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The effects of high-energy feeding on energy balance and growth in infants with congenital heart disease and failure to thrive

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Failure to thrive (FTT) in infants with congenital heart disease (CHD) can be attributed to their low energy intakes and high resting energy expenditures. Energy intake, energy expenditure and growth were studied in infants with CHD on normal formula feeds and then on feeds supplemented with glucose polymer to see whether supplementation improved energy retention and growth. Mean gross energy intakes increased by 31·7% on high-energy feeding and mean weight gain improved from 1·3 g/kg per d on control to 5·8 g/kg per d on high-energy feeding. Resting oxygen consumption ($\dot{V}_{O_2}$ ml/kg per min) was not significantly different on the two feeding regimens, although respiratory quotient rose on high-energy feeding reflecting the increased carbohydrate intake. Estimated energy costs of growth on high-energy feeding fell within the previously described range for normal infants. It is recommended that infants with CHD known to be associated with FTT are fed on high-energy diets from the time of diagnosis in order to optimize growth.

Congenital heart disease: Energy balance in infancy: Failure to thrive: High-energy feeding

Children with congenital heart disease (CHD) commonly fail to thrive (Mehziri & Drash, 1962; Feldt *et al.* 1969; Menahem, 1972; Menon & Poskitt, 1985). They present in infancy as chronic malnutrition with reduced subcutaneous fat, wasted muscles and linear growth retardation. Histopathological studies show liver changes suggestive of malnutrition in infants who die (Naeye, 1965). Failure to thrive (FTT) usually results from the combination of inadequate nutrient intakes, malabsorption, failure to utilize food for growth in the presence of gross anoxia, and increased nutrient requirements. Several studies have shown that FTT associated with high resting metabolic rates is particularly likely in infants with pulmonary hypertension and congestive cardiac failure (Krauss & Auld, 1975; Baum *et al.* 1980; Menon & Poskitt, 1985). These infants may consume energy intakes within recommended requirements for weight but consistently less than requirements for both expected weight-for-age and expected needs for catch-up growth (Lees *et al.* 1965; Menon & Poskitt, 1985).

Various methods have been used to increase the energy intakes of infants with CHD and to assess whether growth improves as a result. Nocturnal or continuous feeding by gavage are effective short-term methods (Yahav *et al.* 1985; Bougle *et al.* 1986). However, it is neither desirable nor always practical to keep young infants with CHD and FTT in hospital for prolonged periods to increase weight, so feeding methods need to be safe, simple and able to be practised at home. High-energy feeding should also be acceptable to very young infants since the prevention or limitation of FTT by early management is preferable to treatment once FTT has developed. If brain growth and function are at risk from poor nutrition in CHD (Naeye, 1965), the greatest risk is likely to be in early life when brain cell multiplication and development are most rapid (Dobbing, 1981). Prolonged periods of malnutrition may inhibit both brain growth and infants' opportunities to learn from the

environment, thus increasing the probability of developmental delay. Ability to withstand the trauma of surgery should be greatest in infants with least nutritional stress. Avoiding, or at least limiting, FTT in infancy is part of the management of infants with CHD.

We have studied the effect of increasing the energy content of feeds to see how this improves energy retention and weight gain in CHD infants. It seemed possible that where there existed adverse factors for growth such as anoxia and poor peripheral perfusion, energy supplementation might be dissipated as increased metabolism rather than as tissue deposition in growth. We have followed not only energy intakes in these infants but energy expenditures as well.

METHODS

Infants admitted to the Royal Liverpool Childrens Hospital (City Branch), with FTT and CHD were studied when their cardiac condition had been stabilized and changes in medical management or surgery, or both, were not anticipated. FTT was defined as failure to gain weight at the expected rate as indicated by growth standards (Vaughan & Litt, 1987). Most children also had weights below the third centile weight for age at entry to the study.

Infants were studied for two balance periods of 3 d by methods similar to those of Menon & Poskitt (1985). During the balance periods, energy intakes were estimated from weighed food intakes and collection and weighing of all food losses as posset, vomit and spillage. Urine and stools were collected for analysis of energy excreted or not absorbed. Energy expenditure was measured daily during each balance period by indirect calorimetry. During the first period (control), infants continued to be given their usual infant formula (and solids if introduced). Energy density of the feeds was then increased over 3 d to at least 125% standard formula by the addition of glucose polymer (Caloreen; Roussel Laboratories, Uxbridge, Middx). After a further day for stabilization at the new energy level, the energy balance (high-energy) studies were repeated. Fig. 1 outlines the pattern of balance periods in relation to energy density of feeds.

