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CONGENITAL HEART DISEASE AND GROWTH FAILURE IN THE FIRST TWO YEARS OF LIFE

CARDIOPATIA CONGÊNITA E FALHA DO CRESCIMENTO NOS PRIMEIROS DOIS ANOS DE VIDA

CARDIOPATÍA CONGÉNITA E INSUFICIENCIA DEL CRECIMIENTO EN LOS PRIMEROS DOS AÑOS DE VIDA

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ABSTRACT

Introduction: Growth failure is a frequent complication of congenital heart disease (CHD), particularly among infants with cyanotic lesions, pulmonary overcirculation, complex biventricular defects, or functionally univentricular circulation. Its pathogenesis extends beyond inadequate caloric intake and includes altered fetal growth, hypoxemia, increased energy expenditure, intestinal hypoperfusion, fluid restriction, perioperative catabolism, prematurity, genetic syndromes, dysphagia, and prolonged hospitalization. Surgical or catheter-based correction improves hemodynamics but does not invariably result in complete nutritional recovery. Objective: To evaluate growth failure and subsequent nutritional recovery in infants with CHD during the first two years of life, identify the main clinical predictors of persistent impairment, and analyze the effects of nutritional and feeding interventions. Methods: A structured narrative review was conducted using PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library. Longitudinal cohorts, multicenter database studies, randomized feeding trials, systematic reviews, and multidisciplinary clinical guidance were prioritized. Outcomes included weight-for-age, length-for-age, weight-for-length, head-circumference z scores, body composition, feeding disorders, tube dependence, morbidity, mortality, and neurodevelopment. Results and Discussion: The greatest simultaneous reductions in weight and length commonly occur during the first four postoperative months. Complex CHD, single-ventricle physiology, low birth weight, prematurity, genetic disorders,

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prolonged ventilation, diuretic exposure, and tube feeding at discharge predict less favorable trajectories. Feeding or swallowing difficulties affect approximately 43% of children with CHD, aspiration approximately 33%, and tube feeding at discharge approximately 31%. Randomized studies indicate that energy- and protein-enriched feeding can improve short-term weight-related outcomes and nutritional recovery, although intolerance may increase and evidence for sustained linear growth, lean-mass accretion, oral autonomy, or neurodevelopment remains limited. Conclusion: Cardiac correction provides an opportunity for nutritional recovery but should not be considered an automatic nutritional cure. Monitoring should combine weight, length, weight-for-length, head circumference, feeding safety, actual nutrient delivery, and developmental assessment. Nutritional therapy should be individualized according to cardiac physiology, fluid allowance, prematurity, swallowing function, and family capacity. The therapeutic target is safe, proportional catch-up growth rather than rapid weight gain alone.

Keywords: Heart Defects. Congenital. Growth Disorders. Failure to Thrive. Infant Nutrition. Cardiac Surgery. Infant.

RESUMO

Introdução: O fracasso no crescimento é uma complicação frequente da cardiopatia congênita (CC), particularmente entre recém-nascidos com lesões cianóticas, sobrefluxo pulmonar, defeitos biventriculares complexos ou circulação funcionalmente univentricular. Sua patogênese estende-se além da ingestão calórica inadequada e inclui crescimento fetal alterado, hipoxemia, aumento do gasto energético, hipoperfusão intestinal, restrição de fluidos, catabolismo perioperatório, prematuridade, síndromes genéticas, disfagia e hospitalização prolongada. A correção cirúrgica ou baseada em cateter melhora a hemodinâmica, mas não resulta invariavelmente em recuperação nutricional completa. Objetivo: Avaliar o fracasso no crescimento e a subsequente recuperação nutricional em recém-nascidos com CC durante os primeiros dois anos de vida, identificar os principais preditores clínicos de comprometimento persistente e analisar os efeitos das intervenções nutricionais e de alimentação. Métodos: Uma revisão narrativa estruturada foi conduzida usando PubMed/MEDLINE, Scopus, Web of Science e a Biblioteca Cochrane. Coortes longitudinais, estudos de banco de dados multicêntricos, ensaios alimentares randomizados, revisões sistemáticas e orientação clínica multidisciplinar foram priorizados. Os desfechos incluíram escore z de peso-por-idade, comprimento-por-idade, peso-por-comprimento, perímetro cefálico, composição corporal, transtornos de alimentação, dependência de sonda, morbidade, mortalidade e neurodesenvolvimento. Resultados e Discussão: As maiores reduções simultâneas de peso e comprimento comumente ocorrem durante os primeiros quatro meses pós-operatórios. Cardiopatia congênita complexa, fisiologia de ventrículo único, baixo peso ao nascimento, prematuridade, transtornos genéticos, ventilação prolongada, exposição a diuréticos e alimentação por sonda na alta predizem trajetórias menos favoráveis. Dificuldades de alimentação ou deglutição afetam aproximadamente 43% das crianças com CC, aspiração aproximadamente 33% e alimentação por sonda na alta aproximadamente 31%. Estudos randomizados indicam que a alimentação enriquecida em energia e proteína pode melhorar os desfechos relacionados ao peso a curto prazo e a recuperação nutricional, embora a intolerância possa aumentar e as evidências de crescimento linear sustentado, acúmulo de massa magra, autonomia oral ou neurodesenvolvimento permaneçam limitadas. Conclusão: A correção cardíaca oferece uma oportunidade para recuperação nutricional, mas não deve ser considerada uma cura nutricional automática. O monitoramento deve combinar peso, comprimento, peso-por-

comprimento, perímetro cefálico, segurança da alimentação, aporte real de nutrientes e avaliação do desenvolvimento. A terapia nutricional deve ser individualizada de acordo com a fisiologia cardíaca, limite de fluidos, prematuridade, função de deglutição e capacidade familiar. O alvo terapêutico é crescimento de recuperação seguro e proporcional, em vez de ganho de peso rápido isoladamente.

