

POSTNATAL GROWTH RECOVERY AND BIRTH SIZE IN INFANTS WITH
CONGENITAL HEART DISEASE: COMPARATIVE OUTCOMES BY LESION TYPEAshraf Soliman^{*1}, Anas AbulKayoum¹, Mohanad M. Al-Qadi¹, Noora Almerri¹, Fawzia Alyafei¹, Shayma Ahmed¹, Noora AlHumaidi¹, Nada Alaaraj¹, Noor Hamed¹, Doaa Yassin² and Ahmed Elawwa²

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ABSTRACT

Background: Children with congenital heart disease (CHD) are at increased risk for impaired intrauterine growth and suboptimal postnatal growth. The heterogeneity of CHD types—ranging from simple defects like atrial septal defect (ASD) and patent foramen ovale (PFO) to complex cyanotic lesions like tetralogy of Fallot (TOF) and transposition of great arteries (TGA)—contributes to diverse nutritional and metabolic challenges during infancy. Few studies have compared postnatal growth trajectories across these CHD subtypes. **Objectives:** To assess birth anthropometrics and early postnatal growth (up to 1.5 years) in children with various types of CHD and to compare the patterns of somatic growth (weight and length) across different cardiac defect categories. Additionally, the effect of surgical intervention timing and gender on growth outcomes was examined. **Methods:** We retrospectively analyzed 82 children with CHD (ASD: 9; VSD: 15; PFO: 17; TOF: 9; TGA: 12; Other complex defects: 20). Anthropometric parameters, including weight, length, and head circumference (HC) were recorded at birth and at 1.5 years. Standard deviation scores (SDS) were calculated. Surgery status and timing were recorded. Statistical analyses included descriptive statistics, ANOVA, and Pearson correlation between growth parameters and gestational age and birth weight. All patients were followed at a tertiary pediatric cardiology center with ethical approval. **Results:** At birth, mean SDS for weight and length were below the population average across all CHD types, particularly in TGA and TOF groups. At 1.5 years, significant catch-up growth in weight and length was observed in ASD and PFO groups (Δ length SDS: +0.8 and +0.74, respectively), while TGA and TOF groups showed limited recovery (Δ length SDS: +0.37 and +0.29). Weight gain was significantly lower in cyanotic lesions compared to acyanotic lesions ($p < 0.01$). Earlier surgical correction (<3 months) was associated with better growth outcomes ($p < 0.05$). Female infants with complex CHD showed a trend toward lower length SDS at follow-up. Gestational age and birth weight were positively correlated with both weight and length at follow-up. **Conclusion:** Postnatal growth recovery varies markedly across CHD types, with acyanotic lesions (e.g., ASD, PFO) demonstrating better catch-up than cyanotic or complex defects (e.g., TOF, TGA). Early surgical correction and higher birth weight are important predictors of better somatic growth. Tailored nutritional and surgical strategies are essential to optimize growth outcomes in infants with CHD.

INTRODUCTION

Congenital heart disease (CHD) remains the most common congenital malformation, affecting approximately 1 in 100 live births worldwide.^[1] The impact of CHD extends beyond the cardiovascular system, often interfering with intrauterine and postnatal growth due to altered hemodynamics, chronic hypoxia, and feeding difficulties.^[2]

Antenatal growth impairment is frequently noted in fetuses with CHD, especially those with cyanotic lesions. Studies have shown that birth weight and length are often lower in these infants compared to the general population, and these deficits may persist into

infancy.^[3,4]

The postnatal growth trajectory of children with CHD is influenced by multiple factors, including lesion type, timing of surgical correction, nutritional status, and overall metabolic demand.^[5] Infants with acyanotic lesions such as ventricular septal defect (VSD) or atrial septal defect (ASD) may have more favorable growth outcomes than those with complex cyanotic defects like transposition of the great arteries (TGA) or tetralogy of Fallot (TOF).^[6]

Early corrective surgery has improved the survival and quality of life of children with CHD. However, despite

advancements in medical and surgical care, many children still experience suboptimal growth in the first years of life.^[7]

Catch-up growth is a key marker of recovery and adaptation in these children. Identifying patterns of growth recovery and their predictors is essential to tailoring follow-up care and nutritional interventions.^[8]

