Abstracts From Other Journals


Infusion of ascitic fluid into systemic circulation (peritoneovenous shunt) can cause disseminated intravascular coagulation (DIC). Nine patients with cirrhosis and refractory ascites studied developed laboratory evidence of DIC. The total collagen infused, as measured by ascitic hydroxyproline concentration, correlated significantly with prolongation of the partial thromboplastin time with kaolin (p<0.005) and the decrease in platelet count (p<0.05). The changes in the coagulation profile were reversible on stopping the infusion, returning to baseline levels within 12 h. Four patients studied 48 h after beginning antiplatelet therapy with aspirin and dipyridamole did not show any changes in coagulation profile. It was concluded that the DIC that complicates ascites infusion into the systemic circulation is largely related to ascitic fluid collagen and can be prevented by aspirin and dipyridamole.


Seven hundred and seventy biopsy specimens from 10 different gastric sites each in 77 patients the stomach were studied for the presence of *H pylori* and active chronic gastritis (ACG). Forty-eight patients with ACG were *H pylori* positive; 20 patients with chronic gastritis (without activity) and 9 patients with normal mucosa were negative for *H pylori*. A strong positive correlation between *H pylori* colonization (Warthin-Starry stain) and ACG was noted; the frequency of *H pylori* colonization was similar in the antrum and body. The average grade of gastritis was distributed evenly throughout the antrum. However, in the body mucosa, the incidence of ACG declined significantly proximal to the borderline between the antrum and body (pylorocardial progression of type B gastritis). The presence and progress of chronic gastritis in the antrum and body differ, despite *H pylori* colonization of both regions.


Portal hypertensive gastropathy (PG) was studied in 107 patients (cirrhosis 35; non-cirrhotic portal fibrosis (NCPF) 24; extrahepatic portal vein obstruction (EHPVO) 46; Budd-Chiari syndrome 2). Before sclerotherapy, although intravariceal pressure was similar, 4 cirrhotics but none with NCPF or EHPVO had PG. After sclerotherapy, 21 (20.3%) additional patients developed PG during a follow up of 23.2±3.4 months. The incidence of PG was higher in cirrhotic patients (37.1%) than in NCPF (16.7%) or EHPVO (8.7%) patients. Development of PG correlated with the severity of liver disease, being more common in Child's C than in Child's A (13%) patients. PG was seen more often in patients with gastroesophageal varices than in patients with esophageal varices alone.


Erythromycin mimics the effects of motilin on gastrointestinal motility probably by acting as a motilin agonist, and is recommended to improve delayed gastric emptying of patients with diabetic gastroparesis. The effect of 200 mg erythromycin on postprandial motility of the stomach and upper small intestine was studied in 13 normal subjects. Erythromycin significantly increased the amplitude of antral contractions during the 2 h postprandial study period. The total number of contractions was not affected but the contractions could be recorded manometrically higher than after placebo (9±12 or 5-6 cm above the pylorus). Antroduodenal coordination was significantly improved during the first postprandial hour.


29 patients with epidemic acute viral hepatitis and 9 with sporadic acute disease were investigated to determine whether the aetiologic agent for the two types of non-A, non-B hepatitis the same or not.

25 (86%) of 29 patients with epidemic and 5 (56%) of 9 with sporadic hepatitis were diagnosed by exclusion as having enterically transmitted NANB hepatitis. Virus like particles (VLP) of 30-34 nm were detected in the stool of one patient each with epidemic and sporadic hepatitis. The VLPs cross reacted serologically and a specific IgM response was seen in acute epidemic and sporadic serum samples. After inoculation with infected stool, tenus monkeys had a mild rise in liver enzymes, and bile samples contained VLPs. It was concluded that the aetiologic agent in epidemic and sporadic NANB hepatitis is the same.