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Original Article

Catch-up growth in children after repair of Tetralogy of Fallot

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Abstract *Purpose:* To evaluate the growth of children after repair of Tetralogy of Fallot, as well as the influence of residual lesions and socio-economic status. *Methods:* A total of 17 children, including 10 boys with a median age of 16 months at surgery, were enrolled in a retrospective cohort, in a tertiary care university hospital. Anthropometric (as z-scores), clinical, nutritional, and social data were collected. *Results:* Weight-for-age and weight-for-height z-scores decreased pre-operatively and recovered post-operatively in almost all patients, most markedly weight for age. Weight-for-height z-scores improved, but were still lower than birth values in the long term. Long-term height-for-age z-scores were higher than those at birth, surgery, and 3 months post-operatively. Most patients showed catch-up growth for height for age (70%), weight for age (82%), and weight for height (70%). Post-operative residual lesions (76%) influenced weight-for-age z-scores. Despite the fact that most patients (70%) were from low-income families, energy intake was above the estimated requirement for age and gender in all but one patient. There was no influence of socio-economic status on pre- and post-operative growth. Bone age was delayed and long-term-predicted height was within mid-parental height limits in 16 children (93%). *Conclusion:* Children submitted to Tetralogy of Fallot repair had pre-operative acute growth restriction and showed post-operative catch-up growth for weight and height. Acute growth restriction could still be present in the long term.

Keywords: Cardiac surgery; nutrition; congenital heart disease; paediatric cardiology

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It is well known that children with congenital heart disease are often small and undernourished.[1–6] Malnutrition results in reduced muscle function,[7] poor wound healing,[8] and impaired immunity,[9] which may increase post-operative morbidity and mortality. Some possible explanations for undernourishment include hypoxia,[10] intestinal venous congestion resulting in malabsorption,[11] genetic factors, and frequent infections of upper and lower respiratory tracts.[1] In addition, feeding problems,[12] inadequate energy intake,[12] and increased energy expenditure[13] have been suggested as contributing factors. Delay in surgical repair can lead to deterioration of nutritional status and growth of children with congenital heart disease, whereas early surgical correction has been associated with better growth outcome.[5,6] In some situations, however, surgical repair may have little influence on growth, because of significant residual lesions or surgery sequelae. Moreover, low socio-economic status probably has an impact on the genesis of growth restriction in patients with congenital heart disease, because it may limit nutrient intake before and after surgical repair. Children with congenital heart disease are reported to present catch-up growth within 3–12 months after surgery in both developed and developing countries.[1–6,14]

Cyanosis is the most prominent clinical manifestation of Tetralogy of Fallot. Chronic hypoxaemia has been shown to contribute to anorexia, feeding

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problems, and may affect the digestive tract by decreasing enzyme activities.[12,15] In addition, there is evidence that serum insulin-like growth factor I concentrations are reduced in patients with cyanotic congenital heart disease,[16] which may intensify growth restriction in these patients. There are a few studies evaluating long-term growth of patients with Tetralogy of Fallot after surgical intervention;[6,17,18] only two studies, performed in developed countries, included a specific population of children with Tetralogy of Fallot.[17,18] Thus, the objective of this study was to determine whether children submitted to total surgical repair of Tetralogy of Fallot in a Brazilian tertiary care university hospital presented catch-up growth after the intervention, and whether the presence of residual lesions and patients' socio-economic status influenced this process.

Materials and methods

Subjects

This is a longitudinal study of a cohort of children with Tetralogy of Fallot submitted to surgical correction between January, 1998 and January, 2006 in our institution. All operated children were eligible. This study was approved by the institutional review board for human research, and informed consent was obtained. We declare no conflicts of interest. Patients were selected by the database of the hospital electronic system for surgery booking. Health records were reviewed, and both clinical and anthropometric data were collected. Patients over 5 years of age at repair, as well as those with genetic, chromosomal or extracardiac abnormalities, or peri-operative death, were excluded.

