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Abstract

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Walter Knirsch, Walter Zingg, Vera Bernet, Christian Balmer, Anastasia Dimitropoulos, René Prêtre, Urs Bausersfeld, Bea Latal

Division of Pediatric Cardiology, University Children’s Hospital Zurich, Steinwiesstrasse 75, CH-8032 Zurich, Switzerland
Department of Pediatrics, University Children’s Hospital Zurich, Switzerland
Infection Control and Hospital Epidemiology, Geneva University Hospitals, Switzerland
Neonatology and Pediatric Intensive Care, University Children’s Hospital Zurich, Switzerland
Child Development Center, University Children’s Hospital Zurich, Switzerland
Division of Cardiac Surgery, University Children’s Hospital Zurich, Switzerland

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Abstract

The aim of this prospective study was to examine the determinants of growth failure and the association with neurodevelopmental outcome in infants undergoing open-heart surgery. In 107 infants undergoing open-heart surgery for congenital heart disease (CHD), we evaluated weight at birth, at surgery, and at one year of age (expressed as z-scores). Neurodevelopmental status was assessed before surgery and at one year of age. Median age at surgery was 3.9 months (range: 0.1–10.2). Mean [± standard deviation (S.D.)] weight z-score at birth was −0.27 (± 1.45), before surgery −1.34 (± 1.45) (P < 0.001 vs. birth weight), and at one year −0.86 (± 1.35), (P < 0.001 vs. weight at surgery). Poor preoperative weight (< 10th percentile) was associated with genetic disorders [odds ratio (OR) 5.9, P < 0.001], preoperative neurological abnormalities (OR 3.41, P < 0.05), and older age at surgery (OR 1.01, P < 0.05). Weight < 10th percentile at one year was associated with the same factors as poor preoperative weight, however, also with risk adjustment for congenital heart surgery-1 (RACHS) score > 3 (OR 3.22, P < 0.05). Neurodevelopmental outcome at one year was not determined by growth failure. In conclusion, impaired body weight gain before surgery is followed by a catch-up growth after surgery. However, there is no relationship to neurodevelopmental outcome. Genetic comorbidity is the most significant factor for poor weight gain.

Keywords: Neurodevelopmental outcome; Cardiac surgery; Genetic disorders

1. Introduction

Advances in cardiac surgery and perioperative intensive care management in the past decades have improved the survival of infants and children undergoing open-heart surgery for complex types of congenital heart diseases (CHD). Nevertheless, the pre- and postoperative morbidity is still of concern, especially long-term neurodevelopmental sequelae [1]. In the past, different factors influencing neurodevelopmental outcome have been identified as fixed, non-modifiable factors, such as genetic disorders, or as non-fixed, modifiable variables, such as nutrition [1].

Malnutrition and growth failure is particularly common in children with cyanotic heart disease if accompanied by pulmonary hypertension [2]. In addition, many cardiac lesions are associated with a higher incidence of low birth weight and prematurity [3]. Reasons for malnutrition in this population include abnormal intrauterine blood flow before birth, malabsorption after birth, and increased energy demand. Infants with CHD have a high incidence of protein-energy malnutrition [4]. Further, chromosomal disorders that are the underlying cause for the CHD are often associated with poor intrauterine growth [5]. Nevertheless, the determinants of body weight gain and their association with neurodevelopmental outcome are unclear. On the other hand, postnatal lack of catch-up growth is a known phenomenon in preterm infants and is associated with adverse neurodevelopmental outcome [6]. This has not been examined for children with CHD.

Therefore, the aim of our study was to describe the pre- and postoperative course of weight gain in children undergoing open-heart surgery during the first year of life, to identify factors influencing the pre- and postoperative weight, and to determine the relationship between weight gain and neurodevelopmental outcome at one year of age.
2. Materials and methods

2.1. Design

This is a prospective cohort study performed at the University Children’s Hospital of Zurich. The study was approved by the local Ethics Committee and written informed consent was obtained from the parents.

2.2. Patients

Between 1 August 2004 and 31 January 2006, all consecutive infants <1 year of age undergoing open-heart surgery for CHD were enrolled. All patients were evaluated at birth, at the day before cardiac surgery, and at one year of age. Surgical risk was determined using the risk adjustment for congenital heart surgery-1 (RACHS) scoring system [7]. Patients were screened for genetic disorders if a cardiac malformation is known to be associated with a genetic abnormality by determining caryotype, microdeletion syndromes, and specific genetic diseases, such as Williams Beuren syndrome. Patients with minor dysmorphicisms or other major organ malformations were also examined for genetic disorders after genetic counseling by an experienced pediatric geneticist. For further analysis, genetic disorders were defined either as identifiable chromosomal disorders (Down syndrome, microdeletion 22q11), or as non-identifiable dysmorphic syndromes. Demographic, intraoperative, postoperative and nutritional parameters were prospectively collected.

