HEADACHES IN CHILDREN

THESIS

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BY

HANEN MAHMOUD HASSAN ALI

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PROFESSOR DR. SAADIA ABD EL FATTAH

PROFESSOR OF PAEDIATRICS

FACULTY OF MEDICINE

AIN SHAMS UNIVERSITY

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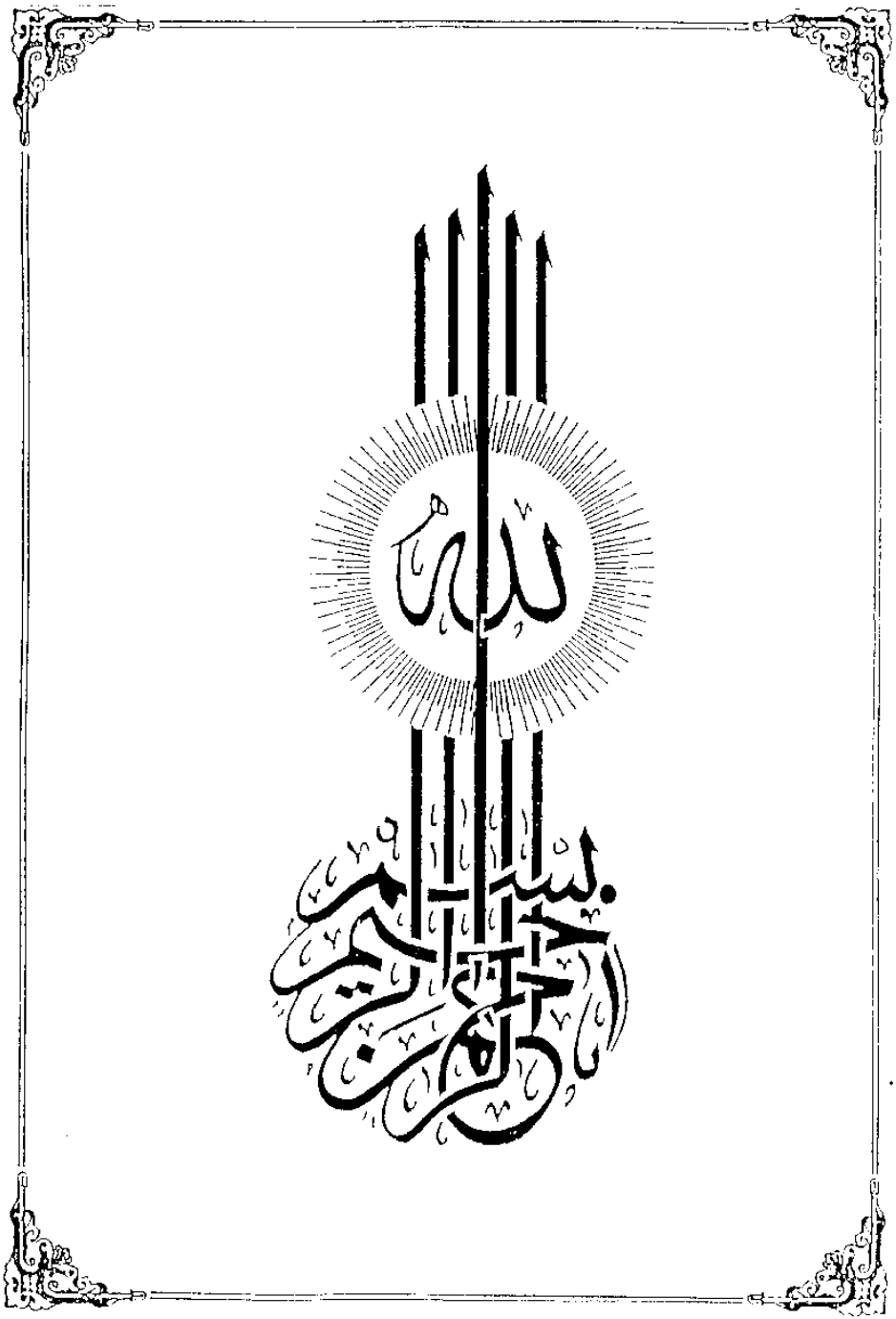

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TO MY MOTHER

CONTENTS

INTRODUCTION

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AIM OF ESSAY

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HEADACHE is a frequent symptom in paediatric practice.