Stools were separated from urine by using preweighed nappy liners positioned into tightly fitting disposable nappies. The beginning and end of each stool collection were marked by feeding sterile carmine marker (BDH, Poole, Dorset) with the first feed of the balance period and the first feed after completion of the balance period. The time between the first appearance of these markers represented the stool collection period. Gross stool output was calculated from reweighing the soiled liners. All pads, urine soaked nappies and soiled liners were stored in labelled plastic bags at $-20°$ until analysis.

Energy content of feeds, stools, urine, posset and vomit

One extra formula feed was prepared for each infant's 24 h batch of feeds. Sample weaning solids were reconstituted to the fed composition using the sample formula. Representative stool samples were achieved by scraping stools from the individual liners. Soiled absorbent pads and urine-soaked nappies were macerated with a known volume of water and allowed to stand for 24 h to equilibrate, and then crudely filtered. Portions of the filtrate were finely filtered under vacuum. Portions of feeds, stools and combined urine–posset–vomit samples were frozen at $-20°$ and freeze-dried, and the energy contents determined by ballistic bomb calorimetry (Miller & Payne, 1959).

Respiratory gas exchange

Infants were studied on two occasions in each 24 h during the balance periods: for 1 h before a feed, and for 1·5 h after a feed. Infants lay on a mattress with a Perspex chamber covering head and thorax. Room air was drawn through a loosely fitting plastic hood using constant negative pressure and into the Perspex chamber (Fig. 2). Flow-rates were at least

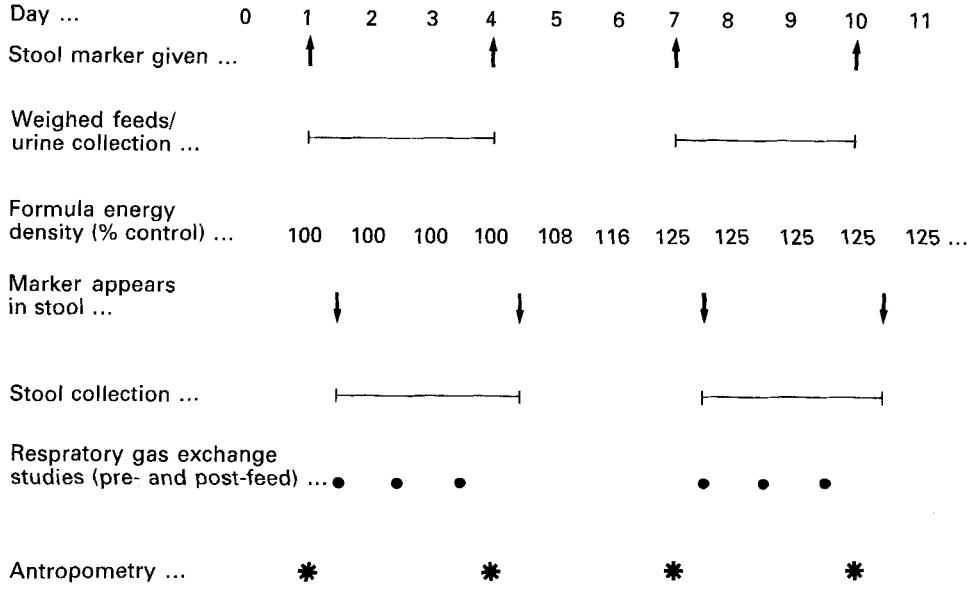

Fig. 1. Plan of energy balance periods on standard and high-energy feeds.