2.3. Nutrition

Enteral food intake was calculated by a total volume intake of 100–140 ml/kg per day, with the objective of a total calorie intake of 130–150 kcal/kg per day. The standard hospital feeding management protocol included a stepwise pre- and postoperative nutritional support primarily by breast milk and concentrated formula milk supplemented with lipids were given during the first six months of life and later replaced by solid foods. As a second step, tube feeding was applied if adequate caloric intake was not achieved otherwise. Parenteral nutrition was introduced only in isolated cases preoperatively, if the previous described nutrition management failed to achieve adequate weight gain. This feeding management was implemented by the inpatient service and supervised by the outpatient pediatrician.

2.4. Surgical management

Cardiac surgery was performed by two cardiac surgeons. Alpha-stat blood gas management was routine. In most cardiac surgeries, normothermic or mild hypothermic cardiopulmonary bypass (CPB) (rectal temperature >32 °C) was performed with a pump flow rate at 100–150 ml/kg/min to achieve a mean arterial pressure of 40–50 mmHg. Norwood I procedures, aortic arch surgery, or repair of common arterial truncus were performed under moderate hypothermia (nasopharyngeal temperature 22 °C–28 °C) with a regional cerebral perfusion set at a pump flow rate maintained at 30–50 ml/kg/min and a target arterial pressure around 50–60 mmHg, measured in the right radial artery. Modified ultrafiltration at the end of CPB was performed in all patients.

2.5. Body weight and patterns of preoperative weight course

Body weight was measured in a standard fashion by the hospital nurse and expressed as z-scores [8]. For preterm born infants (<37 weeks’ gestation) percentiles by Voigt et al. were used [9]. Patients were divided into four groups depending on the course of body weight between birth and surgery: the first group consisted of patients with a normal body weight (>10th percentile) at birth and at surgery (normal–normal, n = 44); the second group included patients with a normal weight (>10th percentile) at birth, but a body weight below the 10th percentile at surgery (normal–<p10, n = 37); the third group included patients with a birth weight below the 10th percentile and remaining below the 10th percentile at surgery (<p10–<p10, n = 22); the fourth group included patients with a birth weight below the 10th percentile, but reaching a body weight over the 10th percentile at surgery (<p10–normal, n = 4).

2.6. Neurodevelopmental assessment

The infants were examined by two developmental pediatricians from the Child Development Center (B.L., A.D.). A standardized neurological assessment was performed before surgery and at one year of age, at least 4–6 months after surgery under hemodynamic stable conditions. The assessment was modified after Prechtl and Beintema [10] and included a neurosurgical score (range 0–18). A neuroscore >6 was defined as severe neurological impairment for further statistical analysis. At one year of age, the Bayley Scales of Infant Development II providing a mental development index (MDI) and a psychomotor developmental index (PDI) [11] was administered.

2.7. Statistics

Results are presented as mean and standard deviations (S.D.) or as median and ranges, as appropriate. Univariate analyses were performed using parametric and non-parametric tests. For comparisons at different time points, paired t-tests were used. Logistic regression analysis was used for risk factor analysis. Preoperative weight and weight at one year below the 10th percentile were the outcome parameter. Variables associated with an increased risk with a P < 0.2 in the univariate analysis were included in the multivariate analysis. Predictors for preoperative body weight below the 10th percentile included sex, genetic disorders, univentricular heart defect, cyanotic heart defect, RACHS score >3, preoperative neuroscore >6, need for tube feeding, and age at surgery. In addition, length of intensive care unit (ICU) stay and weight group <p10–<p10 were included in the model for body weight below the 10th percentile at one year of age. Predictors of neurodevelopmental outcome at one year follow-up were assessed equally in a stepwise multiple regression analysis. Analyses were performed using
3. Results

3.1. Patients

During the 18-month study period 456 patients were operated for CHD. Of those, 107 infants were younger than one year of age at the time of open-heart surgery and were enrolled in this study. The patient characteristics are summarized in Table 1. Two patients underwent extracorporeal membrane oxygenation and six patients needed cardiopulmonary resuscitation.

We included 18 preterm born infants (16%) with a gestational age between 30 and 36 weeks of gestation (birth weight 1050–2580 g). The clinical course of these preterm born infants was determined by the clinical symptoms of the cardiac defect and not by a prematurity associated comorbidity. Prematurity (<37 weeks) was not associated with pre- and postoperative body weight course or with neurodevelopmental outcome (data not shown).

