The Portal Circulation in Cirrhosis

S.	Sheila	Sherlock
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London

Two techniques have been used in the evaluation of the portal venous circulation in cirrhosis – percutaneous trans-splenic veno-graphy and hepatic vein catheterisation.

Trans-splenic venography shows an increase in the spleno-hepatic circulation time in portal cirrhosis, with anastomoses between the

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portal vein and the oesophageal veins. The distortion of the intra-hepatic vascular tree in cirrhosis, described by McIndoe, is confirmed.

If a radio-opaque catheter is advanced into the hepatic vein until it encounters resistance, it blocks the vein and the pressure recorded through it may be called the intrasinusoidal portal venous pressure. This approximates to the portal venous pressure measured directly. The portal venous pressure measured in this way is inversely proportional to the size of the nodules in the cirrhotic liver measured on histological sections. If the nodules are greater than 4 mm. in diameter, portal hypertension is minimal; if less than 2 mm., pressure is very high. The smaller the nodule the greater the proportion of fibrous tissue and the greater the portal vascular distortion, the vessels being crowded together in the dense connective tissue separating adjacent nodules. Fibrosis with distortion of the portal vascular tree is important in the production of portal hypertension.

Another possible factor in the pathogenesis of portal hypertension is easier transmission of the hepatic arterial pressure to the portal vein in the cirrhotic liver, as suggested by Herrick and later by Dock. This is said to follow the opening of hepatic arterial-portal anastomoses in the cirrhotic liver. If the anastomoses were of any magnitude, the oxygen saturation of the hepatic venous blood should be increased. However, there was no significant difference between the hepatic venous oxygen saturation in the cirrhotic as compared with the non-cirrhotic subject. The hepatic blood flow was but little reduced in the cirrhotic liver. This evidence does not suggest that the hepatic arterial flow is proportionately increased over the portal venous flow, in cirrhosis of the liver.

The major factor in the production of portal hypertension in patients with cirrhosis of the liver is distortion of the portal intrahepatic venous circulation as a result of nodule formation and fibrosis.