At the time of writing only two grafts remained patent, after up to two years' follow-up (see table). One of the patent grafts was aneurysmal. No other graft remained patent for more than six months, and four patients had to undergo major amputation. The remaining four patients were severely disabled by claudication or ischaemic ulceration. Other complications apart from graft occlusion and aneurysm formation have occurred. One graft was insufficiently mature for use at six weeks, although maturation was satisfactory at 12 weeks. Both early failures (in cases 4 and 5) followed reoperation for reactionary haemorrhage, probably resulting from fraying of the graft at the proximal anastomosis. One of these grafts was removed two months after operation following a secondary haemorrhage. Infection was not otherwise encountered.

Outcome of Sparks mandril graft

Case No	Age (yrs)	Preoperative ulceration	Fate of graft	Present state of limb
1	63	Yes	Thrombosed at 3 months	Severe claudication
2	44	No	Thrombosed at 4 months	Healed above-knee amputation
3	65	No	Patent at 18 months but aneurysmal	Satisfactory
4	59	Yes	Thrombosed at 24 hours	Healed Gritti-Stokes amputation
5	70	Yes	Thrombosed at 24 hours	Healed Gritti-Stokes amputation
6	59	Yes	Patent at 16 months	Satisfactory
7	50	No	Thrombosed at 4 months	Severe claudication
8	63	Yes	Thrombosed at 6 months	Recurrent ulceration
9	61	Yes	Thrombosed at 2 months	Claudication
10	64	Yes	Thrombosed at 1 month	Healed below-knee amputation

Comment

This series is small but the results correspond with those of the only other reported series.^{4 5} There can be little doubt that the Sparks mandril compares unfavourably with more conventional materials, although it must be remembered that seven of our patients were threatened by amputation and that a completely satisfactory alternative reconstruction was not available. Nevertheless, it is difficult to escape the conclusion that the thrombogenicity and lack of inherent strength of the mandril make it unsuitable for use in the femoropopliteal segment and that some of our patients might have been better served by earlier amputation.

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Two congenital neurological abnormalities caused by thalidomide

Thalidomide (alpha-phthalimidoglutarimide) is a known teratogen. Ingestion of the drug during early pregnancy led to a variety of congenital abnormalities, especially limb deformities, defects of the external ears and duodenal atresia. Though it was stated that the central nervous system was not affected,¹⁻³ more recent reports suggest this is not so.^{4 5} We describe a girl with two congenital neurological abnormalities presumably due to thalidomide.

Case report

A 14-year-old girl presented with lacrimation from the right eye while eating and a lateral gaze palsy. She had had a normal birth and at routine neonatal examination was found to have bilateral hypoplastic thumbs. Her

mother had taken thalidomide regularly during the first trimester for night sedation. At the age of 2 the child had been admitted to hospital for operation on her thumbs. The admission notes state that she had "abnormal eyes." Nevertheless, the abnormal eye movements had been noticed by her mother only for about four years before the present admission. She had not complained of any difficulty in moving her eyes, and had obviously learned to compensate by head movements. The lacrimation from the right eye had first been noticed by her mother after she had been weaned on to solid food.

She had a horizontal gaze palsy to both sides, an amblyopic right eye with gross visual impairment, a minimal right-sided ptosis, production of tears from the right eye during eating, and bilateral hypoplastic thumbs. The results of extensive investigations were all normal. X-ray films of the hands showed abnormalities of the scaphoid, trapezium, trapezoid, and the first metacarpal bone of both hands. They also showed abnormal articulation of the metacarpophalangeal joint of the left thumb. A cervical spine x-ray film showed fusion of the second and third cervical vertebrae. Electronystagmography showed no movements of the eyes in a horizontal direction, but vertical movements were within normal limits.

Comment

Cranial nerve palsies have been reported as a thalidomide effect, but these were attributed to osseous changes within the bony foramina or canals through which the nerves passed.¹⁻³ Recent reports have shown a high incidence of cranial nerve abnormalities, abnormal tear production and epilepsy, attributed directly to thalidomide.^{4 5} In our patient the gaze palsy cannot be explained by the entrapment theory, but must be due to a more central lesion in or around the sixth nerve nucleus. The abnormal right-sided lacrimation could also be explained by a midbrain lesion. The association of rudimentary thumbs, two neurological lesions, and the history of ingestion of thalidomide is further evidence that thalidomide can cause congenital neurological defects.

