primary course of three injections, and samples of blood were obtained from 17 both before and after the primary course. Since the agglutinin response in most children was poor, a booster dose was offered and a final sample of blood was obtained in 12 cases. From Table II it can be seen that after the primary course most of the children did not have detectable antibody to all of the three pertussis agglutinogens, and there was no clear distinction between the three batches of vaccine. After a booster dose, however, most of the children had responded to antigens 1 and 2, but only vaccine X had stimulated a response to antigen 3. Corresponding differences between the three vaccines were found in vaccinated rabbits (Table III). Agglutinins 1 and 2 were produced in all three groups of rabbits; but agglutinin 3 was produced in high titre by vaccine X, in lower titre by vaccine Y, and not at all by vaccine Z.

Discussion

Though the numbers of children and animals in this investigation are small the results give a clear indication of the lines along which further study may be profitable. Perhaps the most striking result was the poor agglutinin response in children who were given three doses (at monthly intervals) of pertussis vaccine made in 1967.

If these agglutinins are important in immunity (Preston, 1966) it would be desirable to determine the agglutinin response to current vaccines by an investigation similar to the one reported here. It would also be possible to compare plain vaccines with those containing adjuvant and to assess the relative efficacies of different immunization schedules. Moreover, it may be possible to predict, from the agglutinin response obtained in the rabbit, the likely response of the child to a batch of vaccine.

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MEDICAL MEMORANDA

Hepatitis-associated Antigen in Liver Disease in Kenya

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The presence of hepatitis-associated antigen in the serum of patients with acute viral hepatitis (Blumberg *et al.*, 1967; Prince, 1968) and of patients with cirrhosis (Wright *et al.*, 1969; Sherlock *et al.*, 1970) suggests that there may be an aetiological relationship between these conditions. In Kenya viral hepatitis is a severe disease (Jindani *et al.*, 1970) and cirrhosis is often encountered. The following is a preliminary report of the occurrence of hepatitis-associated antigen in sera from patients with acute and chronic liver disease in Kenya.

Present Study

Sera from 225 patients attending the Kenyatta National Hospital with a variety of liver diseases and from 200 apparently healthy Nairobi blood donors (Table I) were tested for the presence of hepatitis-associated antigen. A diagnosis of viral hepatitis was made in the presence of jaundice of recent onset associated with raised serum

alanine aminotransferase and normal or only slightly raised serum alkaline phosphatase (up to 36 King-Armstrong units). The diagnosis was supported by a clinical course consistent with viral hepatitis. Twenty-six patients were tested during the acute illness and 49 after complete recovery. A diagnosis of cirrhosis was confirmed on liver histology in 31 patients; in 18 needle biopsy was contraindicated or was unsuccessful. The diagnosis of primary carcinoma of the liver was confirmed histologically in all cases. Other diseases studied included primary extrahepatic portal hypertension, schistosomal hepatic fibrosis, and idiopathic tropical splenomegaly (Pitney, 1968). All patients with primary extrahepatic portal hypertension had normal liver function and normal liver histology.

Sera were tested for hepatitis-associated antigen by the gel-diffusion technique with tris agarose medium as described by Prince (1968). Antibody used for routine testing came from two Kenyan patients and gave a positive reaction of identity with specific sera. The results of this study are summarized in Table I.

Discussion

The reported incidence of the antigen in acute viral hepatitis ranges from 12.3% of 358 patients in Japan (Okochi and Murakami, 1968) to as high as 80% of 49 patients in the U.S.A. (Gocke and Kavey, 1969). This variation may reflect

TABLE I—Occurrence of Hepatitis-associated Antigen in Liver Disease and in Blood Donors in Nairobi. Figures in Parentheses indicate Total Number Tested

Diagnosis	No. Positive	% Positive
Acute viral hepatitis (26)	14	54
Recovered from viral hepatitis (49)	1	2
Cirrhosis (39)*	8	20.5
Primary liver cancer (22)	3	14
Other conditions (89) (see text)	5	5.6
Nairobi blood donors (200)	12	6

*Including five patients with chronic active hepatitis.

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the sensitivity of the method used or the stage of the illness at which the serum was tested. It has been shown that the antigen commonly disappears within a few weeks of the onset of the illness (Wright *et al.*, 1969). In our patients there was no correlation between the duration of symptoms and the presence of the antigen. The varying incidence of hepatitis-associated antigen in acute viral hepatitis may also indicate different epidemiological forms of the disease.