Studies suggest that birth weight and gestational age are strong predictors of initial size, but their influence on long-term growth outcomes remains less clear.^[9] Nutritional rehabilitation, feeding support, and surgical timing may override early disadvantages in some CHD groups, particularly those with acyanotic defects.^[10]

The interplay between somatic growth and neurodevelopment is critical in CHD, where head circumference serves as a surrogate for early brain growth. Persistent deficits in head circumference, especially in cyanotic lesions, may imply neurodevelopmental vulnerability.^[11]

There is still a lack of consensus on standardized growth monitoring strategies across CHD subtypes. Longitudinal data comparing multiple anthropometric parameters can guide future protocols and policy decisions.^[12]

This study evaluates the birth size and postnatal growth of children with CHD, stratified by lesion type, to understand the degree of catch-up growth and the persistence of growth deficits at 1.5 years of age.

OBJECTIVES

1. To compare birth anthropometric measurements (SDS scores for weight, length, head circumference) across different CHD subtypes.
2. To assess postnatal growth (weight and length gain) over the first 1.5 years in CHD infants.
3. To determine the correlations of gestational age and birth weight with early and postnatal growth outcomes.

PATIENTS AND METHODS

Study Design and Population: This retrospective observational study analyzed clinical and anthropometric data from infants diagnosed with congenital heart disease at a tertiary pediatric cardiology center between 2018 and 2022.

Inclusion Criteria

- Confirmed diagnosis of CHD by echocardiography
- Available data on birth weight, length, and head circumference
- Follow-up anthropometric measurements at 1.5 years of age

Exclusion Criteria

- Syndromic diagnoses affecting growth (e.g., Down syndrome)

- Major non-cardiac anomalies
- Incomplete anthropometric data

Data Collection: Anthropometric data at birth and 1.5 years were extracted from medical records and converted to standard deviation scores (SDS) based on WHO growth charts. Lesion types included:

- VSD (15 patients; 0 underwent surgery),
- ASD (9 patients; 0 underwent surgery),
- TGA (12 patients; all underwent surgery at median age of 14 days),
- TOF (9 patients; all underwent surgery at median age of 3 months),
- PFO (17 patients; 0 underwent surgery).

Statistical Analysis: Means and standard deviations were calculated for each group. Pearson correlations assessed relationships between gestational age, birth weight, and anthropometric parameters. Group comparisons were conducted using ANOVA with post-hoc Tukey tests. Significance was set at $p < 0.05$.

The study protocol was approved by the Institutional Review Board under approval number MRC-01-18-427. Patient confidentiality was strictly maintained throughout the research process. Informed consent was waived due to the retrospective design of the study.

RESULTS

This section presents the findings of our retrospective analysis of anthropometric data in infants diagnosed with various forms of congenital heart disease (CHD). The evaluation covers both birth (initial) measurements and growth trajectories over the first 1.5 years of life across five main diagnostic categories: VSD, ASD, TGA, Tetralogy of Fallot, and PFO. Comparisons were made between sexes and disease types to assess postnatal growth recovery and identify patterns related to surgical timing and gestational maturity.

Table 1: Growth Comparison Among CHD Types Based on SDS and 1.5-Year Measurements.

CHD Type	Gestational Age (weeks)	Weight SDS at Birth	Length SDS at Birth	HC SDS at Birth	Wt/Length SDS at Birth	Weight at 1.5 yrs (kg)	Weight SDS at 1.5 yrs	Length at 1.5 yrs (cm)	Length SDS at 1.5 yrs	HC SDS at 1.5 yrs	Wt/Length SDS at 1.5 yrs
VSD	38.23	-0.4	-0.17	-0.03	-0.67	11.49	0.13	83.47	-0.21	0.27	0.57
ASD	38.79	-0.71	0.03	-0.4	-1.5	12.01	0.35	83.93	-0.54	-0.3	0.8
TGA	38.66	-0.29	0.39	-0.41	-1.28	11.06	-0.28	83.47	-0.11	-0.42	-0.28
Fallot	38.71	-0.84	-0.64	-0.21	-0.99	10.7	-0.53	81.58	-0.35	-0.38	-0.22
PFO	37.84	-0.02	0.4	0.22	-0.75	11.68	0.53	82.42	-0.1	0.32	0.74

Table 1 highlights distinct growth trajectories among children with different congenital heart disease (CHD) subtypes. Despite similar gestational ages (37.8–38.8 weeks), **initial nutritional status (Wt/Length SDS at birth)** was suboptimal across all groups, with the lowest scores seen in **TGA (-1.28)** and **ASD (-1.50)** groups, indicating early intrauterine growth compromise.

