Lofenalac, it was necessary to increase total protein intake to over 5 g/kg/day. The amino acid imbalance which this caused was clearly related to the amino acid pattern of the feeds since both cows' milk and Lofenalac are low in cystine and taurine and relatively high in methionine and branched-chain amino acids. We suggest that the tyrosine content of Lofenalac should be increased by at least 20% to enable it to be used with whey based milks, thus avoiding an unnecessarily high protein intake and unbalanced amino acid intake.

The data presented here show that when our patient was growing at 20 g/kg/per day, which was necessary to achieve catch up growth, she required rather less than 90 mg/kg of phenylalanine and between 270 and 290 mg/kg of tyrosine. In infants without phenylketonuria, who are able to convert phenylalanine to tyrosine, the requirements for these two amino acids are best considered together. We conclude that a combined intake of at least 350 mg/kg, necessitating a protein intake of 3.5 to 4 g/kg from a whey adapted milk, is required for catch up growth in preterm infants. This estimate is in agreement with other studies.^{6,7} It has been pointed out that the volumes of milks such as Gold Cap SMA needed to achieve such protein intakes are needlessly high and could be avoided by using milks of higher protein content. We would add that the energy intake provided by large volumes of SMA is

also high and may lead to reluctance to feed (and therefore prolonged tube feeding) and excessive fat deposition.

We thank Dr Gerald McEnery for referring this patient to us and for providing details of the first two weeks of life. Dr Smith is in receipt of financial support from the Medical Research Council.

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Fatal hepatitis B in infant born to a HBsAg carrier with HBsAb

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SUMMARY Fulminant hepatic failure occurred in an 11 week old baby of a Caucasian mother who was hepatitis B surface antigen positive, B e antigen negative, and B e antibody positive. Infants of hepatitis B e antigen positive mothers receive immunoprophylaxis against hepatitis, unlike those born to mothers who are B e antibody positive.

Case history

The baby was admitted to hospital at 10 weeks of age having become jaundiced over a 24 hour period. She was passing dark urine and pale stools. She had been born after a normal delivery at 36 weeks' gestation, and weighed 2.5 kg. Apart from an

episode of transient tachypnoea during the first 48 hours of life, the neonatal period was uneventful. Both parents were Caucasian, there was no history of contact with blood products or drug addiction, and the mother had remained well throughout her pregnancy. The baby had not been exposed to hepatitis nor had she received any blood transfusions.

On examination she was pale and slightly icteric. Investigations showed blood sugar 1.3 mmol/l (23.4 mg/100 ml); prothrombin time greater than 90 seconds (control 11.5 seconds); kaolin cephalin time greater than 3 minutes (control 42 seconds); haemoglobin 9.8 g/dl, reticulocytes 5%, fibrinogen nil detected; total serum bilirubin 268 mmol/l (15.7 mg/100 ml) (all unconjugated); alkaline phosphatase 170 KA units %, aspartate transaminase 113 IU/l,

serum alanine transferase 198 IU/l; ammonia 105 $\mu\text{mol/l}$ (179 $\mu\text{g}/100\text{ ml}$); the blood film showed spherocytes and acanthocytes.

Subsequent management depended on clotting studies (see Figure) as well as continuous parenteral infusion of 10 to 20% dextrose to maintain a satisfactory blood sugar concentration. The baby's condition suddenly deteriorated 10 days after admission when she developed profound hypoglycaemia and progressed to grade IV hepatic failure. In spite of intensive support including lactulose, neomycin, and continued correction of anaemia, clotting disarray, and hypoglycaemia, she died. Consent for necropsy was not granted.

Screening for hepatitis B virus was carried out and as part of investigations to explain the jaundice. The baby was hepatitis B surface antigen positive and, as expected in early acute infection, was also B e antigen positive. Mother was found to be B surface antigen positive, B e antigen negative and B e antibody positive. Father was hepatitis B surface antigen negative. To determine whether the mother at the time of the birth of her baby had acute hepatitis B or was already a carrier of B surface

antigen the specimen of blood taken from her, three months after delivery, was tested for hepatitis B core antibody IgM and was found to be negative. It was concluded that she was a long term carrier of hepatitis B surface antigen and was almost certainly hepatitis B e antibody positive at the time of delivery.