Palavras-chave: Cardiopatias Congênitas. Transtornos do Crescimento. Fracasso no Crescimento. Nutrição do Recém-nascido. Cirurgia Cardíaca. Recém-nascido.

RESUMEN

Introducción: El retraso del crecimiento es una complicación frecuente de la cardiopatía congénita (CC), particularmente en lactantes con lesiones cianóticas, sobrecirculación pulmonar, defectos biventriculares complejos o circulación univentricular funcionalmente. Su patogénesis va más allá de la ingesta calórica inadecuada e incluye crecimiento fetal alterado, hipoxemia, aumento del gasto energético, hipoperfusión intestinal, restricción de líquidos, catabolismo perioperatorio, prematuridad, síndromes genéticos, disfagia e hospitalización prolongada. La corrección quirúrgica o intervencionista mejora la hemodinámica pero no siempre resulta en una recuperación nutricional completa. Objetivo: Evaluar el retraso del crecimiento y la subsecuente recuperación nutricional en lactantes con CC durante los primeros dos años de vida, identificar los principales predictores clínicos del deterioro persistente, y analizar los efectos de intervenciones nutricionales y de alimentación. Métodos: Se realizó una revisión narrativa estructurada utilizando PubMed/MEDLINE, Scopus, Web of Science y la Biblioteca Cochrane. Se priorizaron cohortes longitudinales, estudios de bases de datos multicéntricos, ensayos aleatorizados de alimentación, revisiones sistemáticas y guías clínicas multidisciplinarias. Los resultados incluyeron puntuaciones z de peso para la edad, talla para la edad, peso para la talla, perímetro cefálico, composición corporal, trastornos de la alimentación, dependencia de sonda, morbilidad, mortalidad y neurodesarrollo. Resultados y Discusión: Las reducciones simultáneas más grandes en peso y talla comúnmente ocurren durante los primeros cuatro meses postoperatorios. La CC compleja, fisiología univentricular, bajo peso al nacer, prematuridad, trastornos genéticos, ventilación prolongada, exposición a diuréticos y alimentación por sonda al alta predicen trayectorias menos favorables. Las dificultades de alimentación o deglución afectan aproximadamente al 43% de los niños con CC, la aspiración aproximadamente al 33%, y la alimentación por sonda al alta aproximadamente al 31%. Los estudios aleatorizados indican que la alimentación enriquecida en energía y proteína puede mejorar los resultados relacionados con el peso a corto plazo y la recuperación nutricional, aunque la intolerancia puede aumentar y la evidencia de crecimiento lineal sostenido, acumulación de masa magra, autonomía oral o neurodesarrollo sigue siendo limitada. Conclusión: La corrección cardíaca proporciona una oportunidad para la recuperación nutricional pero no debe considerarse una cura nutricional automática. El monitoreo debe combinar peso, talla, peso para la talla, perímetro cefálico, seguridad de la alimentación, aporte real de nutrientes y evaluación del desarrollo. La terapia nutricional debe individualizarse según la fisiología cardíaca, el límite de líquidos, la prematuridad, la función de deglución y la capacidad familiar. El objetivo terapéutico es un crecimiento de recuperación seguro y proporcional en lugar de solo ganancia rápida de peso.

Palabras clave: Cardiopatías Congénitas. Trastornos del Crecimiento. Retraso en el Crecimiento. Nutrición Infantil. Cirugía Cardíaca. Lactante.

1 INTRODUCTION

Congenital heart disease (CHD) is the most common major congenital anomaly and represents an important cause of infant morbidity and mortality. A global meta-analysis of 260 studies and more than 130 million live births estimated a prevalence of 9.41 cases per 1,000 births between 2010 and 2017. Improvements in prenatal diagnosis, neonatal surgery, catheter-based treatment, intensive care, and interstage surveillance have shifted clinical attention from survival alone toward growth, neurodevelopment, and quality of life.[1]

Growth impairment may begin before surgery and, in some patients, before birth. A longitudinal matched cohort showed that children requiring single-ventricle palliation or complex biventricular repair experienced simultaneous early reductions in weight-for-age z score (WAZ), length-for-age z score (LAZ), and head-circumference-for-age z score (HCZ). The steepest deterioration occurred during the first months of life, with the largest average deficit near four months and residual abnormalities extending into the second and third years.[2]

Growth failure in CHD is not explained solely by insufficient caloric intake. Tachypnea, sympathetic activation, recurrent infection, myocardial workload, and feeding effort may increase energy expenditure. Pulmonary overcirculation may cause respiratory distress and feeding fatigue, while cyanosis can reduce systemic oxygen delivery. Mesenteric hypoperfusion, venous congestion, gastrointestinal dysmotility, and fluid restriction may further limit the volume and tolerance of enteral feeding.[3]

Cardiac surgery introduces additional nutritional stress. Preoperative fasting, systemic inflammation, cardiopulmonary bypass, postoperative fluid shifts, mechanical ventilation, delayed enteral feeding, and protein catabolism may intensify an existing nutritional deficit. Nutrition must consequently be integrated into hemodynamic and perioperative management rather than considered a separate intervention initiated only after severe malnutrition develops.[3]

Feeding performance constitutes another major pathway. Infants with complex CHD may have impaired endurance, immature suck–swallow–breathe coordination, vocal-cord dysfunction, recurrent laryngeal nerve injury, prolonged intubation, gastroesophageal symptoms, or oral aversion after repeated invasive procedures. Supplemental enteral feeding may preserve nutrient delivery, but it does not automatically correct impaired swallowing or promote the acquisition of oral feeding skills.[4]

A scoping review of 15 studies involving 1,107 participants estimated that feeding or swallowing difficulties affected 42.9% of children with CHD. Aspiration was identified in 32.9%, and 31.3% required tube feeding at discharge. The estimated prevalence of feeding or swallowing difficulty was higher in predominantly complex CHD than in predominantly acyanotic disease, emphasizing that feeding impairment is part of the clinical phenotype of severe cardiac disease.[5]