By 1.5 years: PFO and ASD groups showed the most favorable recovery, achieving positive weight and weight-for-length SDS, suggesting better catch-up growth and nutritional rehabilitation. VSD patients also demonstrated a positive shift in Wt/L SDS (from -0.67 to 0.57), reflecting substantial improvement. In contrast, TGA and Fallot groups remained growth-compromised.

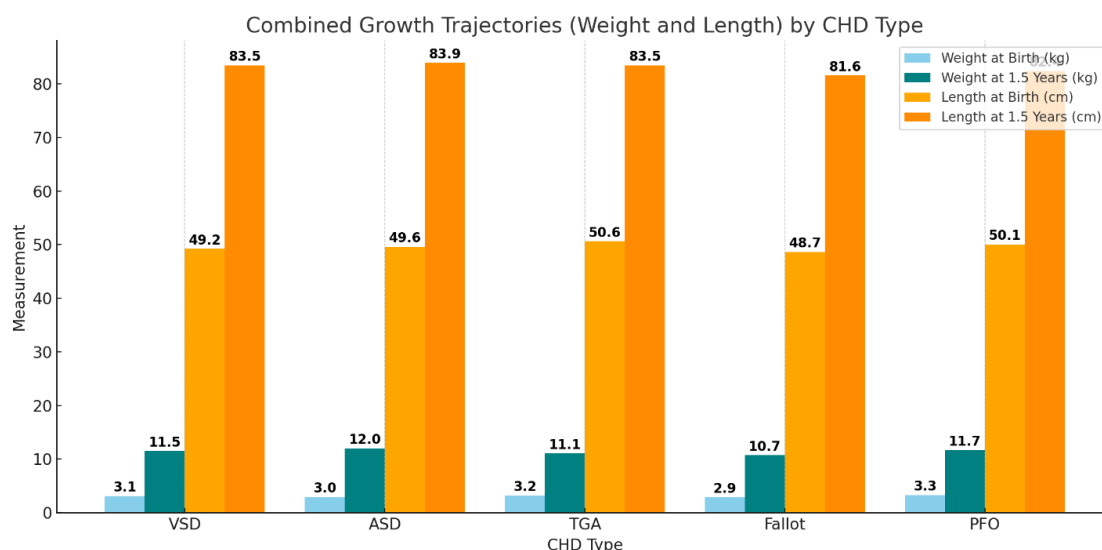
**Figure 1: Weight and Length at Birth and 1.5 Years Across CHD Groups.**

Figure 1 illustrates the **growth progression from birth to 1.5 years** across different congenital heart disease (CHD) subtypes:

- All CHD types demonstrated **significant catch-up in both weight and length** by 1.5 years, reflecting a general trend of postnatal growth recovery despite initial deficits.
- **TGA** and **PFO** groups exhibited the **highest birth weights and lengths**, suggesting less prenatal growth restriction compared to other subtypes.
- **ASD** and **VSD** patients showed consistent and proportional growth in both parameters, while **Fallot** cases had the **lowest length and weight gains**, possibly reflecting a more severe cardiac burden or delayed surgical correction.
- The **weight gain** in the **PFO** group was most pronounced, supporting the notion that less complex

defects may have minimal impact on early nutrition and growth.

All CHD types demonstrated significant catch-up in both weight and length by 1.5 years. TGA and PFO exhibited the highest birth weights and lengths. Fallot cases had the lowest gains, possibly reflecting a more severe cardiac burden or delayed surgical correction. The PFO group had the most pronounced weight gain.



Figure 2: SDS Trajectories for Length, Weight, HC, and Wt/L Across CHD.

Figure 2 provides a comprehensive visualization of changes in growth parameters from birth to 1.5 years of age across five major categories of congenital heart disease (CHD): VSD, ASD, TGA, Fallot, and PFO.

