and Van Wieringen.[11] Bias resulting from self-reporting of weight at age 20 must, however, be taken into account when considering the lower prevalence at 20, and when adjustment was made for this the decrease became smaller for the female subjects and was no longer apparent for the male subjects. After the age of 20 the prevalence of overweight rose again, and 64% of the overweight 26-year-old men and 57% of the women had not been overweight at 20.

Our findings suggest that even if treatment of overweight children during the first decade of life could prevent them from becoming overweight in young adult life the resulting reduction in the number of overweight adults would be less than 10%. From the point of view of preventing adult overweight the second decade appears to be more important because 28% of men and 45% of women who were overweight at 26 years had been overweight at the age of 14 years.

To our knowledge this is the first study based on national data on the development of overweight between the ages of 6 and 26 years. The results suggest that there is no optimal age during childhood for the prediction of overweight in adult life and that excessive weight gain may begin at any time during childhood, adolescence, or young adult life. No specific pattern could be identified for the progression of overweight once established. The study confirmed that the overweight child carries a much greater risk of remaining overweight than his contemporaries of normal weight carry of becoming overweight and is more likely to become severely overweight. Because at the ages of 20 and 26 weight and height were self-reported the prevalence of overweight at these ages may have been underestimated. Nevertheless, the data presented probably reflect the general trends in weight over the period covered by the survey.

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SHORT REPORTS

Hemiballismus treated with sodium valproate

Hemiballismus (proximal unilateral chorea) is a rare disorder characterised by involuntary proximal flinging movements of the limbs on one side of the body. Various treatments have been suggested. We present a patient who did not respond satisfactorily to conventional treatment but whose recovery was associated with the use of sodium valproate.

Case report

A 77-year-old man presented with the sudden onset of gross flailing movements of his right leg and, to a lesser extent, his right arm. On examination he was mentally alert but very distressed by the movements. There were distinct choreoathetotic movements of his right leg with less frequent uncontrolled movement of his right arm. His face was not affected. The movements were less apparent during sleep. Tendon reflexes were brisker in the right arm and leg than in the left, but both plantar responses were flexor. There was hypotonia on the right side. Blood pressure was 220/110 mm Hg supine. Right-sided hemiballismus was diagnosed, due to a presumed vascular lesion affecting the subthalamic nucleus (corpus Luysii) of the opposite side or its connections.

Tetrabenazine 25 mg thrice daily produced no improvement in the abnormal movements, and excessive sedation became a problem. After three days tetrabenazine was replaced with haloperidol 5 mg thrice daily increasing to 15 mg thrice daily. Once again excessive sedation resulted without control of the choreoathetosis. Six days later thiopropazate was substituted with only slight therapeutic effect, and after five days' treatment with this drug at 5 mg thrice daily, sodium valproate 200 mg thrice daily was added. Within 24 hours his clinical signs were distinctly improved and he could walk with support. After five days of combined treatment thiopropazate was stopped and the improvement was fully maintained. He was discharged taking sodium valproate, walking independently and with almost complete control of his hemiballismus, two weeks after this drug was started.

After three months sodium valproate was reduced to 200 mg twice daily; he remained independent and well, noticing occasional uncontrolled movements of his right leg when performing intricate movements with his hands —for example, when shaving.

Comment

Hemiballismus may occur as a result of any disease process affecting the area of the subthalamic nucleus. By far the most common lesion is cerebrovascular, especially in elderly patients. The movements tend to be severe with exhaustion of the patient,[1] damage to the skin, and bruising of deeper tissues owing to repeated trauma. Hemiballismus may also occur as an adverse reaction to stereotactic surgery for Parkinson's disease. Tetrabenazine, haloperidol, thiopropazate, reserpine, and stereotactic surgery have been used to treat this condition with varying degrees of success. We have been unable to find a reference to the use of sodium valproate, but a closely related compound, dipropylacetic acid, has been used unsuccessfully in the management of Huntington's chorea.[2] γ-Amino butyric acid is reduced in the basal ganglia of patients with Huntington's chorea, and dipropylacetic acid raises central nervous system γ-amino butyric acid in animals.[3]

There is disagreement about the natural history of hemiballismus.[1 4] The observed recovery in the case described may have been spontaneous, but we think that this is unlikely. The patient's hemiballismus had not responded to conventional drug treatment, which, moreover, led to excessive sedation. There was a rapid and dramatic improvement, however, with the introduction of sodium valproate, and the number of abnormal movements increased when the dose of sodium

valproate was reduced to 200 mg twice daily even after treatment for three months.

We suggest that sodium valproate should be considered among the drugs of first choice in the management of hemiballismus caused by a vascular lesion in view of its apparent efficacy in this patient and the low incidence of side effects associated with its use, especially the absence of unwanted sedation.

