بسم الله الرحمن الرحيم

وما أوتيتم من العلم إلا قليلا

صدق الله العظيم

EMERGENCY MANAGEMENT OF BLEEDING OESOPHAGEAL VARICES

ESSAY

Submitted in Partial Fulfilment for

MASTER DEGREE

(General Surgery)

By

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M.B., B.Ch.

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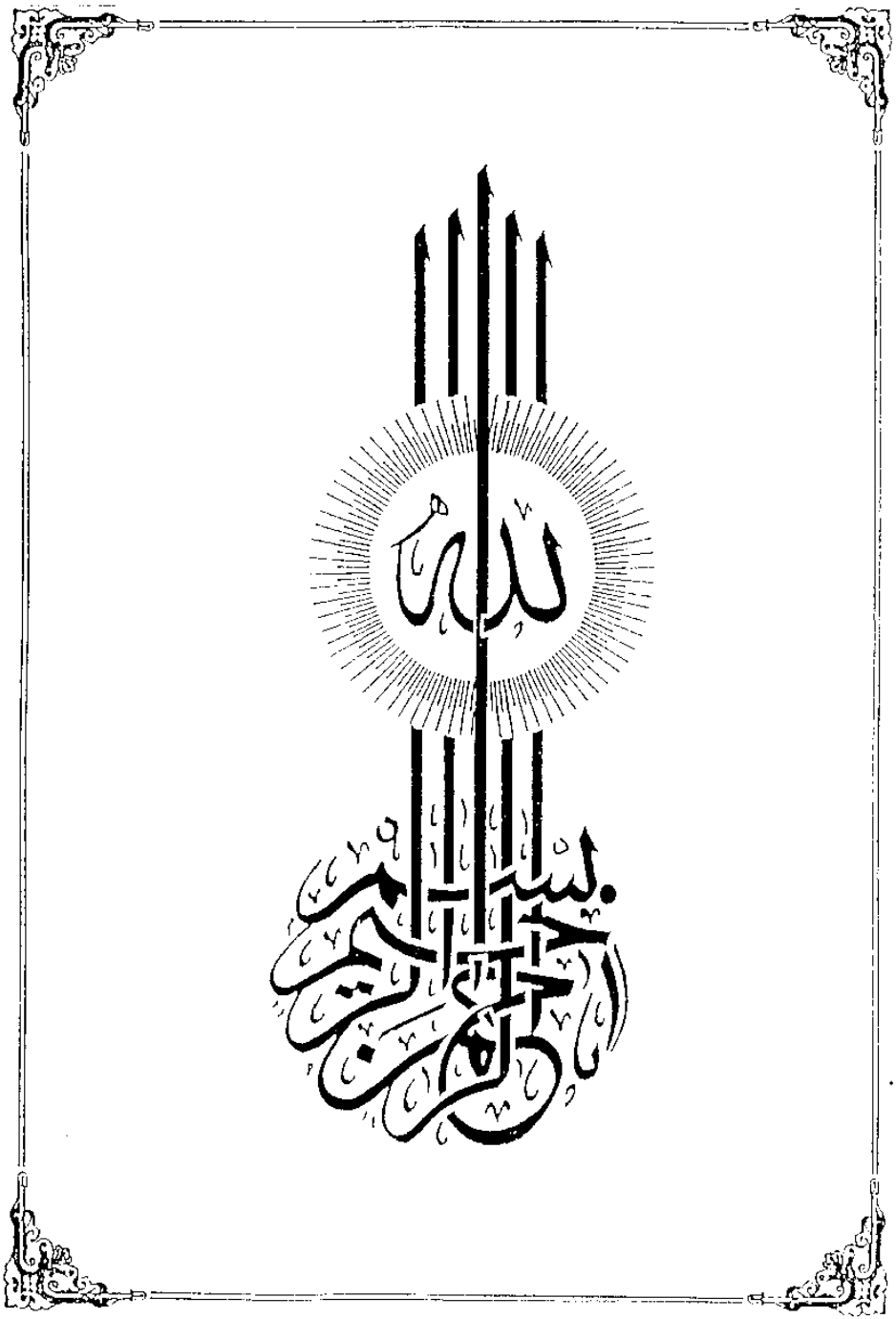
1986



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ACKNOWLEDGEMENT

First and foremost I should like to express my utmost gratitude to Prof. Dr. MOHEY EL-DIN SIDKI whose fatherly guidance and example was the basic foundation of this work.

