NON SURGICAL MANAGEMENT OF OESOPHAGEAL VARICES

Essay

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in

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By

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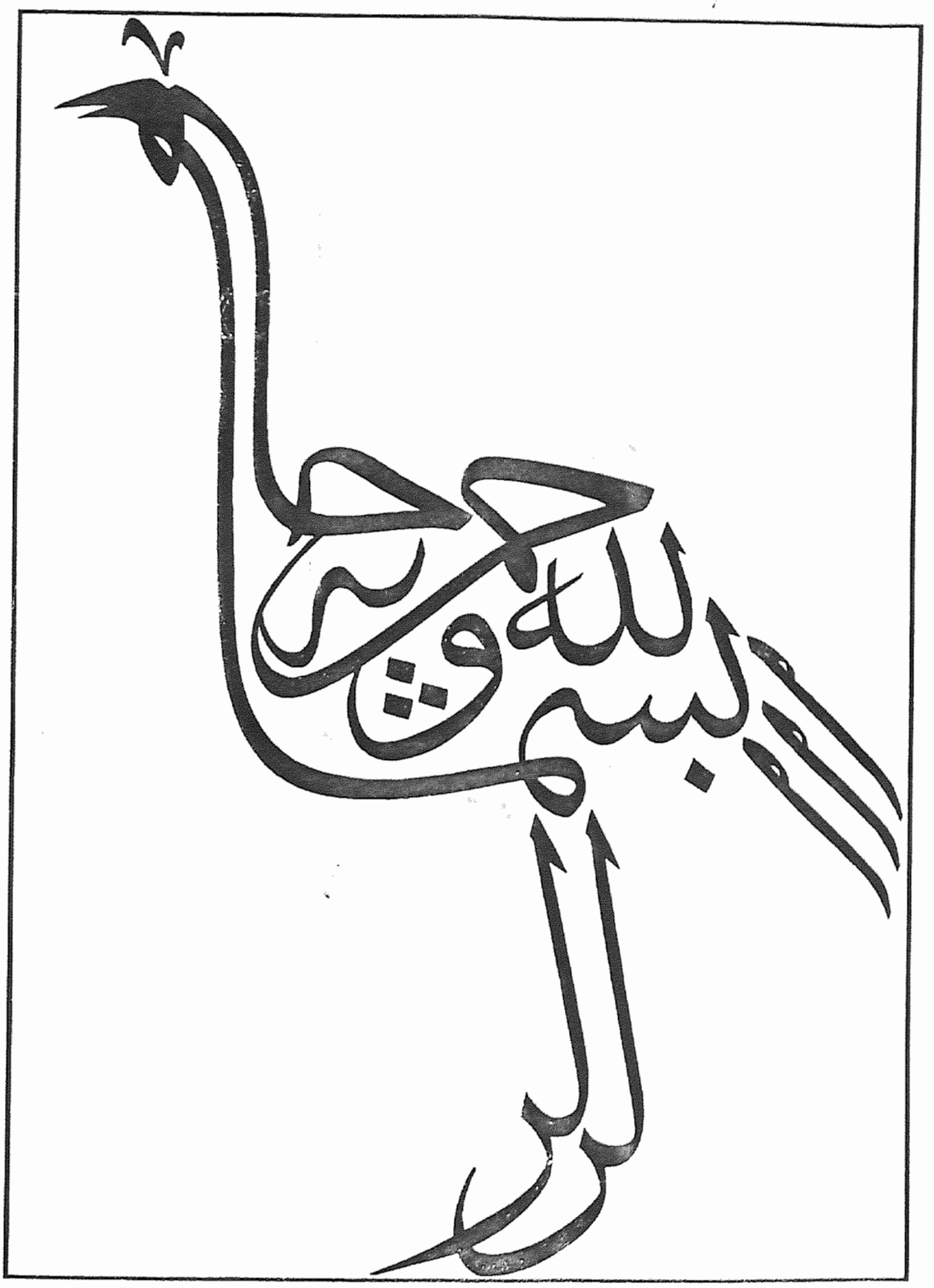
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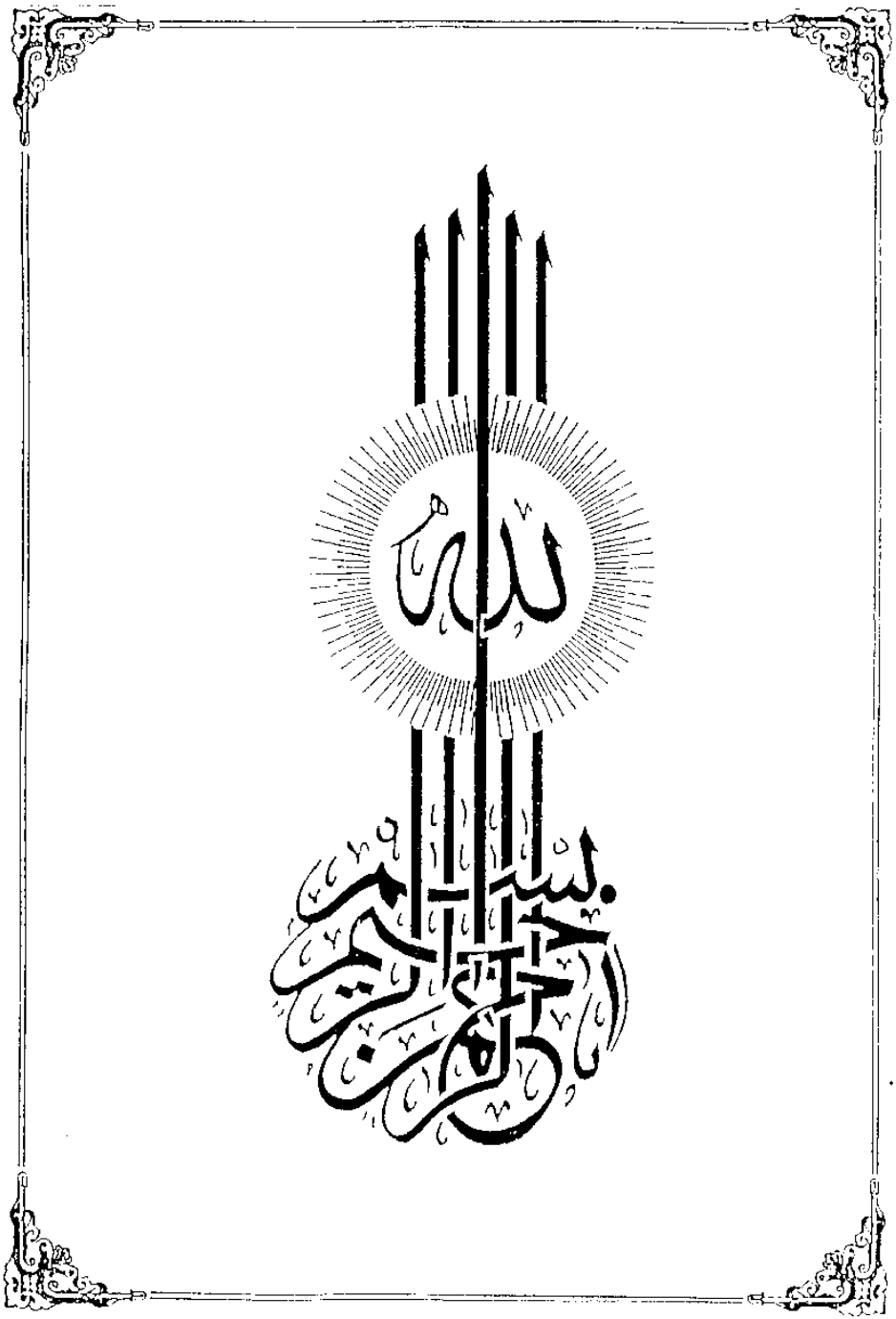