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Nocturnal wheezing in children: management with controlled-release aminophylline

Wheezing at night is an appreciable problem for asthmatic children and their parents. The short duration of action of most bronchodilator drugs and the physiological early-morning dip in pulmonary function exacerbate nocturnal symptoms. Although oral theophylline is an effective bronchodilator,[1] its half life is short and variable,[2] so that therapeutic values cannot be maintained while a child sleeps. Use of controlled-release aminophylline in children results in satisfactory serum[3] and salivary[4] theophylline concentrations eight hours after ingestion, coupled with improvement in respiratory function.[5] Controlled-release aminophylline should be of benefit in the management of nocturnal symptoms; a double-blind trial was conducted to determine its efficacy in asthmatic children.

Patients and methods

We studied 25 asthmatic children aged between 5·2 and 15·3 years (mean age 9·3 years). They were chosen because nocturnal symptoms were a major clinical problem. Twenty-two children completed the study satisfactorily, the records of the three others being inadequate. The dosage of aminophylline (mean 11·3 mg/kg) was determined individually for each child with the aid of salivary theophylline assays.[4]

The study lasted two months: patients were randomly allocated to placebo or controlled-release aminophylline and switched to the alternative preparation after a month. Tablets were taken only at bedtime. Peak flow was recorded on waking (mini Wright peak flow meter). Regular and extra medication was recorded and the early-morning peak flow disregarded if other bronchodilator treatment had been used within six hours. Night symptoms were graded (0-5) and recorded by children or parents, or both, according to a diary card routinely used in our clinic.

Results and comment

The table summarises the results. Data for peak flow and night score were analysed separately by Mann-Whitney tests and showed a significant improvement in morning peak flow in most patients. The small range of possible responses in the night score resulted in most responses being "no change." When the morning peak flow results were combined with the parents or the child's assessment of the night then 16 children improved and six showed no change with controlled-release aminophylline compared with placebo ($p<0·05$). A non-parametric two-way analysis of variance showed that there was a highly significant increase in the number of good nights when controlled-release aminophylline was being used.

We conclude that controlled-release aminophylline is a useful preparation in asthmatic children with nocturnal symptoms; no side effects attributable to it were noted in this study.

Change in peak flow and night score during treatment with controlled-release aminophylline compared with placebo. (Figures are numbers of patients)

	Worse		No change	Improved			
	$p<0·01$	$p<0·05$		$p<0·1$	$p<0·05$	$p<0·01$	$p<0·001$
Peak flow	2	2	4	2	4	1	7
Night score		1	12		5	2	2

We thank the children and their parents for their co-operation; Sister Gardner, Mr Smith, and Mr Pugsley for help and advice; Gill Smith for typing the manuscript; and Napp Laboratories for supplying controlled-release aminophylline (Phyllocontin Continus tablets) and placebo.

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Fatal poisoning with methyl isothiocyanate

Methyl isothiocyanate is substituted for phenyl isothiocyanate in the Edman degradation test to determine the amino-acid sequences of peptides and proteins.[1] The compound donates a cyanide moiety and would therefore be potentially lethal if ingested. In animals the LD_{50} is reportedly 97 mg/kg,[2] which produces corneal, liver, and kidney damage, but little is known about its toxicity in man. We describe a suicide with the compound.

Case report

A 23-year-old chemistry student was brought to the emergency department of Nehru Hospital within 20 minutes after intentionally drinking water containing 50 g methyl isothiocynate (Mann Laboratories Inc, NY). Immediately after ingestion she had noticed severe retrosternal burning and epigastric pain and begun to vomit repeatedly. A few minutes later she began generalised tonic and clonic seizures and became unconscious.

She was admitted deeply comatose and with pulse 98/min and blood pressure 90/60 mm Hg. Chest, heart, and abdomen were normal. The pupils were slightly dilated but equal in size and reacted sluggishly to light. There was complete loss of all reflex and motor activity including oculocephalic reflex. Gastric lavage was performed with sodium thiosulphate, and sodium nitrite and sodium thiosulphate were given intravenously in doses for cyanide intoxication. Seizures were controlled with intravenous diazepam, and she was put on respirator. Peritoneal dialysis was instituted about one hour after admission. She continued to deteriorate with a further fall in blood pressure, however, and died about eight hours after admission. Necropsy a few hours later showed extensive mucosal necrosis of oesophagus, stomach, and proximal part of the duodenum (fig). Other organs showed only evidence of shock.

Comment

The toxic component of methyl isothiocyanate is probably thiocyanate, as our patient's symptoms were closely similar to those seen in acute poisoning with sodium, potassium, and ammonium thiocyanate.[3] Thiocyanates are reduced partly to cyanide in tissues and