I am also in great debt to Lecturer Dr. AMR SHERIF for his valuable remarks and helpful assistance.

I would like to express my deep thanks to all the professors, staff members and my colleagues in the Department of Surgery in Thoudor Bilharz Institute for the encouragement, support and help they gave me during the preparation of this work, especially Dr. N. EDWARD, the Head of the Department and Dr. HESHAM EL ZINI.

Last but not least, I would like to thank my wife for her rapid and accurate typing without which this work would have not been finished on time.

Ahmed Hazem Helmy
1986

CONTENTS

INTRODUCTION

INTRODUCTION

Portal hypertension is a great problem with its main consequence; upper gastrointestinal bleeding. This problem is socio-economic in Egypt. More than 30% of Egyptians in their childhood and youth (ages ranging between 5-35 years) have been attacked by schistosomiasis which is the main cause of portal hypertension and bleeding varices in Egypt. As we have said, it is a socio-economic problem as it affects indirectly the modes of development because one third of the productive mass of people are suffering of this disease directly or its complications mainly acute upper gastrointestinal bleeding which will be discussed in this essay.

In this review, I will start by presenting portal hypertension from the anatomical and pathophysiological point of view which will lead to the main subject that concerns me and has concerned me since I graduated; bleeding oesophageal varices and how to handle it in an emergency situation.

So many centers allover the world have dealt extensively with this subject, trying to find the best way of managing this situation with the least morbidity and mortality rates. In spite of their great efforts, there still remains a dilemma because of the relativity of

the methods they have used i.e. many questions still remain unanswered regarding which patients will benefit from certain forms of therapy. In this essay I will discuss methods and procedures that have been tried in the past decade.

Anatomical considerations of portal hypertension:

The portal system includes all veins collecting blood from the abdominal part of the digestive tube (with the exception of the lower part of the anal canal) and from the spleen, pancreas, and gall bladder. From these viscera blood is conveyed into the liver by the portal vein. In the liver this vein ramifies like an artery and ends in capillary-like vessels termed sinusoids, from which blood is conveyed to the inferior vena cava by the hepatic veins. The blood of the portal system therefore passes through two sets of 'exchange' vessels, the capillaries of the digestive tube, spleen, pancreas and gall bladder, and the sinusoids of the liver. The portal vein and its tributaries have no valves [Gray's Anatomy, 1979].

The portal vein collects blood from the splanchnic area - which includes the abdominal portion of the digestive tube, the pancreas and the spleen - and transports it to the liver. The arteries supplying this blood are the nonhepatic branches of the celiac axis and the superior and inferior mesenteric arteries. There are

frequent variations in the anatomy of the branches of the portal venous system, but the portal vein itself begins rather constantly at the level of the second lumbar vertebra, posterior to the head of the pancreas (Fig. 1), at the junction of the splenic and superior mesenteric veins. It is about 6 to 8 cm long and 1.2 cm in diameter and contains no valves (Fig. 2). At the liver hilum, it separates into a right branch that supplies the right lobe and a left branch that supplies the left, caudate and quadrate lobes. The ligamentum teres joins the left branch of the portal vein and contains within it one or more potential lumens (umbilical or para-umbilical veins) that are remnants of the fetal circulation running from the umbilicus to the left portal vein. The most frequent variations in portal system anatomy are in the inferior mesenteric vein, which may join the superior mesenteric instead of the splenic, and in the left gastric (coronary) vein, which may join the splenic instead of the portal.