Weight alone is an incomplete measure of nutritional recovery. A longitudinal body-composition study found lower weight, length, and head-circumference z scores at three, six, and nine months among infants with complex CHD. Mid-upper-arm circumference was also reduced during early follow-up, while some infants with single-ventricle physiology displayed relatively greater skinfold thickness despite smaller overall anthropometric measurements. Rapid weight gain can therefore conceal persistent deficits in linear growth or lean-tissue accretion.[6]

Anatomical complexity influences growth but does not completely determine it. In a prospective cohort of 801 infants undergoing cardiac repair, complex CHD was associated with lower postoperative WAZ and weight-for-length z score (WLZ) than simple CHD. The interaction between cardiac complexity and preoperative growth was also significant, suggesting that the nutritional trajectory before surgery modifies the probability of postoperative recovery.[7]

Subgroups within complex CHD also follow heterogeneous trajectories. Among 178 infants followed during the first four months after neonatal cardiac surgery, 14% maintained WAZ above zero, 35% remained approximately stable, 28% partially recovered, and 23% never recovered. Persistent failure was associated with longer hospitalization, tube feeding at discharge, and reduced appetite. These findings support repeated assessment of the individual trajectory rather than reliance on a single malnutrition threshold.[8]

The first two years of life constitute a critical period for somatic growth, brain development, feeding-skill acquisition, and family adaptation. Understanding when growth deterioration occurs, which infants remain vulnerable after correction, and which interventions produce clinically meaningful recovery is therefore essential. Nutritional success should be defined by proportional growth and functional development rather than by weight normalization in isolation.

2 OBJECTIVES

2.1 PRIMARY OBJECTIVE

To evaluate growth failure and nutritional recovery among infants with congenital heart disease from birth through the first two years of life, including the effects of surgical or catheter-based correction.

2.2 SECONDARY OBJECTIVES

1. To characterize changes in weight, length, weight-for-length, head circumference, and body composition during the preoperative, postoperative, interstage, and post-repair periods.
2. To compare growth risk among simple acyanotic disease, pulmonary overcirculation, cyanotic lesions, complex biventricular CHD, and functionally univentricular circulation.
3. To evaluate the effects of prematurity, fetal growth restriction, genetic syndromes, hypoxemia, heart failure, prolonged hospitalization, and diuretic exposure on growth.
4. To determine the contribution of dysphagia, aspiration, feeding intolerance, and tube dependence to persistent growth failure.
5. To analyze the effects of human milk, fortification, energy- and protein-enriched formulas, enteral support, standardized nutritional pathways, and home-monitoring programs.
6. To examine the relationship between early growth trajectory and neurodevelopment.

3 METHODOLOGY

3.1 REVIEW DESIGN

A structured narrative review was undertaken because the available evidence includes population databases, prospective and retrospective cohorts, body-composition studies, feeding and swallowing investigations, randomized nutritional trials, quality-improvement programs, and expert guidance with heterogeneous populations and outcomes.

3.2 INFORMATION SOURCES AND SEARCH STRATEGY

PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library were searched from database inception through August 31, 2026. Search terms included combinations of “congenital heart disease,” “heart defect,” “single ventricle,” “cyanotic,” “infant,” “growth failure,” “failure to thrive,” “malnutrition,” “weight-for-age,” “length-for-age,” “weight-for-length,” “head circumference,” “body composition,” “catch-up growth,” “cardiac

surgery," "feeding," "dysphagia," "aspiration," "human milk," "fortification," "high-energy formula," "tube feeding," and "home monitoring."

Reference lists of relevant systematic reviews, longitudinal studies, randomized trials, and professional guidance were also examined. Recent evidence was prioritized, while older studies were retained when they provided foundational longitudinal or interventional data.

3.3 ELIGIBILITY CRITERIA

Studies were prioritized when they:

1. Included infants or children with CHD during the first two years of life.
2. Reported serial anthropometry, nutritional status, body composition, feeding ability, swallowing outcomes, or tube dependence.
3. Evaluated nutritional or feeding interventions.
4. Provided quantitative estimates or adjusted associations.
5. Examined relationships between growth and clinical or neurodevelopmental outcomes.

Adult-only studies, isolated case reports, and studies without an interpretable pediatric growth or feeding outcome were not included as principal evidence.

3.4 OUTCOMES AND CLINICAL PHENOTYPES

Growth outcomes included WAZ, LAZ, WLZ, HCZ, body mass index z score, growth velocity, mid-upper-arm circumference, skinfold thickness, body composition, and malnutrition below −2 standard deviations. Feeding outcomes included oral intake, dysphagia, aspiration, feeding intolerance, nasogastric or gastrostomy dependence, and oral feeding at discharge.

Cardiac phenotypes were classified as:

1. Simple repaired or acyanotic CHD.
2. Pulmonary overcirculation or heart-failure physiology.
3. Cyanotic CHD.
4. Complex biventricular CHD.
5. Functionally univentricular circulation.

Prematurity, low birth weight, fetal growth restriction, genetic syndromes, feeding route, dysphagia, and hospitalization were analyzed as modifiers of growth rather than interchangeable measures of cardiac severity.

3.5 CRITICAL APPRAISAL

Priority was given to prospective design, multicenter enrollment, repeated anthropometric measurements, standardized z scores, adjustment for gestational age and disease complexity, and follow-up beyond the index hospitalization. Randomized allocation strengthened short-term nutritional comparisons, although open-label treatment, selected malnourished populations, intolerance, and limited neurodevelopmental follow-up reduced certainty.

Associations between tube feeding and poor growth were interpreted cautiously because tube dependence frequently reflects greater cardiac complexity or feeding dysfunction. Historical-control studies and home-monitoring programs were considered vulnerable to secular changes in surgical and intensive-care practice.

