# SURGICAL MANAGEMENT OF PORTAL HYPERTENSION

ESSAY

SUBMITTED IN PARTIAL FULFILLMENT OF

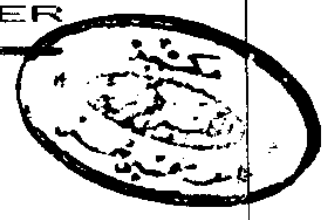
THE MASTER DEGREE IN

GENERAL SURGERY



MOSTFA ALY AWAD NASSER

(M.B., B.Ch.)



SUPERVISORS

PROF. DR. ADEL FAHIM AIN SHOKA

PROFESSOR OF GENERAL SURGERY

AIN SHAMS UNIVERSITY



DR. ABD ALLAH EL SAID

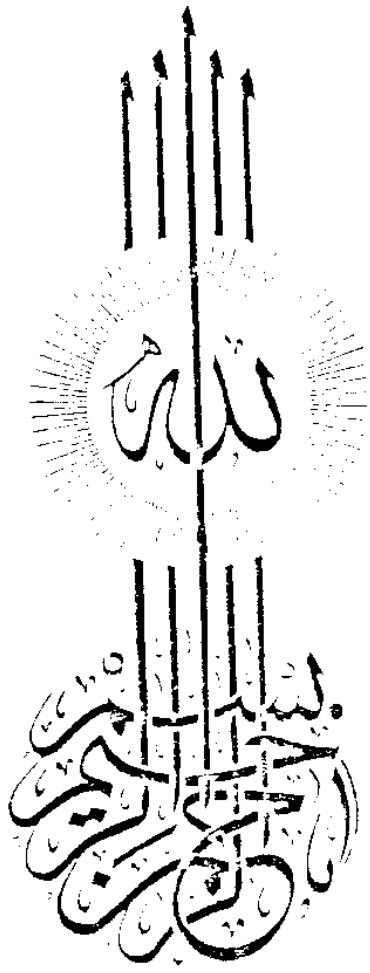
LECTURER OF GENERAL SURGERY

AIN SHAMS UNIVERSITY

FACULTY OF MEDICINE

AIN SHAMS UNIVERSITY

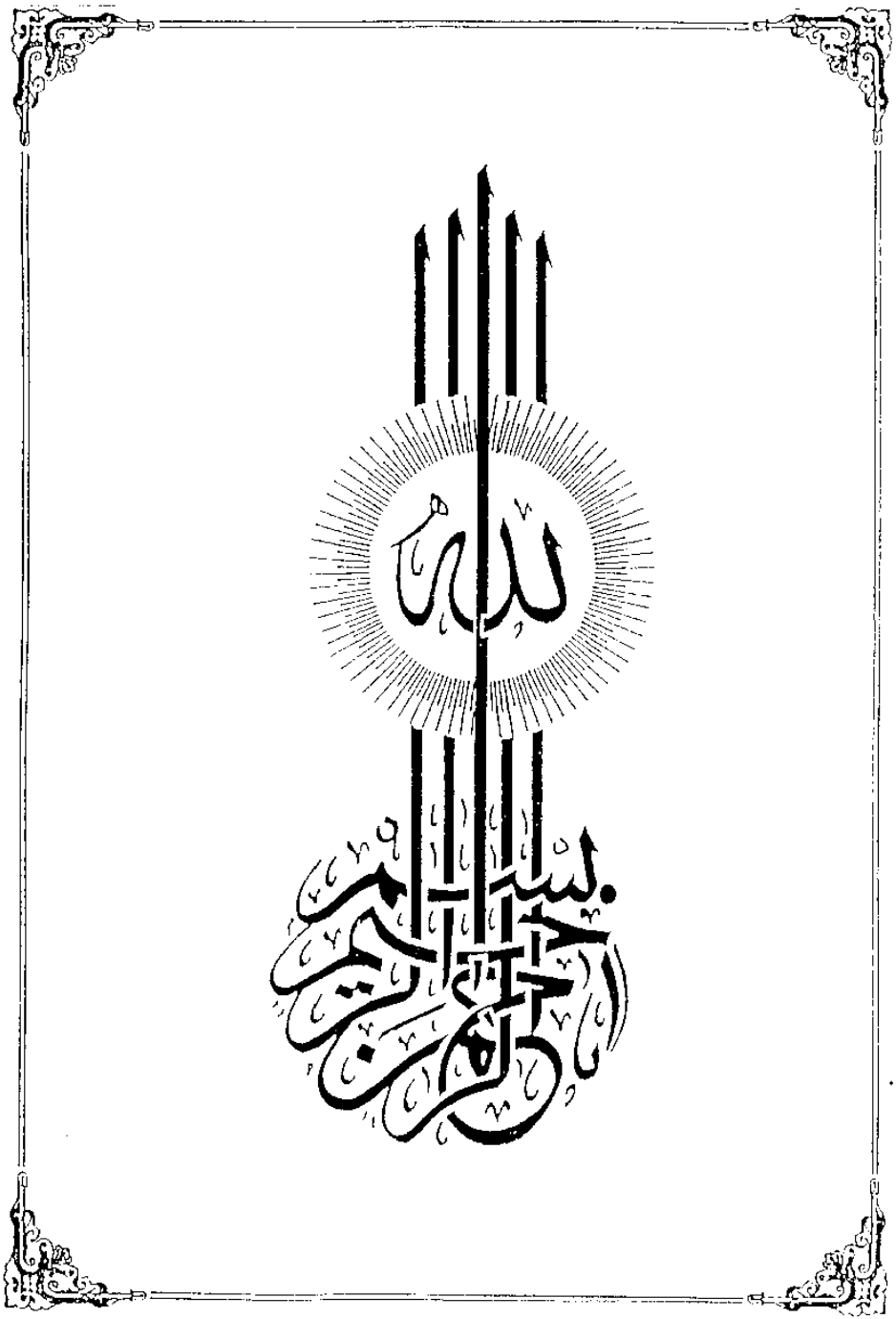
1987



« علم الانسان ما لم يعلم »

صدق الله العظيم

سورة العلق آية ٥



# INTRODUCTION

CONTENTS



* * *

# REVIEW OF LITERATURE

## ACKNOWLEDGMENT

I wish to express my deepest gratitude and sincerest thanks to PROF. DR. ADEL FAHIM AIN SHOKA, Prof. of General Surgery, Ain-Shams University for his very valuable supervision, fruitful guidance and kind help through out the course of the work.

I am also deeply indepted and cordially grateful to DR. ABD ALLAH EL SAID, Lecturer of General Surgery for his endless help.

* * *

# INTRODUCTION

Portal hypertension is one of the most exacting challenge in clinical medicine today, and its alarming complications are still attracting the attentions of surgeons and Physiciens allover the world especially in our country. Bilharzial hepatic fibrosis is the commonest cause of portal hypertension in Egypt, while alcoholic cirrhosis is the commonest cause in Europe and America.

The overriding problem is the management of patients with portal hypertension is the treatment ofits major complications gastro intestinal haemorrhage. Esophageal varices are the commone cause for severe, Persistent upper gastro intestinal bleeding.

Bleeding esophageal varices have long represented a challenge both to the physician and surgeon in the management of severe haemorrhage superimposed on the back ground of chronic liver disease and its metabolic sequle, Therefore two major clinical challenges are present.

1) The control of acute bleeding and the prevention of immediate potential complications.