Data collection

Anthropometric measurements chosen for analysis included those recorded at birth, on the day of the surgery, and 3, 6, and 12 months after intervention. Anthropometric measurements were routinely carried out in the outpatient clinics and wards by trained staff. Children up to 2 years of age were weighed on a digital scale (BP Baby® model, Filizola, Sao Paulo, Brazil) and measured with an anthropometric ruler in the supine position. Children older than 2 years of age were weighed on a digital scale (Personal Line® model, Filizola, Sao Paulo, Brazil) and measured with an anthropometric ruler in the orthostatic position. All patients were examined at the outpatient clinics by the researchers during the data collection period; they also had their current weight and height measured – named "long-term" – and their bone age determined by fist radiography analysis using the Greulich–Pyle method. Bone age was considered delayed when below 2 standard deviations from the mean for age and gender.[19] On the same occasion, parents' heights were measured and they were subsequently used to obtain mid-parental predicted height as previously described.[20] Nutritional status was assessed by z-scores of weight for age, height for age, and weight for height, adopting the Centres for Disease Control and Prevention 2000 curves as reference, using statistical software Epi-Info (Centres for Disease Control and Prevention, Atlanta, United States of America). In pre-term babies, birth z-scores were calculated using appropriate growth charts,[21] whereas pre- and post-operative z-scores were calculated using Centres for Disease Control and Prevention 2000 curves, with corrected age for gestational age until 2 years of life. Chronic growth impairment was defined as a height-for-age z-score below −2 at surgery, and acute growth impairment as a weight-for-height z-score below −2 at the same time point. Catch-up growth was defined as a post-operative rise in z-score above 0.67 standard deviations, which corresponds to the distance between adjacent centile lines of standard growth charts.[22]

Information on the current – long-term – socio-economic status, that is, monthly income, parental educational level, employment status, and housing conditions, was obtained by the social worker during an interview with the patients' parents; families were assigned into 1 out of 5 levels: (1) high, (2) medium–superior, (3) medium–inferior, (4) low–superior, or (5) low–inferior. Current – long-term – energy and protein intake were calculated during interviews by a qualified nutritional therapist using a commercial software package DietSys® (Applied Research, National Cancer Institute, Bethesda, United States of America). Energy intake was expressed in kilocalories per kilogram per day and as the percentage of the resting energy expenditure predicted by the Schofield equation.[23] This formula is known to better correlate with resting energy expenditure measured by indirect calorimetry in children undergoing surgery for both cyanotic and acyanotic congenital heart disease.[24] Protein intake was expressed in grams per kilogram per day.

Pre-operative echocardiographic data were used to determine the Nakata index, which was calculated by dividing the total cross-sectional area of the main pulmonary arteries by the body surface area. The surgical technique for Tetralogy of Fallot repair consisted of transventricular closure of ventricular septal defect and transannular patch reconstruction of right ventricular outflow tract. Echocardiography was performed post-operatively during follow-up with a Hewlett Packard 5500® (Hewlett Packard, Andover, United States of America), and significant residual lesions were defined by the presence of moderate to severe pulmonary regurgitation; systolic pressure gradient between right ventricle and pulmonary artery greater

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than 50 millimetres of mercury; or residual ventricular septal defect with haemodynamic compromise.

Statistical analysis

Results are expressed as median and total range or percentages. All data were analysed using commercially available software package SAS® version 9 (SAS Institute Inc., Cary, United States of America). A mixed-effects linear model was used to investigate the effects of surgical intervention, the presence of residual lesions, and daily energy intake on weight-for-age, height-for-age, and weight-for-height z-scores. Long-term height was compared with mid-parental predicted height, both converted to z-scores, and bone age was compared with chronological age in a within-subject paired analysis using Wilcoxon signed-rank test, because of a non-Gaussian distribution. Patients were then grouped according to pre-term birth, age at repair (above or below the median), socio-economic status, post-operative arterial oxygen saturation (above or below the median) and Nakata index (above or below the median). Between-group comparisons of anthropometric data were made using analysis of variance, Student's t-test, or Fisher's exact test, when appropriate. Results were considered significant when the p-value was less than 0.05.

Results

A total of 17 of 25 patients with Tetralogy of Fallot were included. Their median age was 16 months (range: 0–36 months) at the time of the surgical procedure; this included ten male patients. In all, eight patients were excluded, because of peri-operative death in four children, age at repair above 5 years in two children, and genetic abnormalities in two children. Approximately 23%, that is, four, of the patients were pre-term (range: 33–35 weeks of gestational age). They were followed in the outpatient clinics, and only two patients had a modified Blalock-Taussig shunt previously done. The median duration of follow-up after surgery was 65 months (range: 25–92 months). Demographic data are presented in Table 1, and oxygen saturation and main echocardiographic findings

Table 1. Demographic data.

