DUPLEX SCANNING AND COLOUR FLOW MAPPING OF THE LEFT GASTRIC VEIN IN PATIENTS USING VARIOUS DRUGS FOR PORTAL HYPERTENSION

THESIS

Submitted for partial fulfilment of Master degree in internal medicine

By

Ahmad Mohamad Afifi

MB. B. Ch.

Supervisors

Prof : DR. Mohamad Abdelfattah Taha

Prof. of internal medicine

Ain Shams University

DR. Abdelrahman Abdelhamid Soliman

Lecturer of internal medicine

Ain Shams University

DR. Hassan Galal Ismail Mourad

Lecturer of radiodiagnosis

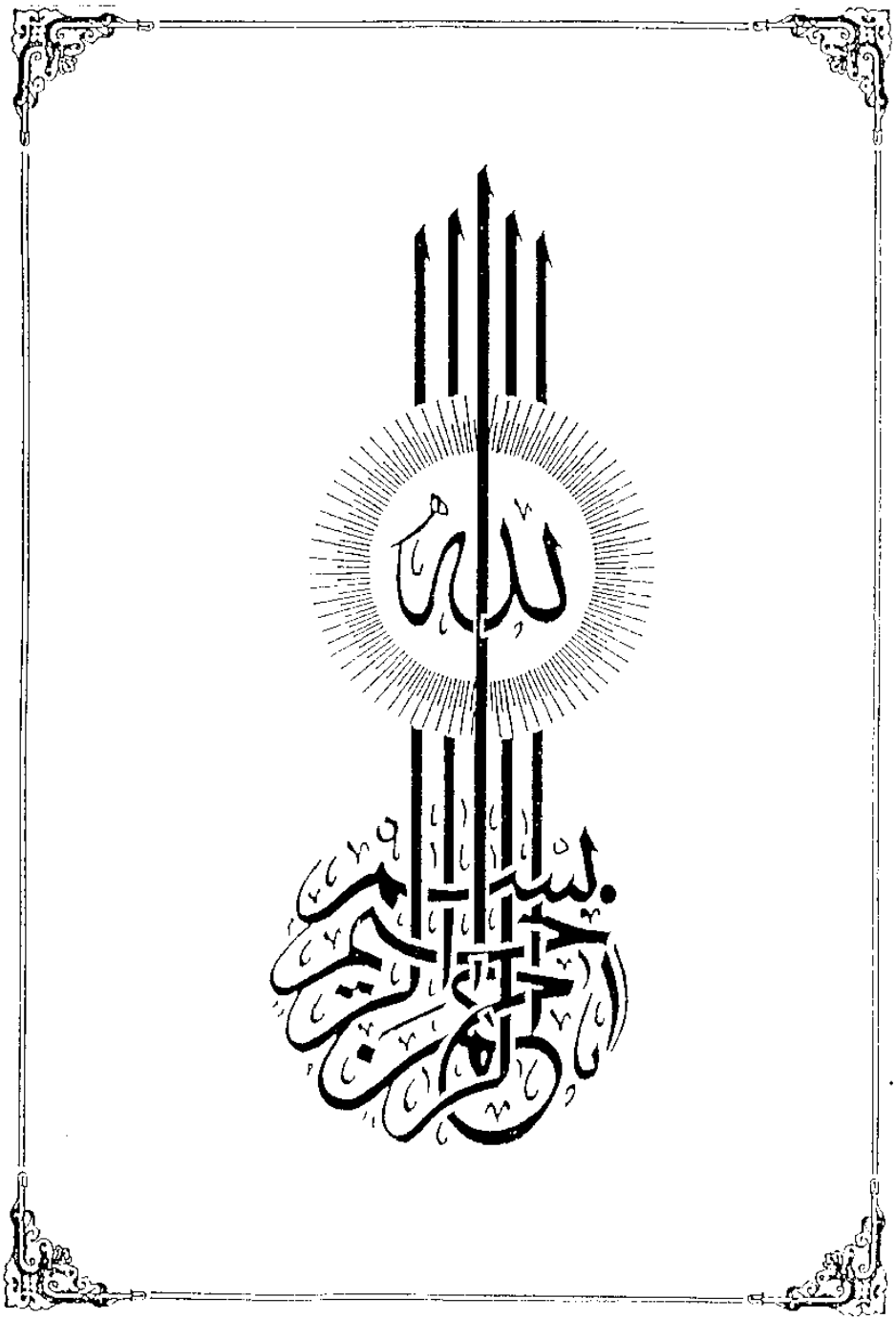
Ain Shams University

FACULTY OF MEDICINE
AIN SHAMS UNIVERSITY
IN 1995

بسم الله الرحمن الرحيم

سبحانك لا علم لنا إلا ما علمتنا

إنك أنت العليم الحكيم



ACKNOWLEDGMENT

I would like to express may sincere thanks and deepest gratitude to **Prof. Dr. Mohamad Abdelfattah Taha**, Professor of internal medicine, Ain Shams University, who offered me the fatherly encouragement, the generous support and many useful criticisms and suggestions throughout this study.
His precious guidauce and continued supervision, which were kindly given, are beyond acknowledgment.

I am honored to thank **Dr. Abdelrahman Abdelhamed Soliman**, lecturer of internal medicine,Ain Shams University, for his cordial support, most appreciable advice and help throughout this work.

I wish to express may deepest gratitude to **Dr. Hassan Galal Mourad**, lecturer of radiodiagnosis, Ain Shams University, for his kind help and true assistance in fulfilling the practical part of this work.

I am deeply grateful to **Dr. Mohamad Azzazi** lecturer of internal medicine Ain Shams University for his great help, co-operation and close guidance in this work.

I am deeply grateful to **Dr. Hossni Rafla**, head of gastro-enterolgy department and endoscopic unit in Mataria Teaching Hospital, for his kind help and True assistance in fulfilling the practical part of this work.

CONTENTS

Introduction And Aim of The Work

INTRUDUCTION AND AIM OF THE WORK

