BRAIN MAPPING
AND LEVEL OF AMMONIA
IN HEPATIC ENCEPHALOPATHY

Thesis

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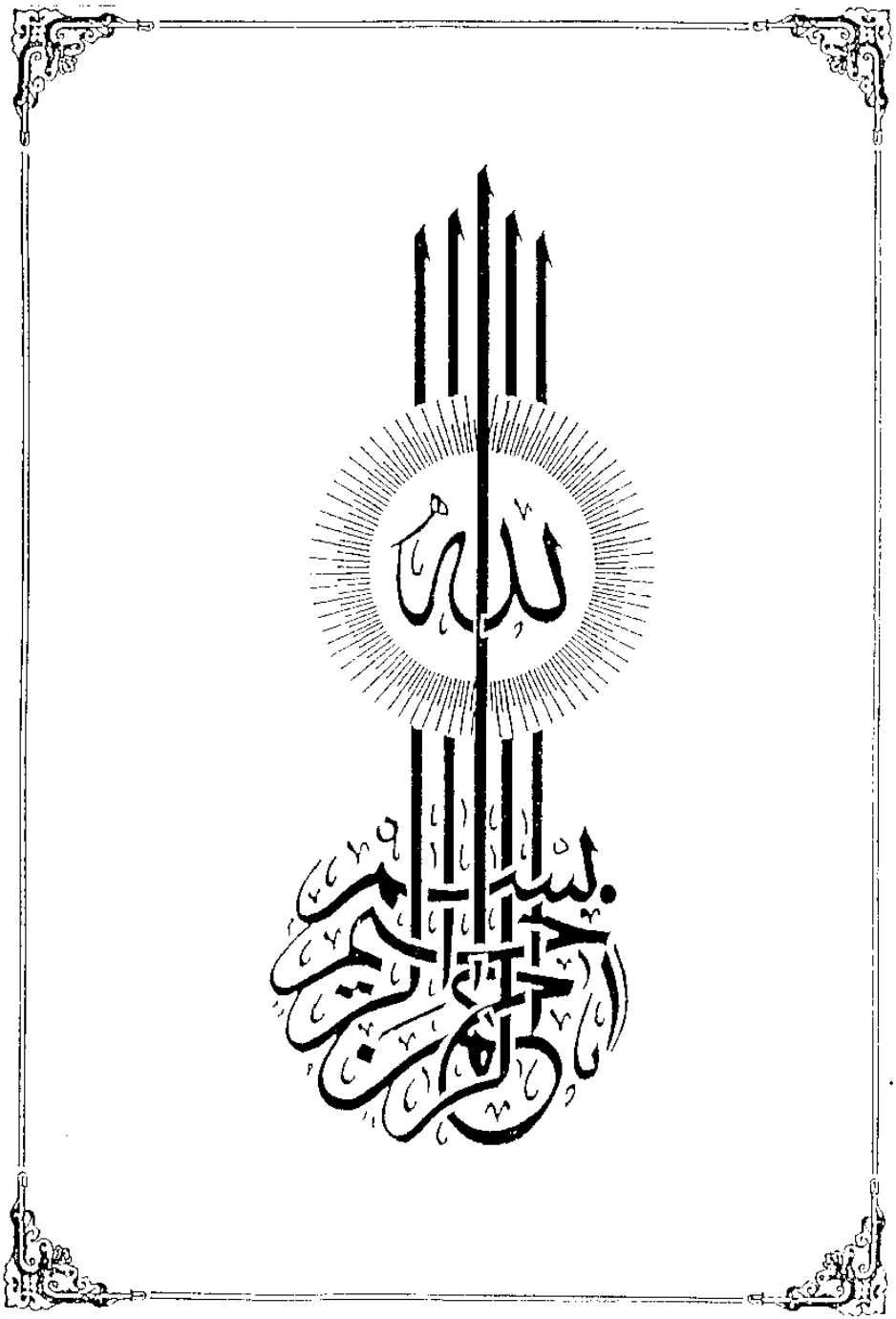

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CONTENTS

Interoduction

INTRODUCTION

The clinical observation of neuropsychiatric changes and EEG recording have been used to differentiate the grades of HE due to liver cirrhosis (Sherlock, 1993), but non of them seems objective or sensitive to quantify the degree of coma nor to detect the SHE (Pappas, 1984).