Birth weight (kilograms)	2.76 (1.85–3.96)
Birth length (centimetres)	47 (42–50.5)
Main symptoms	70% cyanosis, 12% CHF, 17% cyanosis + CHF
Age at surgery (months)	16 (0.3–36)
Weight on the day of surgery (kilograms)	9.09 (2.95–15.2)
Height on the day of surgery (centimetres)	75.5 (50–93)

CHF = congestive heart failure
Numbers are median and range or percentages

Table 2. Oxygen saturation and main echocardiographic findings before and after surgical repair.

	Before surgery			After surgery	
Patient	Weight (kg)	SaO_2 (%)	Nakata index (mm^2/m^2)	Residual lesions	SaO_2 (%)
1	3.5	85	74.3	Moderate to severe PR	96
2	2.9	85	159.2	Systolic transpulmonary gradient >50 mmHg	93
3	7.0	78	69.6	Systolic transpulmonary gradient >50 mmHg	95
4	9.2	84	108.1	Residual VSD with haemodynamic compromise	97
5	13.6	83	129.9	Moderate to severe PR	96
6	9.1	82	109.5	Moderate to severe PR	94
7	11.0	82	63.8	None	98
8	8.1	95	70.8	Residual VSD with haemodynamic compromise	97
9	15.2	89	103.6	None	92
10	11.2	83	130.4	Moderate to severe PR	94
11	9.5	82	251.0	None	96
12	8.4	85	186.5	Systolic transpulmonary gradient >50 mmHg plus moderate to severe PR	97
13	5.6	79	92.6	Systolic transpulmonary gradient >50 mmHg	95
14	12.5	81	120.0	None	98
15	6.8	75	263.1	Moderate to severe PR	96
16	9.3	80	116.9	Systolic transpulmonary gradient >50 mmHg	99
17	8.0	75	88.8	Moderate to severe PR	97

PR = pulmonary regurgitation; SaO_2 = arterial oxygen saturation; VSD = ventricular septal defect

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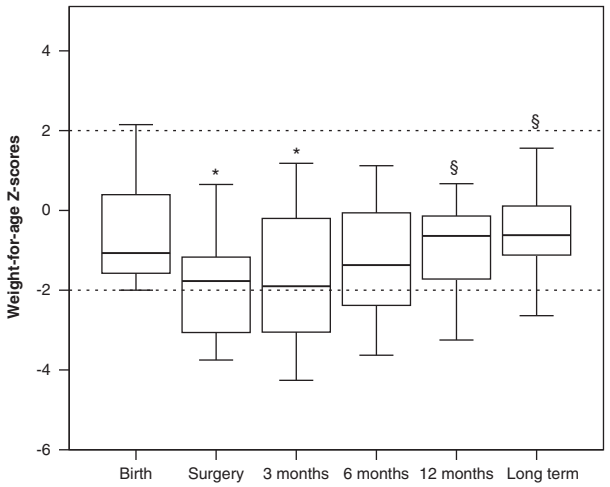

Figure 1.
Median, interquartile, and total range of weight-for-age z-scores at different times before and after surgical repair (= significant difference from birth; § = significant difference from surgery).*

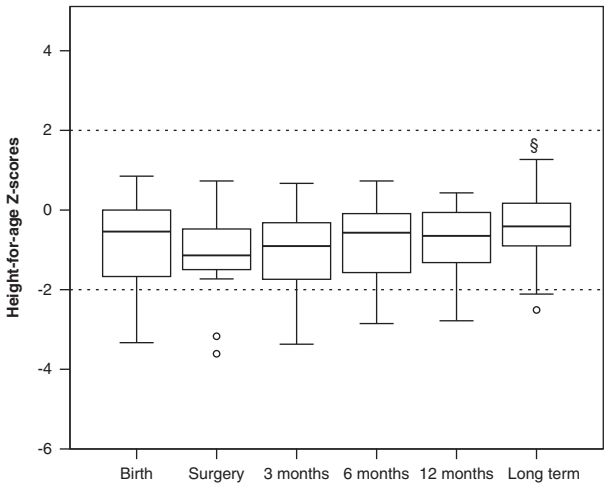

Figure 2.
Median, interquartile, and total range of height-for-age z-scores at different times before and after surgical repair (§ = significant difference from birth, surgery, and 3 months; ○ = outliers).

are shown in Table 2. The median intensive care unit length of stay was 9 (range: 1–18) days, while the median duration of mechanical ventilation was 2 (range: 0–10) days.

