EFFECTS OF BETA-BLOCKERS, VASOPRESSIN AND CALCIUM ANTAGONISTS ON SPLENIC PRESSURE IN CASES OF PORTAL HYPERTENSION.

Thesis

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By

Nabil Anwar Kamel Atalla

M.B.B.Ch, Ain Shams University

Supervised by:

Dr. HASSAN EL-SAYED SOLIMAN
Assistant Prof. of General Medicine,
Faculty of Medicine,
Ain Shams University

Dr. EL-SAID MOSTAFA ABOU-GAMRAH.
Assistant Prof. of General Medicine,
Faculty of Medicine,
Ain Shams University

Dr. MUBARAK MOHAMED HESSEIN
Lecturer of Tropical Medicine,
Faculty of Medicine,
Ain Shams University

Faculty of Medicine,
AIN SHAMS UNIVERSITY
1986

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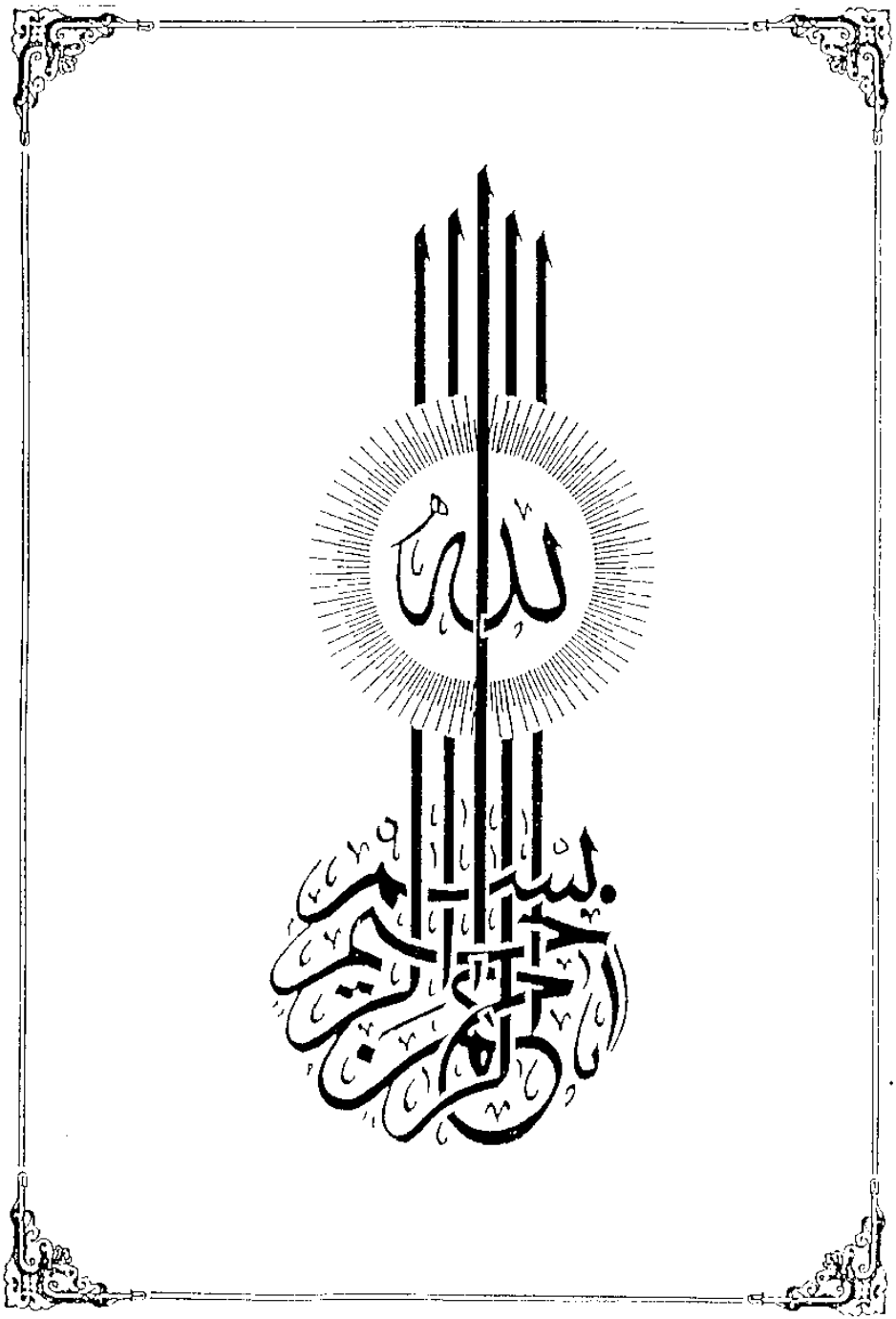

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INTRODUCTION AND AIM OF THE WORK

The use of oral Beta-blockers in cases of portal hypertension is well studied.

No comment was done on the parentral use of it specially in emergency cases.