We thank Dr R G Lascelles for his advice and criticism.

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Return to normal of Argyll-Robertson pupils after treatment

Argyll-Robertson pupils—that is, pupils which do not react to light but do to accommodation—are often associated with neurosyphilis, but sometimes occur as an isolated finding without the disease. Nevertheless, it is essential that a complete physical, neurological (including examination of the spinal fluid), and serological examination should be carried out. I report here a case of Argyll-Robertson pupils in a man with syphilis, which returned to normal after treatment.

Case report

A 46-year-old homosexual man gave a history of a rectal infection in 1973 which was treated with 3.75 megaunits of penicillin, a dose which would almost certainly have arrested any incubating syphilis. Serological tests at the start of treatment for gonorrhoea were negative for syphilis and remained so during his three-monthly follow-up. He was referred by his general practitioner to the special clinic in February 1977 with a three-week history of

blurring of vision and irregularity of pupils. In addition, serological examinations were positive for treponemal infection. He complained of blurred vision and occasional headaches of some three to four weeks' duration, but gave no history of any previous penile or rectal lesions since his previous gonococcal infection in 1973, although he did admit to several homosexual relationships, both active and passive. The results of full general and neurological examinations were normal apart from his right pupil, which was larger than the left, and did not react to light, but reacted to accommodation. The spinal fluid was clear and under normal pressure; the protein content was within normal limits, the cells 30 lymphocytes/mm³, and the Wassermann reaction positive. In the blood the results of four serological tests were positive (WR, VDRC, TPHA, and FTA).

He was treated with daily crystalline penicillin, 1 megaunit four times daily for two days under corticosteroid cover, and thereafter with daily injections of benethamine penicillin G, procaine penicillin, and penicillin G sodium (Triptopen), 1.25 megaunit daily for 14 days. During treatment it was noticed that although the pupils remained unequal, there was a slight definite reaction to light. Two months after treatment the pupils were equal and both reacted satisfactorily to light. When last seen on 11 May 1977, his pupils were equal and reactive to both light and accommodation.

Comment

This represents a satisfactory response to treatment and shows the importance of serological investigations. Stokes *et al*¹ have also seen Argyll-Robertson pupils return to normal after treatment, but this phenomenon must be rare.

I thank Mr N Brown, consultant ophthalmic surgeon and Mr D Hill charge nurse, Coventry and Warwickshire Hospital, for their help.

¹ Stokes, J N, *et al*, *Modern Clinical Syphilology*, 3rd edn. Philadelphia and London, Saunders, 1945.

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Congenital hypothyroid gigantism: a new diencephalic syndrome?

Children with congenital hypothyroidism are almost invariably short, but the patient described here was remarkably large from birth and had congenital hypothyroidism of pituitary origin.

Case report

A boy weighing 5000 g and 57 cm long was born 5 days after term. There was no birth asphyxia. A big tongue, palpable kidneys, a spleen just palpable, and a normal liver were noted. There was no umbilical hernia and there were no ear pits. Visceromegaly was not noted after the first day. The plasma glucose at 12 hours was 2.6 µmol/l (47 µg/100 ml) and repeated testing with Dextrostix showed no hypoglycaemia. The mother's glucose tolerance was normal. Jaundice developed in the first 24 hours and despite phototherapy the plasma bilirubin rose to 408 µmol/l (23.9 mg/100 ml) at 4 days 12 hours and an exchange transfusion was performed. The jaundice, which lasted for 2 weeks, was thought to be due to ABO blood group maternofetal incompatibility.

Thyopac tests done at 6 days and repeated showed evidence of hypothyroidism. Serum thyroxine levels at 6 days, 6 weeks, 10 months, and 11 months were 3.9, 3.8, 4.5, and 3.0 µg/100 ml and plasma thyrotrophin at 11 months was 5 mU/l.