Recurrent head pain is a common, frequently benign symptoms late in childhood. In the young child it is unusual and more often indicative of serious underlying disease.

Deciding whether a child's headache is "organic" or "functional" may be difficult for the most experienced clinician.

A thorough and systematic history and physical examination coupled with selected laboratory test will guide the physician to the correct diagnosis in most cases.

Of all the paediatric patients who have chronic headache, the cause will be organic in only 5% to 13%.

It is essential, therefore, that causes of headaches must be identified as early as possible and their management should be planned promptly.

The aim of the essay is to study headaches in children and their causes.

this may help us to know, causes, early detection and prevention of complication of diseases associated with headache and their management.

REVIEW OF LITERATURE

1. PREVALENCE OF HEADACHE

Data concerning the prevalence of headaches in children of different populations are not readily available, since headache is neither a reportable disorder nor does it often necessitate hospitalization.

Egermark-Eriksson, in 1982, reported on the prevalence of headache in Swedish school children in the age groups 7, 11 and 15 years answered a questionnaire concerning headache.

Recurrent headache was reported by 23% of the children, while 52% said that they had headache occasionally. Headache was more common among the older children than among the younger ones, and in the oldest age-group, headache was more common in girls than in boys.

No significant sex differences were observed in 7-years old and 11-years old children.

The prevalence of migraine in children was 2 - 4.5 %.

2. PATHOPHYSIOLOGY OF HEADACHES

Headaches are actually referred pain to the surface of the head from the deep structures. Many headaches result from pain stimuli arising inside the cranium, but equally as many probably result from pain arising outside the cranium (Goldstein, 1980).

The transmission and modulation of pain appear to depend on discrete neural pathway

Information derived from specific peripheral pain receptors, nociceptors, is processed at SPINAL CORD, BRAIN STEM, and THALAMOCORTICAL levels, although the ultimate cortical receiving region is not known. SEROTONIN has been implicated as an inhibitory transmitter in the brain stem pain modulating system (Basbaum and Fields, 1978).

The most comprehensive idea of how pain is perceived is expressed in the gate-control theory of pain (Melzack and wall, 1965).

The theory proposes that a neural network in the DORSAL HORN of the spinal cord may increase or decrease the flow of information from the PERIPHERAL PRIMARY AFFERENT TERMINALS TO CENTRAL PAIN TRANSMISSION NEURONS.

The "GATING" mechanism, therefore, modulates somatic information before it evokes pain perception. The degree to which the gate affects SENSORY TRANSMISSION is determined by both peripheral and descending influences.

Neural areas responsible for pain perception are activated when the level of activity in pain transmission neurons exceeds a critical level.

The presence or absence of pain, then, is determined by a balance between the INCOMING afferent impulses and CENTRAL influences upon the GATE. Thus a lesion (anatomic or biochemical) that impairs the normal DESCENDING or segmental INHIBITORY influences could OPEN THE GATE, resulting in pain.

Psychological factors such as attention, past experience and state of mind may also influence pain perception through the same mechanism. (Melzack, 1973).

Pain may result from activation of peripheral nociceptors in the presence of a normally functioning nervous system. Another type of pain is the result of injury to the central or peripheral nervous system, and may occur without activation of peripheral receptors. (Raskin, 1980).

