SDS for bone age did not change. The difference between mean sitting height SDS and mean subischial leg length SDS remained unchanged over the five year study period.

Discussion

The availability of large quantities of biosynthetic growth hormone have stimulated many investigators to use this agent in short children who are or are not growth hormone deficient, such as those with dysmorphic syndromes, skeletal dysplasias, and normal short stature. The effect of growth hormone treatment on body segments therefore becomes an important consideration. We have compared the growth of upper and lower segments in children with isolated growth hormone deficiency. Although caution needs to be exercised in the interpretation of sitting height standards (SM Herber, RDG Milner. Have sitting height standards changed? Abstract G87; 58th Annual Meeting of the British Paediatric Association, York, 1986.), our results confirm that sitting height and subischial leg length react equally to human growth hormone treatment in prepubertal children over many years of treatment. Growth hormone treatment in these children prevented further loss in stature which would ensue if treatment had not been initiated. Growth hormone treatment, however, did not improve the height prognosis at the commencement of treatment, as judged by little change in height SDS for bone age. Instead it ensured that the child's remaining growth was 'normalised'.

These findings have important implications. We

would predict that the response of children who have skeletal dysplasia to growth hormone will not be as much as might be expected because of a lack of either the spinal or lower limb component or both to the growth response to growth hormone treatment. Similar findings have been observed in children who have received craniospinal irradiation and been treated with growth hormone.⁵ Changes in standing height in such individuals should not be used to define response but rather the change in body segments should be calculated.

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Fatty liver and medium chain triglyceride (MCT) diet

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SUMMARY A 12 year old boy with intractable epilepsy developed fatty infiltration of the liver after three years' treatment on the medium chain triglyceride (MCT) diet. This was not associated with any hepatic dysfunction and resolved after discontinuing the diet. Three of four other patients on the same diet had evidence of hepatic steatosis.

Sills *et al*, working at Leeds General Infirmary, reported the beneficial effect of the medium chain triglyceride (MCT) diet on the treatment of epilepsy in childhood.¹ Adverse side effects have included mild abdominal pain and diarrhoea, and one patient suffered transient blindness when vitamin supplementation was omitted. More recently we have been aware of the problem of fatty infiltration of the liver.

Case report

The patient, a boy, had been noted at birth to have

Ketogenic diets for the treatment of intractable epilepsy have been used for over 60 years. In 1986

Table Clinical details of the five epileptic patients on the MCT diet who had abdominal ultrasound scans

Case No	Age (years)	Diagnosis of epilepsy	Drugs	Duration of diet (years)	Results of investigations		
					Ultrasound scan	Liver function tests	Triglycerides and cholesterol
1	12	Astatic myoclonic	Clobazam	3	Abnormal†	Normal	Normal
2	8	Astatic myoclonic	Sodium valproate, clobazam	2	Abnormal†	‡	‡
3	4	Astatic myoclonic	None	3	Abnormal†	Normal	‡
4	8	Astatic myoclonic	Carbamazepine	2	Abnormal†	Normal	Normal
5	20	Grand mal	Primidone	3*	Normal	‡	‡

*This patient had very poor dietary compliance.

†Abnormal ultrasound scan with increased echogenicity of the liver.

‡Test not performed.

prune belly syndrome and had required surgery at the age of 2 months to reimplant his left ureter. At the age of 6 he had presented with tonic clonic seizures that were initially controlled with sodium valproate. At the age of 7½, however, he had developed astatic myoclonic epilepsy that was drug resistant. At 9 years of age he was started on the MCT diet with considerable improvement in his seizure control.

At 12 years of age an abdominal ultrasound examination, which was performed after a urinary tract infection, showed evidence of bilateral hydronephrosis and hydroureters that was associated with a thick walled distended bladder indicating bladder outflow tract obstruction. At the same examination the liver was noted to show mild enlargement and an abnormally fine and highly reflective echo pattern. This incidental finding was strongly suggestive of fatty infiltration. Further investigation of hepatic function showed normal liver function tests and normal concentrations of serum triglycerides and cholesterol. The MCT diet was discontinued and four weeks later a repeat ultrasound scan showed diminution of the echogenicity of the liver though the scan was still abnormal. A laparotomy was performed for surgery on the renal tract and a liver biopsy specimen taken at this time showed diffuse fatty infiltration of the liver. Repeat abdominal ultrasound scans one month and three months after surgery showed complete resolution of the previous hepatic abnormalities.