Weight-for-age z-scores decreased from birth to the time of surgery and at 3 months post-operatively, and subsequently increased over time at 6 and 12 months post-operatively, and in the long term. We found that values measured at surgery ($p = 0.0012$) and 3 months post-operatively ($p = 0.0016$) were significantly lower than those at birth. Values measured 12 months after surgery ($p = 0.0082$) and in the long term ($p = 0.0003$) were significantly higher than those at surgery (Fig 1). The presence of residual lesions had a significant influence on weight-for-age z-scores ($p = 0.03$), but not daily energy intake ($p = 0.06$).

Height-for-age z-scores showed a slight decrease before repair and improved over time. Values measured in the long term were significantly higher than those measured at birth ($p = 0.032$), at surgery ($p = 0.005$), and 3 months after repair ($p = 0.007$; Fig 2). Neither the presence of residual lesions ($p = 0.17$) nor daily energy intake ($p = 0.39$) influenced height-for-age z-scores.

Weight-for-height z-scores decreased from birth to the time of surgery and 3 months following repair, and subsequently increased at 6 and 12 months post-operatively. Values measured at surgery ($p < 0.0001$) and after 3 months ($p < 0.0001$), 6 months ($p < 0.0001$), and 12 months post-operatively ($p = 0.0004$), as well as in the long term ($p = 0.0015$), were all significantly lower than those at birth (Fig 3). However, values measured at 12 months post-operatively ($p = 0.026$) and in the long term ($p = 0.042$) were significantly higher than those at

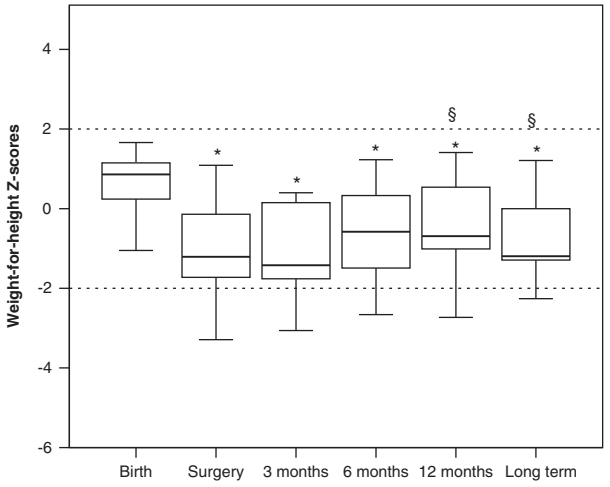

Figure 3.
Median, interquartile, and total range of weight-for-height z-scores at different times before and after surgical repair (= significant difference from birth; § = significant difference from 3 months).*

3 months following repair. There was no influence of residual lesions ($p = 0.08$) or daily energy intake ($p = 0.16$) on weight-for-height z-scores.

On individual analysis, at the time of surgery, three patients (17.6%) had acute growth impairment, one patient (6%) had chronic growth impairment, and two patients (11.7%) had both acute and chronic growth impairment. A total of 12 patients (70.5%) presented catch-up growth for height for age, mainly after 12 months post-operatively; 14 patients (82.3%) exhibited catch-up growth for weight for age, mostly 6 months after surgery; and 12 patients (70.5%) showed catch-up growth for weight for height, four after 3 months, and four in the long term.

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In all, 13 patients (76%) had significant residual lesions identified by echocardiography; it was characterised as severe pulmonary regurgitation in six children, systolic pressure gradient between right ventricle and pulmonary artery above 50 millimetres of mercury in four children, both conditions in one child, and residual ventricular septal defect with haemodynamic compromise in two children. Patients with residual lesions had lower birth weight ($p = 0.02$) and length ($p = 0.02$) when compared with those without residual lesions. In our study, only weight-for-age z-scores were influenced by the presence of residual lesions on multivariate analysis.

Despite the fact that 12 patients (70%) were from families of low socio-economic levels (Table 3), all but one patient had energy intake above the estimated resting energy expenditure for age and gender (median 148%, range: 95–369% of resting energy expenditure; median 79, range: 40–185 kilocalories per kilogram per day). Median daily protein intake was 3.5 (range: 1.7–6.3) grams per kilogram per day. Only two patients had protein intake below 2 grams per kilogram per day. Energy intake did not influence any anthropometric measurements. There were no differences on pre- and post-operative weight-for-age, height-for-age, and weight-for-height z-scores and daily energy and protein intake among patients from different socio-economic levels.