Headache may have its origin in stimulation of extracranial or intracranial structures. The extracranial pain sensitive structures of the head include the scalp, extracranial arteries, mucous membranes of the nasal and paranasal spaces, external and middle ear, teeth and muscles of the scalp, face and neck (Fig.1). The basic extracranial headache mechanisms are distention, pulsation and stretching of arterial walls, or the contraction of skeletal

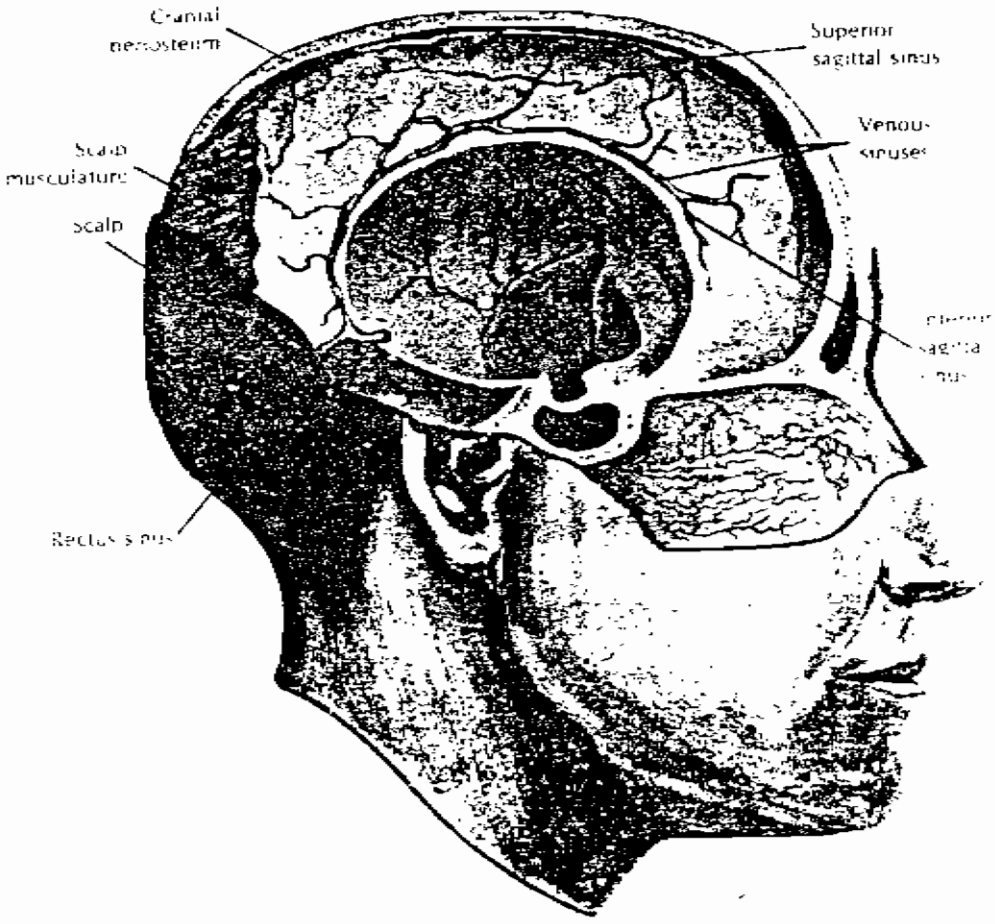

Fig. 1 - Structures sensitive to pain

muscle which stimulate peripheral receptors in these tissues, resulting in the perception of pain.

Intracranial pain-sensitive structures in the head include the intracranial venous sinuses and their tributaries; parts of the dura at the base of the skull; dural arteries (anterior and middle meningeal); proxinal portion of the cerebral arteries leading to and coming from the circle of Willis; upper cervical nerves; fifth, ninth and tenth cranial nerves. (Fig.2)

The brain parenchyma, much of the dura, arachnoid and pia matter, the ependyma of the ventricles, the choroid plexus and the cranium are insensitive to pain. The periosteum is locally sensitive to stretch.

The basic mechanisms in intracranial diseases are TRACTION due to direct or indirect DISPLACEMENT of pain sensitive structures, DISTENTION AND DILATION of intracranial arteries, INFLAMMATION in or about the pain-sensitive structures of the head, DISTORTION of pain-sensitive areas due to increased intraventricular pressure caused by lesions that obstruct cerebrospinal fluid flow, DIRECT PRESSURE by an intracranial mass on certain cranial and cervical nerves. One or more of the mechanisms may be operating in any given patient with headaches. Raised intracranial pressure by itself does not cause headache. (Fig.3)

Headache cannot be provoked by raising the pressure