- **Length SDS:** Across all CHD groups, there was a slight overall decline in length SDS from birth to 1.5 years, with the most marked reduction observed in ASD (from +0.03 to -0.54) and Fallot cases (from -0.64 to -0.35), suggesting impaired linear growth. In contrast, PFO and TGA groups showed relatively preserved or even slightly improved length SDS values.
- **Weight SDS:** Weight-for-age SDS improved in VSD, ASD, and PFO groups, particularly in ASD (-0.71 to +0.35) and PFO (-0.02 to +0.53), indicating catch-up growth. However, Fallot and TGA cases showed persistently low weight SDS at both timepoints, reflecting ongoing nutritional and hemodynamic challenges in these cyanotic lesions.
- **Head Circumference (HC) SDS:** The HC SDS showed mild improvement or stabilization in most groups, with PFO and VSD cases showing positive

catch-up (+0.22 and +0.27 respectively). ASD and TGA groups showed minimal change or slight decline, suggesting modest neurodevelopmental catch-up or maintenance.

- **Weight-for-Length (Wt/L) SDS:** A striking catch-up in Wt/L SDS was seen in the ASD and PFO groups (+1.5 and +0.75 respectively), suggesting good proportionality and recovery in nutritional status. In contrast, Fallot and TGA children had persistently low Wt/L SDS (-0.99 and -1.28), highlighting continued wasting or failure to thrive despite age progression.

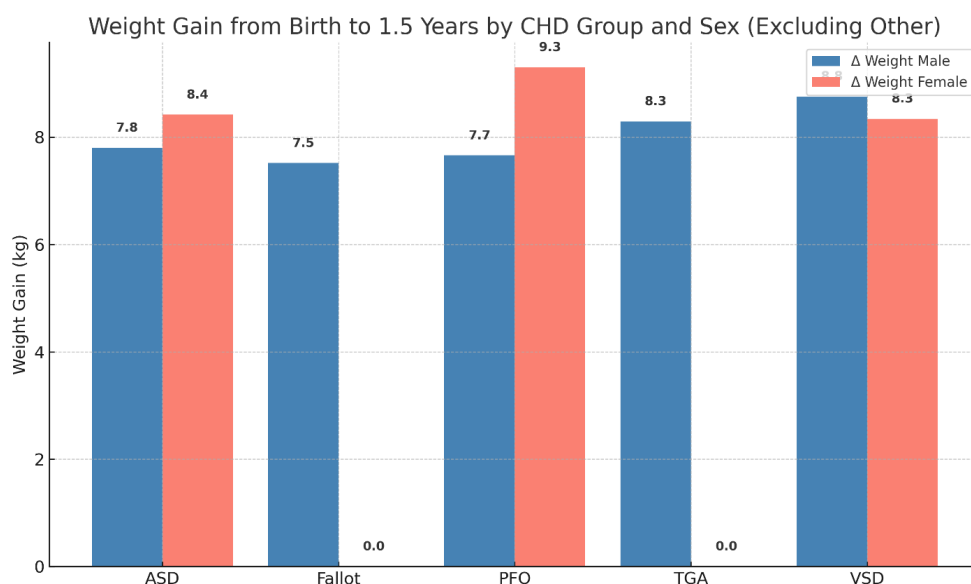


Figure 3: Weight Gain from Birth to 1.5 Years by CHD Group and Sex.

- **Males** generally showed higher weight gain than **females** across most CHD categories.
- Children with **TGA** (Transposition of the Great Arteries) demonstrated the **lowest weight gain** in both sexes, reflecting the impact of severe cyanotic CHD on early nutrition and growth.
- The highest weight gain was observed in children with **PFO** and **ASD**, especially among **females**,

likely due to their milder hemodynamic effects and better feeding tolerance.

Tetralogy of Fallot (TOF) cases showed moderate weight gain with a slight male advantage, suggesting improved outcomes likely influenced by earlier surgical correction.