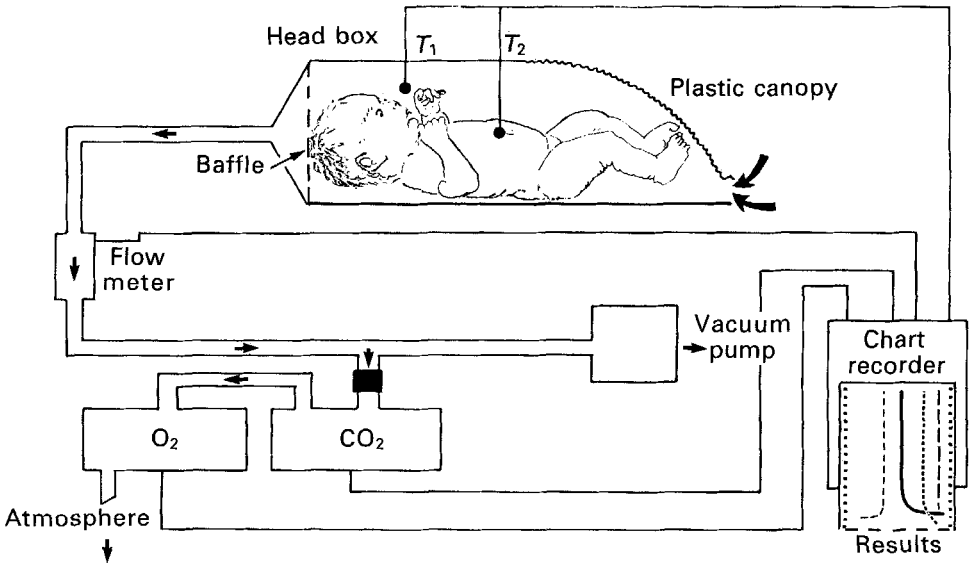

Fig. 2. Equipment and configuration used in the measurement of infant oxygen consumption. Air is drawn under a plastic canopy and into the system by a vacuum pump. Flow-rate is measured, and a sample of the gas analysed for carbon dioxide and O_2 fraction. All values, including box and abdominal temperature (T_1 and T_2) are directed to the chart recorder for later analysis.

ten times the estimated minute volumes of the infants (Lister *et al.* 1974), ensuring an adequate oxygen supply and rapid removal of carbon dioxide. The exit gases were drawn through a mass flow meter (Chell Instruments; Amersham, Bucks) which provided an accurate measure of flow-rate corrected to standard temperature and pressure. Some of the mixed exit gases were dried by passing through an excess of calcium chloride before

estimation of gas fractions. Volumetric changes as a consequence of drying were likely to be small and were ignored. Respiratory gases were measured by infra-red CO_2 analyser (PK Morgan Ltd, Chatham, Kent), and serially connected paramagnetic O_2 analyser (Servomex, Crowborough), both previously calibrated with room air and test gases spanning the expected range. Chamber air temperature and infants' abdominal skin temperature were monitored continuously; temperatures did not vary by more than 0·5° throughout the study periods.

Gas exchange studies included the full range of infant activity, scored as 0 sleeping, 1 awake and quiet, 2 awake and active and 3 crying. These findings were recorded together with exit gas fractions, temperatures and flow-rates on an on-line multichannel chart recorder (Chessell Ltd, Worthing, Sussex). Mean O_2 consumption ($\dot{V}_{O_2}$) and CO_2 production ($\dot{V}_{CO_2}$) for each of the activity levels were calculated by planimetry of the area beneath the curves of the relevant gas fractions and flow-rates.

Differences in diffusion coefficient at the respiratory membrane were accounted for by application of Haldane's correction (Cotes, 1968). Respiratory quotient (RQ) was calculated as mean $\dot{V}_{CO_2}$ divided by mean $\dot{V}_{O_2}$ for each activity level. Energy expenditures at each activity level were calculated by multiplication of the relevant $\dot{V}_{O_2}$ by the appropriate energy equivalent for the observed RQ (Lentner, 1981). Total daily energy expenditure (TDEE) was estimated by weighting energy expenditure for each activity as a proportion of total study time and extrapolating results to 24 h.

Anthropometry

Nude weight was recorded daily throughout the balance periods. Weight changes on days in hospital preceding the control balance, and days in hospital following the high-energy balance (when high-energy feeding continued) were also recorded. Supine length, occipito-frontal circumference (OFC), and biceps, triceps, suprailiac and subscapular skinfolds were measured (Cameron, 1984) at the beginning and end of each balance period.

Percentage body mass index (% BMI; Cole, 1979) was calculated as weight/(length)^2 divided by weight at the 50th centile-for-age/(length at the 50th centile-for-age)$^2 \times 100$ %. Expected weight-for-length, weight-for-age and length-for-age were also calculated. Standards from the National Center for Health Statistics were used throughout (Vaughan & Litt, 1987). Skinfolds are expressed as the logarithm of the summed four skinfolds measured (log TSFT).

