almost no domestic facilities could be of some importance since serological markers were low in children under 1 year of age, and rose sharply in the 1–4 and 5–14 year age-groups. Nevertheless, it is also possible that some children were perinatally infected and that their mothers subsequently became negative for HBeAg since the prevalence of HBeAg decliness with age. The low prevalence of HBeAg may reflect a favorable prognosis for chronic liver disease. However, the absence of this marker may not mean absence of HBV replication but merely reflects a lower level of viral replication (4).

We conclude that all seronegative Kurdish immigrants, especially children, and all newborns should be vaccinated against HBV.

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HEPAT 01135

Synthesis and clearance of atrial natriuretic factor in cirrhosis

The physiology and pathophysiology of atrial natriuretic factor (ANF) clearance has become the subject of increasing interest (1,2). In their recent study Moreau et al. (3) found a negative correlation between liver function as indicated by serum bilirubin concentrations and splanchnic ANF extraction. Since two of their patients with severe liver failure had renal, hepatic or forearm venous concentrations that were higher than the arterial ANF concentrations, the authors conclude that liver failure may limit ANF clearance through the induction of ANF synthesis or other mechanism and lead to increased ANF release. However, the data presented in this paper show that there is no correlation between peripheral arterial or venous ANF plasma levels and the severity of liver disease as indicated by the Child-Pugh score. Furthermore, patients with an ICG clearance of $<10\%$ and presumably a very poor splanchnic clearance exhibit both low renal and pulmonary ANF clearances and low renal, pulmonary and forearm ANF extraction ratios. Remarkably, three of these patients also exhibit both very low peripheral venous and arterial ANF concentrations. Therefore the data of Moreau and colleagues seem to indicate that some patients with severe cirrhosis exhibit a decrease in ANF synthesis which is possibly even more marked than the decreased ANF clearance rates which result in low peripheral venous concentrations. This is an interesting finding and might support our hypothesis of decreased ANF synthesis in severe liver disease: a hypothesis based on the observation that increases in plasma ANF following volume stimulation is blunted in patients with decompensated cirrhosis (4). It seems an interesting task to reconcile this aspect of the Moreau et al. findings with the observation made by Gines et al. (5), of increased cardiac release of ANF in cirrhotic patients.

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HEPAT 01141

Frequent sporadic hepatitis E in West Africa evidenced by characterization of a virus-associated antigen in the stool

Hepatitis E is the enterically transmitted form of non-A, non-B hepatitis in developing countries. During point-source epidemics, the clinical infection rate does not exceed 10% of people living in the area. There is a tendency for young adults to be infected and the rate seems to be higher in people from higher socioeconomic groups (1). These observations suggest that the infectious agent, hepatitis enteric virus (HEV), circulates in the population between epidemics and that the infection, presumably acquired early in life, may be associated with long-lasting immunity. Under these conditions, subjects affected by epidemics may represent a population which has not yet acquired immunity to HEV. In this case, sporadic cases of acute hepatitis E should occur between epidemics and also in developing countries where epidemics are unknown.

No epidemics of hepatitis E have yet been observed in Dakar where effective epidemiological control of hepatitis infections has been performed since 1978, in association with a vaccination campaign against hepatitis B virus (HBV) (2). For these reasons, the Dakar area seems to be an appropriate location for the investigation of sporadic cases of HEV infection, as a potential starting point of possible epidemics in exposed countries.

Thirty patients hospitalized in Dakar were selected. All had a diagnosis of acute infectious hepatitis with jaundice and elevated serum aminotransferase levels. All patients recovered within 6 months after the onset of symptoms. Classical IgM antibody tests were used for viral acute hepatitis diagnosis. The HEV-associated antigen was detected in the stools by an antigen capture ELISA technique recently developed by us, with IgM from an HEV-infected monkey on the solid phase and β-galactosidase-labelled IgG from the same source for antigen detection (3). A Fab-binding glycoprotein, termed 'protein Fv', discharged from the injured liver in stools and shown to induce false-positive reactions was systematically absorbed with insoluble monoclonal IgM (4). Serological markers of acute HAV, HBV, CMV and EBV infections could be detected in 4, 5, 1 and 1 case respectively. Four patients with acute hepatitis had HBsAg in their serum and lacked anti-HBc IgM and anti-delta antibodies, and 3 patients had an anti-HCV antibody. Thus, by a process of elimination, 12 patients seemed to be suffering from hepatitis E.

HEV-AAg was found in 5 of 12 stools of patients without any infectious hepatitis markers and in 1 of 4 patients with anti-HAV IgM. When the sera of patients with acute hepatitis of other etiologies were tested by blocking assay of the ELISA used for HEV-associated antigen detection, only serum from patients with hepatitis E caused significant inhibition, thus confirming the specificity of the test.

HEV appears to be implicated in at least 20% of acute infected hepatitis patients hospitalized in Dakar. Since the same antigen was previously found in an African epidemic (Ivory Coast) (5), the same agent would seem to be responsible for both sporadic and epidemic forms of hepatitis E in West Africa.

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