POST TRAUMATIC CEREBRAL DYSFUNCTION

Thesis

Submitted for Master Degree

in Neurology and Psychiatry

By

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M.B. , B.Ch.

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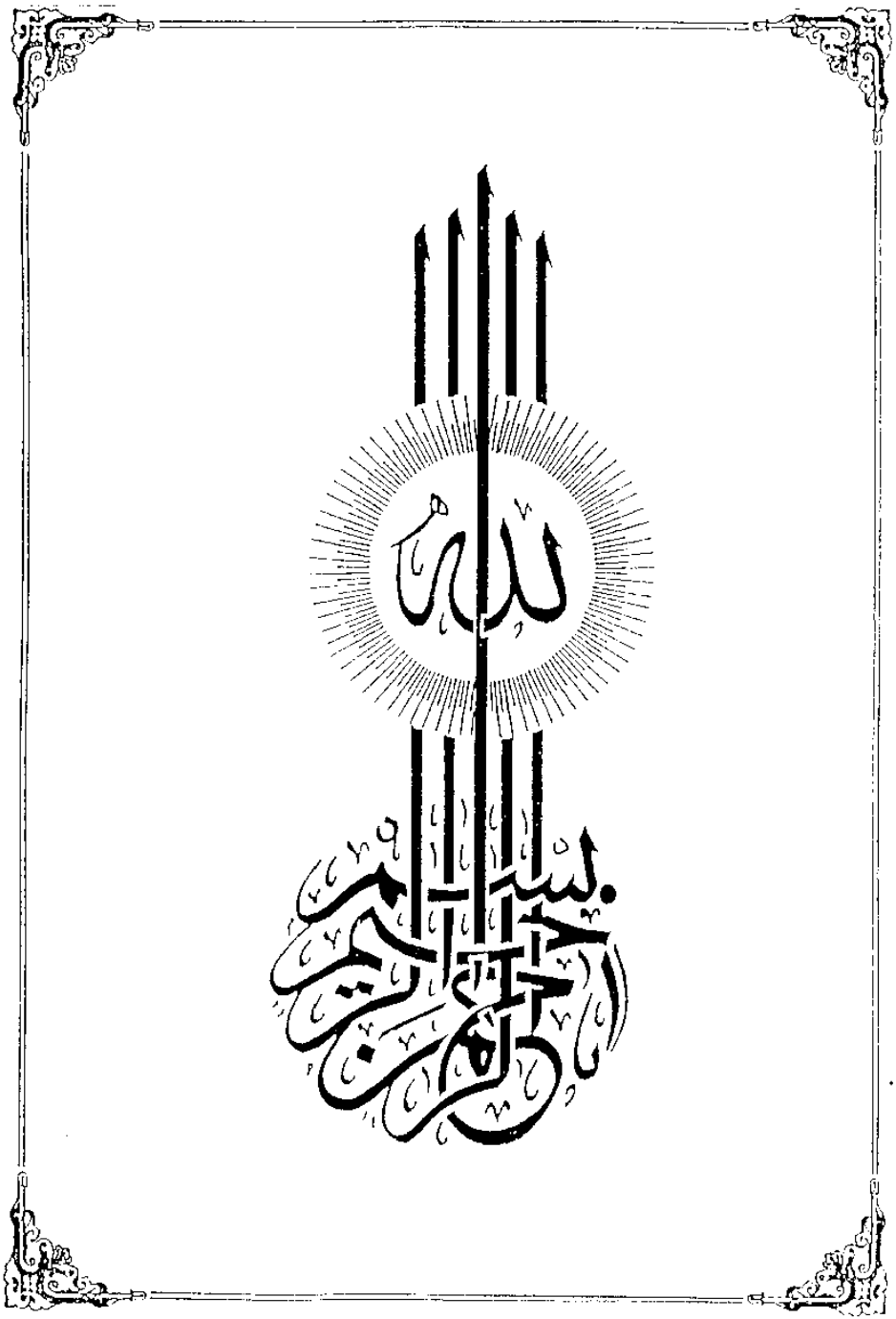

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Salwa Samy Farag

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INTRODUCTION

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Post-traumatic cerebral dysfunction results from head injuries which are caused by falls, accidents, hand to hand combat and birth injuries.

The technical civilization, the increasing speed of transportation and wars have multiplied the number of cranial traumata and their sequelae.

As a result of growth in motor traffic in the last decade, there has been an enormous increase in the number of serious civilian head injuries which resulted in a large number of severe disabled survivors.

Head injuries are now the most important cause of death in the first half of life in the developed countries of the western world.

The aim of work is to delineate the early and delayed effects of head injuries and their pathogenesis in order to plan the way of management on scientific basis.

REVIEW OF LITERATURE

HISTORICAL CONSIDERATION

Head injuries have been undoubtedly among the earliest experience of man, and their treatment has always been indebted to human ingenuity.

Gurdjian and Webster (1958) stated that the Ancient Egyptians were the pioneers who firstly wrote and worked about head injuries.