Statistical analysis

All data were initially tested for normality of distribution. Non-Gaussian data were transformed using logarithmic corrections. The matched pairs Student's t test was used where appropriate. Data untransformable to Normal distributions were compared using the Mann-Whitney U test for ranked scores. Regression analysis was performed using the method of least squares.

RESULTS

Infants

Table 1 lists diagnoses in the fourteen CHD infants (three male, eleven female). Mean age of the group was 0·23 (SE 0·10) years.

Energy intake and weight gain

Except for one infant receiving a soya-bean-based formula, all infants were fed on standard cow's-milk-based formulas. Only two infants received 'solid' foods in addition to formula.

Table 2 shows gross energy intakes and energy losses in stools, urine, posset and vomit for all infants during the control and high-energy studies. On high-energy supplementation,

Table 1. *Age, sex and diagnosis of infants studied*

Patient no.	Age (years)	Sex	Diagnosis
1	0·23	Female	VSD, PHT, CCF
2	0·24	Female	VSD, PHT, CCF
3	0·08	Female	VSD
4	0·33	Male	VSD, PHT, Holt Oram syndrome
5	0·27	Female	AVSD, PHT, Downs syndrome
6	0·19	Female	AVSD, PHT, CCF
7	0·39	Female	AVSD, PHT
8	0·13	Male	VSD, PHT
9	0·24	Female	VSD, CCF
10	0·40	Female	VSD, PHT
11	0·29	Female	Tetralogy of Fallot, CCF
12	0·11	Female	VSD, PHT, Downs syndrome
13	0·09	Male	Truncus arteriosus
14	0·19	Female	AVSD, PHT, Downs syndrome

VSD, ventricular septal defect; PHT, pulmonary hypertension; CCF, congestive cardiac failure; AVSD, atrioventricular septal defect.

mean gross energy intake increased by 31·7% ($P \leqslant 0{\cdot}001$) from 88·5 (SE 4·5)% of recommended daily allowance (RDA) (Department of Health and Social Security, 1979) to 116·8 (SE 5·5)% of RDA. Energy intakes expressed as percentages of RDA correlated significantly with weight gains over the high-energy balance periods (Fig. 3). Energy losses in stools and combined urine–posset–vomit (Table 2) were not significantly different between control and high-energy studies. Metabolizable energy intakes (MEI) expressed both in absolute terms and as percentages of gross energy fed improved on high-energy feeds: from 362 (SE 23) kJ/kg per d to 505 (SE 31) kJ/kg per d ($P \leqslant 0{\cdot}001$), and from 83 (SE 4)% to 87 (SE 3)% ($P \leqslant 0{\cdot}01$), respectively.

During the control balance, MEI was not significantly correlated with weight gain (r 0·28). During the high-energy balance, a highly significant relationship (r 0·83) was demonstrated (Fig. 4). From this, the energy requirement for zero weight gain (maintenance energy) could be calculated at 327 kJ/kg per d and each g weight gain was associated with the retention of 31 kJ energy.

Growth

Table 3 shows mean weight, length, OFC, log TSFT and % BMI for each infant during the two balance periods. Mean expected weight-for-age was 61·3 (SE 2·2)%, height-for-age 91·8 (SE 1·3)% and weight-for-height 75·1 (SE 1·9)% at the time of the control study. Mean weight gains were increased significantly ($P \leqslant 0{\cdot}001$) from 1·3 (SE 0·7) g/kg per d (control) to 5·8 (SE 1·2) g/kg per d (high-energy). Weight-for-age, height-for-age and weight-for-height increased (%) with high-energy feeding: 62·5 (SE 2·3), 91·9 (SE 2·3) and 77·6 (SE 2·1) respectively but only differences in weight-for-height were significant ($P \leqslant 0{\cdot}001$). Outside the periods of intensive study, mean daily weight gain to mid control balance period from a median of 8 d (range 1–15) before the balance was −0·6 (SE 0·7) g/kg per d, and mid high-energy balance period to a median of 8 d (range 1–56) after the balance was 4·8 (SE 0·6) g/kg per d ($P \leqslant 0{\cdot}001$).

Mean % BMI also increased between control and high-energy studies (Table 3) but the increase was not statistically significant. Mean OFC between the balance periods increased ($P \leqslant 0{\cdot}001$) and mean change in log TSFT was increased following supplementation ($P \leqslant 0{\cdot}05$).

