Clinical Aspects of Liver Diseases

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Due to its twofold vascular supply (portal vein, hepatic artery), the liver is closely integrated into the systemic circulation. The total blood flow through the liver is 1,500 ± 300 ml/min. Oxygen consumption amounts to 6 ml/min/100g LWW. (s. pp 17, 244) The portal vein supplies about two-thirds of the hepatic flow volume and is thus responsible for 50% of the oxygen requirement of the liver. The ratio between portal and arterial perfusion is about 2:1; this may, however, be reversed by compensatory autoregulation in cirrhosis patients. Blood from the portal and arterial systems reaches the sinusoids, where both mixing and pressure compensation occur. The hepatic blood flow is mainly regulated by inflow from the hepatic artery, which can be doubled when more oxygen is required. With continued demand for oxygen, the high capacity of the liver cell to extract oxygen (up to 95%) compensates for a limited blood flow. The hepatic flow volume is about 20–25% of the cardiac output (CO). • In addition, the pressure in the lesser circulation is transferred directly to the pressure in the hepatic vein, so that the latter is almost equal to the pressure in the right atrium. The oxygen saturation of the arterioportal mixed blood in the sinusoids decreases continually with the flowing of the blood from the periportal zone to the central vein. The venous blood is then carried from the central veins and the venous branches into the inferior vena cava, usually through three (valveless) hepatic veins. • This anatomical proximity explains disturbances of the liver function as well as morphological damage resulting from shock situations, acute flow block in the hepatic veins and global heart failure. • **Cardiovascular diseases** may thus affect the heart or each of the three vascular systems:

1. Impaired cardiac function
2. Diseases of the hepatic vein
3. Diseases of the portal vein
4. Diseases of the hepatic artery
5. Hereditary haemorrhagic telangiectasia

1 Cardiocirculatory disorders of the liver

► The functional relationship between the heart and the liver has been known since ancient times. In animal experiments conducted in 1914, C. Bolton examined the effects of a ligation of the inferior vena cava on liver histology for the first time. • N. Jolliffe (1929) detected clinically measurable changes of the hepatic function in congestive heart failure. (10) • Systematic investigations carried out by S. Sherlock (1951) showed the consequences of passive liver congestion on hepatic morphology: complete cirrhosis was observed in 25.5% and an increase in reticular fibres in 27.5% of cases; the liver was without pathological findings in 47% of cases. • Autopsies carried out by P. Kotin (1951) showed "cardiac cirrhosis" in 10% of patients suffering from right heart insufficiency. (13) • Subsequently, the effects of cardiocirculatory disturbances on various partial functions and on the morphology of the liver, including the effects of drug metabolism, were presented in numerous publications.

Pathophysiology

► The close functional relationship between the heart and the liver is based upon **two physiologic facts:**

(*1.*) The *hepatic flow volume* is 20–25% of the CO and is thus directly dependent on the cardiac ejection volume.

(*2.*) The *pressure* in the lesser circulation is transferred directly to the valveless hepatic veins and may thus also reach the central veins.

► Even though the pathophysiology of hepatic changes during circulatory shock and in chronic liver congestion is very complex, **two single mechanisms** may be held jointly responsible for the impairment of metabolic and excretory liver functions:

(*1.*) Reduction in blood flow through the liver with hypoxia.

(*2.*) Increase in hepatic vein pressure with centrilobular hyperaemia.

► Circulatory disturbances within the hepatic lobules are of central pathophysiological importance; **three different forms** can be differentiated:

(*1.*) *Disturbed outflow:* the microscopic correlate is centrilobular hyperaemia; macroscopically, the liver is enlarged and has a dark red colour.

(*2.*) *Disturbed inflow:* hypoperfusion due to arterial, portovenous or combined oligaemia results in centrilobular necrosis or even anaemic liver infarction.

(*3.*) *Disturbed flow:* primarily, this occurs intralobularly as a result of various disorders (e.g. DIC, intrasinusoidal fibrin precipitation, centrilobular increase in fibres).

The liver may be involved in numerous ways due to acute, short-term disturbances of the cardiocirculatory function or due to chronic, long-term heart failure:

Acute heart insufficiency	Chronic heart insufficiency
1. Shock liver 2. Acute liver congestion	1. Chronic liver congestion 2. Congestive fibrosis 3. Cardiac cirrhosis