164 ARTERIAL HYPERTENSION IN CIRRHOSIS. ARTERIAL COMPLIANCE, VOLUME DISTRIBUTION, AND CENTRAL HAEMODYNAMICS

J.H. Henriksen[1], S. Fuglsang[1], F. Bendtsen[2], S. Møller[1]. [1]*Department of Clinical Physiology & Nuclear Medicine, Hvidovre Hospital, Hvidovre, Denmark;* [2]*Department of Gastroenterology 439, Hvidovre Hospital, Hvidovre, Denmark*

Background and Aim: Arterial hypertension is a common disorder. Thus a prevalence of 10–15% hypertension could be expected in patients with cirrhosis. Vasodilatation, hyperkinetic circulation, and reduced effective volaemia are central elements in the haemodynamic dysfunction in cirrhosis. The aim of the present study was to investigate whether cirrhotic patients with arterial hypertension are normokinetic and normovolaemic or whether they reveal the same circulatory dysfunction as their normotensive counterparts.
Material and Methods: Thirty-three patients with arterial hypertension (5%) were identified among 648 patients with cirrhosis: 14 in Child class A, 12 in class B, and 7 in class C. The controls were 130 normotensive cirrhotic patients. Both groups underwent a haemodynamic investigation with determination of cardiac output (CO), mean arterial pressure (MAP), plasma volume (PV), central blood volume (CBV), hepatic venous pressure gradient (HVPG), hepatic blood flow (HBF), arterial compliance (AC), and systemic vascular resistance (SVR) in the supine position.
Results: Liver function, as evaluated on the galactose elimination capacity, indocyanine green clearance, HBF, and Child score, was significantly better in the hypertensive cirrhotics than in their normotensive counterparts ($p < 0.05$–0.01), but portal pressure was similar (HVPG 13 vs 15 mmHg, ns). MAP was 33 mmHg higher in the arterial hypertensive group (115 vs 82, $p < 0.001$) and inversely correlated to AC in both groups ($r = -0.43$ and -0.61, $p < 0.02$). AC was significantly lower and normal in the arterial hypertensive group (1.07 vs 1.39 mmHg/ml $p < 0.02$), and SVR significantly higher and normal (1475 vs 1020 dyn.sec/cm5, $p < 0.01$). The arterial hypertensive cirrhotic patients were hyperdynamic (CO = 6.80 vs 7.14 l/min, ns) and central hypovolaemic (CBV = 19.8 vs 20.6 ml/kg, ns) as were the normotensive patients, but different relations were found to the arterial blood pressure. Whereas arterial pressure was inversely correlated to CO, PV, and Child score in the normotensive group ($p < 0.01$), the same relations were either direct or insignificant in the arterial hypertensive cirrhotics.
Conclusion: Arterial hypertension is uncommon in patients with cirrhosis. Arterial hypertensive cirrhotic patients are hyperkinetic and central hypovolaemic like their normotensive counterparts, but vasodilatation is reduced and the regulation of arterial blood pressure may be less deranged.

165 PROLONGED QTC-INTERVAL IN PRE-PORTAL HYPERTENSIVE CIRRHOSIS

H. Ytting[1], J.H. Henriksen[2], S. Fuglsang[2], F. Bendtsen[1], S. Møller[2]. [1]*Department of Gastroenterology 239/Hvidovre Hospital, University of Copenhagen, Copenhagen, Denmark;* [2]*Department of Clinical Physiology/Nuclear Medicine 239/Hvidovre Hospital, University of Copenhagen, Copenhagen, Denmark*