Portal venous blood passes through one capillary system in the splanchnic viscera and leads to another

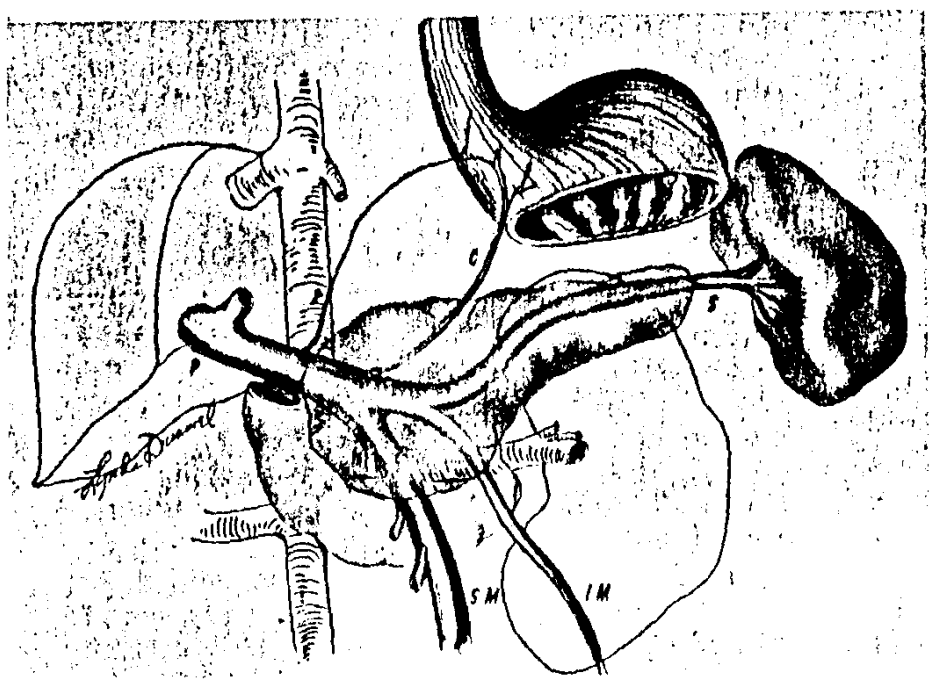

Fig. (1): Portal vein and its major tributaries.
C = coronary (left gastric); IM = inferior mesenteric; P = portal; S = splenic; SM = superior mesenteric.