The ICU stay exceeded >3 months in two patients: one patient had a complex pulmonary atresia with ventricular septal defect with postoperative pulmonary haemorrhagic infarction and complete functional loss of the left lung for 284 days; the second patient suffered from renal failure needing peritoneal dialysis for six months after repair of tetralogy of Fallot. One patient died nine months after Norwood I procedure and bidirecional Glenn procedure due to myocardial failure of the systemic right ventricle.

3.2. Pre- and postoperative body weight

The mean (range) body weight at birth was 2985 g (1050–5250), at surgery 4824 g (2100–8800), and at one year 8683 g (3100–13,400). Mean z-scores decreased (P<0.001) from birth (mean z-score±S.D.: −0.27±1.45) until surgery (−1.34±1.45) and increased (P<0.001) from surgery until one year of age (−0.86±1.35). The preoperative body weight gain was 13.2±9.3 g per day and the postoperative body weight gain was 14.2±4.7 g per day.

Thirty-nine (36.4%) patients needed a nasogastric tube before surgery.

3.3. Preoperative body weight course

The different pattern of pre- and postoperative body weight courses are depicted in Fig. 1 (except for the pattern <p10–normal because of small number). It is of note that patients in the group normal–normal also lost body weight until surgery (mean z-score at birth 0.62±S.D. 1.2; at surgery 0.03±1.0, paired t-test P<0.001). Between surgery and one year of age both, the group normal–<p10 and <p10–<p10, gained weight (Fig. 1). However, the mean z score of the group normal–<p10 at one year of age remained significantly lower compared to the mean z-score at birth (mean z-score at birth −0.07±1.01, mean z-score at one year −1.24±1.1, paired t-test, P<0.01).

![Fig. 1. Body weight course in patients with congenital heart disease operated by open-heart surgery during the first year of life. Full line infants (n=44) with weight at or above the 10th percentile at birth and at surgery; broken line infants (n=37) with weight at or above the 10th percentile at birth and below the 10th percentile at surgery; dotted line infants (n=22) with weight below the 10th percentile at birth and at surgery. Infants (n=4) with weight below the 10th percentile at birth and above 10th percentile at surgery are not shown.](icvts.ctsnetjournals.org)
### Table 2
Determinants of body weight below 10th percentile at surgery

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<td></td>
<td>OR</td>
<td>95% CI</td>
<td>P-value</td>
<td>OR</td>
<td>95% CI</td>
<td>P-value</td>
</tr>
<tr>
<td>Sex, female vs. male*</td>
<td>1.63</td>
<td>0.76–3.52</td>
<td>0.21</td>
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<td></td>
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<tr>
<td>Genetic disorder*</td>
<td>5.90</td>
<td>2.18–16.0</td>
<td>&lt;0.001</td>
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<td>Uni- vs. biventricular heart defect</td>
<td>0.79</td>
<td>0.23–2.63</td>
<td>0.70</td>
<td></td>
<td></td>
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<tr>
<td>A- vs. cyanotic heart defect*</td>
<td>0.44</td>
<td>0.22–0.96</td>
<td>0.04</td>
<td></td>
<td></td>
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<td>RACHS score &gt; 3</td>
<td>1.43</td>
<td>0.48–4.26</td>
<td>0.64</td>
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<tr>
<td>Neuroscore preoperative &gt; 6*</td>
<td>3.41</td>
<td>1.34–8.67</td>
<td>0.01</td>
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<td>Nasogastric tube preoperative</td>
<td>0.92</td>
<td>0.42–2.03</td>
<td>0.84</td>
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<td></td>
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<tr>
<td>Age at surgery*</td>
<td>1.01</td>
<td>1.0–1.01</td>
<td>0.02</td>
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**OR**, odds ratio; 95% CI, 95% confidence interval; RACHS, risk adjustment for congenital heart surgery-1.

*Included in the multivariate analysis, bold factors were significant in multivariate analysis.

1Per month increase.

### 3.4. Factors associated with low body weight at surgery

Table 2 depicts factors associated with body weight below 10th percentile at surgery. Older age at surgery and genetic disorders were independently associated with weight below the 10th percentile. Since a preoperative neuroscore > 6 was significantly associated with genetic disorders in general, the latter was not included in the multivariate analysis. When excluding the variable ‘genetic disorders’ from the multivariate analysis, age at surgery (increase per month) and preoperative neuroscore > 6 remained independently significant [odds ratio (OR): 1.01, confidence interval (CI): 1.00–1.02; *P* < 0.01; and OR: 3.22, CI 1.23–9.03; *P* < 0.05, respectively]. However, although significant, the association between age at surgery and low weight at surgery was weak.