2) The prevention of any subsequent bleeding and thus possibly The improved survival of patients who have bled.

The aim of this study is to review the experience of many authors to find the current views and the ideal treatment to this critical patients with less attendant mortality and morbidity.

## ANATOMY OF THE PORTAL CIRCULATION:

The portal vein is formed by the confluence of the splenic and superior mesenteric veins at the level of the second lumbar vertebra behind the neck of the pancreas.

The portal vein lies in front of the inferior vena cava, passes upwards behind the pancreas and the first part of the duodenum and enters between the two layers of lesser omentum.

It runs for 8-9 cm. from its origin to the Hilum of the liver where it divides into two lobar branches right and left branches.

Below the 1st part of the duodenum, the common bile duct curves away from it to the right behind the pancreas, and the Hepatic artery curves to the left infront of the pancreas to the coeliac artery [Last (1979).

The portal system conveys between 60-80% of the afferent blood to the liver, together with the products of digestion from the alimentary canal and the internal secretions, insulin and glucagon from the pancreas.

No less than one-fifh of the portal blood comes from the spleen **(Rain & Ritchie 1982).**

The portal vein receives tributaries from the lower Esophagus and most of the stomach. It receives:-

* Left gastric vein (coronary).
* Rt. Gastric vein.
* Superior pancreatico duodenal vein.
* Right gastro-epiploic vein.
* Para umbilical vein.
* Cystic vein (Last 1979)

At the free border of the lesser omentum, the portal vein has the following relations:-

* Bile duct in front and to its right side.
* Hepatic artery in front and to its left side.
* Inferior vena cava behind it, but separated from it by the opening of the lesser Sac. (foraman of Winslow) (Last 1979).

The coronary (left gastric vein ) usually enters the portal vein on its anteromedial aspect just proximal to the margin of pancreas,in which case it usually must be ligated during the surgical construction of a porto caval shunt.

