almost no domestic facilities could be of some impor-
tance 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 declines with age. The
low prevalence of HBeAg may reflect a favorable progno-
sis 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 immi-
giants, especially children, and all newborns should be
vaccinated against HBV.

M. Kulstrunk*, D. Evequozb, U.C. Dubachc, W.
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Synthesis and clearance of atrial natriuretic factor in cirrhosis

The physiology and pathophysiology of atrial natri-
uretic 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 pre-
sented 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 splanch-
ic extraction 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.

Alexander L. Gerbes
Medizinische Klinik III, University of Munich, Klinikum Grosshadern,
8000 Munich 70, Germany

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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.

J. Pillot*, Y. Lazizi*, Y. Diallo* and B. Leguenno*

*Unité d'Immunologie Microbiennne, Institut Pasteur de Paris, 28 rue du Dr Roux, 75724 Paris, France and *Laboratoire de Virologie, Institut Pasteur de Dakar, Dakar