A STUDY IN HAEMODYNAMICS OF PORTAL HYPERTENSION

THESIS

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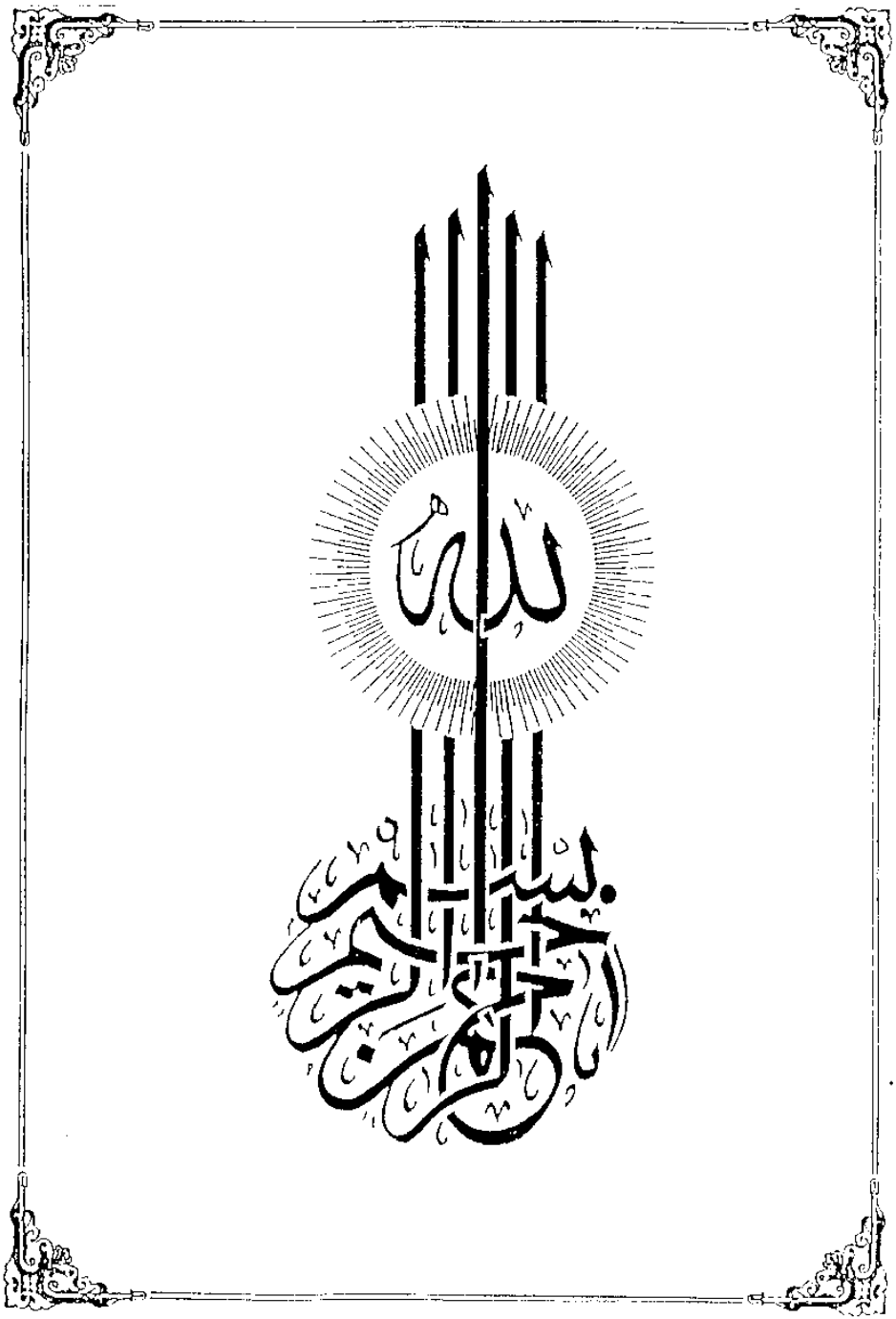

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Portal hypertension with its potentially lethal complication, still constitutes not only a national problem but also a challange to the efficiency of the medical and surgical treatment .

Haemodynamic studies were carried out, as well as blood gases were, estimated the main aim was to detect, reversal of blood flow, in portal vein, as well as the possibility that intrahepatic shunting may increase the state of liver ischaemia in Bilharzial hepato-splenomegaly.

The haemodynamic aspects of the study comprise visualisation of the portal circulation through direct splenoportography, percutneously, combined with splenic pulp manometry, a procedure whose value has repeatedly recognised by many authers. Endoscopic examination of the aesophagus has been made before, splenoportography to diagnose oesophageal varices.

Hepatic angiography have been made to asses the state of liver vascularity , and the size of the hepatic artery and if there is any attenuation or relative diminution in its diameter and size, that may contribute to liver ischaemia.

