Symptoms and Syndromes

14 Portal hypertension

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► Aristoteles (384–322 BC) first described the portal vein within the venous system. Herophilos (ca. 300–250 BC) was the first to recognize the portal vein system and its importance as the discharge zone for all resorbent intestinal veins. A description of the portal vein system with its intrahepatic branches and separate bloodstream was given by Galenus (129–199 AD). The independence of the portal vein circulation from the overall blood circulation was demonstrated by F. Glisson (1597–1677). (s. pp 6, 7, 9)

► In 1905 the **cause** of portal hypertension was regarded by R. Kretz as being the mechanical constriction of the hepatic veins resulting in a shunt between the arterial and venous circulation. • The term **portal hypertension syndrome** was defined by A. Gilbert and M. Villaret (1906) and taken to encompass ascites, opsiuria, splenic tumour, haemorrhoids, gastrointestinal bleeding and the development of hepatofugal collaterals. The underlying disease consisted of cirrhosis and portal vein thrombosis. (43) • **Measurement of portal venous pressure** was taken by L. M. Rousselot in 1936. Pressure measurement by puncturing oesophageal varices was first performed by P. Allison (1951). A. Paton et al. (1953) reported on the indirect determination of portal venous pressure with the aid of hepatic venous pressure measurements. A correlation between pressure in the portal vein and pressure in the splenic vein was established by M. Atkinson and S. Sherlock (1954).

1 Definition

A persistent pressure elevation of >12 mmHg in the portal vein circulation, dilation of the portal vein to >13 mm or an increase in the portal pressure gradient of >7 mmHg (difference between the pressure of the portal vein and that of the inferior vena cava) is termed portal hypertension. At pressure values of more than 20 mmHg, collaterals generally develop. • *Portal hypertension is regarded as a systemic disease which affects a number of organ systems.*

2 Pathogenesis

The portal vein is 5–8 cm long with a diameter of 1.2 ± 0.2 (or 0.97) cm. The portal venous pressure is 3–7 (–12) mmHg. It is dependent on several *criteria*: posture, intra-abdominal pressure (e.g. coughing, compression), respiratory phase, Valsalva's manoeuvre and a number of biochemical mediators. (20)

Hepatic circulation: About 70–75% of the hepatic circulation (1,500 ± 300 ml/min, or 1.4–1.5 ml/min/1.73 m^2 body surface) pass through the portal vein (25–30% via the hepatic artery). Hepatic circulation increases after the ingestion of food, but decreases by about 30% after physical exertion as a result of sympatheticotonia. The oxygen content of portal venous blood is lower than that of arterial blood, but is significantly higher than in the rest of the venous system. The portal vein supplies about 10–12 ml O_2/min × 100 g, i.e. 50–60% of the liver's oxygen requirement. The liver is very adaptable in this respect and balances an increased or decreased oxygen supply by decreasing or increasing oxygen extraction (by almost 100%) from the portal and arterial blood. (4, 10, 17, 87, 99) (s. pp 826, 833, 836)

Hepatic blood flow: In addition to the autoregulation of the arterial hepatic system, the hepatic blood flow is mainly regulated via neural (sympathetic) stimulation and the effects of hormones, mediators or pharmaceuticals. • Agents such as α-agonists, histamine, serotonin, noradrenaline, endothelin and angiotensin have a constrictive effect on the portal venous system. A vasodilatory effect on the arterial system is brought about by β-agonists, nitric oxide (NO) (51, 118), glucagon, prostaglandins, pentagastrin, etc. • Under pathological conditions (e.g. lowered response to vasoconstrictors), individual factors of these biochemical regulation systems may be present to a greater or lesser extent and thereby lead to pathophysiological mechanisms. This also applies to the action of endotoxins with regard to an elevation of the portal pressure. • **Ito cells** are key targets for vasodilators: they regulate the hepatic microcirculation. (118)

Hepatic resistance: Various areas of resistance can regulate the circulation of blood in the liver: (*1.*) arterioles of the hepatic artery, (*2.*) arterioles in the region of the splanchnic vessels, (*3.*) presinusoidal portal venules, (*4.*) sinusoids and postsinusoidal sections of vessels, and (*5.*) portosystemic collaterals.

Forward-flow hypothesis: When hepatic resistance is constant, increased blood supply causes an elevation of the portal pressure. • **Backward-flow hypothesis:** This primarily requires an increase in hepatic vascular resistance. The subsequently reduced circulation of blood in the liver is compensated by increased circulation in the splanchnic vessels.

The portal hypertensive syndrome is caused by (*1.*) increased resistance in the portohepatic circulation and (*2.*) an increase in the splanchnic vein blood supply.

► The *increase in vascular resistance* is the decisive factor and, in the majority of cases, is even the sole cause. It can be *functional and reversible* as well as *structural and irreversible.* Blood flow correlates directly with vessel diameter to the 4^{th} power, i.e. small radial changes cause large changes to vessel resistance. An increase in the blood supply may favour the occurrence of portal hypertension or enhance its clinical development. The persistent interference with biochemical mechanisms which regulate the blood circulation in the liver and the impact of pathological substances may have further pathogenic effects. (23, 62, 72, 81, 118, 128) (s. fig. 14.1)

Despite the development of *portosystemic collaterals*, which ought to lead to a fall in portal hypertension, the *hyperdynamic circulation* accompanied by splanchnic vasodilation (= increased cardiac output, decreased systemic vascular resistance, hypervolaemia, systemic arteriolar vasodilation) maintains portal hypertension in both the splanchnic and systemic vascular systems. (10, 47, 87) The hyperdynamic circulation is either the cause