Krugman *et al.* (1967), and Prince (1968) believe that the antigen occurs only during serum hepatitis—that is, when infection is due to the long-incubation agent (MS-2). Contrary to previous belief Krugman *et al.* (1967) have shown that the infective agent of long-incubation-type hepatitis can be transmitted by oral ingestion of infected material. Prince *et al.* (1970) believe that this agent is mainly responsible for sporadic cases of hepatitis in adults in the U.S.A. The finding of the antigen in 54% of our cases of acute viral hepatitis may indicate that these patients had long-incubation-type hepatitis.

Before drawing conclusions from the apparent association of hepatitis-associated antigen with cirrhosis and primary hepatic carcinoma in our series several factors must be taken into consideration. Firstly, a 6% incidence of the antigen was found in apparently healthy blood donors. Secondly, liver disease is common in Kenya, and the possibility of a chance association cannot be excluded in the small numbers studied so far. Thirdly, patients with portal hypertension are likely to have received blood transfusion after haemorrhage from oesophageal varices. On this last point it would have been tempting to suppose that the increased incidence of hepatitis-associated antigen in these patients had resulted from transfusion of blood containing the antigen. In fact, there was no obvious relationship between blood transfusion and the presence of the antigen (Table II).

Hepatitis-associated antigen was present in 8 out of 39 patients with hepatic cirrhosis and 3 out of 22 with primary hepatic carcinoma. Alpha-fetoprotein was detected in the serum of 14 (64%) of the 22 patients with primary hepatic carcinoma, and the three patients with hepatitis-associated antigen were all alpha-fetoprotein positive. Though only two patients with primary hepatic carcinoma were known to have

Histological Diagnosis of Burkitt's Lymphoma: A Cautionary Tale

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In 1969 the World Health Organization published a monograph listing histological criteria for the diagnosis of Burkitt's lymphoma. This was intended to help differentiate this neoplasm from the tumours with which it is most commonly confused: acute lymphoblastic leukaemia, acute myeloblastic leukaemia or the blastic phase of chronic myelogenous leukaemia, reticulum-cell sarcoma, and lymphocytic lymphosarcoma (Berard *et al.*, 1969). The diagnosis is facilitated when air-dried Romanovsky-stained imprint preparations are available for study (Wright, 1967). We recently saw a patient

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TABLE II—Comparison of Occurrence of Hepatitis-associated Antigen in All Patients with That Found in Patients who had Not Received Blood Transfusion

Diagnosis	Total Series		Not Transfused	
	No. Tested	No. Positive	No. Tested	No. Positive
Cirrhosis	39	8 (20.5%)	32	8 (25%)
Primary liver cancer	22	3 (14%)	20	2 (10%)
Other conditions (see text)	89	5 (5.6%)	60	4 (6.6%)
Nairobi blood donors	200	12 (6%)	200*	12 (6%)

*It is assumed that none of the donors had received a blood transfusion.

cirrhosis, it must be acknowledged that without post-mortem examination cirrhosis cannot be excluded in the other patients. The numbers studied so far are small, but the results do suggest an association of cirrhosis and primary hepatic carcinoma with hepatitis-associated antigen.

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in whom the histological differential diagnoses ranged much farther afield than other lymphoproliferative or myeloproliferative disorders before being recognized as Burkitt's lymphoma.

Case Report

A 4-year-old girl was admitted to the Lymphoma Treatment Centre on 19 September 1969 with a history of abdominal distension and paraplegia of three months' duration. Physical examination was remarkable only for two hard abdominal masses 10 by 12 cm, one arising from each iliac fossa, and flaccid paralysis of both lower limbs. At laparotomy on 21 September a greatly enlarged right ovary was excised; the left ovarian tumour was left in place. Frozen section of the mass showed a neoplasm made up of cells arranged in tubules and pseudorosettes. An imprint preparation showed small dark cells with little cytoplasm (Fig. 1), and there appeared to be a second population of paler, larger cells intermixed with them. A tentative diagnosis of neuroblastoma was suggested.

A paraffin section prepared from tissue which had been placed in 10% formalin for 16 hours showed a malignant tumour whose cells were arranged in cords and clumps (Fig. 2). A tentative diagnosis of dysgerminoma was made, pending section of better fixed material. This tumour forms 4 to 10% of the neoplasms found in East African children (J. N. P. Davies, personal communication, 1969). A subsequent section of material from the edge of the block of tissue which had been in formalin for 64 hours showed all the features of Burkitt's lymphoma—that is, fat-containing phagocytic histiocytes interspersed among uniformly primitive lymphoblastic