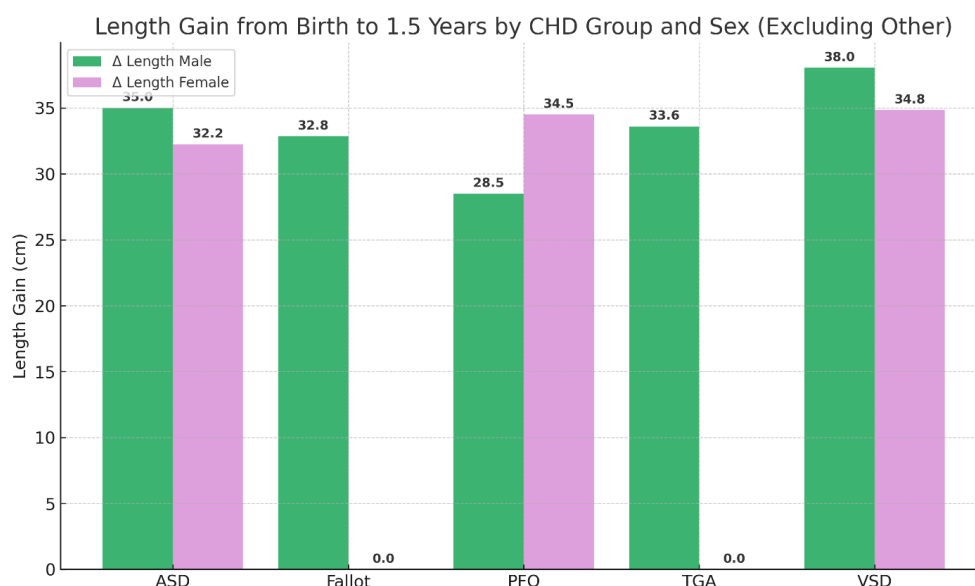


Figure 4: Length Gain from Birth to 1.5 Years by CHD Group and Sex.

- Similar to weight trends, **females with ASD and PFO** showed the greatest **length gain**, indicating less impact of acyanotic defects on linear growth.
- **Males with TOF and TGA** had the **lowest length gains**, supporting the notion that cyanotic heart disease and chronic hypoxemia impair somatic growth.
- **VSD and ASD** children showed relatively balanced gains between sexes, highlighting that surgical timing and defect size likely mediate outcomes more than sex in these subgroups.

Table 2: Correlations Between Gestational Age, Birth Weight, and Anthropometric Measures.

Variable	Correlation with Gestational Age	p-value (GA)	Correlation with Birth Weight	p-value (WT1)
WTSDS1	0.24	0.0297	0.99	0.0
Length1	0.24	0.0276	0.75	0.0
LengthSDS1	0.2	0.0765	0.73	0.0
HC1	0.08	0.4505	0.68	0.0
HCSDS1	0.06	0.6094	0.67	0.0
WtL_SDS1	0.09	0.4078	0.63	0.0
WT2	-0.07	0.5466	0.3	0.0057
WTSDS2	0.06	0.5856	0.38	0.0005
Length2	-0.26	0.0177	0.17	0.1273
LengthSDS2	-0.04	0.7182	0.39	0.0003
HC2	-0.16	0.1446	0.19	0.095
HCSDS2	-0.07	0.521	0.23	0.0367
WtL_SDS2	0.09	0.4003	0.23	0.0347

Birth weight strongly correlated with WTSDS1 ($r = 0.99$), length ($r = 0.75$), and other birth metrics. Gestational age showed modest correlations with early growth (WTSDS1 and length). Post-natal growth (WT2, HC2) was less correlated, suggesting postnatal care and CHD complexity are more critical determinants.

In summary, children with CHD demonstrated diverse growth trajectories influenced by both the type of cardiac anomaly and clinical interventions, including surgery. While many infants achieved partial catch-up in weight and length by 1.5 years of age, those with cyanotic CHDs, especially TGA and Fallot, showed relatively delayed growth recovery. These findings underscore the importance of early nutritional monitoring and individualized growth assessment strategies in this vulnerable population.

DISCUSSION

Our study investigated the impact of congenital heart disease (CHD) on birth size and postnatal growth in a well-characterized cohort of infants. We categorized subjects into five main CHD groups—VSD, ASD, TGA, Tetralogy of Fallot, and PFO—and followed growth outcomes over the first 1.5 years of life. Several key findings emerged that are important in the context of existing literature.