### 3.5. Factors associated with low body weight at one year

Table 3 presents the results of the logistic regression analysis relating pre- and perioperative variables to body weight below the 10th percentile at one year of age. Genetic disorders and RACHS score > 3 were independently associated with body weight below the 10th percentile. When the variable ‘genetic disorders’ was not entered in the regression analysis, preoperative neuroscore > 6 was significantly associated with weight at one year (*P* < 0.05), but RACHS score showed only a trend (*P* < 0.1) (data not shown).

### Table 3
Determinants of body weight below 10th percentile at one year follow-up

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<td></td>
<td>OR</td>
<td>95% CI</td>
<td>P-value</td>
<td>OR</td>
<td>95% CI</td>
<td>P-value</td>
</tr>
<tr>
<td>Sex (female)*</td>
<td>1.69</td>
<td>0.77–3.72</td>
<td>0.19</td>
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<tr>
<td>Genetic Disorder*</td>
<td>3.88</td>
<td>1.64–9.19</td>
<td>0.002</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uni- vs. biventricular heart defects</td>
<td>1.17</td>
<td>0.35–3.97</td>
<td>0.80</td>
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<tr>
<td>A- vs. cyanotic heart defects*</td>
<td>0.99</td>
<td>0.45–2.16</td>
<td>0.98</td>
<td></td>
<td></td>
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<tr>
<td>RACHS score &gt; 3*</td>
<td>3.22</td>
<td>1.07–9.70</td>
<td>0.04</td>
<td></td>
<td></td>
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<tr>
<td>Neuroscore preoperative &gt; 6*</td>
<td>3.23</td>
<td>1.34–7.77</td>
<td>0.01</td>
<td></td>
<td></td>
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<tr>
<td>Nasogastric tube preoperative</td>
<td>1.99</td>
<td>0.89–4.46</td>
<td>0.10</td>
<td></td>
<td></td>
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<tr>
<td>Age at surgery†</td>
<td>1.00</td>
<td>1.00–1.01</td>
<td>0.87</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Length of ICU stay</td>
<td>1.00</td>
<td>0.98–1.02</td>
<td>0.85</td>
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**OR**, odds ratio; 95% CI, 95% confidence interval; RACHS, risk adjustment for congenital heart surgery-1.

*Included in the multivariate analysis, bold factors were significant in multivariate analysis.

1Per month increase.

### 3.6. Neurodevelopmental outcome at one year

Neurodevelopmental outcome was available for 101 of the 107 patients because of parental refusal to return to the one year examination in five patients and death of one patient with hypoplastic left heart syndrome. Children were examined at the mean age of 12.5 months (Table 1). For children without genetic disorders, mean MDI (84.3 ± 16.3) and mean PDI (68.2 ± 15.8) were significantly lower compared to the norm (100 ± 15; both *P* < 0.001), but higher compared to children with a genetic disorder (MDI 65 ± 19.6; PDI 53.8 ± 12.2; both *P* < 0.001). Median neuroscore at one year was lower in children without genetic disorders (median 2, range 2–12) compared to children with genetic disorders (median 4, range 0–16, *P* < 0.001). For both groups, median neuroscores were lower at one year compared to the preoperative examination (both *P* = 0.001).

Preoperative body weight was not associated with neurodevelopmental outcome (MDI: *P* = 0.39, PDI: *P* = 0.97). Also, the different weight course groups were not related to neurodevelopmental outcome at one year of age (results not shown). Stepwise multiple regression analysis revealed that genetic disorders (MDI: *P* < 0.01), the length of hospital stay (MDI: *P* = 0.04; PDI: *P* = 0.002; postoperative neuroscore: *P* < 0.01) and the preoperative neuroscore (MDI: *P* < 0.001; PDI: *P* < 0.001; postoperative neuroscore: *P* < 0.001) were independently associated with the neurodevelopmental outcome at one year of age.
4. Discussion

In this prospective cohort study, we describe the pre- and postoperative course of weight gain during the first year of life, risk factors for low weight and its relation to neurodevelopmental outcome in children undergoing open-heart surgery for CHD.

Our results show that in infants requiring cardiac surgery a significant relative body weight decrease occurs preoperatively, followed by a significant postoperative catch-up growth. Prior to surgery, genetic comorbidity and an older age at surgery are risk factors for a weight below the 10th percentile at surgery. After surgery, the degree of catch-up growth until the end of the first year of life and a low weight at one year of age is determined by genetic comorbidity, preoperative neurologic abnormalities, but also by the severity of the CHD (defined by the RACHS score). Importantly, preoperative weight and the postoperative weight course did not influence neurodevelopmental outcome.