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To
My Father
My Mother

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Introduction

INTRODUCTION

In Egypt Schistosomiasis has been an endemic disease since ancient times **(Ruffes, 1910)**. Accordingly, the problem of portal hypertension consequent upon schistosomal hepatic fibrosis constitutes current national and economic problem. Bleeding from oesophageal varices is the most dreadful complication of portal hypertension where it occurs to many people endangering their lives **(Johansen and Helton, 1992)**.

Now non operative methods take the upper hand in treating the acute variceal bleeding **(Sauerbruch and Discher, 1991)**, Sclerotherapy being the most popular primary treatment. It can control variceal haemorrhage in most of patients **(Treblanche, 1990)**. Also balloon tamponade has been proved to be a successful temporising technique in controlling acute variceal bleeding **(Paquet and Feussner, 1985)**. In addition there is evidence that pharmacological measures have the same effect as endoscopic sclerotherapy **(Jenkins *et al.*, 1988)**.

Trans Jugular Intrahepatic Portosystemic Shunt (TIPS) seems to be a promising treatment of portal hypertention with low rates of operative mortality ,rebleeding and low incidence of hepatic encephalopathy **(Rössle and Hagg, 1992)**.

Although non surgical methods have proved to be effective in controlling acute bleeding episodes from oesophageal varices, yet their role in prevention of initial or recurrent attacks of bleeding is not that satisfactory **(Johansen and Helton, 1992)**.

Aim of the Work

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The aim of this work is to review the voluminous literature as well as the update knowledge about the non surgical management of oesophageal varices.

Etiology of Portal Hyper Tension

AETIOLOGY OF PORTAL HYPERTENSION

Portal pressure is determined by portal venous blood flow and the resistance to portal venous inflow to the liver. The major component subject to alteration is portal venous flow, which in turn is regulated by splanchnic blood flow (**Benoit and Granger 1986**). In normal circumstances, physiological stimuli including food ingestion and exercise, may alter splanchnic blood flow and thereby influence portal flow (**Richardson and Withrington, 1981**). Homeostatic mechanisms involving neurohormonal factors compensate for any change in portal blood flow and serve to maintain portal pressure within a normal range (less than 8 mmHg) by modulating portal vascular resistance (**Richardson and Withrington, 1981**).

Portal hypertension results when the compensatory mechanisms are inadequate as consequence of pathological increase in either portal venous inflow or resistance (**Witte and Witte, 1982**).

Causes of portal hypertension: (Boyer, 1982)

I. **Increased resistant to flow:**

A. Prehepatic (Portal vein obstruction):

1. Congenital atresia or stenosis
2. Thrombosis of portal vein.
3. Thrombosis of splenic vein.

4. Extrinsic compression.

5. Cavernomatous transformation

B. Hepatic:

1. Cirrhosis:

- Portal (alcoholic-nutritional-laennec's)
- Post-necrotic (post hepatitic, idiopathic)
- Others (Wilson's disease, late schistosomiasis)

2. Schistosomiasis.

3. Acute alcoholic liver disease.

4. Congenital hepatic fibrosis.

5. Idiopathic portal hypertension (hepato-portal sclerosis)

C. Post hepatic

1. Budd-chiari syndrome

2. Veno-occlusive disease.

II. Increase portal blood flow:

A. Arterial portal venous fistula.

B. Increased splenic flow.

1. Banti's syndrom.

2. Splenomegaly (not due to liver disease).

(After Boyer, 1982)

Haemodynamic studies allow further more accurate classification depending on the predominant sites of resistance. As more than a single resistance site may be present.

(Mac Mathuna *et al.*, 1992)

I. Increased resistance to flow:

i. Presinusoidal

A. Extrahepatic (portal vein thrombosis)

a. Omphalitis

b. Pancreatitis.

c. Trauma.

d. Malignancy.

e. Polycythemia.

f. Peripheral lymphadenopathy.

B. Intrahepatic

a. Biliary atresia.

b. Primary biliary cirrhosis.

c. Sarcoidosis.

d. congenital hepatic fibrosis.

e. Myeloproliferative disorders.

f. Hepatoportal sclerosis.

ii. Sinusoidal

A. Cirrhosis

B. Toxic hepatitis

C. Fatty metamorphosis.

iii. Post sinusoidal

A. Intra hepatic.

1. Cirrhosis

a. Post necrotic

b. Portal

c. Hemochromatosis.

2. Veno-occlusive disease.

B. Extra hepatic

1. Budd-chiari syndrome

2. Congestive heart failure/constrictive peri cardi-

tis

II. Increase blood flow

A. Arterial portal venous fistula.

B. Increased splenic flow (Banti's syndrome)