Comatosed, reflexes absent, respiratory depression with cyanosis, and circulatory failure or shock.	4

Adapted from [Barry, H.R., and John, E.O., 1976 and Silver, H.K. et al, 1980].

NEUROPHYSIOLOGIC CONSIDERATIONS

In recent times there have been some clarifications and amplifications of earlier neuropathologic observations in the mid-brain and thalamus associated with protracted coma. [Adams, R.D., 1980] Maintenance of consciousness implies intact function of the ascending reticular activating system (ARAS). This is a highly complex polysynaptic region in the core of the upper pons and the mid brain. It divides into both thalamic regions and ultimately becomes wide spread within the hemispheres [Sabin, T.D., 1981].

Anatomic studies show the reticular activating system of the upper brain-stem to receive collaterals from the specific sensory pathways and to project, not just to the sensory cortex of the parietal lobe as do the thalamic relay nuclei for somatic sensation, but to the whole of the cerebral cortex.

The later has corticofugal connections which feed back nerve impulses to the reticular formation. Sensory stimulation, it would seem then, has the double effect of conveying to the brain information about the outside world and also of providing some of the energy for activating those parts of the nervous system on which consciousness depends. [Adams, R.D., 1980].