'Windswept deformity' is commonly seen in children in Africa but residual deformities are not as often found in adults, suggesting that a considerable proportion must correct spontaneously. Because of this caution should be exercised when recommending the more drastic method of treatment for this condition.

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Pancreatitis during sodium valproate treatment

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SUMMARY A girl aged 1 year died of acute haemorrhagic pancreatitis while taking sodium valproate. Necropsy showed widespread vascular disease that may have contributed to the onset of pancreatitis. Previous reports of pancreatitis in children receiving valproic acid are reviewed and although the association is rare, a causal relation between pancreatitis and valproic acid seems to have been established.

Although some 1 000 000 patients have been treated with sodium valproate,¹ serious or fatal reactions are rare and most of the published reports describe liver damage.¹ Acute pancreatitis has, however, been reported in 7 children receiving valproic acid, 1 of whom died.²⁻⁶ We report on a child who died of pancreatitis while taking sodium valproate.

Case report

A normal baby girl aged 3 months suffered brief right sided clonic fits during a short febrile illness. The cause of the fits was not known but they stopped after treatment with phenobarbitone. Her subsequent development was normal and phenobarbitone was stopped at age 9 months. After another febrile illness at 1 year, the right sided fits recurred, together with a right hemiparesis and frequent vacant episodes. These seizures were completely controlled with 25 mg/kg per day of sodium valproate.

Ten days after starting treatment with sodium valproate the baby was readmitted to hospital with a 4 day history of drowsiness, anorexia, and vomiting. Abdominal distension and peripheral circulatory failure were found. Resuscitation failed

and she died shortly after admission. Investigations before death showed a serum valproate concentration of 5.9 mg/100 ml (therapeutic range 5-10 mg/100 ml) and a serum amylase of 3520 IU/l (normal range 100-400 IU/l).

Necropsy showed extensive haemorrhagic pancreatitis that had been present for several days. More recent areas of necrosis were found throughout the intestinal mucosa. In addition there was a widespread long standing non-inflammatory endothelial proliferation of many medium sized arteries. Obliteration of some of these arteries had resulted in areas of ischaemic necrosis of varying ages, particularly in the kidney and left cerebral hemisphere. The aetiology of the vascular lesions was unknown.

Discussion

The clinical details of the children in whom valproic acid has been associated with pancreatitis are shown in the Table. A relation between pancreatitis and valproic acid is established in the 4 patients who again developed pancreatitis on re-exposure to valproate. In 1 patient 4 separate courses of treatment resulted in 4 episodes of pancreatitis.⁶ Four patients were receiving other anticonvulsant agents. In 2 patients who were taking phenytoin the pancreatitis resolved when valproate was stopped, despite the continuation of phenytoin treatment. Re-exposure to valproate again resulted in pancreatitis in 1 of these patients.⁵ There does not seem to be any relation between pancreatitis and the patient's sex, type of fits, past or present medical history, or the dose of valproic acid. Pancreatitis occurred within the first 6 months of treatment in all the children. One patient died after laparotomy

Table Details of children with valproate induced pancreatitis

Source	Age (years)	Sex	Type of fits	Other diseases	Recurrence on re-exposure to valproate	Dose of valproate mg/kg	Duration of treatment to onset of pancreatitis	Other drugs given	Outcome
This report	1	F	Absence	Vascular disorder	Not tested	25	6 dys	Nil	Died
Camfield <i>et al</i> ²	11	F	Focal Absence	Meningitis at 2 yrs	Yes	45	3 mth	Nil	Survived
Camfield <i>et al</i> ²	3½	M	Akinetic	Premature; Perinatal asphyxia; Developmental retardation; Infantile spasms	Not tested	25 55	1½ mth 3 mth	Phenytoin	Survived
Batalden <i>et al</i> ³	8	M	Petit mal	Nil	Yes	30 30	5 wk 6 mth	Nil	Survived
Sasaki <i>et al</i> ⁴	11	F	Absence	Nephritis at 7 yrs	Yes	30 20	4 mth 6 mth	Phenobarbitone	Survived
Coulter and Allen ⁵	8	M	Myoclonic Akinetic	Nil	Yes	65 20	4 mth 4 mth	Phenytoin Clonazepam	Survived
Coulter and Allen ⁵	10	F	Not known	Not known	Not tested	30	3 mth	Nil	Died
Parker <i>et al</i> ⁶	16	M	Psychomotor	Nil	Yes	40 40 40 40	5 mth 5 mth 4 mth 6 wk	Carbamazepine Methsuximide Acetazolamide	Survived

had shown acute haemorrhagic pancreatitis, but no other details of this patient are given.⁵

Our patient developed pancreatitis at a younger age, after a shorter exposure to valproate than any other, and she was the only patient who was given sodium valproate rather than valproic acid. Necropsy confirmed acute haemorrhagic pancreatitis. The short time (6 days) between taking valproate and the onset of symptoms suggests a causal relation but the contribution, if any, of the vascular disease to the development of pancreatitis in this patient is not clear.

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Circumstances surrounding deaths from accidental poisoning 1974-80

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SUMMARY The deaths of 99 children under 5 years of age were studied through the coroners' records. Sixty nine deaths were caused by medicines—most of which were prescribed for mother, were recently dispensed, and currently in use. Household products accounted for 11 deaths, lead poisoning for 6, carbon monoxide for 5 and 'medical mishaps' for 8. Prevention lies in both education and more effective and selective use of child resistant containers.

Each year accidental poisoning in children accounts for roughly 24 000 hospital admissions,¹ but fortunately few deaths. Much effort had been put into reducing this number but until the selective introduction of child resistant closures (CRCs) on drug containers in 1976 there was little change.² The use of CRCs has now been extended by voluntary agreement to solid dose medicines dispensed by pharmacists but as there has been opposition from both the public and the medical and