Abdominal ultrasounds were undertaken in four other epileptic patients who were on the MCT diet. Three of these patients showed a diffusely bright liver on abdominal ultrasound scans suggestive of fatty infiltration of the liver. The fifth patient had a normal scan but it was notable that she had poor dietary compliance (table).

Discussion

For many children the MCT diet has had a profound

effect on the control of their epilepsy. Side effects previously reported have been minimal—namely abdominal pain and diarrhoea—though Livingston reported that children on the diet failed to grow.² We have been unable to find any other reports of the effects of the ketogenic MCT diet on hepatic function in children.

The mechanism of fatty infiltration of the liver is unclear. Potentially there are three mechanisms whereby fatty infiltration may occur. Firstly from increased dietary intake, secondly from the direct metabolic changes within the liver, and thirdly from breakdown of the fatty acid pool of adipose tissue. In other disease processes where the MCT diet has been used hepatic steatosis has not been described. The quantity of MCT used in these diets, however, is not sufficient to produce ketosis. Medium chain triglycerides are absorbed directly from the gastrointestinal tract in the form of medium chain fatty acids. These are carried directly to the liver by the portal vein where they are converted to carbon dioxide, acetate, ketones, and long chain fatty acids.³ Previous studies of the effect of the MCT ketogenic diet in epileptic children have shown no effect of the diet on liver function tests, serum cholesterol, lipoproteins, or low density lipoproteins.⁴ This is in contradistinction to the standard ketogenic diet where hyperlipidaemia has been described.⁵ The ketogenic diet does increase the concentrations of the ketones, betahydroxybutyrate, and acetoacetate without any change in the blood pH.⁵ One of the final common pathways for the production of fatty change in the liver is the accumulation of excess hydrogen ion within the cell.⁶ It is possible that the mechanism of fatty infiltration that we describe is due to a fall in the intracellular pH secondary to the chronic ketosis produced while on the diet.

Despite the fatty infiltration noted in our patients there was no noticeable effect on the liver function tests of these children, and more importantly when

the diet was discontinued the abnormal changes disappeared. We report these changes so that clinicians using this diet may be aware of the abnormal abdominal ultrasound findings and their importance.

We would like to thank Dr WI Forsythe for permission to study the patients under his care.

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HIV infection in Zimbabwe

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SUMMARY A total of 188 children with positive serology for HIV were identified during an 18 month period. Two seronegative children with clinical features of AIDS had seropositive mothers. Ten children were asymptomatic on initial testing; one has since died with infection. The commonest presenting features were generalised lymphadenopathy, failure to thrive, chronic diarrhoea, and pneumonia. Thirty four children are known to have died.

AIDS is reported to be endemic in parts of Africa and is thought to be largely heterosexually transmitted.¹ The vertical transmission of this disease to the children of mothers who are seropositive is now well recognised.^{2,3} During 1985 patients began to present in Zimbabwe with illnesses suggestive of HIV infection. Young adults were presenting with severe herpes zoster, generalised lymphadenopathy, and severe respiratory infections. Children were seen who appeared to be suffering from malnutrition but whose socioeconomic histories were inconsistent with this, and they were failing to respond to standard nutritional and medical treatment. Other children were seen with recurrent infections or infections that were resistant to conventional treatment, suggesting an immunodeficiency state. Routine testing of donated blood began in August 1985 at the Harare Blood Transfu-

sion Service and HIV testing has been available to clinicians since then.

Patients and methods

The Harare Blood Transfusion Service serves the northern half of Zimbabwe, covering a population in excess of three million. HIV testing was performed using the ELISA test,⁴ and confirmation of all positive results was obtained using the Western blot technique.⁵ All HIV request forms on seropositive children between October 1985 and March 1987 were reviewed. This accounts for all positive HIV serology in children documented in the north and eastern part of the country as no other facility was testing at the time. Clinical data was obtained from a questionnaire incorporated into the request form and additional information, when available, was extracted from some case notes in the central hospitals. The questionnaire was introduced only in the second half of the period under review and the quantity and quality of the information supplied was quite variable. In view of this the data represent only minimal estimates of each feature.

Results

A total of 188 children were found to be seropositive and of these 145 were identified in the central hospitals in Harare. Two other children who had clinical features suggestive of AIDS but negative