In 25% of cases, the left gastric vein joins the splenic vein. The inferior mesenteric vein generally drains into the splenic vein several centimeters to the left of the junction with superior mesenteric vein (way 1981).

In the hepato duodenal ligament, the portal vein lies dorsal and slightly medial to the common bile duct, here a large lymph node is often present lateal to the vein and must be dissected off before a shunt can be performed.

In adult, the portal vein and its tributaries are free from valves, but in the foetus and soon after birth, valves can be demonstrated in the Tributaries of portal vein which (disappear) in the adult age but sometimes they persist in a degnerate form.

Anatomy of collateral circulation: (Fig.1)

The aim of the collateral circulation in cases of portal hypertension is to divert blood away from the liver to decongest the stagnant portal syste due to Intra Hepatic or extra-hepatic obstruction of the portal blood flow.

Sites:

a) Around the Cardio Esophageal area:

Between the left gastric and short gastric (portal system) oesophageal, azygos, Diaphragmatic, intercostals (systemic circ) producing (Esophageal) varices.

b) Around the anus:

Between the superior rectal (portal system), middle and inferior retctals (systemic circ.)------producing piles.

c) Around the umbilicus:

The para umbilical vein extends from the hilum of the liver to the umbilicus where it anastomoses with the veins of the abdominal wall, as they distend and become varicose they produce-caput medosae.

d) Retroperitoneal:

i) Reteroperitoneal vein of Retzius which connects the intestinal veins and the inferior vena cava.
ii) Accessory portal system of sappy between the bare area of the liver and the diaphragm.

Physiology of the Portal circulation:

A large share of cardiac output flows through the vessels of the intestine through thespleen, finally coursing into the portal venous system and then through the liver this is called the portal circulatory system and plus the arterial blood flow into the liver is called splanchnic circulation (Guyton 1981).

Approximately 350 litres of blood flow through a normal spleendaily (Philpott & Ballinger 1977).

Total hepatic blood flow is about 1500 ml/mint. and constitutes 29% of the cardiac output. Two thirds of the flow enters through the portal vein and one-hird through The hepatic artery (Gyton 1981) The splanchnic circulation is composed mainly of 3 parts namely:

Mesenteric circulation, splenic circulation and the hepatic circulation. The intraluminal pressure of the Mesenteric vein is about 15 mm.Hg.

The hepatic artery and portal vein converge on the hepatic sinusoids which drain into the central lobular vein and then to the hepatic vein and then to theinferior vena cava.

The portal venous pressure is about 3-13 mm.Hg., while hepatic venous pressure is about 5 mm.Hg.

The pressure in the hepatic artery branches that converge in the sinusoids is 90 mm.Hg., while the pressure in the sinusoids is less than the pressure in the portal vein 3-13 mm. Hg. which is considerably higher than the almost zero presure in the vena cava.

This marked drop of pressure between the arterial and sinusoidal pressure can be explained due to the presence of the hepatic arterioles which are innervated by vasoconstrictor fibres reaching the hepatic artery via the Hepatic sympathetic plexus.

There are no known vasodilator nerve fibres reaching the liver. also there are no known functioning hepatic vein sphincters in human

The regulatory mechanism governing sinusoidal blood flow are not well understood, but the normal sinusoidal bed can accomodate large variatons in portal flow without significant changes in portal pressure (Lautt, 77).

The endothelium of the hepatic sinusoids is generally much more permeable to proteins than any endothelium elsewhere in the body.

The hepatic artery after it supplies the structural elements of the liver empties into the hepatic sinuses to mix with the portal blood The pressure in the intestinal venules and capillaries have a much greater tendency to become abnormally higher than any capillaries elsewhere in the body due to considerably higher portal venous pressure than that in vena cava.

**Pathophysiology of portal Hypertension:**

Portal blood pressure is normally from 3-13 mm.Hg. (10-15 cm. water) The portal vein carries) two thirds of the hepatic blood flow which carry with it 25% of the oxygen reaching the liver. The portal blood pressure varies markedly during the day, it varies with changes in position, phases of respiraion and intra abdominal pressure (Maingot, 1985).

In all instances of portal hypertension, intra splenic pressure is elevated (normaly the pressure within the spleen is usually 27-81 cmm.saline higher than that of the portal vein per se.

Usually, portal hypertension results from increased resistance to flow and very rarely due to massively by increased portal inflow.

Sudden occlusion of the portal vein results in an immediate 60% rise in hepatic arterial flow, within weeks, total flow gradually returns toward normal.

On the other hand, sudden reduction of hepatic arterial supply are not immediately met by significant increase in the portal vein flow.

In both normal subjects and cirrhotics total hepatic flow and portal pressure drop following hepatic arterial occlusion.

Arterial collaterals develop over months and arterial perfusion is ultimately restored (Marks 1969).