The left gastric vein is a major pathway to oesophageal varices in portal hypertension, it reflects the haemodynamic changes in oesophageal varices. Measurement of blood flow in it by doppler ultrasonography is a useful non invasive technique for evaluation of variceal hemodynamics.

Bleeding from oesophageal varices, gastric varices and portal hypertensive gastropathy is a major cause of morbidity and mortality in cirrhotic patients.

Much effort has been paid to the research of new and more effective forms of medical treatment of portal hypertension, new pharmacological agents have been introduced both for the control of acute variceal bleeding and for the prevention of variceal bleeding *(Burrough, et al., 1992)*.

The aim of this work is to evaluate the effect of, propranolol verapamil and captopril on hemodynamics of LGV and hence on hemodynamics of oesophageal varices in cirrhotic patients with portal hypertension.

Review of Literature

REVIEW OF LITERATURE

ANATOMY OF PORTAL CIRCULATION

The portal system includes all veins that carry blood from the abdominal part of the alimentary trace, the spleen, pancreas and gall bladder. The portal vein enters the liver at the portahepatis and is divided into two main branches, one to each lobe, it has no valves in its larger channels *(Sherlock, 1993)* .

The portal vein is formed by union of the superior mesentric vein and the splenic vein, just anterior to the head of pancreas at about the leve of the second lumbar vertebra. It extends slightly to the right of the mid-line for a distance of 5.5-8 cm. to the porta hepatis. It has a sigmental intrahepatic distribution accompanying the hepatic artery *(Sherlock 1993)*.

The superior mesentric vein is formed by tributaries from the small intestine, colon, head of pancreas and irrigularlyfrom the stomach via the right gastro-epiploic vein.

The splenic venous channels (5-15) originate at the splenic hilum and join near the tail of pancreas with the short gastric veins to form the main splenic vein. This proceeds in a transverse direction in the body and head of pancreas, lying below and in front of the splenic artery. It receives numerous tributaries from the head

