

## Short reports

### Growth retardation in asthma: role of calorie deficiency

J J COGSWELL AND M M EL-BISHTI

Poole General Hospital, Dorset, and Department of Paediatrics, Guy's Hospital

**SUMMARY** Height and weight were recorded and nutrient intake assessed during a 1-year period in 8 short asthmatic children who subsequently received energy supplements for a further year. None received systemic steroids. Total energy intakes in 6 of the children were reduced but no relationship between intakes and linear growth was found. Linear growth performance did not improve despite the fact that energy intake was normalised by supplementation, although some children showed an accelerated weight gain. We suggest that calorie supplementation is not effective for promoting optimal linear growth in short asthmatic children.

Children with chronic asthma may show growth retardation<sup>1,2</sup> and this effect is accentuated by prolonged systemic administration of corticosteroid drugs.<sup>3</sup> Severe growth retardation is restricted to a small group of asthmatic children with severe chest deformity and persistent airways obstruction.<sup>4</sup> The causes of growth inhibition in children with chronic asthma not receiving steroids are unknown. Chronic hypoxaemia and poor appetite have been suggested<sup>2</sup> but the role of calorie deficiency has not been studied.

The purpose of this study was to examine whether growth velocity in a group of asthmatic children was related to nutrient intake, and whether an increase of energy intake by dietary supplements would affect growth performance.

#### Patients and methods

Eight prepubertal asthmatic children were studied. There were 7 boys who were on or below the 3rd centile for height and one girl whose height was just above the 3rd centile.

The clinical severity of the children's asthma was variable: 6 had severe asthma, with more than one wheezy episode weekly during the preceding year. All 6 had rhonchi and reduced peak expiratory flow rates ( $-2$  SDs below mean for height) between exacerbations, and all had missed some schooling

despite prophylaxis with inhaled beclomethasone. The remaining 2 children had mild intermittent asthma with fewer than one wheezy episode a week in the preceding year, normal respiratory function between attacks, and no missed schooling. None of the 8 children received oral corticosteroid drugs either before or during the study.

The children were observed for one year. Height (using a Harpenden stadiometer) and weight (using a beam balance) were measured every 3 months by the same observer. Height age was calculated from the data of Tanner *et al.*<sup>5,6</sup> Height velocity in cm/year was expressed as number of standard deviations (SDs) from the mean in relation to chronological age.

Dietary intakes of total energy and protein were assessed from prospective 3-day weighed records performed by the parents at home every 3 months. No supervision of the diet in the home was attempted. Nutrient intakes were calculated from the diet records using standard tables<sup>7</sup> and expressed as percentages of recommended daily allowances (% RDA) for height age of the child.<sup>8</sup>

For a further year, the children's diet was supplemented with varying amounts of energy supplement based on a glucose polymer.\* No fixed amount was given but parents recorded the quantity taken. The dietary intakes during the second period were recorded and analysed as during the first.

#### Results

Clinical data and growth data are shown in Table 1. Height velocity was reduced in 6 of the 8 asthmatic patients in the first period, and did not alter significantly ( $t = 1.049$ ; paired  $t$  test) in the second period during energy supplementation.

Weight velocity increased in 4 of the 8 children after supplementation but this did not reach statistical significance ( $t = 1.162$ ; paired  $t$  test).

Mean nutrient intakes of total energy and protein in absolute amounts, and as percentages of recommended daily allowance (%RDA) for children of the same height during first and second periods are

\*Caloreen. Roussel Laboratories Ltd, London.

Table 1 Growth data for patients during the first period compared with the energy supplementation period

At start of study			First period				Second period (energy supplemented)	
Case	Age (years)	Sex	Height (cm)	Weight (kg)	Height velocity (SD score)	Weight velocity (kg/year)	Height velocity (SD score)	Weight velocity (kg/year)
1	7.1	M	109	15	+0.58	2.1	+0.79	0.7
2	9.4	M	120	22.5	-0.52	1.6	+1.01	4.0
3	3.6	F	91.4	12.4	-2.48	1.0	-0.95	1.6
4	6.3	M	107	16.9	-0.54	1.1	-2.56	0.9
5	12.5	M	136	26	-1.25	2.3	-2.11	4.9
6	14.5	M	103	15.6	+0.93	1.5	-1.3	2.1
7	9.1	M	123	21.4	-0.79	1.9	-1.5	1.4
8	9	M	121	22.3	-0.52	1.9	-2.52	2.3
Mean $\pm$ 1 SD	8.9 $\pm$ 3.4	—	113.8 $\pm$ 13.9	19.01 $\pm$ 4.7	-0.57 $\pm$ 1.05	1.67 $\pm$ 0.46	-1.14 $\pm$ 1.38	2.24 $\pm$ 1.4

Table 2 Mean nutrient intake of patients during the first period compared with the energy supplementation period