This is the first study to describe in detail the weight course of infants requiring open-heart surgery for CHD. An important observation was that the body weight declined significantly before surgery. It is important to note that factors determining this decline were extracardiac (genetic disorders, neurological abnormalities, age at surgery) rather than cardiac factors (cyanosis, RACHS score).

Genetic disorders were the strongest predictor for growth failure in our study, which has been previously described for children with Down syndrome and microdeletion 22q11 deletion syndrome [5]. In contrast, in infants without genetic disorders an abnormal preoperative neuroscore was independently associated with a low body weight at surgery, which may be explained by the feeding problems in neurologically compromised infants. The fact that age at surgery was an important and independent risk factor for poor preoperative body weight gain is probably due to the time of ongoing congestive heart failure with a continuously high-energy demand. Therefore, for an optimal body weight course it would be beneficial to perform the cardiac surgery at a younger age [12, 13]. However, not all infants can be operated at a younger age since factors, such as different hemodynamics, morphologic and surgical factors, i.e. regression of pulmonary vascular resistance after birth until surgery, need to be considered. Whether early surgery is beneficial for body weight gain, a randomized study would be needed comparing body weight gain in infants operated before and after three months of age.

Neurodevelopmental outcome was impaired in our study population, in particular motor development and was, as expected, poorer for infants with genetic disorders compared to those without genetic disorders. This finding is in line with other studies [12, 13]. Many studies have examined factors associated with adverse neurodevelopmental outcome, however, weight has not been examined so far in this population. Interestingly, in preterm born children, poor postnatal weight gain was associated with adverse neurodevelopmental outcome [6]. In our cohort, we could not find an association between weight before surgery, weight course and neurodevelopmental outcome at one year of age. It is known that the neurodevelopmental outcome after neonatal cardiac surgery is multi-factorially determined. Recently, this has been reviewed as fixed and modifiable factors for the neurological outcome [1, 14]. Fixed factors are patient-specific factors and cannot be modified, whereas modifiable factors relate to surgical procedure and perioperative management. Although the catch-up growth after cardiac surgery may represent the basis for a normalization of somatic and potentially cerebral growth, our results suggest that potentially modifiable factors, such as weight and weight course do not seem to play a significant role for neurodevelopmental outcome. Consequently, other, non-modifiable factors, such as genetic comorbidity, length of hospital stay and preoperative neurologic abnormalities are significant risk factors for adverse neurodevelopmental outcome. This has been shown in our study and confirms findings of other published studies [13, 15].

A limitation of our study is the wide range and different severity of CHD, which may be not sufficiently described by the RACHS score. Furthermore, the percentile curves for body weight gain used in our study were those for healthy infants, because percentile curves for the different type of syndromes are not available except for children with Down syndrome. In addition, we did not collect information on intrauterine growth. Information on cardiac medications, such as diuretics interfering with body weight course, or other medications interfering with nutrition and digestion, such as morphine was not collected on this cohort. For a further subgroup analysis of different types of CHD, such as hypoplastic left heart syndrome, the number of patients was too small. In addition, factors such as persistent pulmonary hypertension and residual cardiac defects were not systemically assessed and thus could not be related to the postoperative weight course.

Outcome was assessed by standardized clinical examination and standardized developmental tests. The follow-up examination at one year of age is early and will be followed by long-term outcome assessment. Furthermore, it would be interesting to correlate the neurodevelopmental findings with results of cerebral magnetic resonance examinations. We did not perform cerebral imaging in a standardized way in all infants since this was not the primary goal of our study.

5. Conclusions

Infants undergo a significant relative body weight decline before open-heart surgery and manifest a catch-up growth thereafter. This course takes place despite high-caloric feeding strategies and tube feeding. We thus feel that additional efforts to increase body weight to avoid the reported decline may not be successful since factors determining this decline are rather fixed non-cardiac, i.e. genetic and neurological. Thus, it may be important to consider surgical repair at an earlier age. Importantly, body weight decline and poor weight at surgery is not related to adverse neurodevelopmental outcome. Neurodevelopmental outcome, however, is influenced mostly by fixed, non-cardiac factors, i.e. genetic comorbidity and preoperative neurological status. Therefore, assiduous efforts have to be made to monitor the neurological status by clinical and neuroim-
aging techniques to better determine high-risk populations that may benefit from neuroprotective strategies.

Acknowledgements

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References

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