Table 2. *The 24 h energy balance results in infants* with congenital heart disease and failure to thrive given normal feeds or feeds with increased energy level*

Patient no.	Gross energy intake (kJ/kg)	Stool energy losses (kJ/kg)	u/p/v energy losses (kJ/kg)
Control			
1	469	29	16
2	543	35	19
3	432	26	17
4	431	31	11
5	368	62	19
6	452	47	14
7	340	28	12
8	346	30	15
9	634	82	75
10	302	31	7
11	488	52	29
12	431	241	11
13	462	34	13
14	466	104	7
Mean	440	60	19
SE	23	15	5
High-energy			
1	546	26	20
2	572	38	25
3	587	19	26
4	496	35	15
5	539	52	28
6	642	40	21
7	441	56	6
8	796	52	9
9	716	69	17
10	379	17	7
11	567	70	13
12	603	277	10
13	672	42	7
14	562	42	6
Mean	580	60	15
SE	29	17	2
Statistical significance of difference	$P \leqslant 0{\cdot}001$		

u/p/v, Urine, posset and vomit.
* For details of subjects, see Table 1.

Respiratory gas exchange

Resting heart rate, resting respiratory rate and the proportion of each balance period spent sleeping during the gas-exchange studies were not significantly different between the control and high-energy balance periods.

Table 4 lists sleeping $\dot{V}_{O_2}$ and RQ for each infant during the control and high-energy balance periods. Mean $\dot{V}_{O_2}$ whilst asleep and during activity (control 12·1 (SE 0·5) ml/kg per min, high-energy 12·0 (SE 0·5) ml/kg per min) did not change significantly between control and high-energy studies. Mean sleeping $\dot{V}_{O_2}$ tended to be higher in those infants with documented pulmonary hypertension.

Mean sleeping RQ rose significantly ($P \leqslant 0{\cdot}001$) following carbohydrate supplemen-

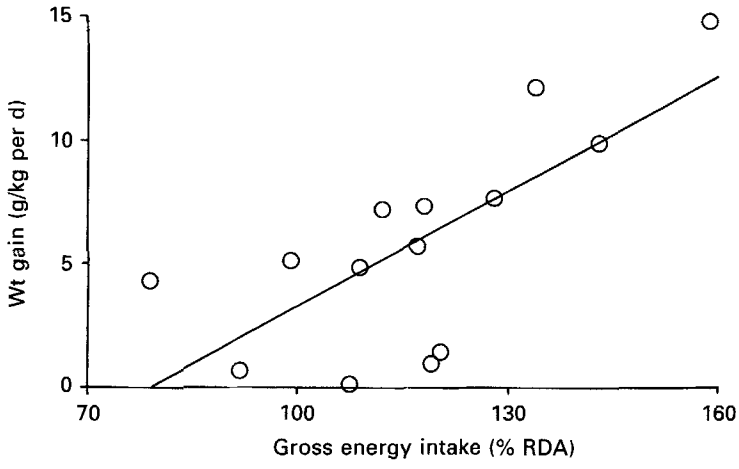

Fig. 3. Regression relationship of gross energy intake (expressed as percentage recommended daily allowance (RDA); Department of Health and Social Security, 1979), against weight gain for the high-energy balance period in infants with congenital heart disease and failure to thrive. For details of experimental procedures, see p. 132. $y = 0{\cdot}1582x + 12{\cdot}6691$, r 0·72; SE of estimate 3·22; $P \leqslant 0{\cdot}005$.

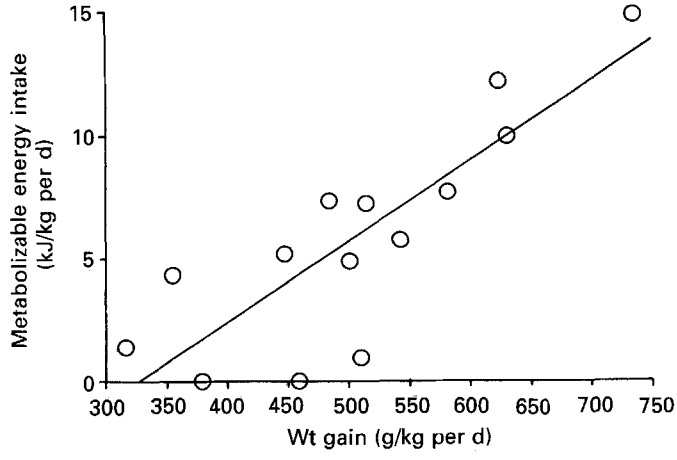

Fig. 4. Regression relationship of weight gain against metabolizable energy intake for the high energy balance period in infants with congenital heart disease and failure to thrive. For details of experimental procedures, see p. 132