Background and Aims: The QTc-interval is prolonged in a substantial fraction of patients with cirrhosis, indicating delayed repolarisation of the myocardium. However, no information is available on the pre-portal hypertensive stage. The aim of the present study was therefore to determine the QTc-interval in cirrhotic patients without significant portal hypertension.
Material and Methods: Fourty-six patients with cirrhosis and a hepatic venous pressure gradient (HVPG) below 10 mmHg (mean 6.6 mmHg) underwent a haemodynamic study. They were compared to 38 cirrhotic patients with substantial portal hypertension (mean HVPG: 18 mmHg) and controls without liver disease (mean HVPG: 3 mmHg).
Results: The fraction with prolonged QTc-interval (>0.440 s) was similar in the two cirrhotic groups (49% vs 50%, ns) and significantly above that of the controls (5%, $p < 0.005$). Likewise, mean QTc was 0.447 and 0.449 s in the two cirrhotic groups (ns), values which are significantly above that of the controls (0.410 s, $p < 0.01$). In a subset of patients with a hepatic venous pressure gradient below or equal to 5.0 mmHg ($n = 14$), the QTc was mean 0.420 ± 0.007 s, a value not significantly higher than that of the controls, and this group had well-preserved liver function. In the pre-portal hypertensive cirrhotic group, the QTc-interval was inversely related to indicators of liver function like the indocyanine green clearance ($r = -0.34$, $p < 0.02$), but no relations of the QTc-interval to systemic haemodynamics were present. Frequency correction of the QT-interval by several different principles (Bazett, Ashman, Fridericia) gave essentially the same results.
Conclusion: Delayed repolarisation of the myocardium is present in a substantial fraction of patients with cirrhosis without significant portal hypertension. In these pre-portal hypertensive cirrhotic patients the prolonged QTc-interval is related to the decreased liver function, rather than to haemodynamic dysfunction.

166 DETECTION OF EARLY CENTRAL CIRCULATORY TRANSITS IN PATIENTS WITH CIRRHOSIS BY GAMMA VARIATE FIT OF INDICATOR DILUTION PROFILES

J.H. Henriksen[1], S. Møller[1], S. Fuglsang[1], F. Bendtsen[2]. [1]*Department of Clinical Physiology & Nuclear Medicine 239, Hvidovre Hospital, University of Copenhagen, Copenhagen, Denmark;* [2]*Department of Gastroenterology 439, Hvidovre Hospital, University of Copenhagen, Copenhagen, Denmark*

Background and Aims: Patients with cirrhosis have a hyperdynamic circulation with abnormally distributed blood volume and widespread arteriovenous communications. The aim of the present study was to detect possible very early (i.e. below 4 s) and early (i.e. above 4 s) central circulatory transits and their potential influence on determination of the central blood volume.
Material: Thirty-six patients with cirrhosis and 19 control subjects without liver disease undergoing haemodynamic catheterisation were given central bolus injections of albumin with different labels. Exponential and gamma variate fits were applied to the indicator dilution curves, and relations between flow, circulation times, and volumes were established according to kinetic principles.
Results: No significant very early central circulatory transits were identified. In contrast, early (i.e. 4 s to maximal) transits corresponding to a mean of 5.1% (vs 0.8% in controls, $p < 0.005$) of cardiac output (equivalent to 0.36 vs 0.05 l/min, $p < 0.01$) were found in patients with cirrhosis. These early transits averaged 7.7 s vs 12.7 and 17.2 s of ordinary central transits of cirrhotic patients and controls, respectively ($p < 0.001$). The early transits were directly correlated to the alveolar-arterial oxygen difference in the cirrhotic patients ($r = 0.46$, $p < 0.01$), but not in the controls ($r = 0.04$, ns). There was a good agreement between the central blood volume determined by the conventional indicator dilution method and that determined by separation of early and ordinary transits by the gamma variate fit method (1.51 vs 1.53 l, ns).
Conclusion: No very early central circulatory transits were identified in patients with cirrhosis. A significant part of the cardiac output undergoes an early transit, probably through pulmonary shunts or areas with high ventilation–perfusion ratios in patients with cirrhosis. Composite determination of central blood volume by the gamma variate fit method is in close agreement with established kinetic method. The study brings further evidence of an abnormal central circulation in cirrhosis.