The aim of this work is to study the effect of I.V. use of Beta-blockers, vasopressin, and calcium antagonists in cases of portal hypertension within the first hour of injection hoping to find a suitable treatment in casesof bleeding portal hypertension.

Review of the literature

ANATOMY OF THE PORTAL CIRCULATORY SYSTEM

The liver has two routes for its blood supply, the hepatic artery and portal vein. The hepatic artery arises from the coeliac trunk. It gives the gastro-duodenal artery and then it becomes the main hepatic artery. It passes below the medial end of the epiploic foramen; then it crosses infront of the portal vein and ascends between the layers of the lesser omentum to the porta hepatis where it divides into right and left branches to the corresponding lobes of liver (Sherlock, 1981).

The Portal vein (Fig. 1); is formed by the junction of the superior mesenteric and splenic veins, their union taking place in front of the vena cava and behind the upper border of the head of pancreas. Passing upwards through the right border of the lesser omentum to the under surface of the liver, it enters the transverse fissure, where it is somewhat enlarged, forming the sinus of the portal vein and divides into two branches which accompany the ramifications of the hepatic artery and hepatic duct throughout the substance of the liver, of these two branches, the right is the larger, but the shorter, of the two (John A. Crocco, 1977).

It is about 6 - 8 cm long and 1.2 cm in diameter and has no valves in its larger channels (Reynolds, 1969).

The portal system is composed of four large veins which collect the venus blood from the viscera of digestion (stomach, intestine, and pancreas) and from the spleen.

The veins forming the portal system are: superior mesenteric vein, splenic vein, inferior mesenteric vein, gastric veins, and cystic vein (John A. Crocco, 1977).

Anastomosis between the portal and systemic venous systems:

(1) Plexus around lower end of oesophagus: (Fig. 2)

Oesophageal branches of left gastric vein (portal) and lower systemic oesophageal veins (systemic).

(2) Around umbilicus:

Paraumbilical veins accompany the round ligament of liver (portal) and superficial veins of the anterior abdominal wall (systemic).

(3) Plexuses around lower 1/3 of rectum and anal canal:

Superior haemorroidal vein (portal) and middle and inferior haemorroidal veins (systemic).

(4) Extraperitoneal surfaces of abdominal organs:

Tributaries of superior and inferior mesenteric veins (portal) and subdiaphragmatic and retroperitoneal veins (systemic) (Harding Rains, 1977).

THE PORTAL VEIN AND ITS TRIBUTARIES

(Marten Seler Kleckner, 1960)

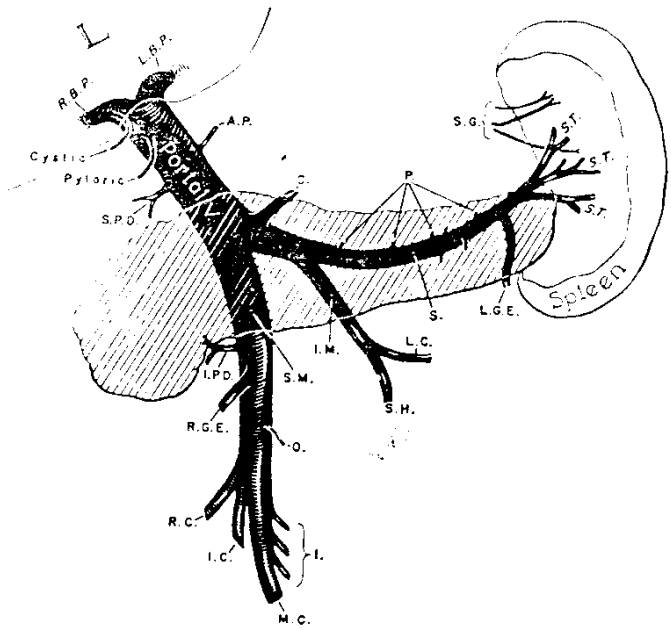

Fig. (1): The extrahepatic portal system of veins, anterior aspect.

A.P.	accessory pancreatic vein;
C.	coronary vein ; cystic - cystic vein;
I.	intestinal veins;
I.C.	ileocolic vein;
I.M.	inferior mesenteric vein;
L.C.	left colic vein;
L.G.E.	left gastroepiploic vein;
M.C.	middle colic vein
O.	omental vein;
P.	pancreatic veins; pyuloric - pyloric vein;
R.B.P.	right branch of portal vein.
R.C.	right colic vein
R.G.E.	right gastroepiploic vein S.- splenic vein;
S.G.	short gastric veins; S.H. - superior haemorrhoidal vein;
S.M.	superior mesenteric vein.
S.P.D.	superior pancreaticoduodenal vein.
S.T.	splenic trunks.