An attempt has been made to viscualise intrahepatic arterio portal shunting which may be a factor augmenting portal hypertension.

The blood studies were carried out to assess liver function, patients were categorized, according to the status of liver function. The measurement of O_2 tension in portal circulation as well as arterial blood has been made, to evaluate the importance of intrahepatic or intrasplenic arterio-venous shunts.

Wedged hepatic venography and W.H.V.P studied with correlation to trans-splenic pressure.

Ultrasonography were carried out to detect its value in diagnosing portal hypertension and liver affection.

REVIEW OF LITERATURE

ANATOMY

PORTAL SYSTEM OF VEINS

The portal system includes all veins which carry blood from the abdominal part of the digestive tube, (with exception of the lower part of the anal canal) the spleen, pancrease and gall bladder to the liver. From these viscera, the blood is conveyed to the liver via the portal vein entering the liver at the porta hepatis dividing into two main branches one to each lobe. In the liver each vein ramifies into hepatic sinusoid from which blood is conveyed to the inferior vena cava by the hepatic veins. The portal system pecuilar in that it ramifies into two capillary beds between the abdominal viscera and inferior vena cava. In the adults it is devoid of valves. Valves can be easily seen in the newborn infants, however, with increase age these valve become insufficient so that cusps no longner oppose. (Gilfillan 1950).

The Portal Vein :

Developmental anatomy:

There are three basic types of veins in the embryo :

1. Paired umbilical veins in the lateral abdominal wall to the sinus venosus .

2. Paired vitelline veins from the yolk sac to the umbilical veins at the sinus venosus.

3. Paired anterior cardinal veins lateral to the nervous system joining with paired posterior, cardinal veins to form the common cardinal vein or duct of Curvier.

The primitive septum transversum envelops the paired vitelline and umbilical veins as they approach to each other to form with the ducts of curvier at the caudal end of the heart (Sinus Venosus). A tubular ventral evagination from the gut, the hepatic diverticulum, invades the septum transversum.

The vitelline veins are the first to be invaded by the hepatic tissue. The invaded region develops into the hepatic sinusoids and divides vitelline vein into proximal and distal portions. The proximal portions give rise to hepatic veins. Later, three transverse anastomoses take place in the distal portions of the vitelline veins :

1. A cranial anastomosis in the liver ventral to the duodenum .
2. An intermediate anastomosis dorsal to the duodenum .
3. A caudal anastomosis ventral to the duodenum .This gives two venous rings which are soon interrupted :

between the two layers of the lesser omentum to the oesophageal veins. It then turns, backwards and passes downwards and to the right behind the lesser sac of peritoneum and ends in the portal vein at the upper border. of the superior part of the duodenum, sometimes it terminate in the splenic vein. It's length is 5.5 cm. to 6 cm. and it's width is 0.13-0.38 cm. It transmits the high portal pressure to oesophageal veins at the cardio-oesophageal junction (Khairy 1960).

4. The Right Gastric Vein.

It is of small size, it runs from left to right along the pyloric portion of the lesser curvature of the stomach, between the two layers of the lesser omentum and ends in the portal vein. It is joined by prepyloric vein which ascends in front of the pylorus and usually marks the site of the pyloric opening.

5. The para umbilical veins.

These establish an anastomosis between the veins of the abdominal wall and the portal vein. They are found in the ligamentum teres of the liver.

6. Cystic veins

The veins draining the gall bladder vary considerably. These from it's upper surface lie in the areolar tissue between the gall bladder and liver and usually run directly into the liver through the fossa for the gall bladder to join hepatic veins. Those from other parts of the gall bladder join to form one or two cystic veins on the neck of the gall bladder, and these commonly enter the liver either directly or after joining with veins draining the hepatic ducts and the upper part of the bile duct. Only rarely does a single or double cystic vein drain into the right branch of the portal vein (Last,1974).

AETIOPATHOLOGY OF PORTAL HYPERTENSION

Although the liver appears to be relatively solid organ, about a liter of portal venous blood flows through at each minute at a low pressure of (4-6 mmHg) indicative of the liver's low resistance to blood flow. The liter of portal blood joins at the sinusoidal level with about 500 ml of arterial blood that has been reduced to a similarly low pressure by a high resistance in the arteriolar bed. Via a unique arrangement of sinusoid with markedly fenestrated walls, the combined portal and arterial blood streams make extensive contact with the microvilli of the hepatocytes in the space of disse to enable the liver to perform its numerous metabolic taks . (Reynolds, 1983) .

The overwhelming majority of cases of portal hypertension are related to intra hepatic obstruction . The major reason for that is the increased mechanical resistance to blood flow through the liver due to architectural disturbance. The impilicated other pathogenic factors are intrasinusaoidal collagen deposition in perivenular area as in alcoholic liver injury, portal zone fibrosis as in chronic hepatitis and nodular regeneration