$y = 0{\cdot}0325x + 10{\cdot}6323$, r 0·83; SE of estimate 2·58; $P \leqslant 0{\cdot}001$.

tation. Mean RQ during periods of activity was similarly affected (control 0·83 (SE 0·01), high-energy 0·90 (SE 0·02), $P \leqslant 0{\cdot}001$).

Energy available for growth and activity

Table 5 shows MEI, TDEE, 'spare' energy and weight gain for each infant during control and high-energy balances. Energy expenditures showed no significant differences between the balance periods. 'Spare' energy available for growth, calculated as MEI minus that energy estimated as utilized in basal and activity energy expenditure from the gas-exchange studies, was significantly increased by energy supplementation ($P \leqslant 0{\cdot}001$).

Table 3. *Weight, length, occipito-frontal circumference, logarithm of the sum of four skinfolds measured and percentage body mass index during control and high-energy balance periods for infants with congenital heart disease and failure to thrive*

Patient no.	Wt (kg)	Length (mm)	OFC (mm)	log TSFT (log.mm)	% BMI (%)
Control					
1	3·86	563	385	1·27	81·0
2	3·58	566	376	1·23	74·0
3	2·78	497	345	1·34	81·6
4	3·68	623	392	1·24	57·7
5	3·02	557	352	1·07	63·8
6	3·29	542	364	1·33	75·6
7	4·12	594	380	1·34	75·2
8	2·70	506	328	1·22	70·6
9	2·45	484	326	1·40	69·1
10	3·47	569	380	1·15	66·3
11	3·06	526	351	1·34	71·8
12	2·27	482	330	1·11	78·0
13	3·01	510	350	1·24	78·8
14	3·20	560	362	1·13	73·2
Mean	3·18	541	358·6	1·24	72·6
SE	0·14	11	5·9	0·03	1·8
High-energy					
1	4·13	569	385	1·32	84·0
2	3·55	566	376	1·28	72·6
3	2·93	503	345	1·32	87·6
4	3·91	626	397	1·25	60·5
5	3·13	557	353	1·09	65·6
6	3·48	547	368	1·32	77·8
7	4·34	594	380	1·33	76·1
8	2·81	516	334	1·27	69·4
9	2·70	492	335	1·43	73·0
10	3·49	573	382	1·14	65·6
11	3·18	531	354	1·33	73·0
12	2·38	491	332	1·10	69·2
13	3·29	517	354	1·27	83·9
14	3·47	571	366	1·20	76·5
Mean	3·34	547	361·5	1·26	73·9
SE	0·15	11	5·6	0·03	2·1
			$P \leqslant 0{\cdot}001$	$P \leqslant 0{\cdot}05$	

OFC, occipito-frontal circumference; log TSFT, logarithm of the sum of four skinfolds measured; % BMI, percentage body mass index (see p. 134).

DISCUSSION

The present study confirms what several studies have shown previously, namely that some infants with CHD have high resting metabolic requirements as well as low energy intakes (Lees *et al.* 1965; Stocker *et al.* 1972; Huse *et al.* 1975; Menon & Poskitt, 1985). FTT is thus explained as a consequence of deficiency between energy retention and energy requirements. Our study shows that it is practical to increase energy retention by increasing the energy density of feeds with glucose polymer. Energy supplementation in this way does not lead to increased nutrient losses through urine, posset and vomit or malabsorption. More significantly, weight gain improves, although even with MEI at 140% of control levels, few children achieved the 10 g/kg per d weight gain desirable for catch-up growth in this age group (Ashworth & Millward, 1986). Changes in length in these children were small and

Table 4. *Sleeping oxygen consumption* ($\dot{V}_{O_2}$; *ml/kg per min*) *and respiratory quotient* (*RQ*) *for the control and high-energy balance periods for infants with congenital heart disease and failure to thrive*