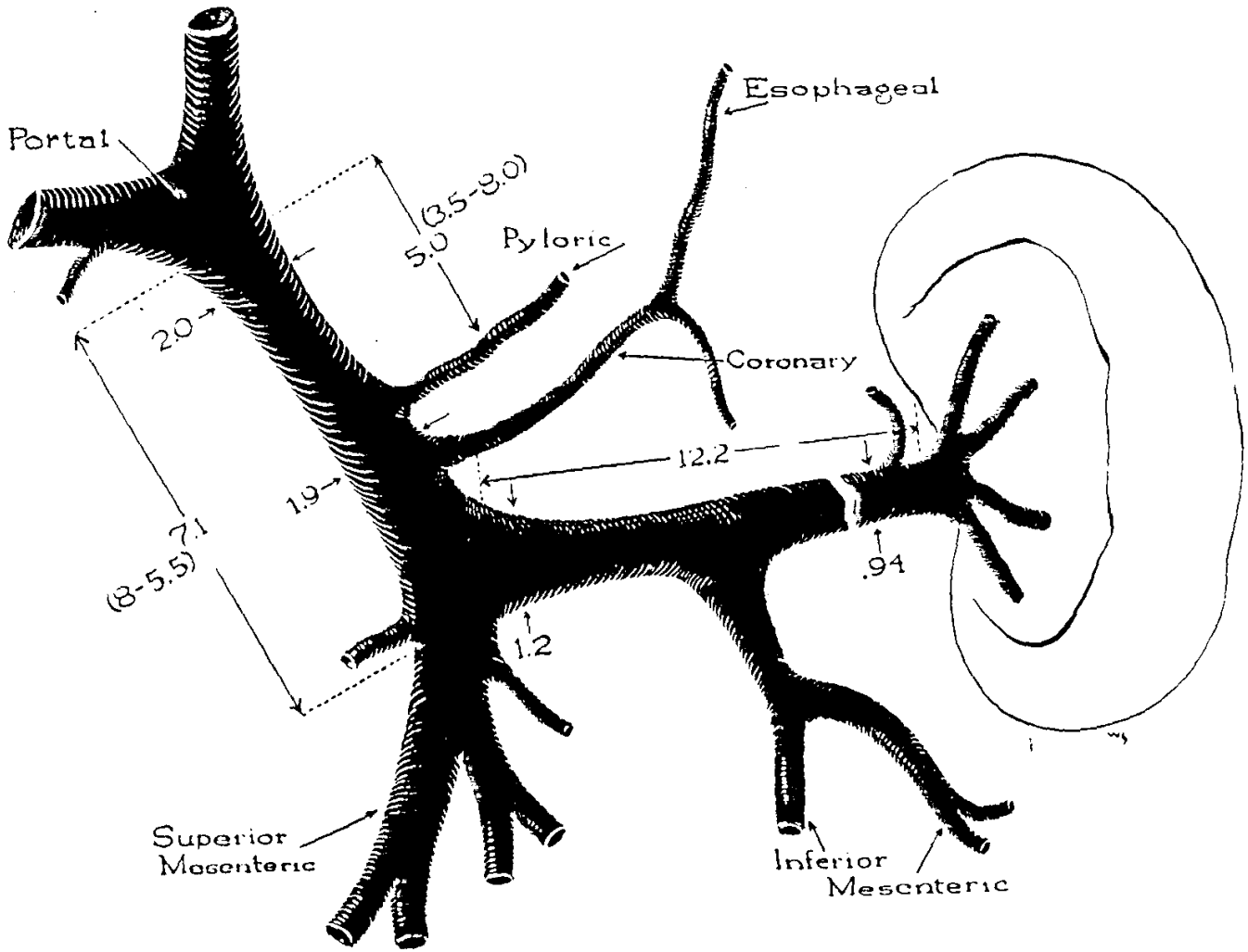

Fig. (2): Mensuration of the portal vein and its main branches. Figures are in centimeters.

capillary system, the hepatic sinusoids. Portal venous blood differs from most other venous blood in being under slightly higher pressure in order to overcome the resistance of the hepatic sinusoids, in being less depleted in oxygen because of the relatively high blood flow through the splanchnic area, and in containing many nutrients and bacterial waste products from the digestive tube that are en route to the liver.

Normal fasting hepatic blood flow approximates 1500 ml/minute. The high-pressure hepatic arterial and low-pressure portal venous streams unite at the level of the hepatic sinusoid. The best estimates available indicate that about two-thirds of the hepatic blood flow and about half of the total oxygen consumption is supplied by the portal vein while the hepatic artery contributes the remainder.

A natural consequence of stasis and increased pressure in any venous bed is the development of connections to neighbouring low pressure veins. The collateral circulation in long-standing portal hypertension is well developed, though the size and location of the major collaterals vary considerably from patient to patient. The stimulus for the development of collateral vessels is portal hypertension [T. Reynolds, 1983a].

The natural sites for the development of portal collateral vessels are areas where veins draining into the portal stream are in juxtaposition to veins draining into the caval system. The major locations for this are (Fig. 3); at the submucosa of the oesophagus where anastomoses from between the tributaries of the coronary vein (portal drainage) and azygos vein (superior vena cava drainage). This results in submucosal varices of the lower oesophagus and upper stomach. Collaterals from the spleen to the stomach contribute to this anastomotic plexus. At the submucosa of the rectum where the lower portion of the rectum normally drains into the inferior vena cava through the haemorrhoidal veins, whereas the upper portion of the rectum drains into the portal system through the middle and superior haemorrhoidal veins. Anastomosis between these venous systems result in haemorrhoids. At the anterior abdominal wall where the umbilical vein remnant of the fetal circulation in the falciform ligament normally carries little or no blood but remains probe-patent. In portal hypertension, it can serve as an anastomosis between the main left portal vein and the normotensive epigastric veins of the anterior abdominal wall that drain ultimately into the superior and inferior vena cava. At the parietal peritoneum