It was reported in Adwin Smith Papyrus (1930) that different cases of head injuries were translated from Ancient Egyptian language to hieroglyphic language and lastly to english. The papyrus classified head injuries into laceration fractures, compound fractures and compound commonuted fractures and described each type carefully. It was reported in the papyrus about facial distortion, motor defects in the extremities and loss of speech from a wound in the temporal region. Also the direction for examining a person with a head injury, include palpation of the wound , observation of the patients movements and postures and his ability to speak. Even the examiner's sense of smell was called on (Harveys et al., 1982).

Greek medicine inherited many of Egyptian methods, and hippocrates methods of treating a dislocated mandible. Hippocrates held that no head injury was trifling since even wounds involving only the integument, and it was proved to be dangerous if neglected. They also divided skull injuries into simple fracture (fissure), contusion without fracture or depression and fracture with depression (Harveys et al., 1982).

The crusades of the eleventh and twelfth centuries were the principal means in spreading Islamic Knowledge to Europe (Harveys et al., 1982).

In the 16th century the physicians ascribed concussion or comotio cerebri to intracranial bleeding, and this opinion found support in the centuries later (Harveys et al., 1982).

Webfer (1681) recognised a chronic subdural haematoma.

Peyronie (1699) reported the first case of subdural abscess after head injury and after fracture of left parietal region. He used trephine and then incised the dura.

In the 18th century the physician discussed contusions and laceration of the scalp, and skull fractures. They divided it into fissure, contrafissure and depression. They described the possible extravasions of the blood into the cranial cavity after a violent blow and believed that these could cause the unconsciousness or coma, paralysis, vomiting and could be associated with bleeding from the mouth, ears and nose(Harveys,1982).

John Abernethy (1831) described extradural and subdural haematomas. Also he pointed out that trephination was successful only in the treatment of extradural collection of blood.

Cooper (1824) discussed the mechanism of concussion and suggested that it might be due to a change of function rather than to a disorganization of the brain. In slight concussion, it was possibly that only the cerebral circulation was disturbed, where as in severe concussion the brain might be torn.

Pathologically, Strich (1956) stated that the work of Schamus (1890-1899) who demonstrated that secondary degeneration of nerve fibres occur above and below the

level of injury. Schamus inferred that nerve fibres die as a result of mechanical change.

This early contribution was extended by Strich's classic studies of injury to the cerebral white matter. Case studies of recovery after closed head injury were published during the later half of the 19th century and the early part of the 20th century (Levin et al.,1982).

The two world wars and the greatly improved techniques of experimental study of our own century have brought with them a great expansion in the knowldge (Harvey's et al., 1982).

Holland and Brews (1980) reported that the incidence of serious intracranial trauma during delivery has fallen considerably over the past decade with the improvement in obstetric care.

CLASSIFICATION OF HEAD INJURIES

Grinker (1966) classified head injury according to:

(a) The type of fracture which may be linear fracture, fracture of the base of skull and depressed fracture.

(b) Cerebral injury with or without fracture which include cerebral concussion, cerebral contusion, laceration, subpial haemorrhage, subarachnoid haemorrhage, extradural haemorrhage, subdural haemorrhage and cranial nerve injury.

(c) The sequelae of injury: Post traumatic syndrome and Post traumatic epilepsy.

Jennet and Teasdale (1981) classified head injuries into (a) clinical aspects which include concussion or coma and open or closed. (b) Pathological aspects: at site of impaction (fracture of the skull, contusion of the grey matter and diffuse white matter lesions) and secondary brain damage (raised intracranial pressure, intracranial haematoma and ischemic brain damage).

Harveys et al. (1982) classified head injury according to the pathological aspect after closed head injury. They classified it into (1) Primary brain injury

(immediate on impact) which contains: (a) Macroscopic lesions as contusion (coup and contrecoup) and laceration. (b) Microscopic lesions such as wide spread shearing and stretching of the nerve fibres. (2) Secondary mechanisms of brain injury (intracranial haemorrhage, oedema in white matter adjacent to focal mass lesions, ischaemic brain damage, raised intracranial pressure, brain shift and herniation.

A suggested classification will be adopted,through this work to be more clinicopathological and practical is:

I. Early Effects of Head Injury

The head injuried patient may present with many symptoms and signs which indicate the pathology of the brain damage that leads to cerebral dysfunction.

a) Disturbances in consciousness:

1) Unconsciousness for few seconds to few minutes and seldom to more than ten minutes, indicates cerebral concussion.

2) Unconsciousness for few minutes and after recovery of consciousness, the patient remains confused and

exhibits other symptoms of cerebral dysfunction. It is reasonable to indicated cerebral concussion or contusion.

3) Unconsciousness which lasts hours, days or weeks, indicates severe form of contusion, laceration and intracerebral haemorrhage.

4) Deepening coma indicates cerebral compression, as extradural haemorrhage, subdural haemorrhage and intracerebral haemorrhage.

5) Deepening coma that follows a lucid interval after concussion is very important as it indicates cerebral compression.

6) Unconscious patient with contracted pupil on one side, then the pupil dilates and fails to react to light, and the same sequence of events subsequently occuring on the opposite side (Hutchinson pupil). These indicate tentorial herniation with pressure upon the trunk of one or both third cranial nerves. Smaller pupil indicates incipient uncal herniation and may also pontine damage "if bilateral".

b) Venous congestion in the optic discs and fundi, must raise the possibility of an extradural haematoma, but