Her baby daughter was born at term by a spontaneous delivery, weighing 2·75 kg. The sutures were widely separated, the fontanelle large, and the skull bones soft and easily indented. There was no swelling at the wrists or ankles, but the rib ends seemed enlarged. The clinical diagnosis of rickets was confirmed by a wrist x-ray (Fig.) and the results of biochemical investigations showed a low serum calcium and raised alkaline phosphatase levels. These and subsequent investigations are shown in the Table. Her level of 25-HCC was

TABLE

Biochemical findings in a baby with congenital rickets

Age (mth)	Calcium (mg/100 ml)	Phosphorus (mg/100 ml)	Alkaline phosphatase (IU/l.)
4 dy	6·7	5·5	227
10 dy	7·9	6·0	222
1	9·0	6·0	265
3	8·9		284
4	10·5	6·5	225
5	9·5	6·1	188
6	9·5	5·7	150

8·3 ng/ml. The infant was treated with 1000 units calciferol daily, later increased to 3000 units, calcium gluconate 300 mg three times a day for a fortnight, and was fed on S.M.A. After 6 months the rickets had healed.

Comment

The baby had clinical, biochemical, and radiological features of rickets detected in the first few days of life, and her mother had biochemical and clinical evidence of osteomalacia. The mother's dietary intake of vitamin D was lower than that required in pregnancy and the level of 25–HCC considerably below normal, which has not previously been shown in a case of neonatal rickets. These findings suggest that the mother had simple nutritional osteomalacia causing rickets to develop in the fetus before birth: true congenital rickets. This supports the suggestion made by Purvis *et al.* (1973) that maternal vitamin D deficiency is a cause of hypocalcaemia in the neonate.

Over the last few years a number of Asian neonates have been seen in Derby with prolonged hypocalcaemia and were regarded as examples of transient idiopathic hypoparathyroidism (Prader and Fanconi, 1969). Rickets was not suspected and the babies were treated with large doses of vitamin D. In retrospect, some of the cases were probably examples of congenital rickets. We agree with Ford *et al.* (1973) that rickets should always be considered as a cause of hypocalcaemia in an Asian neonate, and that biochemical survey for osteomalacia should be

performed routinely in antenatal care of Asian women.

Summary

A newborn Asian baby with congenital rickets is described. Her mother had osteomalacia, which, in view of her low dietary intake of vitamin D and low level of circulating 25–HCC, was probably nutritional in origin. Biochemical screening of pregnant Asians for osteomalacia should be a routine part of antenatal care.

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Hyperglycaemia and convulsions in children

It is common practice to measure blood sugar content in patients with acute episodes of convulsions. However, very few reports deal with blood glucose levels during fits in infancy. Millichap (1968) cited studies of 86 patients with febrile convulsions who had normal values. Zellweger (1948) found raised values (from 117 to 296 mg/100 ml) in 6 out of 12 children examined during or immediately after a febrile convulsion. Those authors cited by Millichap (1968) who found raised CSF glucose levels in children do not report the corresponding blood values. We wish to report our experience.

Short reports

Material and methods

Thirty-nine children were seen during an acute episode of convulsions. In none was there any other symptom of an acute inflammation of the CNS. All had apparently been healthy up to the beginning of the convulsions. Their ages were from 10 months to 12 years (mean 2½ years). There were 25 boys and 12 girls. None of the children had taken food for at least 4 hours before the blood glucose measurement.

In 33 children the final diagnosis was 'febrile convulsions' and in the remaining 6 'epilepsy'. In all children blood was drawn from the cubital vein during the fit or immediately after. Glucose was examined by the orthotoluidin method (Dubowski, 1962).

Results

The Fig. shows the values of blood glucose in these children. Column 1 shows the values of glucose in the 39 children during or immediately after the fit. In 22 children the values were higher than 120 mg/100 ml. In 13 of them the values were
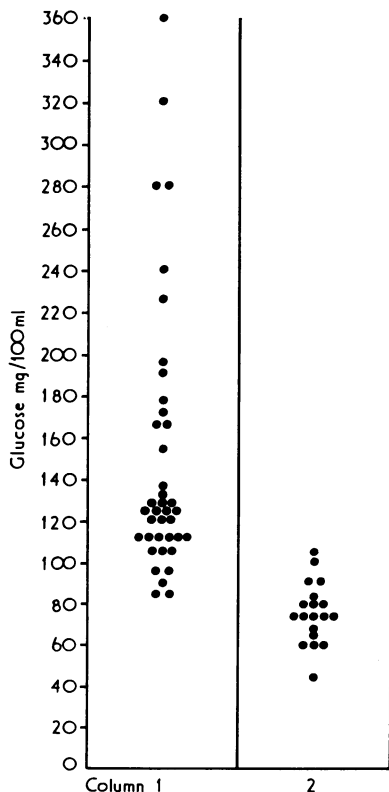

FIG.—*Blood glucose values. Column 1, during convulsive episode. Column 2, 2 hours after cessation of the convulsions in the 19 children whose original values were above 120 mg/100 ml.*

upward of 140 mg/100 ml, and in 6 were over 200 mg/100 ml. The highest value found was 360 mg/100 ml. In 19 of the children with a glucose level of over 120 mg/100 ml, a second blood sample was examined 2 hours after convulsions stopped. These values are shown in column 2 of the Fig. Their range is from 45 to 105 mg/100 ml.

In 8 of the children with blood glucose values over 180 mg/100 ml, an oral glucose tolerance test was performed before their discharge. The peak glucose level was 117 mg/100 ml in one case, but lower in the other 7.