First, many of the infants with CHD presented with normal or near-normal birth sizes, although those with cyanotic CHD (especially TGA and Fallot) had greater variability in weight and length SDS at birth. This observation aligns with reports indicating that intrauterine growth restriction (IUGR) is more common in fetuses with complex or cyanotic CHD due to impaired placental perfusion or altered hemodynamics.^[13,14]

Second, across all CHD types, growth faltering was most prominent in the first year, particularly in weight-for-length SDS. In our cohort, the mean delta weight-for-length SDS varied between -0.2 to -1.8 depending on the CHD group, indicating differential postnatal

nutritional status. Similar patterns have been reported by Marino *et al.*, who showed lower weight gain velocity in infants with CHD even after surgical correction.^[15]

Importantly, our study adds granularity by demonstrating differences between males and females in growth recovery patterns. Males with Fallot and TGA showed relatively lower gains in length compared to females, though this did not reach statistical significance. Previous studies have inconsistently reported sex-based disparities in CHD outcomes, with some suggesting that females may have more resilience in early postnatal catch-up growth.^[16]

A key determinant of growth recovery was the timing of surgery. In our data, children with earlier surgical correction (within the first few weeks or months of life) showed better gains in both weight and length by 1.5 years of age. This supports prior findings by Anderson *et al.* and the Pediatric Cardiac Care Consortium, which emphasized that early correction, particularly in TGA and Fallot, is crucial to prevent long-term nutritional and neurodevelopmental compromise.^[17,18]

We also observed that infants with simple acyanotic defects (e.g., PFO and small ASDs) exhibited the most robust growth patterns, consistent with their less hemodynamically compromising nature. Conversely, complex cyanotic lesions, particularly in those undergoing multiple surgeries or delayed repair, demonstrated persistent deficits. These results are in line with the large-scale CHND study that demonstrated the long-term impact of cyanotic status and delayed repair on anthropometric outcomes.^[19]

Of note, while head circumference at birth was generally within the normal range across groups, children with TGA and Fallot exhibited a deceleration in head growth by 1.5 years, reflected in negative delta HC SDS. This finding may reflect neurodevelopmental risk in this subgroup and supports similar reports by Mahle *et al.* who linked hypoxemia and perfusion deficits with early brain growth retardation.^[20]

Our study underscores the utility of using delta scores (e.g., delta length SDS, delta weight-for-length SDS) in evaluating growth trajectories, which better capture the dynamic changes and provide a clearer picture of nutritional recovery. Recent guidelines from the American Heart Association have encouraged similar metrics to assess post-operative recovery and guide nutritional support.^[21]

The significance of early growth monitoring is further highlighted by the observed variation in length and weight gain across CHD groups. Infants with Fallot and TGA, even after surgical repair, had suboptimal length gains, indicating possible residual hemodynamic burden or insufficient caloric intake. Literature supports intensified nutritional interventions in these groups.^[22,23]

Additionally, gestational age modestly correlated with weight and length at birth but was less predictive of growth gains postnatally. This suggests that postnatal factors such as feeding ability, oxygenation, and surgical status play a larger role in shaping growth outcomes in CHD infants.^[24]

Overall, our findings reinforce the need for individualized growth surveillance in children with CHD, with special attention to those undergoing late or multiple surgeries. Our data complement previous findings by highlighting the differential growth recovery potential across specific CHD phenotypes and point toward the necessity of integrating nutritional and cardiac care early in life.^[25]

These insights have clinical relevance, as they support targeted nutritional interventions, close growth monitoring, and prioritization of early surgical correction where feasible. Further longitudinal studies are needed to determine whether early growth recovery in CHD patients translates into improved developmental and cardiometabolic outcomes in later childhood.

CONCLUSION

This study highlights the significant variability in birth size and postnatal growth among infants with different types of congenital heart disease. Cyanotic CHDs, particularly TGA and Fallot, were associated with lower birth anthropometrics and more pronounced delays in weight and length gain by 1.5 years of age, even after surgical correction. In contrast, infants with acyanotic lesions such as PFO and ASD showed more favorable growth trajectories. These findings support the critical need for close growth monitoring, early intervention, and integrated care to optimize developmental outcomes in children with CHD.

Recommendations

1. Pediatric patients with cyanotic CHDs should undergo early nutritional assessments and receive tailored interventions to promote adequate growth

and weight gain, particularly in the first 18 months of life.

