harmony presaged the decline from favour of the notion of allogeneic inhibition. Lymphocyte trapping, the subject of a communication by Frost and Lance on page 101 of this issue of *Nature*, is similarly a phenomenon widely thought about but of which the biological significance is unclear.

In earlier papers Lance and his colleagues have described how, when mice are injected with ^{51}Cr-labelled syngeneic lymphocytes during an immune response, the label appears preferentially in the responding lymphoid organ. Other immunologists have shown a small drop in lymphocyte output from lymphoid organs in the early stages following antigenic stimulus, and it seemed to Lance and his colleagues reasonable to suppose that during an immune response there were alterations in the flow of lymphocytes through the responding organ such that more cells were, in the Lance terminology, trapped. It has been found that although trapping can occur during an immune response, the mechanism is more or less independent of the immunological status of the organism in which trapping is demonstrated.

In their new paper Frost and Lance describe how trapping is much reduced in both the spleen and lymph nodes during a response to antigen in mice which also carry certain transplanted ascites tumours. Ascitic fluid injected into non-tumour bearing recipients had a similar depressing effect on the trapping system. As it was shown in other experiments that the same tumours growing in a solid condition failed to alter the capacity to trap—which indeed was augmented in lymph nodes draining the site of implantation of the tumour—the authors could not argue that malignancy *per se* was having an immunosuppressive effect. Nevertheless, they postulate that part of the immunosuppression sometimes found in advanced cases of malignancy could be due to failure of trapping.

It will be of considerable interest to find out whether the mice with ascitic tumours had similar factors in their serum to those found in shock plasma which can inhibit some of the activities of the reticuloendothelial system. It could be that trapping is a manifestation of stimulation of the macrophages and that in various 'stress' situations trapping is reduced because of partial failure of excitation of macrophages.

From a Correspondent

EPIDEMIOLOGY

Sexual Transmission of Hepatitis B

from our Medical Virology Correspondent

THE epidemiological concepts of hepatitis B infection have recently undergone a significant change. The demonstration that the hepatitis B agent is infective by mouth and the finding that the infection is endemic in closed institutions, the prevalence of infection in adults in urban communities, the carrier rate and age distribution of hepatitis B antigen in different geographical regions and the relatively high incidence in poor socio-economic environments, have altered the epidemiological dogma that hepatitis B is spread exclusively by blood and blood products through the direct parenteral or percutaneous route. Some evidence is now also available for the transmission of hepatitis B by intimate personal contact and possibly by the venereal route.

In 1971, Hersh and colleagues (*New Engl. J. Med.*, **285**, 1363) presented the case history of six patients with hepatitis B infection. Although the precise mode of transmission from a man to his close female companion or companions was not clearly defined, a non-percutaneous route was strongly implicated. The first patient developed hepatitis 3 months after blood transfusion, hepatitis B antigen persisted in his blood and 3 months later his wife developed antigen-positive infection. The second patient was a drug addict who contracted acute hepatitis B; his wife, who was not an addict, developed hepatitis without jaundice 4 months later and the antigen was detected transiently in her serum. The third patient suffered from hepatitis and his girl friend developed a similar illness 3 months later. The fourth patient was a technician working in a renal dialysis unit and hepatitis B antigen was detected in his serum during routine examination. Three of his intimate female contacts developed acute hepatitis B infection. Another patient developed acute hepatitis B; her husband had chronic hepatitis and the antigen was repeatedly found in his serum. The sixth patient in this series was a drug addict, who died of acute fulminant hepatitis. His girl friend, who did not take drugs, suffered from acute hepatitis 2 months later.

CEA-like Substance on Erythrocyte Membranes

IN 1965 Gold and Freedman described an antigen which was thought to be specific for human tumours and foetal tissues of endodermal origin. Called carcinoembryonic antigen (CEA), it was a finding of potentially great importance. But it was not until about four years later, when the first CEA radioimmunoassays were reported, that the CEA story really began to interest the scientific community. Such methods suddenly gave rise to the possibility of being able to perform early screenings for the deadly colonic carcinomas, at a time when the disease would not yet be clinically apparent. Sadly, more recent experience has shown that this hope was over-optimistic; for colonic CEA, or substances cross-reactive with it, has been detected by various methods to occur in the urine, plasma, or faeces of patients with both neoplastic and non-neoplastic (usually inflammatory or regenerative) disorders, and even, albeit in much smaller amounts, in apparently healthy subjects.

It is conceivable that the occurrence of CEA (a glycoprotein molecule), in non-neoplastic conditions, could be, in part, due to the release of glycoprotein moieties from the various types of cells which have cross-reacting antigenic specificities with known colonic carcinoma CEA. This possibility is strengthened by results of Nery, Bullman and Barsoum described in *Nature New Biology* next Wednesday (November 14).

Noting that the levels of CEA in the plasma of fresh blood specimens may increase on storage of the whole specimen, Nery *et al.* set about to see if CEA-like cellular components are gradually released into the plasma by red cells. Using isolated red cell membranes they indeed detected CEA by radioimmunoassay; furthermore, red cells from cancerous and non-cancerous patients as well as healthy control donors were positive and all gave similar mean values. Further analysis of the CEA-like substances demonstrated that they have glycoprotein and glycolipid structures, partially cross-react immunologically (as defined by double diffusion in agarose) with known colonic carcinoma CEA, and have molecular weight characteristics similar to colonic carcinoma CEA. The CEA activity was assessed to be probably more than a single structural entity.

It would seem, as Nery *et al.* cogently point out, that what is known as 'immunologically defined CEA' may in fact represent a family of antigenically related molecules, possessing subtle structural differences, perhaps resulting from impaired cellular metabolism. For those who are concerned with assay of CEA, or indeed of any purified tissue antigen, the implications are self-evident.