There was no influence of pre-term birth on long-term height-for-age (−1.1 plus or minus 1.0 versus −0.2 plus or minus 1.0, $p = 0.15$), weight-for-age (−0.8 plus or minus 0.8 versus −0.4 plus or minus 1.1, $p = 0.55$), or weight-for-height (0.3 plus or minus 1.3 versus −1.1 plus or minus 0.8, $p = 0.07$) z-scores. Age at surgery (above or below 16 months) did not influence long-term height-for-age (−0.6 plus or minus 1.2 versus −0.3 plus or minus 1.0, $p = 0.65$), weight-for-age (−0.3 plus or minus 1.2 versus −0.7 plus or minus 0.9, $p = 0.39$), or weight-for-height (−0.2 plus or minus 1.5 versus −1.0 plus or minus 0.8, $p = 0.29$) z-scores. Arterial oxygen saturation (above or below 96%) also did not influence long-term height-for-age (−0.2 plus or minus 1.5 versus −0.6 plus or minus 0.7, $p = 0.54$), weight-for-age (−0.2 plus or minus 1.4 versus −0.8 plus or minus 0.7, $p = 0.23$), or weight-for-height (−0.2 plus or minus 1.3 versus −0.9 plus or minus 1.1, $p = 0.44$) z-scores. Lastly, the Nakata index (above or below 109 square millimetres per square metres) did not influence long-term height-for-age (0.0 plus or minus 0.9 versus −0.8 plus or minus 1.1, $p = 0.12$), weight-for-age (−0.4 plus or minus 1.0 versus −0.7 plus or minus 1.2, $p = 0.52$), or weight-for-height (−1.7 plus or minus 0.8 versus −0.4 plus or minus 1.1, $p = 0.17$) z-scores.

Median bone age was significantly lower than median chronological age in the long term (median 72, range: 24–108 months versus median 81, range: 26–120 months, respectively, $p = 0.002$). Only five patients had bone age higher than chronological age.

Table 3. Socio-economic distribution.

Socio-economic level	n (%)
(1) High	0
(2) Medium–superior	0
(3) Medium–inferior	5 (29%)
(4) Low–superior	8 (47%)
(5) Low–inferior	4 (23%)

There was no significant difference between median mid-parental predicted height z-scores and observed long-term height-for-age z-scores (median −0.36, range: −1.51–1.03 versus median −0.39, range: −2.51–1.27, respectively, $p = 0.92$). Approximately 93% of patients had their long-term height-for-age z-scores within mid-parental predicted height z-score limits.

Discussion

The present study showed that Brazilian children undergoing surgical correction of Tetralogy of Fallot exhibited growth restriction before surgery, with recovery mainly 12 months after repair. Previous reports in a large cohort of children with several types of congenital heart disease in a developing country showed catch-up growth within 3 months after surgical repair.[5,6] In children undergoing surgery for Tetralogy of Fallot, previous studies in developed countries showed that catch-up growth occurred within 6–12 months after surgery.[17,18] In our study, weight-for-age z-scores were significantly impaired pre-operatively and 3 months after surgery. Cheung et al[17] also reported pre-operative weight-for-age impairment with post-operative catch-up growth in a specific population of children with Tetralogy of Fallot, but they did not evaluate the presence of post-operative residual lesions. We showed that they influenced weight-for-age z-scores in our study. In contrast to the latter, in the present study we did not observe pre-operative decreases on height-for-age z-scores, although three children (17%) had values below −2 at surgery, indicating chronic growth impairment. Interestingly, long-term height-for-age z-scores were significantly higher than those measured at birth, at surgery, and 3 months post-operatively. Indeed, 70% of children showed catch-up growth for height for age. Moreover, in our study, weight-for-height z-scores were severely impaired pre-operatively, when compared with birth values, recovering only later after surgery, indicating pre-operative acute growth impairment. However, only five patients (29%) had acute growth impairment at

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surgery when considering a lower limit of −2 for the z-score. It is noteworthy that all post-operative values, including the long-term measures, were significantly lower compared with birth. In addition, despite the fact that 70% of our children showed catch-up growth for weight for height they did not reach the birth values, suggesting that acute growth impairment could still be present. In a heterogeneous cohort of children with congenital heart disease in a developing country, weight-for-age, height-for-age, and weight-for-height z-scores were below −2 in 59%, 26%, and 56% of children, respectively.[5] Schuurmans et al[18] observed low pre-operative mean weight-for-age and height-for-age z-scores in 18 Dutch children with Tetralogy of Fallot, which improved after the introduction of adequate nutritional therapy. The mean pre-operative weight-for-height z-score was normal, suggesting the presence of chronic rather than acute growth compromise. Australian children with Tetralogy of Fallot were reported to have both acute and chronic growth impairment pre-operatively.[17]