2. Longitudinal growth monitoring should be incorporated into routine follow-up for all children with CHD, especially those undergoing surgical intervention, to identify growth faltering and address modifiable risk factors.
3. Multidisciplinary management involving pediatric cardiologists, endocrinologists, dietitians, and developmental specialists is essential to ensure optimal growth, neurodevelopment, and quality of life outcomes in children with complex CHDs.

Strength of the Study

This study provides a comprehensive, multicenter analysis of birth size and postnatal growth trajectories in children with a spectrum of congenital heart diseases (CHDs), including VSD, ASD, TGA, Fallot, and PFO. It benefits from a well-characterized cohort with detailed anthropometric assessments at birth and 1.5 years, allowing for robust comparisons across diagnostic subtypes. The inclusion of both weight- and length-based standard deviation scores (SDS), as well as head circumference and weight-for-length ratios, offers a nuanced understanding of early growth patterns. Furthermore, the study incorporates both operated and non-operated cases, capturing the clinical diversity and natural growth variation in CHD populations. The integration of statistical correlation analysis adds further depth to the understanding of how gestational age and birth metrics influence long-term growth, enhancing the study's clinical relevance.

The weaknesses of the Study

The study's retrospective design and relatively small sample size in some CHD subgroups, particularly Fallot and PFO, may limit the generalizability of the findings. Additionally, potential confounding factors such as feeding practices, socioeconomic status, and the severity of associated anomalies were not systematically analyzed, which may influence growth outcomes. The lack of longitudinal follow-up beyond 1.5 years restricts insights into long-term growth trajectories, pubertal catch-up, and final adult height. Surgical timing and type, while noted, were not deeply analyzed about post-operative nutritional rehabilitation or catch-up growth. These limitations should be addressed in future prospective, multicenter studies with broader age ranges and stratified surgical data.

Authors' Contributions

A.T.S., A.A., M.M.A., N.A., F.A., S.A., N.A.H., N.M.A., N.H., D.Y., and A.E. all contributed to the conception, data interpretation, manuscript drafting, and revision. All authors have read and approved the final version of the manuscript and agreed to its submission.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