4 RESULTS

4.1 GROWTH TRAJECTORIES

Early growth failure was most pronounced among infants undergoing complex neonatal surgery or staged single-ventricle palliation. Weight and length frequently declined together during the first month, reached their greatest average deficit near four months, and then demonstrated variable recovery. Length and head circumference often recovered more slowly than weight.[2]

Preoperative nutritional status influenced postoperative trajectory. In the prospective study of 801 infants, complex CHD was associated with an adjusted postoperative WAZ reduction of −0.72 and an adjusted WLZ reduction of −0.93 compared with simple CHD. The interaction between preoperative growth and disease complexity suggested that cardiac correction did not erase the nutritional deficit present at the time of surgery.[7]

Low birth size was associated with operative risk across the full birth-weight distribution. In 25,244 neonates and young infants undergoing cardiac surgery, operative mortality was 8.6% and major complications occurred in 19.4%. Relative to a birth-weight z score between 0 and 0.5, adjusted mortality odds were 1.4 for mild growth restriction, 1.7 for moderate restriction, and 2.4 for severe restriction.[9]

Single-ventricle populations displayed meaningful catch-up but remained vulnerable to persistent linear-growth impairment. In the Single Ventricle Reconstruction Trial, growth improved after staged palliation; however, at three years, 6% of children with hypoplastic left

heart syndrome had WAZ below −2 and 18% had LAZ below −2. Weight therefore recovered more completely than length in many children.[10]

Persistent growth failure was not limited to single-ventricle disease. Among 331 children assessed between six and 12 months after cardiac surgery during infancy, 16% had at least one growth measurement below −2 standard deviations and 14% had a weight-for-height value below −2. Genetic syndromes, birth weight of 3 kg or less, complex CHD, surgery after 30 days, prenatal diagnosis, and continued diuretic use were associated with greater risk.[11]

Table 1

Principal quantitative studies of early growth in congenital heart disease

Study	Sample	Design and period	Outcomes	Principal findings
Daymont et al., 2013[2]	856 children with CHD, matched 10:1 with controls	Longitudinal cohort through 36 months	WAZ, LAZ, HCZ	Surgical groups had simultaneous weight and length decline during the first month, maximal deficits near four months, and persistent separation through 24–36 months
Shi et al., 2021[7]	801 infants undergoing cardiac repair	Prospective simple-versus-complex CHD cohort	WAZ, WLZ, LAZ	Complex CHD was associated with adjusted WAZ β −0.72 and WLZ β −0.93; preoperative trajectory modified postoperative growth
Lisanti et al., 2022[8]	178 infants after neonatal surgery for complex CHD	Latent WAZ trajectories during four months	Growth-trajectory class	14% maintained WAZ above zero, 35% remained stable, 28% partially recovered, and 23% never recovered
Steurer et al., 2021[9]	25,244 infants undergoing cardiac surgery	Multicenter surgical database	Mortality and major complications	Adjusted mortality odds increased progressively from 1.4 with mild to 2.4 with severe birth-weight restriction
Burch et al., 2014[10]	Infants with hypoplastic left heart syndrome	Longitudinal Single Ventricle Reconstruction Trial analysis	WAZ and LAZ	At three years, WAZ was below −2 in 6% and LAZ below −2 in 18%
Brief et al., 2022[11]	331 infants operated before one year	Assessment 6–12 months after surgery	Growth failure and weight-for-height	Growth failure occurred in 16%, and weight-for-height below −2 occurred in 14%
Irving et al., 2022[6]	120 infants, 58% with complex CHD	Longitudinal anthropometry to nine months	WAZ, LAZ, HCZ, arm circumference, skinfolds	CHD was associated with lower WAZ, LAZ, and HCZ at three, six, and nine months and lower arm circumference early in follow-up

Study	Sample	Design and period	Outcomes	Principal findings
Tseng et al., 2021[24]	69 infants after bidirectional Glenn surgery	Head growth through 12 months after surgery	HCZ and motor outcomes	Mean HCZ increased from −1.13 to 0.35; 66.7% increased by more than one z score, but accelerated growth did not predict motor scores

CHD: congenital heart disease; HCZ: head-circumference-for-age z score; LAZ: length-for-age z score; WAZ: weight-for-age z score; WLZ: weight-for-length z score.
Source: Prepared by the authors.

4.2 FEEDING AND SWALLOWING OUTCOMES

Feeding and swallowing impairment was common, especially after complex neonatal surgery. The pooled prevalence of feeding or swallowing difficulty was 42.9%, aspiration 32.9%, and tube feeding at discharge 31.3%.[5] Silent aspiration was an important concern because the absence of cough or choking did not reliably exclude unsafe swallowing.

A systematic review found that approximately 30%–50% of infants undergoing neonatal surgery for CHD failed to achieve complete oral feeding by discharge. Preoperative feeding difficulty, prolonged fasting, extended mechanical ventilation, vocal-cord dysfunction, and low early postoperative oral intake were recurrent predictors of tube dependence.[17]

Tube-assisted feeding supported adequate nutrient delivery but was also a marker of greater illness and feeding complexity. In a single-ventricle cohort, children discharged with tube-assisted feeding had an adjusted mean WAZ approximately 0.56 lower during subsequent follow-up than orally fed children. This association cannot be interpreted as evidence that the feeding tube itself caused poor growth.[18]

4.3 NUTRITIONAL INTERVENTIONS

Standardized nutritional pathways were associated with attenuation of early growth decline. In a pathway study comparing 44 intervention infants with 38 historical controls, the reduction in WAZ from birth to four months was −0.9 in the intervention group and −1.5 among controls. At 12 months, WAZ and LAZ were also more favorable in the pathway group, and pediatric intensive-care stay was shorter.[19]