Patient no.	$\dot{V}_{O_2}$ (ml/kg per min)	RQ
Control		
1*	9·58	0·90
2*	10·96	0·78
3	8·74	0·88
4*	11·05	0·73
5*	9·83	0·73
6*	12·55	0·78
7*	11·70	0·71
8*	9·34	0·71
9	10·99	0·91
10*	14·76	0·71
11	10·68	0·77
12*	8·69	0·89
13	8·71	0·82
14*	9·79	0·78
Mean	10·53	0·79
SE	0·45	0·02
High-energy		
1*	9·75	0·88
2*	10·26	0·90
3	9·87	0·81
4*	10·36	0·88
5*	12·02	0·83
6*	12·85	0·91
7*	13·07	0·83
8*	8·83	0·90
9	11·54	0·97
10*	11·53	0·77
11	9·67	1·01
12*	9·89	0·91
13	8·66	0·77
14*	8·13	0·96
Mean	10·46	0·88
SE	0·41	0·02
		$P \leqslant 0·001$

* Pulmonary hypertension.

due to difficulties in making precise measurements in this age group, we cannot comment on the effect of high-energy feeding on linear growth.

Continuous nasogastric infusion of energy at 1·4–1·8 times measured metabolic rate has demonstrated a good anabolic response without change in $\dot{V}_{O_2}$ in adults with congestive heart failure (Heymfield & Casper, 1989). Similarly, high-energy feeding did not significantly alter resting O_2 consumption in our infants. RQ rose on high-energy feeding, indicating a relatively greater proportion of carbohydrate being used as the energy substrate.

We could detect no measurable post-prandial increase in thermogenesis during either control or high-energy feeding. There are several possible explanations for this. Others have pointed out that, because of frequent feeds, infants never achieve basal metabolism before feeding (Freymond *et al.* 1986). Thus, any post-prandial increase in $\dot{V}_{O_2}$ will be relatively

Table 5. *Metabolizable energy intake (MEI), total daily energy expenditure (TDEE), 'spare' energy available for growth and weight gain for the control and high energy balance periods for infants with congenital heart disease and failure to thrive*

Patient no.	MEI (kJ/kg)	Estimated TDEE (kJ/kg)	'Spare' energy (kJ/kg)	Balance wt gain (g/kg)
Control				
1	424	358	66	4·3
2	489	336	153	−1·9
3	388	283	105	−1·1
4	389	364	25	5·4
5	287	365	−78	−0·7
6	391	381	11	1·0
7	300	425	−125	−1·1
8	301	343	−42	−0·6
9	477	378	99	−0·1
10	264	299	−35	−0·1
11	408	352	55	4·4
12	178	258	−80	0·0
13	415	294	121	6·7
14	356	344	12	1·6
Mean	362	341	20	1·3
SE	23	12	23	0·7
High-energy				
1	500	335	165	4·9
2	510	310	200	1·0
3	543	313	229	5·7
4	447	351	96	5·1
5	458	393	66	0·0
6	581	421	160	7·7
7	379	408	−29	0·0
8	735	312	423	14·8
9	630	446	184	9·9
10	355	266	89	4·3
11	484	295	189	7·3
12	316	304	12	1·4
13	623	341	282	12·2
14	515	332	182	7·2
Mean	505	345	161	5·8
SE	31	14	31	1·2
	$P \leqslant 0{\cdot}01$		$P \leqslant 0{\cdot}001$	$P \leqslant 0{\cdot}001$

small compared with premeal $\dot{V}_{O_2}$. The infants in our study were being fed at least every 4 h or more frequently. Differences between premeal and post-meal O_2 consumption might be expected to be 3–9% of the meal's metabolizable energy in infants fed every 3 h (Freymond *et al.* 1986). On high-energy feeding these increases might be in the region of 2–6 kJ/kg per 3 h. Differences in activity of the infants before and after feeding could mask such small changes. Brooke & Ashworth (1972) showed increased thermogenesis post-prandially in proportion to the rates of weight gain in children recovering from malnutrition. Only children growing at rates of greater than 4 g/kg per d continued to show increased post-prandial thermogenesis in their study.

From the regression of MEI and weight gain it is possible to make some estimate of the energy required to maintain basal metabolism and the energy cost of growth in these children. We do not have the information to relate each child's weight gain to different levels of energy intake. Regressing energy intakes and weight gains of the infants on high-

energy feeding provides an estimate of energy needs for nil weight gain (maintenance energy requirement) of 327 kJ/kg per d. The estimated energy cost of growth (31 kJ/g) is within the range 8–45 kJ/g quoted by Ashworth & Millward (1986). Our relatively high value for the energy cost of growth might suggest predominant deposition of fat. This is corroborated by the significantly increased skinfold thicknesses and is to be expected in early catch-up growth (Ashworth & Millward, 1986).