Also, in general the level of ammonia tend to rise in direct proportion to the severity of mental changes although the correlation may not be close and can not be quantified with HE or in patients with cirrhosis (Stahli, 1963).

Recently brain mapping (automated EEG analysis) is reported to have a reliable and specific role in evaluation of neurological changes of HE (Duffy, 1984).

Aim of The work

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The aim of this work was for study the value of topographic brain mapping, and comparing it to the level of ammonia, in detection of clinical and subclinical HE in patients with liver cirrhosis.

REVIEW OF Literature

CHAPTER (1)
HEPATIC ENCEPHALOPATHY

1. Definition

Hepatic encephalopathy (HE) is a neuropsychiatric syndrome that develops secondary to liver disease or porto-systemic shunts or both (Sherlock, 1993).

Various terms used to define these disorders such as porto-systemic encephalopathy, hepatic encephalopathy, hepatic coma, however, the term (hepatic encephal) is more appropriate since encephalopathy also occurs in cirrhotic patients without presence of large blood shunting as direct consequence of hepatocellular failure (Ferenci, 1991).

2. Classification

(1) *Subclinical Hepatic Encephalopathy*: They are considered normal as there is no gross alteration in mental function but early detectable by neuropsychological testing such as, brain mapping and neuropsychiatric tests (Ferenci, 1991).

(2) *Acute or Subacute Hepatic Encephalopathy*: Patients with acute liver failure, rapid onset, short course for few days, bad prognosis and difficult to reverse (Zieve, 1987).

(3) *Acute or Subacute Recurrent*: If there is more than one acute episode usually there is precipitating factors

e.g., gastrointestinal bleeding, drugs, infections. This is usually associated with good prognosis and subside usually without specific treatment (Capocaccia, 1984). The main difference in outlook for patients with acute and acute recurrent hepatic encephalopathy that the first is due to mainly hepatocellular failure alone, whereas the second is associated with readily correctable precipitating factors (Ferenci, 1981).

(4) *Chronic Recurrent Hepatic Encephalopathy*: They are cirrhotics with extensive portal collateral circulation if untreated will take a periods of months or years but reversible and controlled with suitable therapy (Summerskill *et al.*, 1976).

(5) *Chronic Permanent Hepatic Encephalopathy*: Mostly in cirrhotic with portal collateral circulation and developed permanent neurological abnormalities resembling that seen in Wilson's disease (Victor *et al.*, 1988).

(6) Rarely is one of myelopathy which is almost reversible (Conn, 1993).

Clinical Grading of Hepatic Encephalopathy

- Stage 0 normal: No symptoms, no signs of HE, normal psychomatric tests.
- Subclinical: Will be discussed in details.
- Stage I, II, III, IVa and b.

Stage I

It is the most difficult to recognize (Read *et al.*, 1977). Usually there is mild confusion, euphoria or depression, decreased attention, slowing of ability to perform mental tasks, irritability, disorder of sleep pattern (Conn, 1979).

Stage II

The early symptoms of altered mental status become more pronounced and sleep pattern are greatly disturbed. The characteristic flabbing termor (astrixis) is generally present which occur in the form of tremulousness after a latent period of 2-30 seconds, this tremulousness has 2 separate components, one is a varying oscillation of the fingers, usually in the antero-posterior direction, but with a rotatory component at the wrist. The second component consists of tiny, but clearly defined, random motions of the fingers at the metacarpal - phalangeal joints from 0-2 times/second, and increase in amplitude when the hands remained dorsiflexed (Leavitt, 1965).

Also, there is cerebellar disturbances which manifested by dysartheria, intention tremors, , abnormal gait. Basal ganglia involvement produces: Parkinsonian features as resting tremors, choreoathetoid movement (Summerskill *et al.*, 1976; Read *et al.*, 1977).

Stage III

Manifested by increased obtundation, sleeping for most of the day but able to be aroused when shaken (Zieve, 1987).

Stage IV

(a) Comatosed with some response to painful stimuli.

(b) Comatosed with no response to any stimuli.

(Sherlock, 1993)

In stage III and IV there is Glasgow scale as an additional useful clinical parameter:-