Randomized trials of high-energy feeding demonstrated improved nutrient delivery and short-term weight-related outcomes. In one trial involving 50 infants, protein- and energy-enriched formula allowed adequate intake and positive nitrogen balance to be achieved earlier. However, diarrhea occurred more frequently, with an adjusted hazard ratio of 3.16.[20]

Among 100 infants after surgery for cyanotic CHD, malnutrition at six months occurred in 18% of those receiving calorie-enriched formula and 36% of those receiving standard formula. Although this represented an absolute difference of 18 percentage points, the finding originated from a single study and requires replication.[21]

The largest randomized trial enrolled 244 infants younger than six months with moderate or severe malnutrition after cardiac surgery. Early high-energy feeding improved WAZ and weight-for-height z scores at intensive-care discharge. Nutritional recovery at three months occurred in 19.4% of the intervention group and 6.5% of controls, while postoperative infection occurred in 3.2% and 12.5%, respectively.[22]

Table 2

Nutritional, feeding, and surveillance interventions

Study	Population	Intervention or exposure	Principal result	Interpretation
Norman et al., 2022[5]	15 studies; 1,107 participants	Review of feeding and swallowing disorders	Feeding/swallowing difficulty 42.9%; aspiration 32.9%; tube feeding at discharge 31.3%	Feeding safety and competence are major determinants of nutritional risk
Elgersma et al., 2022[15]	16 studies	Human-milk exposure	Exclusive human milk was associated with lower necrotizing enterocolitis risk; growth evidence was heterogeneous	Human milk should be protected but may require fortification
Marino et al., 2020[19]	44 pathway infants and 38 historical controls	Multidisciplinary nutritional pathway	Birth-to-four-month WAZ change −0.9 versus −1.5; more favorable WAZ and LAZ at 12 months	Standardization may reduce preventable nutritional deterioration
Cui et al., 2018[20]	50 postoperative infants	Enriched versus standard formula	Earlier adequate intake and positive nitrogen balance; diarrhea HR 3.16	Higher density improves delivery but may reduce tolerance
Zhou et al., 2021[21]	100 infants after surgery for cyanotic CHD	Calorie-enriched versus standard formula	Malnutrition at six months: 18% versus 36%	Medium-term weight benefit is possible but requires confirmation
Chen et al., 2021[22]	244 malnourished infants after surgery	Early high-energy feeding versus usual care	Recovery at three months: 19.4% versus 6.5%; infection: 3.2% versus 12.5%	Selected malnourished infants may derive short-term clinical benefit
Scott et al., 2025[23]	Single-ventricle cohorts	Home-monitoring program	Similar average interstage growth velocity but less early deceleration and later weight restoration	Monitoring may facilitate earlier identification and intervention

CHD: congenital heart disease; HR: hazard ratio; LAZ: length-for-age z score; WAZ: weight-for-age z score.
Source: Prepared by the authors.

5 RESULTS AND DISCUSSION

5.1 GROWTH FAILURE BEGINS BEFORE SURGICAL CORRECTION

The conventional explanation that cardiac surgery alone causes growth failure is incomplete. Fetal growth restriction, placental abnormalities, altered cerebral and systemic oxygen delivery, genetic conditions, and prematurity may establish nutritional vulnerability before the first postnatal feeding. Birth size therefore reflects clinically relevant prenatal biology as well as gestational age.[9]

After birth, the direction and magnitude of cardiac shunting influence energy requirements and feeding tolerance. A large left-to-right shunt may cause tachypnea, sweating, hepatomegaly, and feeding fatigue even in the absence of cyanosis. Cyanotic lesions may combine reduced oxygen delivery with increased metabolic and respiratory demands. Duct-dependent systemic circulation and functionally univentricular physiology may additionally compromise systemic and mesenteric perfusion.[3]

The same prescribed caloric intake consequently does not represent the same biological exposure in every lesion. One infant may absorb and use an enteral prescription efficiently, while another expends a considerable proportion of the delivered energy on respiratory and cardiac work. Actual nutrient delivery must therefore be interpreted in the context of physiology rather than compared with a single universal caloric target.[3]

Intensive-care measurements may also be misleading. Short-term weight changes can reflect edema, fluid resuscitation, delayed chest closure, diuresis, or removal of invasive support rather than tissue accretion. Length and head circumference are more difficult to measure but provide essential context. Growth assessment should employ repeated measurements, corrected age for premature infants, and the same reference standard over time.[6]

5.2 GROWTH TRAJECTORY ACCORDING TO CARDIAC PHENOTYPE

Infants with simple lesions and limited hemodynamic burden may grow near population expectations and demonstrate prompt catch-up after definitive repair. Persistent deterioration after successful repair of a simple lesion should not automatically be attributed to CHD. Residual hemodynamic abnormalities, feeding dysfunction, gastrointestinal disease, allergy, endocrine disorders, genetic conditions, and adverse social determinants should be reconsidered.[7]

Pulmonary overcirculation creates a mismatch between increased energy requirements and limited feeding capacity. Respiratory distress can make feeding prolonged and exhausting, while fluid restriction prevents the delivery of sufficient energy using ordinary milk density. Management requires coordination of energy density, protein, sodium, medication timing, feeding duration, and route.[3]

Cyanotic and complex biventricular disease frequently combines reduced oxygen delivery, repeated procedures, prolonged hospitalization, and preoperative growth deterioration. The prospective cohort of 801 infants showed that complexity and preoperative growth interacted in determining postoperative outcomes. Nutritional protection should therefore begin before surgery through avoidance of unnecessary fasting, quantification of actual intake, and early escalation when the trajectory deteriorates.[7]

Functionally univentricular disease requires the most intensive surveillance. Growth must be maintained between staged operations in a circulation with limited physiological reserve. Reduced intake or declining weight velocity may precede more obvious signs of interstage cardiovascular deterioration. Nevertheless, single-ventricle physiology does not imply irreversible growth failure, as meaningful catch-up can occur after improved hemodynamics and staged palliation.[10]