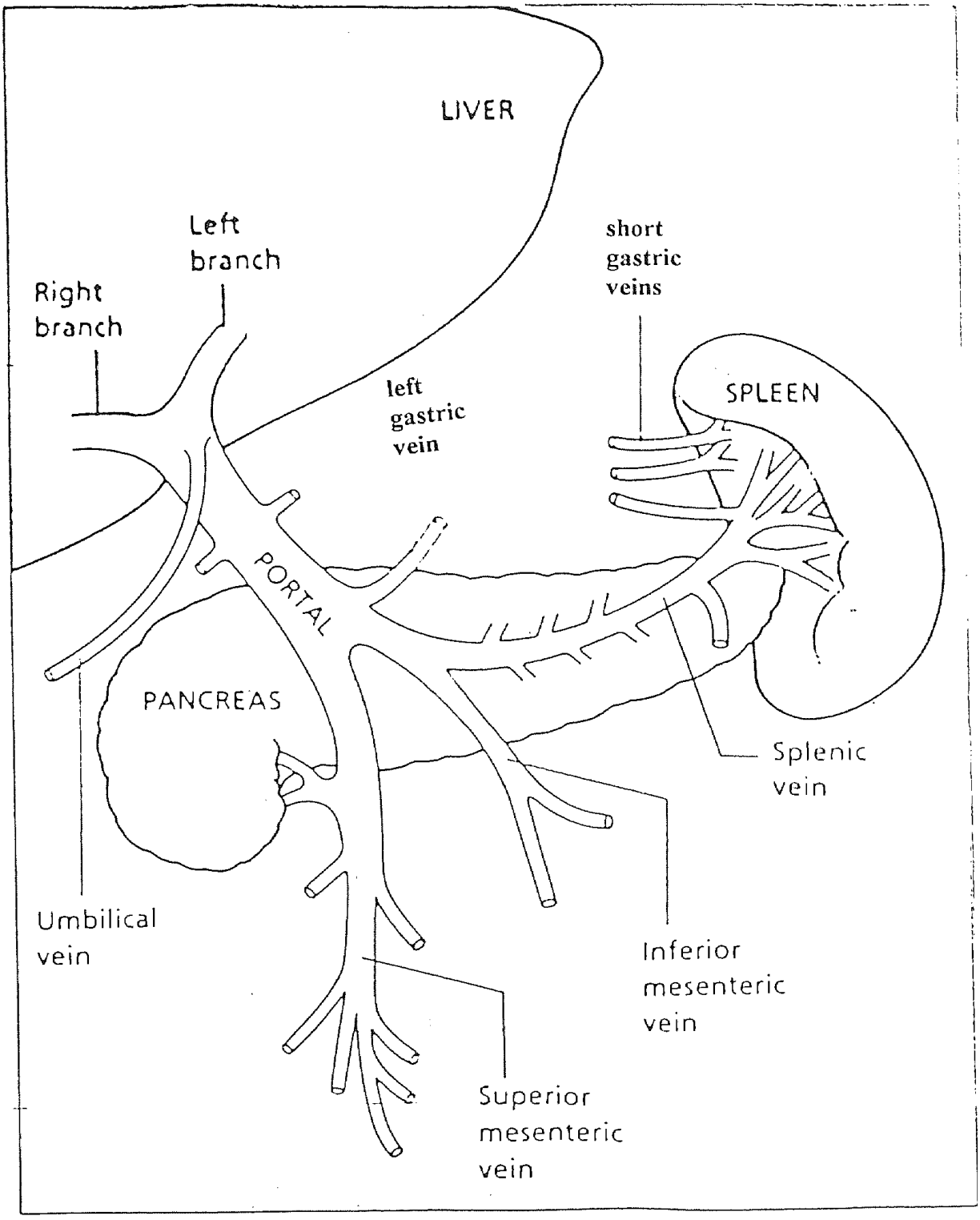
of pancrease, and the left gastro-epiploic vein enters it near the spleen *(Sherlock, 1993)*.

The inferior mesentric vein is bringin blood from the left part of the colon and rectum usually enters the splenic vein at its medial third. Occasionally, however it enters the junction of superior mesentric and splenic veins.

The left gastric vein usually originates from the portal splenic vein junction or its vicinity and runs to the oesophago-gastric junction *(Matsutani, et al., 1993)*. It runs to the left along the lesser curvature up to the oesophagus, then passes around behind the peritoneum of the lesser sac with the left gastric artery. It passes down to the right above the hepatic artery and joins the portal vein at the upper border of pancrease.