(Marten Seler Kleckner, 1960)

1. Sup. vena cava
2. Azygos
3. Hemiazygos
4. Coronary
5. Gastric
6. Vasa Brevia
7. Varicose periesophageal plexus.

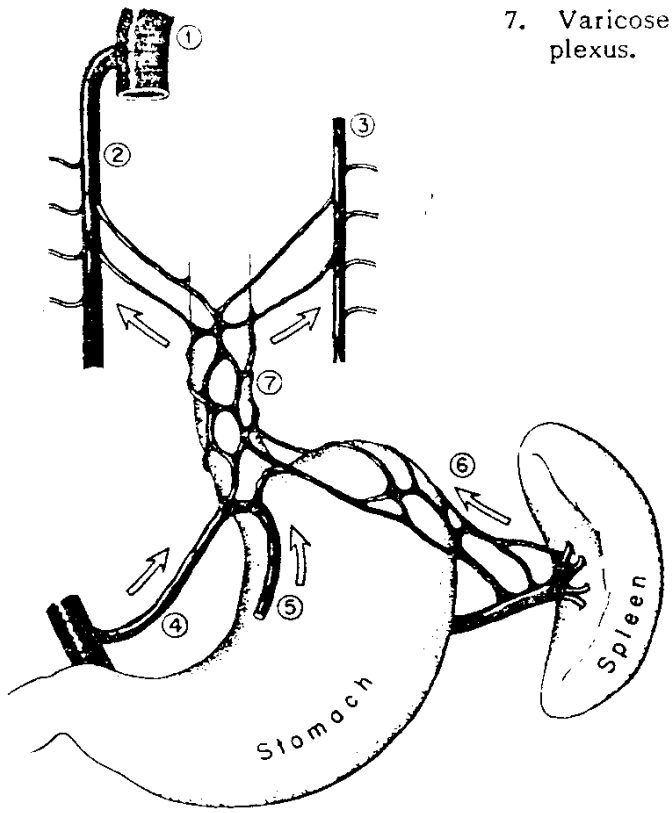

Fig. (2): Portal Hypertension. Collateral portal shunts are established anastomosing the vasa brevia and left gastric veins of the valveless portal veins with the azygos and hemiazygos veins.

PHYSIOLOGY OF THE PORTAL CIRCULATORY SYSTEM

Approximately 75% of the blood flowing through the liver comes from the portal vein. This blood, while too inadequately oxygenated for the liver, is neverthless, rich in nutrient absorbed from the small intestine. The remaining 25% of the blood is derived from the hepatic artery, which furnishes, principally, oxygen and maintains a blood pressure gradient in the hepatic sinusoids, resulting in resistance to flow of blood from the portal vein (Martin Seler Kleckner, 1960).

The portal vein drains blood from the sinusoids of the spleen and from the capillaries of the splanchnic vessels in the pancreas, stomach, and intestine. After penetrating the liver, the portal vein ramifies and divides progressively into smaller and smaller branches, which finally end in the hepatic sinusoids. These sinusoids are a network of intercommunicating endothelial-lined spaces between the one-cell thick cords of the liver cells in the hepatic lobule. The subdivisions of the hepatic artery also empty into the sinusoids. Blood exists from the sinusoids into the central vein of the respective lobule and eventually empties via the hepatic veins into inferior vena cava (Charles I. Wagner, 1978).

Animal studies have shown that the portal vein contains alpha-adrenoceptors only and no beta-receptors and its tone is

partly maintained by adrenoceptor stimulation. Normally the portal vein shows a linear pressure-blood flow relationship with no evidence of autoregulation, such as is seen in the heart or kidney, whereby blood flow is maintained over a range of pressure changes (Mills, 1984).

The intrahepatic portal vein radicles have smooth muscle in their walls that is innervated by adrenergic vasoconstrictor nerve fibers reaching the liver via the third to 11th thoracic ventral roots and the splanchnic nerves. The vasoconstrictor innervation of the hepatic artery comes from the hepatic sympathetic plexus. There are no known vasodilator fibers reaching the liver (Williams F. Ganong, 1977).

PORTAL PRESSURE AND PORTAL HYPERTENSION

The normal portal pressure is 5 - 10 mmHg (Harding Rains, 1977) and 10 - 12 Cm H_2O (Reynolds et al., 1970).

Mousa and El-Garim in 1959 measured the intrasplenic pressure in more than forty patients and stated that the upper normal level was 20 cm. saline and that pressures above this were considered as cases of portal hypertension. An increase in the pressure in the portal system, rising above the normal of 5 - 10 mmHg, occurs when there is increased resistance to the flow of blood in the portal or hepatic veins (Jan A.D. Bouchier, 1982). This increased resistance to flow within the liver leads to opening of extrahepatic collaterals between the portal and systemic circulations, which allows blood to the liver (Charles I Wagner, 1978).

The naturally occurring shunts are ineffective in lowering portal hypertension and preventing oesophageal haemorrhage (Marten Seler Kleckner, 1960).

The main causative factor is an outflow block caused by compression of the hepatic vein tributaries by regenerative nodules (Jan A.D. Bouchier, 1982). The important role of the regenerative nodule in increasing tissue tension, compressing and