THE ROLE OF ANAESTHIOLOGIST IN THE INTENSIVE CARE UNIT IN CASES OF COMA DUE TO HEAD INJURY

THESIS

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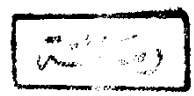

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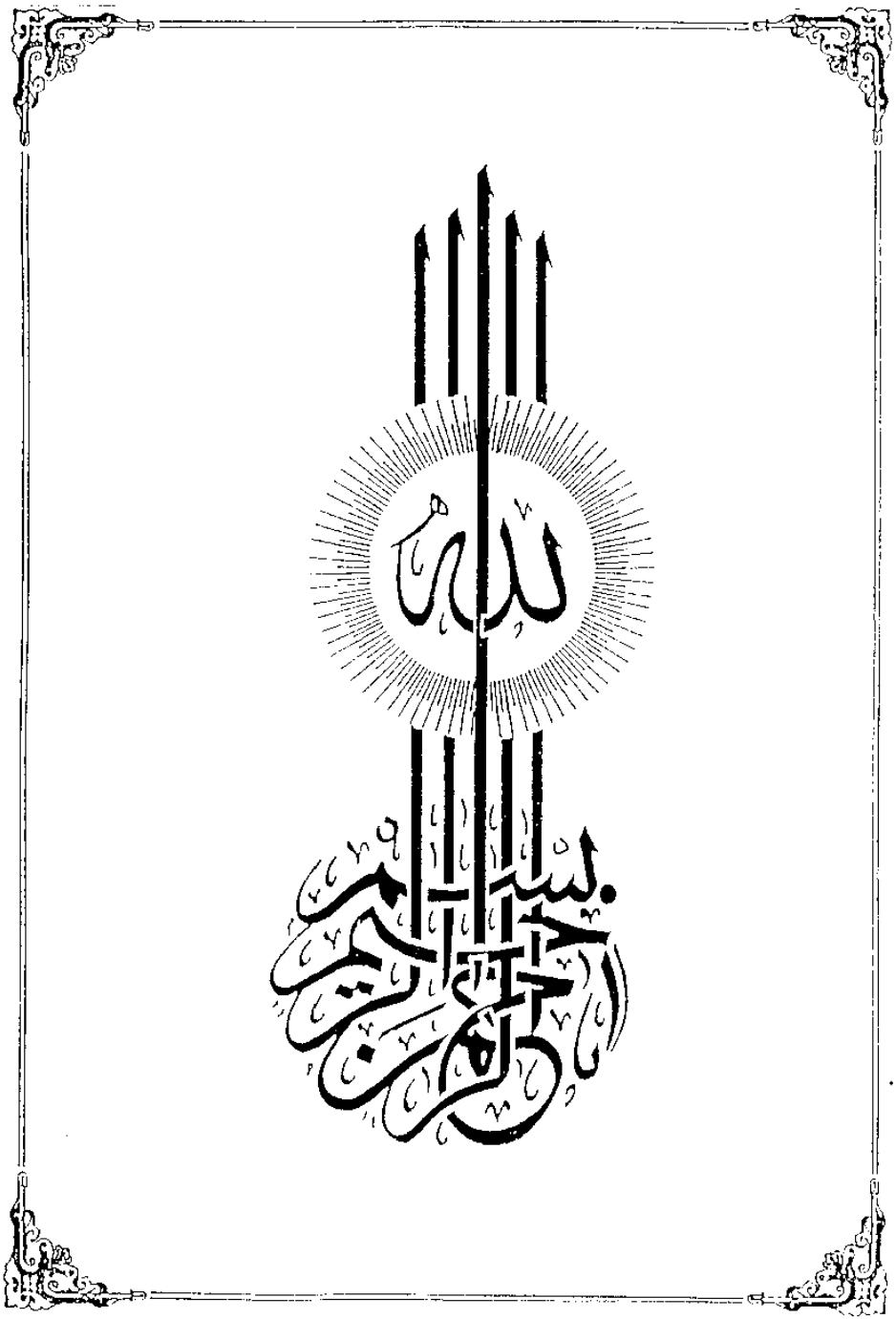

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The anaesthetist's contribution to the management of head injured patients has expanded considerably in the past few years, reflecting change in the total management of these patients.