Pattern of hepatic distribution of portal inflow :

There is no consistent pattern of hepatic distribution of hepatic inflow. Sometimes, splenic blood goes to the left lobe and sometimes to the right lobe. Crossing over of the blood stream can occur in the human portal vein. Blood flow is probably stream-lined rather than turbulent *(Sherlock, 1993)*.



The anatomy of the portal venous system.

(Sherlock, 1993)

PORTAL HYPERTENSION

Portal pressure is normally about 7 mm Hg in man. Portal hypertension is the elevation of the pressure in portal vein to about 10-12 mm Hg *(Sherlock, 1993)*.

Causes of portal hypertension

Portal hypertension usually follows obstruction to the portal blood flow anywhere along its course *(Reynolds, 1982)*.

Sherlock (1986) classified portal hypertension into two groups, presinusoidal extrahepatic or intrahepatic and a big general group of hepatic causes.

Classification of portal hypertension

Type	Cause	Underlying disorders
I. Presinusoidal a. Extrahepatic	blocked portal vein	1. Intra-abdominal infection 2. Clotting diseases. 3. Tumour invasion. 4. Trauma.
b. Intrahepatic	Portal zone infelteration	1. Schistosomiasis. 2. Congenital hepatic fibrosis 3. Sarcoidosis. 4. Lymphomas. 5. Primary biliary cirrhosis.
II. Hepatic a) Intrahepatic	Cirrhosis	1. Alcholism. 2. Chronic active hepatitis.
b) Postsinusoidal	Hepatic venous obstruction	1. Clotting diseases. 2. Tumour invasion. 3. Oral contraceptives. 4. Cytotoxic drugs.

(Sherlock, 1986)

I. Presinusoidal :

In presinusoidal extrahepatic portal venous obstruction, the obstruction may be at any point in the course of the portal vein *(Webb, 1979)*.

1. Aetiology of extrahepatic obstruction :

A) Infection :

Umbalical infection with or without catheterization of the umbalical vein may be responsible in neonates *(Thompson et al.,1964)*.

Acute appendicitis and peritonitis are causative in older children. Ulcerative colitis and crohn's disease could be complicated by portal vein obstruction. Portal vein obstruction may be secondary to biliary infection, for instance to gall stones or primary sclerosing cholangitis *(Sherlock, 1985)*.

B) Post-operative :

The portal and splenic veins commonly blocked after splenectomy, specially when, preoperatively, the patient had normal platelate count, which however rises postoperatively. The thrombus spreads from the splenic vein into the main portal vein. It is specially likely in patients with myeloid metaplasia *(Broe et al.,1981)*.

C) Trauma :

Portal vein injury usually follows car accidents and is rare *(Sherlock, 1986)*.

D) Hypercoagulable state :

Enhanced clotting may be a factor, particularly in the older age group. Myeloproliferative diseases specially polycythemia rubra vera are the commonest association.

E) Invasion and compression :

The classic example is hepato-cellular carcinoma. Carcinoma of head of pancreas, of the body and of the adjacent organs may lead to portal vein block.

F) Congenital :

Congenital obstruction could be produced any where along the line of the right and left vitellin veins from which, the portal vein developed. The portal vein may be absent with visceral venous return passing to systemic veins particularly the inferior vena cava *(Morse et al., 1986)*.

G) Miscellaneous :

Portal vein thrombosis has been associated with pregnancy and with contraceptive pells, especially in older women and with long use *(Capron et al., 1981)*.

H) Unknown causes :

In some patients , the aeitology of portal hypertension after the fullest investigations, remain obscure.

2. <u>Presinusoidal intrahepatic obstruction</u> :

A) Schistosomiasis :

Ova pass into the portal blood stream and lodge as emboli in the intrahepatic portal venules.

Elwi and Attia (1962) described the intrahepatic vascular changes in the course of schistosomal fibrosis.

B) Congenital hepatic fibrosis :

Portal hypertension is probably due to deficiency of terminal branches of the portal vein in the fibrotic portal zones *(Kane and Katz, 1982)*.

C) Sarcoidosis :

Vilinsb et al., (1970) considered portal hypertension to be a mani-festation of increased inflow through the large spleen. It has been postulated that the granulomatous lesions obstruct the portal venules hepatic venules or sinusoids.