First period					Second period (energy supplemented)				
Case	Total energy		Protein		Case	Total energy		Protein	
	Kcal/day	% RDA	g/day	% RDA		Kcal/day	% RDA	g/day	% RDA
1	1359	78.1	35.9	83.4	1619	93	34	79.1	
2	1240	62.6	38	77.5	1793	90.5	52.4	106.9	
3	1541	102	36.4	98.3	1438	95.8	33.3	90	
4	1656	83	49.3	114.6	1873	107.6	53.8	125	
5	1285	56	45.9	80.5	2461	107.9	73.9	129.6	
6	1694	108.5	58.3	135.5	1974	113.4	61.5	143	
7	1587	80	63.5	129.5	1764	89.1	52.3	106.7	
8	1683	85	45.2	92.2	1897	95.8	46	93.8	
Mean $\pm$ 1 SD	1505 $\pm$ 184	81.9 $\pm$ 17.6	46.5 $\pm$ 10.2	101.4 $\pm$ 22.5	1852 $\pm$ 298.8	99.1 $\pm$ 9.1	50.9 $\pm$ 13.3	109.3 $\pm$ 21.8	

RDA = recommended daily allowance.

shown in Table 2. Before energy supplementation 6 of the 8 children had reduced total energy intake (mean 82% RDA) but protein intake was normal (mean 101% RDA). After energy supplementation mean energy intake increased significantly compared with the first period ( $t = 2.75$   $P < 0.05$ ; paired  $t$  test). No significant difference was found in protein intake between the two study periods ( $t = 0.978$ ; paired  $t$  test).

No statistically significant correlation was found between height velocity and either energy or protein intake during the first or second period. Similarly, there was no significant relationship between weight velocity and nutrient intakes during either period.

## Discussion

The causes of poor growth in asthmatic children are unknown. Possibilities include hypoxia,<sup>2</sup> and nutritional inadequacy. Since no previous studies on the nutritional status of asthmatic children had been reported, this study was designed to assess the nu-

tritional intake in a group of short asthmatic children. A second objective was to examine the relationship between energy intake and growth performance in asthmatic children.

Our results have shown that the mean energy intake in half of the patients was below that recommended for height age. Despite this reduction in energy intake no clear association could be established between growth velocity and energy intake. We were able to increase with supplementation the mean energy intake from 82% RDA in the first period to 99% RDA in the second period. Nevertheless, this energy supplementation had no effect on linear growth velocity, but did result in accelerated weight gain in 4 of the 8 asthmatic children.

Although the number of patients in this study was small, the evidence suggests that stunting of growth in asthmatic children not taking systemic corticosteroids is unlikely to be due to energy deficiency. Calorie supplementation is not an effective way to promote optimal linear growth in short asthmatic children.

## References

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Correspondence to Dr J J Cogswell, Department of Paediatrics, Poole General Hospital, Longfleet Road, Poole, Dorset BH15 2JB.

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## Effect of a rocking bed on apnoea of prematurity

S J TUCK, P MONIN, C DUVIVIER, T MAY, AND P VERT

*Service de Médecine Néonatale, Maternité Universitaire, Nancy, France*

**SUMMARY** We describe a rocking bed for use in incubators. Its effect was studied in 12 preterm infants with idiopathic apnoea, using each as his own control. All but one had less apnoea when the bed was rocking than when it was still. Apnoea associated with a significant fall in transcutaneous  $PO_2$  was less frequent, and fewer interventions were needed to terminate apnoeic attacks.

Various forms of repeated sensory stimulation—such as tactile stimulation or the use of irregularly oscillating waterbeds—can reduce apnoea in preterm infants. This approach to the management of recurrent apnoea avoids the potential hazards of treatment with continuous positive airways pressure (CPAP) or methylxanthines.

We have constructed a bed which imparts a regular cephalo-caudal rocking movement, not exceeding  $3^\circ$  in either direction, in the hope of preventing apnoeic attacks by vestibular stimulation.

### The apparatus

The rocking bed consists of a rectangular aluminium tray, mounted on pivots near the midpoints of its 2 longer sides. The regular rocking movement is obtained by alternate inflation and passive deflation of a small bellows beneath one end of the tray. The bellows is connected by a tube to a control unit

beside the incubator. This contains a reservoir of compressed air, maintained by an electric pump, and a valve through which the bellows are automatically connected in turn to the reservoir, for inflation, and to the atmosphere, for deflation. Variable resistances are included in both parts of the circuit, so that inflation time and deflation time, as well as the cycle rate, can be altered separately.

### Patients

We studied 12 infants with recurrent apnoea for which no neurological, metabolic, or infective cause could be found. All had had at least 3 episodes of apnoea ( $>12$  seconds) with bradycardia ( $<100$ /minute) in the preceding 24 hours, or 2 in the preceding 6 hours. Gestation ranged from 26 to 32 (mean 29.5) weeks, birthweight from 800 to 1700 (mean 1210) g, and postnatal age from 2 to 45 (mean 14) days. In no case did the sum of gestational and postnatal age exceed 34 weeks.

Four infants were receiving added oxygen ( $F_{iO_2} < 0.34$ ). Only one had received theophylline, 5 days previously.

### Methods

Each infant was studied for two consecutive equal periods. The bed was rocking during one, and was still during the other, the sequence being randomly