We have studied only the effects of increasing energy intakes on growth. It could be argued that we should have given a more balanced mixture of nutrients. However, energy deficiency is probably the most important nutrient deficiency in these children. There is little clinical evidence for protein deficiency and urea levels are often high, suggesting poor utilization of nitrogen due to small amounts of energy available for growth. Also, carbohydrate supplementation is cheaper than supplementation with fat or protein.

Our estimates of TDEE are inevitably incomplete. In order to have more complete estimates of TDEE it would be necessary to monitor respiratory gas exchange over 24 h, or to record activity over the full 24 h and relate this to previously determined $\dot{V}_{O_2}$ at these activity levels. Shorter periods of observation can only give vague estimates of TDEE which must vary in how close they are to real expenditure, depending on how individual activity varies throughout the day. In premature infants estimates over 6 h appear to be practical and have a mean error of estimating TDEE of 0·9% and a mean absolute error of 5·6% (Bell *et al.* 1986). With 2 h measurement periods in premature infants the mean error was −2·8% and mean absolute error 8·6% (Bell *et al.* 1986). Shultze *et al.* (1986), again studying growth of premature infants, concluded that periods of respiratory gas exchange made for less than a complete post-prandial period have little value in estimating long-term $\dot{V}_{O_2}$. Our studies, by spanning the feeding periods, although not including gas exchanges during feeds, covered a full cycle from meal-to-meal during each day of study.

There are few estimates of TDEE in normal infants of this age. A study of normal breast-fed infants where TDEE was estimated by the doubly labelled water method (Lucas *et al.* 1987) showed mean TDEE to be 280 (SE 30) kJ/kg per d at 5 weeks and 300 (SE 10) kJ/kg per d at 11 weeks of age. Weight gains in these two age groups were 36·5 (SE 2·5) g/d and 25·6 (SE 3·1) g/d respectively. Another study estimated TDEE of infants without a tendency to become overweight as 323 (SE 12) kJ/kg per d (Roberts *et al.* 1988). These values are less than those recorded in the infants in our study and refer to infants growing at normal rates and, presumably, normal activity. The contrast with the poor weight gain and higher TDEE of our infants is striking. It would be our subjective impression that our infants were less active than normal in the usual physical activities of crying, voluntary limb movement and feeding.

The children in the present study showed varying degrees of FTT, and had a variety of cardiac conditions. Inherent potential for growth may have varied since the study included some children with Down's syndrome who tend to be short even in the absence of associated CHD. Work in animals, confirmed in man, suggests that maintenance metabolic costs in normal individuals are approximately 1·5 times basal metabolism. Brooke *et al.* (1979), studying premature infants, found maintenance energy expenditure only 1·2 times resting metabolism. In the present study the estimated energy cost of maintenance was only 1·06 times resting metabolism. It would seem that little energy is available for, or directed towards, growth.

Why should infants with CHD have high resting metabolism? One of the problems relating energy intakes to body-weight in these infants is the abnormal body composition as most had a high lean body mass.

Increased cardiac mass may also explain the elevated resting $\dot{V}_{O_2}$. In adults with severe aortic stenosis, $\dot{V}_{O_2}$ by the hypertrophied heart may account for 32·4% as opposed to 10·8% total $\dot{V}_{O_2}$ (Pittman & Cohen, 1964). Infants with corresponding conditions of severe aortic

stenosis and coarctation of the aorta are likely to die or receive surgical correction before they have developed FTT. This may explain why it is particularly infants with *right* ventricular hypertrophy (usually secondary to pulmonary hypertension) who show the most significantly elevated resting O_2 consumption.

The present study suggests that infants with types of CHD known to be associated with FTT require gross energy intakes probably in excess of 120% recommended for body-weight in order to achieve satisfactory rates of growth. Since the daily energy intake necessary for normal growth is less than that required to achieve catch-up growth, early initiation of high-energy feeding seems desirable. Weight and length monitoring should determine whether energy supplementation is appropriate or whether it is leading to excessive fat gain. Transient obesity in some infants with CHD may be a risk worth taking to avoid poor brain growth or unfavourable surgical outcome.

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