Cardiology in the Young

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Growth failure and nutritional-endocrine adaptations in children with cyanotic CHD: a structured narrative review

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Review

Cite this article: Soliman AT, Alyafei F, Abdulkayoum AT, AlQadi M, Ahmed S, Alhemaidi N, Alaaraj N, and Hamed N (2026) Growth failure and nutritional-endocrine adaptations in children with cyanotic CHD: a structured narrative review. *Cardiology in the Young*, page 1 of 6. doi: 10.1017/S1047951126124500

Received: 12 June 2026 UTC
Revised: 7 September 2026 UTC
Accepted: 27 September 2026 UTC

Keywords:
cyanotic CHD; growth failure; natriuretic peptides; insulin-like growth factor 1; malnutrition; Paediatric cardiology

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Abstract

The heart is increasingly recognised as a cardiovascular signalling organ that releases biologically active natriuretic peptides during haemodynamic stress. In cyanotic CHD, chronic hypoxaemia, altered loading conditions, increased energy expenditure, recurrent illness, and delayed repair may disrupt endocrine, metabolic, and nutritional pathways that regulate growth. This structured narrative review synthesises evidence linking cardiac biomarkers, systemic hormonal adaptation, nutritional status, surgical timing, and growth outcomes in children with cyanotic CHD. PubMed, Scopus, and Google Scholar were searched for human studies published from January 1990 to December 2025 that reported cardiac biomarkers, systemic hormonal measures, growth parameters, nutritional outcomes, or postoperative recovery in paediatric CHD. Thirty-six studies were included and narratively synthesised because lesion types, age groups, assays, and outcomes were heterogeneous. The evidence indicates consistent activation of natriuretic peptide signalling, reduced circulating insulin-like growth factor 1 and insulin-like growth factor binding protein 3 in several paediatric cohorts, appetite-related hormonal changes, impaired weight gain, and frequent linear growth faltering. Longitudinal studies suggest partial improvement in growth velocity and anabolic signalling after timely surgical correction, although incomplete recovery may occur when preoperative hypoxaemia and undernutrition are prolonged. Growth failure in cyanotic CHD should therefore be regarded as a multifactorial cardiovascular-nutritional syndrome rather than isolated primary endocrine disease. Early surgical referral, individualised nutrition, and coordinated cardiology, endocrinology, and dietetic follow-up are central to improving long-term outcomes.

Introduction

CHD is not only a structural cardiovascular disorder but also a chronic systemic condition that may influence nutrition, metabolism, endocrine signalling, and somatic growth. These interactions are especially relevant in cyanotic CHD, where persistent hypoxaemia, increased metabolic demand, pulmonary vascular disease, recurrent infection, feeding difficulty, and delayed surgical correction may converge during periods of rapid childhood growth. This issue carries particular significance in low-resource or delayed-referral settings, where prolonged preoperative cyanosis and coexisting undernutrition are more common and may compound the adverse effects on growth.[1–5]

The myocardium releases biologically active natriuretic peptides, particularly atrial natriuretic peptide and B-type natriuretic peptide, in response to stretch, pressure load, and myocardial stress. These peptides are clinically useful markers of cardiovascular burden and may also interact with renal, vascular, adipose, and metabolic pathways. However, in children with cyanotic CHD, their broader endocrine and metabolic significance remains incompletely defined.[6–10]

Growth failure in cyanotic CHD is multifactorial. Linear growth faltering, poor weight gain, and malnutrition cannot be explained solely by low caloric intake. Chronic hypoxaemia, inflammation, impaired hepatic perfusion, increased resting energy expenditure, feeding difficulties, neurohormonal activation, and postoperative recovery all affect growth trajectories. Several studies have described reduced insulin-like growth factor 1 concentrations, altered growth hormone-insulin-like growth factor signalling, changes in appetite-related hormones, and incomplete catch-up growth after delayed repair.[5,11–20]

This review was prepared to provide an integrated paediatric cardiology-focused synthesis of these mechanisms. The objectives were to summarise evidence on cardiac endocrine biomarkers in cyanotic CHD, evaluate secondary systemic hormonal and metabolic adaptations, and relate these changes to growth, nutritional status, and postoperative recovery.