5.3 CARDIAC CORRECTION IS NOT EQUIVALENT TO NUTRITIONAL CURE

Definitive repair or staged palliation can reduce respiratory work, improve systemic output, increase oxygen delivery, and permit liberalization of fluid intake. These effects create a biological opportunity for catch-up growth. They do not eliminate prematurity, genetic disease, acquired neurological injury, vocal-cord dysfunction, oral aversion, gastrointestinal abnormalities, or the cumulative effects of prolonged catabolism.[3]

Different anthropometric dimensions recover at different rates. Weight responds rapidly to increased caloric intake and changes in fluid balance. Length reflects sustained protein adequacy, skeletal growth, endocrine function, and control of chronic disease. Head circumference may accelerate after improved hemodynamics but does not by itself establish normal brain development.[6]

The American Heart Association recommends neurodevelopmental risk stratification and longitudinal evaluation for children with high-risk CHD. Neurodevelopment reflects the combined influence of genetic, fetal, perioperative, nutritional, socioeconomic, and family

factors.[14] Anthropometric recovery must therefore be interpreted as one component of a broader developmental trajectory.

Rapid weight gain without corresponding improvement in LAZ may indicate disproportionate fat accumulation. Conversely, an infant who remains small but follows a stable proportional trajectory may be clinically more secure than one undergoing rapid downward percentile crossing. The therapeutic objective is coordinated improvement in weight, length, head circumference, body composition, feeding competence, and development.[6]

5.4 HUMAN MILK, FORTIFICATION, AND NUTRIENT COMPOSITION

Human milk should be actively supported in infants with CHD because of its immunological and gastrointestinal benefits. A systematic review of 16 studies found that an exclusive human-milk diet was associated with a lower risk of necrotizing enterocolitis. Evidence concerning growth, hospitalization, and postoperative feeding was favorable in some studies but was less consistent and more susceptible to confounding.[15]

Human milk alone may not provide sufficient energy and protein when fluid intake is restricted or metabolic demand is high. Fortification can increase nutrient delivery without excessive volume. The prescription should define final caloric density, protein content, preparation technique, storage, and monitoring.[16]

Increasing caloric concentration is not a purely mathematical intervention. Excessive concentration can alter osmolality, gastric emptying, stool pattern, electrolyte load, and renal solute exposure. Preparation errors may also cause substantial variation in delivered nutrients. Formula or fortification changes should therefore be accompanied by assessment of hydration, tolerance, electrolytes, renal function, and actual intake.[16]

A binary comparison between breastfeeding and formula feeding is clinically inadequate. Expressed human milk can be fortified, direct breastfeeding can be combined with measured supplementation, and formula can be used when human milk is unavailable or insufficient. Feeding composition and route should be reassessed whenever cardiac physiology, fluid allowance, oral competence, or growth velocity changes.[15]

5.5 FEEDING AND SWALLOWING AS COMPONENTS OF GROWTH FAILURE

Feeding difficulty should be separated into endurance, swallowing safety, gastrointestinal tolerance, sensory-motor skill, and caregiver–infant interaction. An infant may

receive insufficient volume because of tachypnea, aspirate despite apparently adequate intake, vomit because feeding is too concentrated or rapid, or resist oral feeding after repeated invasive procedures. These mechanisms require different interventions.[4]

Instrumental swallowing assessment should be considered when feeding is accompanied by coughing, choking, wet breathing, recurrent respiratory infection, unexplained desaturation, vocal-cord dysfunction, or prolonged intubation. It may also be required when clinical concern remains despite an apparently reassuring bedside evaluation because aspiration can be silent.[5]

Management can include changes in nipple flow, pacing, positioning, feeding duration, consistency, or route. Thickening and other modifications must be individualized because they can influence hydration, caloric delivery, feeding effort, and gastrointestinal tolerance. Safe intake takes priority over the immediate objective of complete oral feeding.[4]

Tube feeding can be lifesaving when oral intake is insufficient or unsafe. It can also reduce the energy expenditure associated with prolonged and unsuccessful oral feeding attempts. However, tube use introduces caregiver burden, equipment requirements, and a potential risk of prolonged dependence if positive oral experiences and objective weaning criteria are not maintained.[17]

Associations between tube feeding and poorer growth should not be interpreted as simple evidence of harm. Infants discharged with feeding tubes generally have greater cardiac complexity, longer hospitalization, lower oral endurance, or swallowing impairment. Tube feeding is therefore both a treatment and a marker of disease severity.[18]

5.6 EFFECTS OF HIGH-ENERGY FEEDING

Nutritional intervention studies demonstrate a gradient of benefit rather than a universal effect. Energy- and protein-enriched formulas improve short-term nutrient delivery and may produce better weight-related outcomes in selected patients. The evidence is less convincing for durable normalization of length, lean mass, oral feeding, or neurodevelopment.[20–22]

The enriched-formula trial conducted by Cui and colleagues demonstrated that adequate caloric intake and positive nitrogen balance could be achieved earlier. The greater incidence of diarrhea is clinically important because a feed that is repeatedly interrupted or diluted may deliver less usable nutrition despite its higher prescribed energy density.[20]

In the randomized study of infants with cyanotic CHD, calorie-enriched formula reduced the proportion classified as malnourished at six months from 36% to 18%.[21] If applicable to comparable populations, this absolute difference would correspond to approximately one additional infant avoiding malnutrition for every six treated. However, differences in formula composition, malnutrition definitions, and clinical setting limit generalization.

The 244-infant trial of early high-energy feeding demonstrated improvements in intensive-care-discharge WAZ and weight-for-height, fewer infections, shorter respiratory support, and a higher probability of nutritional recovery at three months.[22] Nevertheless, more than 80% of intervention participants did not meet the study's recovery definition at three months. High-energy feeding improved the probability of recovery but did not overcome all determinants of malnutrition.