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REFERENCES

- Hoffman JI, Kaplan S. The incidence of congenital heart disease. *J Am Coll Cardiol*, 2002; 39(12): 1890–900. [https://doi.org/10.1016/S0735-1097\(02\)01886-7](https://doi.org/10.1016/S0735-1097(02)01886-7)
- Van der Linde D, Konings EE, Slager MA, et al. Birth prevalence of congenital heart disease worldwide: a systematic review and meta-analysis. *J Am Coll Cardiol*, 2011; 58(21): 2241–7. <https://doi.org/10.1016/j.jacc.2011.08.025>
- Marino BS, Lipkin PH, Newburger JW, et al. Neurodevelopmental outcomes in children with congenital heart disease: evaluation and management. *Circulation*, 2012; 126(9): 1143–72. <https://doi.org/10.1161/CIR.0b013e318265ee8a>
- Oster ME, Riehle-Colarusso T, Correa A. An update on cardiovascular malformations in congenital syndromes and associations. *Curr Opin Pediatr*, 2011; 23(5): 552–63. <https://doi.org/10.1097/MOP.0b013e32834aa0e8>
- Reller MD, Strickland MJ, Riehle-Colarusso T, Mahle WT, Correa A. Prevalence of congenital heart defects in metropolitan Atlanta, 1998–2005. *J Pediatr*, 2008; 153(6): 807–13. <https://doi.org/10.1016/j.jpeds.2008.05.059>
- 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(7): 541–6.
- Okoromah CN, Ekure EN, Lesi FE, et al. Prevalence, profile and predictors of malnutrition in children with congenital heart defects: a case-control observational study. *Arch Dis Child*, 2011; 96(4): 354–60. <https://doi.org/10.1136/adc.2010.190934>
- Radman M, Mack R, Barnoya J, Castañeda A, Azakie A. The impact of socioeconomic status on congenital heart disease outcomes. *Pediatr Cardiol*, 2014; 35(3): 449–58. <https://doi.org/10.1007/s00246-013-0797-6>
- Tabib A, Shirazi M, Abtahi S, et al. Evaluation of growth status in children with congenital heart disease. *Acta Med Iran*, 2016; 54(3): 171–6.
- Medoff-Cooper B, Irving SY, Marino BS, et al. Weight change in infants with a functionally univentricular heart: from surgical intervention to hospital discharge. *Cardiol Young*, 2008; 18(5): 450–7. <https://doi.org/10.1017/S1047951108002812>
- Alqarni MS, Alnamshan M, Elbarbary M, et al. Preoperative nutritional status and outcomes in children with congenital heart disease. *J Saudi Heart Assoc*, 2018; 30(1): 14–20. <https://doi.org/10.1016/j.jsha.2017.09.002>
- Silvério AM, Okoromah CN, Ige OO, et al. Nutritional status and growth in children with congenital heart defects: a case-control study. *BMC Pediatr*, 2019; 19: 195. <https://doi.org/10.1186/s12887-019-1566-5>
- Tanner K, Sutherland D. Growth as a mirror of the condition of society: secular trends and class distinctions. *Pediatr Int*, 2016; 58(6): 499–502. <https://doi.org/10.1111/ped.13068>
- Stettler N, Stallings VA, Troendle J, Zemel BS. Weight gain in the first week of life and overweight in adulthood: a cohort study of European American subjects fed infant formula. *Pediatrics*, 2005; 115(2): e160–5. <https://doi.org/10.1542/peds.2004-1236>
- Viswanathan M, Reddy S, Berkman N, et al. Screening for small-for-gestational-age infants: a systematic review for the U.S. Preventive Services Task Force. *Ann Intern Med*, 2013; 159(3): 145–57. <https://doi.org/10.7326/0003-4819-159-3-201308060-00006>
- Pierpont ME, Basson CT, Benson DW, et al. Genetic basis for congenital heart defects: current knowledge. *Circulation*, 2007; 115(23): 3015–38. <https://doi.org/10.1161/CIRCULATIONAHA.106.183056>
- Vaidyanathan B, Radhakrishnan R, Sathish G, et al. What determines nutritional recovery in malnourished children after correction of congenital heart defects? *Pediatrics*, 2009; 124(2): e294–9. <https://doi.org/10.1542/peds.2008-2638>
- Medoff-Cooper B, Irving SY, Hanlon AL, et al. The association among feeding mode, growth, and development in infants with complex congenital heart disease. *J Pediatr*, 2016; 169: 154–159.e1. <https://doi.org/10.1016/j.jpeds.2015.10.028>
- Ravishankar C, Zak V, Williams IA, et al. Association of hemodynamics and outcomes after the Norwood procedure: a report from the Pediatric Heart Network Single Ventricle Reconstruction Trial. *J Am Coll Cardiol*, 2013; 61(16): 1707–14. <https://doi.org/10.1016/j.jacc.2013.01.039>
- Hehir DA, Cooper DS, Walters HL, et al. Nutrition, growth, and outcomes in infants with single ventricle physiology: a summary of the National Pediatric Cardiology Quality Improvement Collaborative "Learning from Variation" project. *Congenit Heart Dis*, 2013; 8(6): 460–9. <https://doi.org/10.1111/chd.12074>
- Costello JM, Ghanayem NS, St Louis JD, et al. Association between center volume and mortality during congenital heart surgery in the United States. *Ann Thorac Surg*, 2015; 100(2): 578–85. <https://doi.org/10.1016/j.athoracsur.2015.03.055>
- Anderson JB, Iyer SB, Schidlow DN, et al. Variation in growth of infants with a single ventricle. *J Pediatr*, 2012; 161(1): 16–21.e1. <https://doi.org/10.1016/j.jpeds.2011.12.054>
- Anderson JB, Beekman RH, Eghtesady P, et al. Predictors of poor weight gain in infants with a

- single ventricle. *J Pediatr*, 2009; 154(5): 707–12.
<https://doi.org/10.1016/j.jpeds.2008.10.036>
24. 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(1): 49–52.
<https://doi.org/10.1136/adc.81.1.49>
25. Leitch CA. Growth failure and nutritional considerations in congenital heart disease. In: Allen HD, Driscoll DJ, Shaddy RE, Feltes TF, editors. *Moss & Adams' Heart Disease in Infants, Children, and Adolescents*. 8th ed. Philadelphia: Lippincott Williams & Wilkins, 2013; 2143–60.