The anaesthetist now has vital responsibilities in the critical care unit. This has occurred through an increasing appreciation of the fact that secondary changes, which are superimposed on the initial cerebral insult, have major impact on the outcome of head-injured patients.

In this study, stress has been made on the pathophysiological changes present in comatose patients following head injury.

Special attention was made to the various lines of management and monitoring of comatose patients with head injury.

Special lights were thrown on sequelae, prognosis of such patients and irreversible brain death after prolonged coma.

Finally, this work is a review of the role of the anaesthetist in the intensive care unit in management and in modifying different factors to the benefit of the critically ill patients following head injuries.

PATHOPHYSIOLOGICAL CHANGES

Anatomical Consideration of the Reticular Formation:-

The reticular formation occupies a central position in the brain stem.

The maintenance of consciousness depends to an important extent upon the central reticular formation.

It is situated in the tegmentum of the medulla, Pons and mid-brain above which it merges into thalamic reticular system. (Bannister, 1978).

The reticular formation consists essentially of two Parts:

I. Ascending fibres.

II. Descending fibres.

I. The ascending fibres recieves afferent connections involving most kinds of sensations, it include the following:

(A) spino-reticular fibres.

(B) Trigeminal-reticular fibres.

(C) Auditory-reticular fibres.

These fibres connect with intralaminar thalamic nuclei, and from here they diffuse as a thalamo-cortical projections to the cortex. (Atkinson et al., 1982).

It was identified an ascending reticular activating system, within the paramedian tegmentum of the mid-brain and extending to the hypothalamus and thalamus. this system diffusely projects to the cerebral cortex and profoundly influences both arousal and the electro encephalographic pattern. (Plum and Brennan, 1982).

II. The descending fibres are made upon on each side of medial and lateral reticualr tracts, continous below with the reticulo-spinal tract in the anterior column of the spinal cord, it affects reflex muscle tone, the medial tract inhibitory, and the lateral tract fascilitatory.

The reticular formation may be considered to be a heterogenous collection of neurons, it does not include neurons belonging to the cranial nerve nuclei, sensory relay nuclei and cerebellar relay nuclei. (Rowbotham and Dott 1964).

Six nuclei had been identified by the previous author (1964), in the bulk of the central part of the brain-stem tegmentum, lesions of which may result in frank coma in man.

These nuclei are paired, one lying in each half of the brain stem, they are named as following:-

- Nucleus gigantocellularis.
- Nucleus pontis caudalis.

- Nucleus oralis.
- Nucleus guneiformis.
- Nucleus subcuneiformis.
- Nucleus tegmenti Pedunculopontinus.

Pathophysiology of Coma and disorder of consciousness after head injuries

Normal consciousness requires an interaction between the reticular activating system of the brain stem and the cerebral cortex.

The reticular activating system is responsible for arousal or alertness and the cerebral cortex for the content of consciousness of the awarness of self and environment. (Bannister, 1978(.

Impairment of consciousness result from lesions affecting the reticular formation, the reticular formation - cortical connections or the cerebral cortex.

Coma resulting from a head injury probably results from a disordered reticualr formation, and drugs produce coma by their effect on the reticular formation. Hypoxia and hypotension may produce coma by diffuse depression of the cerebral cortex.

A unilateral supratentorial lesion such as a subdural or extradural haematoma will only produce coma when there is diencephalic or brain

stem damage resulting from herniation of structures through the tentorial hiatus. (Conway, 1978).

Causes of Disordered consciousness :-

Following injury there is often a natural tendency to assum that impairment of consciousness has resulted from head injury. This is only one of the many factors which can lead to coma after head injury.

Factors affecting consciousness in the patient after trauma:

1- Head trauma:

(a) Primary impact damage.

(b) Complicating cerebral oedema.

(c) Intra cranial haematoma.

2- Hypoxia.

3- Hypotension

4- Drugs:

Analgesics, sedatives.

5- Metabolic disorders.

Renal Failur, hepatic failure.

6- Miscellaneous:

e.g. fat embolism, DIC.

In the early assessment of the patient after trauma only the first three of these factors need to be taken into account, and the first priority is to determine which of this is the main contributing factor. (Holloway, 1979).

Deterioratiang consciousness after trauma is more likely to be due to hypoxia than to an acute intracranial haematoma.