The appropriate clinical principle is consequently to provide sufficient energy and protein within the infant's fluid and gastrointestinal tolerance. Escalation should be accompanied by monitoring of actual intake, emesis, stool pattern, abdominal findings, renal function, electrolytes, and growth proportionality.[16]

5.7 STANDARDIZED NUTRITIONAL PATHWAYS

Nutritional pathways can reduce delays, inconsistent prescriptions, and loss of follow-up. Important components include prenatal counseling when possible, human-milk support, early risk classification, standardized anthropometric measurements, explicit intake targets, fortification criteria, feeding observation, dietitian participation, and post-discharge reassessment.[19]

The consensus-based pathway evaluated by Marino and colleagues was associated with less WAZ deterioration during the first four months and better WAZ and LAZ at 12 months. Pediatric intensive-care stay was also shorter. Because the comparator was historical and the intervention was multifaceted, the observed benefit cannot be attributed to a single component.[19]

The primary value of a pathway may be greater reliability rather than identification of one ideal diet. A standardized process ensures that reduced intake, impaired growth velocity, and feeding dysfunction are recognized before severe malnutrition develops. It also facilitates communication between cardiology, surgery, intensive care, dietetics, nursing, feeding therapy, primary care, and the family.[19]

5.8 HOME MONITORING AND THE INTERSTAGE PERIOD

Discharge represents a high-risk nutritional transition. The prescribed feeding concentration, volume, and route must be translated into a regimen that caregivers can prepare and deliver safely. Written instructions should define the recipe, minimum intake, oral-feeding duration, tube supplementation, medication timing, warning symptoms, and contact pathway.[23]

Home monitoring has been most extensively developed for infants with single-ventricle physiology. Programs may include daily weight, oxygen saturation, intake, vomiting, stool output, and predefined red flags. Declining intake or growth may identify dehydration, infection, shunt obstruction, or worsening hemodynamics before obvious cardiovascular collapse.[23]

A recent observational comparison found similar average interstage weight velocity before and after implementation of a home-monitoring program. However, program enrollment was associated with less early growth deceleration and subsequent weight restoration during the second year. These findings suggest that the value of monitoring extends beyond a change in mean daily weight gain.[23]

Monitoring frequency should reflect physiological risk. Daily weighing may be appropriate during the single-ventricle interstage but can create unnecessary anxiety and overreaction to normal variation in lower-risk infants. Individual measurements should be interpreted according to slope, intake, symptoms, and measurement reliability.[23]

5.9 PREMATURITY, LOW BIRTH WEIGHT, AND GENETIC CONDITIONS

Prematurity compounds the nutritional effects of CHD. Preterm infants have lower nutrient stores, immature feeding coordination, greater respiratory vulnerability, and reduced tolerance of fluid or surgical stress. Corrected age should be used for growth assessment, but correction does not eliminate the additional biological risk.[25]

In preterm infants undergoing early surgery for complex CHD, lower birth-weight z score, single-ventricle anatomy, prolonged ventilation, and cardiopulmonary resuscitation were associated with mortality or adverse clinical outcomes. Neonatal and cardiac nutritional strategies should therefore be coordinated rather than applied independently.[25]

Genetic syndromes and extracardiac anomalies can modify expected growth, muscle tone, feeding function, gastrointestinal health, endocrine status, and operative risk. Trisomy

21, 22q11.2 deletion syndrome, Turner syndrome, and other conditions may produce characteristic growth patterns independent of caloric intake.[14]

Syndrome-specific charts may provide useful context, but they should not replace evaluation of trajectory. Even when short stature is expected, acute percentile crossing, disproportionate weight loss, or failure of head growth requires investigation. The objective is to detect preventable deterioration relative to the child's physiological and genetic potential.[14]

5.10 GROWTH AND NEURODEVELOPMENT

Growth and neurodevelopment share multiple determinants, including placental function, genetic abnormalities, oxygen delivery, brain maturation, cardiac surgery, intensive-care exposure, feeding skill, caregiver stress, and socioeconomic conditions. Poor growth may therefore function as a marker of cumulative biological adversity rather than as an isolated cause of neurodevelopmental impairment.[13]

Lower WAZ and LAZ during early infancy have been associated with less favorable developmental performance at six and 12 months.[12] A systematic review similarly found that impaired postnatal growth, particularly deficits in linear and head growth, was associated with adverse neurodevelopmental outcomes.[13]

The evidence does not establish that increasing weight alone improves neurodevelopment. Nutritional intervention is necessary to support brain growth, but neurodevelopment cannot be reduced to a single anthropometric measurement. Growth faltering should trigger developmental surveillance and early intervention rather than be used as a diagnostic test.[13]

Head circumference also requires careful interpretation. Following bidirectional Glenn surgery, mean HCZ increased by approximately 1.5 standard deviations, and two-thirds of infants demonstrated an increase greater than one z score. Accelerated head growth was not associated with better motor scores in that cohort.[24]

Infants with complex CHD, prematurity, genetic conditions, perioperative neurological injury, prolonged hospitalization, feeding dysfunction, or adverse growth trajectories should receive coordinated nutritional and developmental follow-up. Developmental services should not be postponed until severe delay becomes apparent.[14]

5.11 PRACTICAL FRAMEWORK FOR NUTRITIONAL RECOVERY

Assessment should begin with the cardiac and developmental phenotype. Relevant information includes gestational age, birth measurements, prenatal diagnosis, genetic findings, cardiac physiology, operations, residual lesions, oxygenation, diuretics, fluid allowance, ventilation history, infections, and length of hospitalization.[26]

WAZ, LAZ, WLZ, and HCZ should be plotted at clinically meaningful intervals. Mid-upper-arm circumference or skinfold measurements can be added when body composition is uncertain and measurement technique is reliable. A decline of 0.5–1 z score, particularly across more than one dimension, may be more clinically informative than an isolated value that remains above −2.[27]

The nutritional prescription must be compared with what the infant actually receives. Interrupted feeds, emesis, incomplete oral volumes, medication volume, mixing errors, and feeding duration should be incorporated into the calculation. Observation of at least one complete feeding can identify fatigue, respiratory deterioration, swallowing abnormalities, and caregiver difficulties that are not evident from the written prescription.[26]

Treatment should address the dominant mechanism. Increasing caloric density may help an infant limited by fluid restriction, but it will not correct aspiration. Tube supplementation may solve inadequate delivery but not gastrointestinal intolerance. Surgical correction may reduce energy expenditure but not reverse oral aversion or genetic growth limitation.[26]

Current reviews support early multidisciplinary participation, human-milk preservation, physiology-informed enteral advancement, protein adequacy, judicious parenteral nutrition when enteral intake is insufficient, micronutrient assessment, and reassessment after each surgical transition.[26,27] No single energy target is appropriate for every infant with CHD.