When I first saw the child at the age of 10 months he was of enormous stature, but motor and intellectual development were normal. The radiological bone age was 3 to 4 months (one ossification centre at the wrist). Growth in the first 2 years is shown in the figure. Developmental progress was normal, as were hearing and vision. There was no gross visual field defect and the fundi were repeatedly normal up to 3 years of age.

Plasma urea, electrolyte, alkaline phosphatase, cholesterol, triglyceride, and lipoproteins concentrations were normal. Chromosome analysis showed a normal male karyotype.

Family—There was no family history of endocrine or neurological disease. The mother was 163 cm tall and weighed 66.3 kg, the father was 178 cm tall and weighed 78.1 kg, and the brother—aged 3.1 years—measured

Growth up to 2 years. (Growth chart amended from Gairdner and Pearson.⁶) 10th, 50th and 90th percentiles in a normal population of boys are shown.

101.4 cm (95th percentile) and weighed 16.9 kg (90th percentile). Adjusted for mid-parent height,¹ the patient's height at 2 years was 3 cm above the 97th percentile and his brother's height at 3 years 1 month was between the 90th and 95th percentiles.

Adrenal function—The response to intramuscular tetracosactrin 250 µg was as follows: at 12 months plasma cortisol was 580 nmol/l (21 µg/100 ml) before injection and 980 nmol/l (35.5 µg/100 ml) 30 minutes after injection; at 1 year 8 months concentrations were 625 nmol/l (22.6 µg/100 ml) before injection, 1045 nmol/l (37.9 µg/100 ml) 20 minutes after injection, and 1280 nmol/l (46.4 µg/100 ml) 60 minutes after injection.

Posterior pituitary function—Urine osmolality after overnight fluid deprivation was 855 mOsm/kg.

Hypothalamus-pituitary-thyroid function—At 12 months his thyrotrophin concentrations in response to an intramuscular injection of 100 µg of thyroid-releasing hormone (TRH) were: 8 mU/l before injection, 9 mU/l after 30 minutes, and 9 mU/l after 60 minutes. At 1 year 8 months plasma thyrotrophin concentration was 4 mU/l before an intravenous injection of TRH 200 µg, 5 mU/l after 20 minutes, 4 mU/l after 30 minutes, and 4 mU/l after 240 minutes. Plasma growth hormone concentration (unstimulated) was 3 mU/l at 1 year 3 months and 6 mU/l at 1 year 8 months. Serum somatomedin value at 1 year was 96% of standard (normal for age 40-60%). Serum tri-iodothyronine concentration at 3 years was 2.7 nmol/l (1.8 ng/ml). Plasma prolactin was 250 mU/l. A skull radiograph at 10 months and an EMI scan at 2 years 5 months were normal.

Treatment—Thyroxine 0.0125 mg twice a day was given from the age of 12 months, and the dose was gradually increased to 0.025 mg twice a day. The radiological bone age was 6 to 9 months at 15 months, 1 year 8 months at 2 years, and 2 years 11 months at 3 years.

Comment

This patient had congenital hypothyroidism of pituitary origin as shown by low serum thyroxine values, delayed bone age, normal thyrotrophin,² and lack of thyrotrophin response to TRH. He was also very large. Unstimulated plasma growth hormone values were normal but the serum somatomedin concentration was similar to values found in "cerebral gigantism."³ The weight gain between 2 months and 10 months indicated considerable hyperphagia, which, together with the increased height, suggested abnormal hypothalamic function. There was therefore evidence of a defect in the pituitary gland and possibly in the hypothalamus. The benign course and the normal findings on computerised axial tomography make a tumour unlikely. The best defined disorder of diencephalic function in childhood is Russell's emaciation syndrome due to a tumour in the region of the third ventricle.⁴ Recently, however, the concept of diencephalic syndromes has been expanded to include various disorders⁵ which share features suggestive of diencephalic dysfunction, though in none has diencephalic pathology been shown conclusively. Soto's syndrome and the Wiedemann-Beckwith syndrome are associated with increased stature