Methods

Tests for hepatitis B surface antigen by reverse passive haemagglutination and for hepatitis B e antigen and hepatitis B e antibody by enzyme immunoassay were done at the Public Health Laboratory, Fazakerley Hospital. The results of tests for hepatitis B e antigen and antibody were confirmed by radioimmunoassay at the Virus Reference Laboratory, Colindale, where the tests for hepatitis B core IgM antibody were also performed.

Discussion

Early in acute hepatitis B infection, hepatitis B surface antigen and hepatitis B e antigen appear in the serum. Hepatitis B core antibody appears during the acute phase of the illness followed by B e antibody which signals the cessation of viral replication. Antibody to surface antigen, which is associated with long term immunity, is detected after hepatitis B surface antigen has disappeared.

In certain high risk areas vertical transmission is believed to account for 40% of chronic carriers. Babies at particular risk are those born to hepatitis B surface antigen carrier mothers who are B e antigen positive—a measure of high infectivity. Of the babies who become infected, many develop chronic infection in adult life and this is implicated in the development of primary hepatocellular carcinoma.

The racial origin of the mother affects¹ the risk of transmission from mother to baby. Derso *et al* showed that there was a high carrier rate in Chinese babies born to mothers who were chronic carriers of hepatitis B surface antigen and no carriers were found among the European infants. Similarly, a study in two west London hospitals² of maternal chronic carriers showed that eight of 18 infants with Chinese mothers became B surface antigen positive, but only six of 92 with mothers of other races were affected and that the presence of hepatitis B e antigen in the mother correlated with transmission to the child.

It is unusual for babies born to hepatitis B e antibody positive mothers to develop hepatitis B virus infection but several cases have been recorded.³ Sinatra *et al* reported three cases of

Figure Clotting studies.

perinatally transmitted acute icteric hepatitis B in infants born to B e antigen negative, B e antibody positive mothers, two of Asian and one of Caucasian origin.³

Delaphane *et al*⁴ reported from Chicago on three fatal cases of hepatitis B in infancy, all born to hepatitis B surface antigen positive mothers, one of whom was B e antibody positive. It is suggested that infants born to B e antibody positive mothers have a normal immunological response which may cause hepatic damage.

Studies have recently shown that passive administration of hepatitis B immunoglobulin may be insufficient to protect children from a lifetime risk of exposure.⁵ It is suggested that hepatitis B immunoglobulin be given to the baby for initial protection and, at the same time, vaccine for long term protection from an active immune response. If serological tests at 12 to 15 months detect hepatitis B surface and e antibodies there has been successful passive/active immunisation. On the other hand the presence of B surface antigen at 12 to 15 months reflects failure of the prophylactic regimen.

If the mother of the baby described in this case had been identified antenatally as hepatitis B e antibody positive this would have led to exclusion of her child from immunoprophylaxis.⁶ It would be

appropriate therefore to give immunoprophylaxis to all infants born to B surface antigen positive mothers and not only to those found to be B e antigen positive.

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Scalp changes after fetal monitoring

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SUMMARY We prospectively studied 535 newborn infants who had been monitored during labour with scalp electrodes. Daily examination of scalp changes showed frequent transient mild lacerations, while severe complications were rare: seven (1.3%) had scalp ulceration and one (0.2%) developed scalp abscess.

Fetal heart monitoring during labour is now used in nearly all obstetric units to improve perinatal outcome. Several reports have described complications after the application of the scalp electrodes.¹⁻³ The whole spectrum of neonatal scalp changes associated with fetal monitoring, however, and their precise incidence have not as yet been described. We undertook a prospective study with daily ex-

amination of scalp changes in all newborn infants monitored during labour, and describe their incidence, clinical spectrum, and factors associated with severe complications.

Patients and methods

Study patients. All the infants delivered at the Beilinson Medical Center between 1 August, 1982 and 16 October, 1982 who had direct fetal heart rate monitoring during labour were included in the study. Of the total 660 live births during that period, 535 (81.1%) were monitored by scalp electrodes, and these infants comprised the study group. The 1513 OA fetal monitoring spiral electrodes (Hewlett-Packard Company USA) were used in our patients.

Methods. Direct fetal monitoring was performed