Blood glucose levels one hour after a normal meal were examined in 18 other children and found to range between 72 and 96 mg/100 ml.

Discussion

Hyperglycaemia is a well-known phenomenon in electric convulsive therapy (Gour and Bhargava, 1957). It has been studied in electrically shocked rats, and was found to be related to a rise in the level of catecholamines in the blood and urine of the treated animals. After adrenalectomy, the electric shock is followed by a decrease in the blood level, while vagotomy makes the hyperglycaemic response even more marked (McIlwain and Bachelard, 1971).

It appears that the hyperglycaemia during a convulsive episode is due to stress, is of short duration, and probably has no pathological significance. It may be important to bear in mind because of its possible effects on the glucose level of the CSF. More studies should be undertaken to correlate the blood and CSF glucose levels in convulsive episodes. Millichap (1968) reported raised CSF glucose in 35 out of 68 patients examined, while Phadke in 1957 (cited by Millichap, 1968) found a raised CSF glucose in all 28 cases examined. As we have done lumbar punctures in only 6 of our cases, we have been unable to substantiate these findings. It may be that hyperglycaemia is the cause of raised CSF glucose values in some viral CNS infections.

Knowledge of the possibility of extreme hyperglycaemia in convulsive disorders should help to prevent false diagnosis of diabetes mellitus.

Summary

Blood glucose was determined during or immediately after a fit in 39 children with 'febrile convulsions' and in 6 with other fits.

In 22 of the 39 children blood glucose over 120 mg/100 ml was found. In 6 children the levels exceeded 200 mg/100 ml. 2 hours after the seizures the blood glucose levels returned to normal.

It is important to recognize this phenomenon to prevent an incorrect diagnosis of diabetes mellitus and inappropriate treatment with insulin.

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Neonatal thyrotoxicosis treated with propranolol

Neonatal thyrotoxicosis is a rare transient disorder related to the presence of long-acting thyroid stimulator (LATS) in the infant's blood. Treatment of the disorder has been based on iodides, sedatives, thiouracil drugs, and digoxin. As the condition is self-limiting and subsides spontaneously, control of symptoms through blockade of β-adrenergic receptors may be a more logical mode of therapy. Propranolol has been used in this way in the interval between the administration of radioactive iodine and its suppression of thyroid function (Hadden *et al.*, 1968; Shanks *et al.*, 1969). Recently a case of neonatal thyrotoxicosis has been described in which propranolol was used in conjunction with digoxin, phenobarbitone, propylthiouracil, and Lugol's iodine (Smith and Howard, 1973). This is the first reported case of neonatal thyrotoxicosis treated only with propranolol.

Case report

A male infant weighing 2720 g was delivered on 25 January 1972, by elective caesarean section at 38 weeks' gestation. The pregnancy had been complicated by moderate pre-eclampsia.

The mother, a 26-year-old primigravida, had had a subtotal thyroidectomy in 1963 for thyrotoxicosis. Moderate exophthalmos, present before this operation, was still present. During the pregnancy the eye signs became more marked and the results of thyroid function tests were T_3 red cell uptake 8–10%, protein-bound iodine (PBI) 7·4–11·4 μg/100 ml. The fetus had persistent tachycardia with the heart rate greater than 180 beats/minute. The mother was treated during the last 10 days of pregnancy with carbimazole 20 mg 4 times daily, propranolol 40 mg 4 times daily, and diazepam. During this period the fetal heart rate was less than 150 beats/minute.

At birth the infant was noted to have a small goitre; there was no exophthalmos and he was not overactive. The heart rate was 130 beats/minute and there was no cardiac failure. Estimations of maturity confirmed that the infant was at 38 weeks. Thyroid function tests on the cord blood (Table) were in the thyrotoxic range (PBI

TABLE

Tests of thyroid function in infant

	T_3 red cell uptake (%)	PBI (μg/100 ml)
Birth	38·1	9·1
3 wk	38·1	16·0
7½ wk	15·5	6·2
(Normal)	(11·5–19·5)	

9·1 μg/100 ml, T_3 red cell uptake 38·1%). Further tests of thyroid function, T_4/T_3 ratio, and free thyroxine index were all in the thyrotoxic range.

The baby was kept under close observation. He remained well for the first 2 weeks, was not overactive, and slept for normal periods. His intake of milk (450 ml/day) was in keeping with his calculated requirements, though his weight remained static. However, by the end of the second week, signs suggestive of thyrotoxicosis were appearing and by 3 weeks the infant was obviously thyrotoxic (Fig.). He was hyperactive, irritable, and cried continuously. His appetite was voracious and he took 840 ml milk/day. His skin had become abnormally pink, hot, and velvety in texture. The eyes were more prominent and there was lid retraction. The heart rate was 180 beats/minute but there was no evidence of cardiac failure. EEG showed sinus tachycardia. There was a distinct tremor in all limbs. The results of thyroid function tests were PBI 16 μg/100 ml, T_3 red cell uptake 38·1%.

Treatment with propranolol (Inderal) was started with a dose of 0·5 mg 4 times daily, given as a solution added to his milk at the end of the third week. Daily increments in the dose were made over 4 days, until he was receiving 4 mg 4 times daily. On this treatment his behaviour assumed a more normal pattern and the heart rate slowed to 140–150 beats/minute. Improvement was most marked immediately after each increase in dose but with time this seemed to decline so that the dose had to be further increased over 2 weeks to 8 mg 4 times daily, which gave a plasma propranolol level (Shand, Nuckolls, and Oates, 1970) of 89 μg/l. 2 hours after a dose.