INTRODUCTION

Hepatosplenomegaly is considered to be one of the most important clinical problems in Egypt. Liver fibrosis is the primary pathological feature in this condition. Retrograde embolization of Schistosoma mansoni ova leads to diffuse bilharzial granulomatous reaction in the portal branches of the liver followed by fibrosis (Kamel, 1973).

Splenomegaly is a nearly constant feature of hepatic schistosomiasis. The early stages of all schistosomal infections are accompanied by splenic enlargement due to hyperplasia of its reticulo-endothelial tissue (Elwi & Attia, 1972).

With the advance of hepatic fibrosis, and the development of portal hypertension, the spleen enlarges considerably due to chronic venous congestion, congestive splenomegaly (Kissane, 1990).

Sherlock (1981) stated that an enlarged spleen is the most important diagnostic sign of portal hypertension. If the spleen can not be felt or not enlarged radiologically, the diagnosis of portal hypertension is questionable.

Haemodynamics of portal hypertension has been considerably clarified by the development of animal models such as the rat with ligated portal vein or carbon-tetrachloride induced cirrhosis (Sherlock, 1989).

The fundamental haemodynamic abnormality is an increased resistance to portal flow. As, the portal venous pressure is lowered by the development of collateral's deviating portal blood into systemic veins the portal hypertension is mentioned by increasing the blood flow in the portal system which thus becomes hyperdynamic. This increased flow is achieved by rising cardiac output and by splanchnic vasodilatation. These findings have been confirmed in the rat model and in cirrhotic patients by duplex ultrasound (Sato et al., 1987).

Osler was one of the first surgeons to advocate a splenectomy for oesophageal variceal haemorrhage. Pemberton, in 1945, reported a 50% incidence of recurrent variceal haemorrhage with splenectomy alone, and this technique was soon abandoned with the development of porto-caval shunts (Spencer & Ronald, 1986).

Splenectomy and devascularization without oesophageal transection practiced by Hassab, predominantly in patients with schistosomiasis, has been successful in bilharzial patients. The operative mortality and the frequency of rebleeding have both been low in this group of patients (Berk & Bockus, 1985).

The haemodynamic changes that take place after the portosystemic shunt or distal splenorenal shunt procedure are well decumented, but there have been few reports on the changes that occur after a non-shunting operation for the treatment of oesophageal varices (Steegmuller et al., 1984).

The changes in portal haemodynamics including portal venous flow after devascularization and splenectomy, have not been clarified because of the lack of siutable method for measuring human portal blood flow (Takanaka et al., 1990).

The accuracy, rapidity, low cost and non - invasiveness of duplex ultrasonography have been exploited in the pre and postoperative evaluation of some of the sickest and most fragile patients in contemporary medicine - those with various forms of liver disease. This experience include not only patients with cirrhosis and portal hypertension but also those undergoing liver transplantation (Sherlock, 1994).

The objective of the present study is to investigate the changes in the portal vein haemodynamics, as well as, the role of splenic blood flow after splenectomy and devascularization operation. The color doppler ultrasonography is used for this purpose.

DEVELOPMENTAL ANATOMY OF PORTAL CIRCULATION

In prenatal life the right and left vitelline veins form the yolk sac and the right and left umbilical veins, originally from the allantois but later from the placenta opened independently into the common sinus venosus of the heart (BasMajian, 1980).

The liver developed in the septum transversum from the branchings of a diverticulum of the duodenum. As a result, the portions of the vitelline and umbilical veins which lie in the septum are expanded as the liver grows, and at the same time broken up into many anastomosing channels called sinusoids. Strands of liver cells multiply and invade the sinusoidal blood spaces until they are, in the main, reduced to the size of capillaries, and the liver become a comparatively solid organ (Romanes, 1991).

Thereafter, the prehepatic parts of the right umbilical, left vitelline and the left umbilical veins disappeares, leaving only the prehepatic part of the right vitelline vein to conduct blood from the liver to the heart.