Several studies have suggested that surgical intervention should be carried out early in order to attenuate growth restriction and improve outcome. Encouraging results of early repair of congenital heart disease have been reported in neonates and infants.[6] Although the median age at surgery of our patients was higher than those reported in the literature,[17,18] catch-up growth was not influenced by it, because they presented catch-up within 6–12 months after surgery, and long-term z-scores were within normal range. In fact, we observed a trend for patients operated on later in life to present a more prolonged catch-up period. Therefore, we believe that a higher age at surgery can lead to a delayed catch-up, instead of low stature in adulthood. Nevertheless, we did not study patients operated on after 5 years of age, in whom this effect on long-term growth could be better studied. Interestingly, the presence of residual lesions influenced weight-for-age z-scores, but catch-up growth was not affected. However, because patients with post-operative residual lesions were already lighter – lower birth weight – and shorter – lower birth length – before surgery, we cannot rule out that impaired anthropometric measurements at birth may have prevented the demonstration of the influence of post-operative residual lesions in height recovery. Nevertheless, almost all long-term height-for-age z-scores were within mid-parental predicted height limits. This finding agrees with a previously published study.[17] Furthermore, in our study, socio-economic status did not influence peri-operative or long-term growth, as shown in children from India with several types of cardiac malformations.[6] Interestingly, despite the fact that most patients were from low socio-economic status families, all but one child had their energy intake fairly above the estimated resting energy expenditure for age and gender. All energy exceeding the resting energy expenditure should be used for daily activity and spared for growth.[25] We did not evaluate our patients' physical activity level, but as our children grew we assumed that energy intake was enough for basal metabolism, daily activity, and growth. In addition, we have found that our patients had total daily energy intake above previously reported values of measured total energy expenditure after heart surgery in several countries.[24,26–29] Daily protein intake was also above that recommended for age and gender.[30] Although socio-economic status was evaluated only in the long-term, we assumed it to be the same throughout the study.

The limitations of this study include the relatively small sample size, and lack of physical activity evaluation and direct measurement of total energy expenditure. In addition, as we do not routinely do cardiac catheterisation in patients with Tetralogy of Fallot – only five did – it is possible that patients with low Nakata index and high oxygen saturation had undiagnosed single or multiple aortopulmonary collateral arteries. Lastly, although patients with genetic and chromosomal abnormalities were excluded, those included in the study were not routinely screened for 22q11 deletion.

This is the first study to address the influence of residual lesions and socio-economic status on catch-up growth in a specific population of children submitted to surgical repair of Tetralogy of Fallot in a developing country. Our children had more acute and less chronic growth impairment, and catch-up growth occurred in almost all of them, regardless of socio-economic status. Only weight-for-age z-scores were influenced by the presence of residual lesions.

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References

1. Mehrizi A, Drash A. Growth disturbance in congenital heart disease. J Pediatr 1962; 61: 418–429.
2. Feldt RH, Strickler GB, Weidman WH. Growth of children with congenital heart disease. Am J Dis Child 1969; 117: 573–579.
3. Cameron JW, Rosenthal A, Olson AD. Malnutrition in hospitalized children with congenital heart disease. Arch Pediatr Adolesc Med 1995; 149: 1098–1102.
4. Venugopalan P, Akinbami FO, Al-Hinai KM, Agarwal AK. Malnutrition in children with congenital heart defects. Saudi Med J 2001; 22: 964–967.
5. Vaidyanathan B, Nair SB, Sundaram KR, et al. Malnutrition in children with congenital heart disease (CHD) determinants and

