erythrocytes and of ^{125}I -labelled albumin in the hepatic artery and portal vein of the normal dog's liver. The sinusoidal transit time is longer in the hepatic artery than in the portal vein and the reverse holds for the non-sinusoidal time. The difference suggests that arterial and portal venous blood flows are largely independent of each other within the liver. The experiments also indicate that circulation is slower in sinusoids perfused by the hepatic artery rather than in those perfused by the portal vein. The hepatic artery transit time is very long in patients with hepatic cirrhosis.

DRS SOVAK and KARDEL (Copenhagen) demonstrated an elegant new biplane cinesangiography technique for visualizing the portal venous tree and collaterals and for measuring portal flow in dogs, using a new particulate non-embolizing medium (Mygind, 1966) and small droplets of Lipiodol ultra-fluid. The flow was calculated from the mean velocity of the particles and the mean cross-sectional area of the portal vein. DR W. A. FUCHS and his co-workers (Bern) defined hepatic arterial steal as a redistribution of perfusion in cirrhotic patients in favour of arterial inflow. There is retrograde filling of the pancreatic arcade, gastroduodenal artery, and inter-hepatic arteries. This compensatory mechanism increases hepatic blood flow when coeliac axis inflow is reduced and as such it benefits the liver, but it may do so at the expense of the circulation to the pancreas and small intestine. Hepatic artery steal is noted in those cirrhotics with splenomegaly, raised wedged hepatic vein pressure, and prolonged bromsulphthalein retention.

DRS N. GITLIN and co-workers (London) have studied the splenic blood flow and resistance in patients with well-compensated cirrhosis before and after portacaval anastomosis. This operation led to decreased size of the spleen, decreased splenic resistance, and increased splenic blood flow. A somewhat similar change was noted by DR B. PORRO and colleagues (Milan) in cerebral blood flow after portacaval shunt operations; there was a significantly increased cerebral blood flow and decreased vascular resistance.

DRS L. M. BLENDIS and co-workers (London) have found that in patients with normal or elevated red cell mass there is also an increase in plasma volume resulting in a total blood volume greatly in excess of the predicted value. There was a close correlation between plasma volume and spleen size in patients with blood dyscrasias but not in cirrhotics. Medical treatment or splenectomy in the blood dyscrasia group resulted in little fall in the plasma volume though in the following nine months there was a slow return to normal.

HORMONES

DRS GERDES and STROHMEYER (Marburg) have demonstrated a qualitative and quantitative change in metabolic transformation of cortisol in hepatic cirrhosis, supporting the concept that progressive deterioration in hepatic degradation of cortisol upsets the feedback mechanism of hepatic regulation of pituitary ACTH secretion leading to pituitary suppression, reduced adrenocortical cortisol production, and a diminished diurnal variation of free cortisol and its metabolites.

Comment

Moore (*Gastroenterology*, 1968, **54**, 501-507) showed that if a glass electrode was calibrated only at pH 7 measurements in the range pH 1 to 3 were likely to show large errors. Was this factor taken into account in the study by Lennard-Jones and his colleagues (*Gut*, 1968, **9**, 177-182) on the effect of foods on acidity of gastric contents? If not, how might such errors affect the results?

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As described in the original paper (*Gut*, **6**, 113-177) the pH meter was calibrated at pH 4.0 as well as at pH 7.0. Moore's experiments demonstrate the validity of calculating hydrogen ion concentrations from measured pH values in hydrochloric acid solutions and gastric juice of known ionic strength obtained from fasting subjects. In gastric juice containing food the situation is more complex, as the effect of protein on the liquid junction potential is not known and the ionic strength varies. For these reasons no attempt has been made to derive hydrogen ion concentrations from the pH measurements made on gastric contents after food. The absolute accuracy of the results may have been affected by the fact that the pH meter was not calibrated in the range pH 1 to pH 3. Moore has shown that calibration with commercial buffers produces a constant bias and in these experiments on the effect of food on the acidity of the gastric contents any such bias is likely to have affected results with different diets in the same way.

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European Association for the Study of the Liver—*concluded*

After parenteral administration of testosterone to the male cirrhotic, DR P. GREGERSEN (Copenhagen) noted a reduced recovery of urinary 17-ketosteroids, suggesting abnormal androgen metabolism rather than primary testicular insufficiency as a cause of feminization. DRS DELLA CASA and colleagues (Modena) have investigated oestrogen metabolism in cirrhotics. The basal excretion of oestrogen was significantly higher in cirrhotics than in healthy subjects, but there were no significant differences between cirrhotics with or without feminization. ACTH stimulation and dexamethasone suppression did not influence appreciably the excretion of oestrogen in normal subjects but resulted in significant elevation of urinary oestrogen excretion in cirrhotics with signs of hyperoestrogenism. Cirrhotics without feminization behaved intermedially between normals and feminized cirrhotics.

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