animals (Levine 1967) or in humans (Goudie & Pinkerton 1962, Hume & Roberts 1967).

There are no specific histological features of human rubella infection; however, in tissue culture cytopathic changes simulate those found in this child.

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Mild Hyperparathyroidism in a Girl aged 10 Years 6 Months

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D de G, girl born 19.1.58. West Indian

History: This girl first complained of mild abdominal pain, occasional vomiting, and severe constipation at the age of 9. Her constipation was so unusual and severe that we estimated the plasma calcium, which was 12·0 mg/100 ml. Gradually all her symptoms resolved spontaneously and during the months before the exploration of her neck she felt well and her bowel actions were normal.

She had been given cod liver oil 500 i.u. daily but this was stopped three months after the onset of her symptoms.

Her parents and sisters were well and showed no biochemical abnormality. On examination her height remained on the 75th percentile throughout the period of observation. She was normotensive and there was no corneal calcification.

Investigations: During eighteen months' observation there was sustained hypercalcæmia, mean value 12·4 mg/100 ml; low plasma inorganic phosphate, mean value 3·0 mg/100 ml; and very high urinary calcium, mean value 320 mg/100 ml. An initial hydrocortisone suppression test at the age of 9½ showed a clear fall of the plasma calcium, but when repeated at the age of 10½ there was no suppression of the hypercalcæmia.

Plasma vitamin D levels were normal, there was no hypersensitivity to vitamin D (10,000 i.u. calciferol daily for ten days). Sarcoidosis was excluded by a normal liver biopsy, a negative Kveim test and a positive Mantoux test.

When she was 10½ Mr Selwyn Taylor explored her neck and examined all four parathyroid glands. The right lower, which contained a small adenoma, was removed. She made an uneventful recovery and has remained normocalcæmic with a normal urinary calcium.

Comment

Although there are descriptions of 39 children under the age of 16 with proven parathyroid adenoma (Fanconi & Mieth 1967), we have found only one other description of a child who had only moderate hypercalcæmia and neither radiological bone disease nor renal involvement. He was a boy of 15½ found to be hypertensive at routine examination and subsequently to have a serum calcium of 13 mg/100 ml (Chaves-Carballo & Hayles 1966).

Considerable difficulty was experienced in establishing this diagnosis in the face of mild hypercalcæmia that was initially suppressed by hydrocortisone. The diagnosis was supported first by the basal phosphate excretion index, which was raised when compared with normal girls of her age. Later a bone biopsy showed increased osteocytic osteolysis when compared with a post-mortem biopsy of a specimen from another girl of the same age. Finally, a repeat hydrocortisone suppression test failed to lower the plasma calcium. The value of these tests in childhood is discussed elsewhere (Latham *et al.* 1969).

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Therapeutic Problems of Adolescent Homocystinuria

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J D, boy aged 11

This patient showed the classical features of homocystinuria, including iridodonesis, ectopia lentis, skeletal and joint changes, and high complexion. Inferior vena caval thrombosis with pulmonary infarction occurred two years ago following chickenpox. He showed marked emotional lability. IQ 70. Elevation of plasma homocystine (2·5 mg/100 ml), methionine (3·8 mg/100 ml) and urinary homocystine (85 mg/24 h) confirmed the clinical diagnosis.

Effective reduction of plasma homocystine has been variously achieved by combining a low methionine intake with supplemental cystine and a methyl donor – betaine or choline (Perry *et al.* 1968) or by independent oral administration of pyridoxine (Barber & Spaeth 1967) or folic acid (Carey *et al.* 1968). Unfortunately, dietary treatment in our patient was not successful. The unpalatability of the food regimen soon led to dietary indiscretions. The need for regular hospital attendances for plasma and urine monitor-

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ing became unacceptable to both patient and parents, and blood sampling was eventually refused. The scheme had to be abandoned after four weeks.