short term impact of corrective intervention. Indian Pediatr 2008; 45: 541–546.
6. Vaidyanathan B, Radhakrishnan R, Sarala DA, Sundaram KR, Kumar RK. What determines nutritional recovery in malnourished children after correction of congenital heart defects? Pediatrics 2009; 124: e294–e299.
7. Hill GL. Malnutrition and surgical risk: guidelines for nutritional therapy. Ann R Coll Surg Engl 1987; 69: 263–265.
8. Haydock DA, Hill GL. Impaired wound healing in surgical patients with varying degrees of malnutrition. J Parenter Enteral Nutr 1986; 10: 550–554.
9. Gross RL, Newberne PM. Role of nutrition in immunologic function. Physiol Rev 1980; 60: 188–302.
10. Varan B, Tokel K, Yilmaz G. Malnutrition and growth failure in cyanotic and acyanotic congenital heart disease with and without pulmonary hypertension. Arch Dis Child 1999; 81: 49–52.
11. Sondheimer JM, Hamilton JR. Intestinal function in infants with severe congenital heart disease. J Pediatr 1978; 92: 572–578.
12. Thommessen M, Heiberg A, Kase BF. Feeding problems in children with congenital heart disease: the impact on energy intake and growth outcome. Eur J Clin Nutr 1992; 46: 457–464.
13. Leitch CA, Karn CA, Peppard RJ, et al. Increased energy expenditure in infants with cyanotic congenital heart disease. J Pediatr 1998; 133: 755–760.
14. Manso PH, Carmona F, Jacomo ADN, Bettiol H, Barbieri MA, Carlotti APCP. Growth after ventricular septal defect repair: does defect size matter? A 10-year experience. Acta Paediatr 2010; 99: 1356–1360.
15. Bernstein D, Bell JG, Kwong L, Castillo RO. Alterations in postnatal intestinal function during chronic hypoxemia. Pediatr Res 1992; 31: 234–238.
16. Dundar B, Akcoral A, Saylam G, et al. Chronic hypoxemia leads to reduced serum IGF-I levels in cyanotic congenital heart disease. J Pediatr Endocrinol Metab 2000; 13: 431–436.
17. Cheung MM, Davis AM, Wilkinson JL, Weintraub RG. Long term somatic growth after repair of tetralogy of Fallot: evidence for restoration of genetic growth potential. Heart 2003; 89: 1340–1343.
18. Schuurmans FM, Pulles-Heintzberger CF, Gerver WJ, Kester AD, Forget PP. Long-term growth of children with congenital heart disease: a retrospective study. Acta Paediatr 1998; 87: 1250–1255.
19. Reiter EO, Rosenfeld RG. Normal and aberrant growth. In: Kronenberg HM, Melmed S, Polonsky KS, Larsen PR (eds.). Williams Textbook of Endocrinology. Elsevier, Philadelphia, 2008, pp. 853–854.
20. Tanner JM. The use and abuse of growth standards. In: Falkner F, Tanner JM (eds.). Human Growth. Plenum, New York, 1986, pp. 95–112.
21. Niklasson A, Albertsson-Wikland K. Continuous growth reference from 24th week of gestation to 24 months by gender. BMC Pediatr 2008; 8: 8.
22. Ong KK, Preece MA, Emmett PM, Ahmed ML, Dunger DB. Size at birth and early childhood growth in relation to maternal smoking, parity and infant breast-feeding: longitudinal birth cohort study and analysis. Pediatr Res 2002; 52: 863–867.
23. Schofield WN. Predicting basal metabolic rate, new standards and review of previous work. Hum Nutr Clin Nutr 1985; 39 (Suppl 1): 5–41.
24. Avitzur Y, Singer P, Dagan O, et al. Resting energy expenditure in children with cyanotic and noncyanotic congenital heart disease before and after open heart surgery. J Parenter Enteral Nutr 2003; 27: 47–51.
25. Menon G, Poskitt EM. Why does congenital heart disease cause failure to thrive? Arch Dis Child 1985; 60: 1134–1139.
26. Nydegger A, Bines JE. Energy metabolism in infants with congenital heart disease. Nutrition 2006; 22: 697–704.
27. Steltzer M, Rudd N, Pick B. Nutrition care for newborns with congenital heart disease. Clin Perinatol 2005; 32: 1017–1030.
28. De Wit B, Meyer R, Desai A, Macrae D, Pathan N. Challenge of predicting resting energy expenditure in children undergoing surgery for congenital heart disease. Pediatr Crit Care Med 2010; 11: 496–501.
29. Nydegger A, Walsh A, Penny DJ, Henning R, Bines JE. Changes in resting energy expenditure in children with congenital heart disease. Eur J Clin Nutr 2009; 63: 392–397.
30. Trumbo P, Schlicker S, Yates AA, Poos M. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. J Am Diet Assoc 2002; 102: 1621–1630.