Table 3

Phenotype-based assessment and management

Clinical phenotype	Dominant mechanisms	Priority assessment	Initial management direction
Simple repaired CHD	Usually limited residual energy imbalance	Serial growth, residual lesion, feeding and gastrointestinal history	Investigate persistent faltering rather than indefinitely increasing calories
Pulmonary overcirculation	Tachypnea, sweating, fatigue, high energy expenditure, fluid restriction	Observed feeding, actual intake, respiratory status, electrolytes, WAZ and LAZ	Increase energy and protein density within tolerance; supplement exhausting oral feeds
Cyanotic or complex biventricular CHD	Reduced oxygen delivery, repeated procedures, prolonged hospitalization	Oxygenation, perfusion, intake, swallowing, multidimensional anthropometry	Protect preoperative trajectory and advance postoperative nutrition when stable
Single-ventricle interstage	Limited physiological reserve, dehydration and shunt-related risk	Home weight, intake, saturation, vomiting, predefined red flags	Structured home monitoring and explicit escalation criteria
Prematurity or low birth weight	Low nutrient stores, lung disease, immature feeding coordination	Corrected-age growth, head circumference, micronutrients, swallowing	Coordinate neonatal and cardiac nutrition; fortify human milk when indicated
Genetic syndrome	Syndrome-specific stature, tone, endocrine or gastrointestinal disease	Standard and syndrome-specific trajectories; targeted evaluation	Establish individualized goals and treat correctable comorbidities
Dysphagia or aspiration risk	Unsafe swallowing, silent aspiration, recurrent respiratory disease	Feeding-specialist assessment and instrumental testing when indicated	Modify flow, pacing, position, consistency, or route
Tube-fed infant	Inadequate or unsafe oral intake	Delivered intake, oral skills, tube complications, caregiver capacity	Use tube support while maintaining safe oral exposure and reassessment criteria
Apparent post-repair catch-up	Rapid weight gain with uncertain proportional recovery	WAZ, LAZ, WLZ, HCZ, body composition, development	Reassess energy density after hemodynamic improvement and avoid overfeeding

Source: Prepared by the authors.

6 LIMITATIONS

This was a structured narrative review rather than a prospectively registered systematic review with duplicate screening and de novo meta-analysis. The selected studies were intended to represent the principal growth trajectories, feeding problems, intervention strategies, and clinical outcomes rather than constitute an exhaustive inventory.

The evidence was heterogeneous regarding cardiac anatomy, surgical era, age, gestational-age correction, reference charts, malnutrition thresholds, feeding protocols, and follow-up duration. Weight was measured more frequently and reliably than length, head circumference, or body composition, potentially overstating the completeness of nutritional recovery.

Many pathway, human-milk, tube-feeding, and home-monitoring studies were observational and vulnerable to confounding by indication, center practice, and secular improvements in treatment. Infants requiring tube feeding or intensive nutritional support generally had greater disease complexity, limiting causal interpretation.

Randomized nutritional trials were generally single-center, open label, and focused on short-term postoperative outcomes. Evidence concerning sustained linear growth, body composition, oral autonomy, neurodevelopment, and quality of life remains limited.

Results from single-ventricle, cyanotic, or severely malnourished cohorts cannot automatically be generalized to children with simple repaired CHD. Nutritional targets must be individualized according to anatomy, physiology, prematurity, growth potential, feeding safety, and family capacity.

7 CONCLUSION

Congenital heart disease can impair growth before birth and commonly produces its steepest postnatal deterioration during the first four months of life. Complex biventricular and single-ventricle physiology carry the greatest average risk, but prematurity, fetal growth restriction, genetic conditions, heart failure, hospitalization, and feeding dysfunction produce substantial variation within each anatomical category.

Cardiac correction represents an important biological turning point but does not guarantee complete nutritional recovery. Weight frequently improves after repair or staged palliation, while length, head circumference, lean mass, and oral feeding competence may recover more slowly or remain impaired. Weight normalization alone should therefore not define successful catch-up growth.

Nutritional intervention should be matched to the dominant mechanism. Human milk should be supported and fortified when necessary, while energy- and protein-enriched feeding can improve short-term weight-related outcomes in selected infants. Higher caloric density requires tolerance monitoring, and tube feeding should be used when oral intake is unsafe or insufficient without abandoning oral-skill development.

Clinical monitoring should integrate WAZ, LAZ, WLZ, HCZ, corrected gestational age, cardiac physiology, actual nutrient delivery, body composition when feasible, observed feeding, swallowing safety, and developmental progress. Standardized multidisciplinary pathways and single-ventricle home-monitoring programs can improve the reliability of care and facilitate earlier intervention.

Future multicenter studies should use standardized growth and body-composition outcomes, objective feeding measurements, and follow-up through at least 24 months. Trials must determine whether early nutritional gains result in sustained linear growth, lean-mass accretion, oral autonomy, improved neurodevelopment, and better quality of life. Until such evidence is available, the most defensible goal is safe and proportional individualized growth rather than maximal weight gain.

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