The recognized relationship between increased platelet stickiness and raised plasma homocystine forms the basis of dietary therapy in the adolescent, which aims to prevent the thromboembolic features of the disease. The extent to which increased platelet stickiness is the sole cause of these episodes remains largely speculative, but the role of the generalized vascular change, so frequently observed at autopsy in these individuals, cannot be insignificant. This progressive structural damage is not reversed by dietary treatment.

While the value of dietary therapy in the affected newborn is not in dispute, the benefits for the adolescent would not seem clear. The added practical difficulties of dietary restriction in these mentally retarded children are considerable, while from the humanitarian point of view, this dietary policy seems hard to justify.

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Retinal Vein Thrombosis with Peripheral Venous Thrombosis and Thrombocytopenia

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A B, boy aged 9½ years

Presented in April 1968 with a six-week history of pain followed by swelling of the whole right leg, which showed signs of deep venous thrombosis, confirmed by venogram (Fig 1). No systemic symptoms or signs. Investigation showed high ESR and platelet counts of around 60,000 on various occasions. Bone marrow suggestive of subacute or chronic idiopathic thrombocytopenic purpura. Partial thromboplastin time prolonged, hence anticoagulants were not given. Early in May 1968 developed pain in both eyes, diplopia, impaired vision and slight restriction of visual fields. Bilateral retinal vein thrombosis found. The diagnosis of a diffuse vasculitis involving retinal and peripheral veins was made. Treated with prednisolone 40 mg/day. His vision and fundi returned to normal within four weeks. Ten weeks later, and again on subsequent occasions, he developed superficial thromboses of various veins in the right leg and thigh. Biopsy was not helpful. Six months after starting treatment, and while still on prednisolone 10 mg/day, he still had fresh episodes of thrombosis in the right leg. Detailed hæmatological examination did not reveal any clotting defect, the only persistent abnormality being thrombocytopenia, prolonged partial thromboplastin time and decreasingly elevated ESR. No LE cells or antinuclear factors demonstrated at any stage.

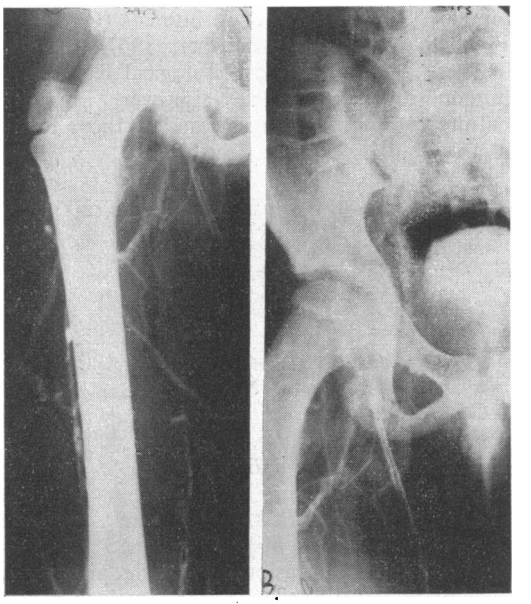

Fig 1 *Venogram of right leg showing a block at the level of the femoral vein. The iliac veins have been filled by the collaterals*

Discussion

The association of peripheral venous thrombosis with retinal venous occlusion has not been described before. In the presence of the bilateral retinal findings, the possibility of an intracranial space-occupying lesion was considered but excluded. Embolism of the retinal veins from the leg veins could not have occurred in the absence of a congenital heart disease; also the lesions were too symmetrical for this possibility.

The usual causes of retinal vein thrombosis are stagnation thrombosis in arteriosclerosis, external compression and primary vasculitis. The first two are usually seen in adults and there was no clinical evidence for these in this child: by exclusion, vasculitis was favoured.

The VI nerve palsy responsible for his diplopia remained unexplained and it lasted about six to eight days with complete recovery.

The other unusual feature was the presence of low platelets with prolonged partial thromboplastin time and prothrombin time. The platelets were particularly low during acute episodes of