In general, it is safer to assume that any disorder of respiration or circulation which present is more likely to be the cause of disordered consciouness rather than the result of associated brain injury.

Massive pituitary damage secondary to brain injury could theoretically produce "neurogenic hypotension" but clinical example of this are extreme rarities. A good working rule is that shock means a major injury elsewhere than to the head.

Pasthophysiology of the head injury:

A head injury may involve the scalp, Skull, dura, blood vessels, brain, spine and spinal cord.

Although injuries to the scalp and skull may not be major consequence to the brain, they served as indicators that the head has been traumatized. A skull fracture occur in only about 65% of patients with a severe injury to the brain. (Jennett and Teasdale, 1981).

The major consequence of any head injury is the injury to the brain. Brain injury occurs in two phases. At the instant of impact, a variable

amount of primary brain injury occurs. Then, secondary process are set in motion which lead to further brain, injury and often death. At the present time, we only have preventive and not therapeutic measures for brain injury. All our therapy is directed at the secondary insult. The rotatory movement of the brain inside the head produce shearing stresses which are responsible for the most significant brain injuries. Mild injuries tend to involve only the cortex while progressivly more severe injuries involve the deeper structures including the brain stem.

Thus injury to the brain stem is uncommon without diffuse injury to the hemisphere.

The brain may be injured in different ways at the moment of impact. (Strich, 1970). These include concussion, contusion, laceration, petechial haemorrhages and white matter lesions. Concussion is a clinical syndrome of loss of consciousness without focal signs, and is completely reversible within 24 hours. It was believed until recently that no permenant brain injury occured with concussion. However, microglial cell clusters, indicating cell damage have been demonstrated in patients with recent concussion who died of other causes. (Oppenheimer, 1968). Other non-observable changes such as nerve fibre stretching, alteration in membrane polarity, and disruption of synapses have postulated in the Pathogenesis of concussion.

Contusion indicate more severe brain damage. They appear grossly as haemorrhagic and necrotic areas which commence at the crest of the gyri

in the superficial cortex and extend into the brain substance. As the brain rotates inside the skull after the impact, it rubs against the rough areas of the floor of the anterior and middle fossae, and the sphenoid wings. Consequentely, the inferior and anterior temporal lobes and the inferior frontal lobes areusually most affected by contusions.

The lesions are often on the side opposite the impact (contrecoup lesions), and occur most frequently with blows to the occipital region. Contusion can also ,be seen under the site of impact on the skull, particularly if there is depressed skull fracure (Artru and Michenfelder 1981).

Lacerations of the brain occur in similar sites to contusions, but represents a more severe injury.

Petechial haemorrhages developing in the white matter and corpus callosum are caused by tearing of blood vessels during brain rotation. These haemorrhages may produce damage by their coalescence into haematomas,or by producing ischaemia distal to the torn vessel.

The same shearing forces which tear blood vessels also tear nerve fibres producing diffuse white matter damage. (Adams et al., 1977). These lesions are most frequently responsible for the persistant vegetative state.

Further, brain injury may result from haematoma formation; cerebral oedema, and cerebral ischemia.

A haematoma may be extradural (ruptured meningeal vessels associated with a fracture) Subdural (often associated with severe brain contusion and/or laceration) or it may be intracerebral ("pulped" temporal lobe). Many patients with an extradural haematoma do not have a severe primary brain injury and, therefore, have a lucid interval before deteriorating. Evacuation of the clot before secondary brain damage occurs should result in a good outcome.

In contrast, subdural and intracerebral haematomas usually occur in people with severe primary brain injury. The final outcome is frequently poor despite evacuation of the haematoma (Hulands, 1980).

Normally, the amount of extracellular fluid in the brain (less than 150 ml) is tightly controlled by an intact blood-brain barrier. "Vasogenic" Cerebral oedema results when intravascular fluid leaks across a disrupted blood-brain barrier into the extracellur space. In addition, swelling of cells injured by trauma and ischaemia leads to "cytotoxic" brain oedema. This combined volume of fluid produces brain swelling which may behave as a mass.

Blood supply of the Brain:

Arterial Supply

The arterial supply of the brain is derived from two Pairs of vessels, the internal carotid arteries(I.C.A.) and the vertebral arteries.