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Materials and methods

This structured narrative review used a transparent search strategy informed by Preferred Reporting Items for Systematic Reviews and Meta-Analyses principles. These principles were applied to improve transparency of study identification and selection, although quantitative meta-analysis was not feasible because of substantial heterogeneity in lesion type, age group, hormonal assay, surgical status, and outcome definitions.[21]

PubMed, Scopus, and Google Scholar were searched for studies published between January 1990 and December 2025. Search terms included combinations of cyanotic CHD, CHD, natriuretic peptide, atrial natriuretic peptide, B-type natriuretic peptide, growth hormone, insulin-like growth factor 1, insulin-like growth factor binding protein 3, endocrine function, growth failure, weight gain, malnutrition, ghrelin, leptin, insulin, cortisol, and surgery. Reference lists of eligible articles were also reviewed.

Eligible studies included original human research involving paediatric patients with cyanotic CHD or paediatric CHD cohorts with relevant cyanotic subgroups. Studies were included when they reported at least one cardiac biomarker, systemic hormonal parameter, metabolic marker, growth outcome, nutritional index, or postoperative recovery outcome. Prospective cohorts, retrospective cohorts, case–control studies, cross-sectional studies, and interventional studies were eligible. Editorials, narrative reviews without original data, non-indexed reports, animal-only studies, and case reports with fewer than five patients were excluded.

Two reviewers screened titles and abstracts, followed by full-text assessment of potentially relevant articles. Extracted data included age group, cardiac diagnosis, cyanosis status, biomarker or hormone assessed, growth or nutritional measure, timing of surgery, and principal findings. Risk of bias was evaluated using ROBINS-I principles for observational studies and Cochrane guidance for trials, with attention to selection bias, confounding, outcome measurement, incomplete data, and selective reporting.[22,23]

Results

Thirty-six studies were included (Figure 1). The evidence base comprised studies of cardiac biomarkers, growth hormone-insulin-like growth factor axis alterations, anthropometric and nutritional outcomes, appetite-related hormones, metabolic markers, inflammatory markers, and postoperative recovery. Most studies were observational, involved relatively small cohorts, and included heterogeneous cardiac diagnoses. The overall methodological quality was low to moderate, mainly because of referral-centre selection, incomplete adjustment for disease severity and nutrition, and variation in surgical timing.

Cardiac endocrine biomarker studies consistently demonstrated activation of natriuretic peptide signalling in CHD. Atrial natriuretic peptide, B-type natriuretic peptide, and N-terminal pro-B-type natriuretic peptide were elevated in fetal, neonatal, paediatric, and adult CHD populations, particularly when ventricular load, myocardial strain, or cyanosis was present. These biomarkers are best interpreted as indicators of haemodynamic stress and myocardial adaptation rather than as primary mediators of growth failure.[10,24–29] Complementary biomarker studies have evaluated cardiac troponin I in congenital defects with pressure or volume overload.[30]

Several studies reported lower circulating insulin-like growth factor 1 or insulin-like growth factor binding protein 3 concentrations in children with cyanotic CHD, sometimes despite normal or mildly elevated growth hormone levels. This pattern suggests impaired anabolic signalling but does not establish primary growth hormone resistance. Reduced insulin-like growth factor 1 probably reflects multiple interacting mechanisms, including chronic hypoxaemia, undernutrition, systemic inflammation, hepatic perfusion abnormalities, and overall cardiac severity.[11–14,31] Fetal CHD research has also examined cord-blood IGF-1 and IGFBP-3 in relation to perinatal brain development.[32]

Growth and nutritional studies consistently showed that linear growth impairment, underweight, and wasting are common in children with CHD and are particularly important in cyanotic or more severe lesions. Stunting appears to reflect chronic illness and prolonged nutritional stress, whereas poor weight gain may be driven by increased energy expenditure, feeding fatigue, recurrent hospitalisation, pulmonary hypertension, and delayed repair. These outcomes are interrelated and should be assessed together rather than as isolated nutritional measurements.[1–5]

Metabolic and appetite-related hormone studies described altered ghrelin, leptin, insulin, cortisol, tumour necrosis factor alpha, and interleukin 6 patterns. Elevated ghrelin may reflect compensatory appetite stimulation during undernutrition, whereas reduced leptin may reflect diminished fat mass and altered energy balance. Abnormal insulin secretion and elevated cortisol suggest physiological stress and impaired anabolic efficiency. However, the available studies are too limited to support routine clinical use of these markers outside research settings.[16–20,33]

Longitudinal studies indicate that surgical correction may be followed by partial recovery in insulin-like growth factor 1 concentrations, growth velocity, weight gain, and nutritional status. Recovery is more likely when hypoxaemia and haemodynamic burden are relieved early and when postoperative nutrition is optimised. Incomplete catch-up growth after delayed repair suggests that prolonged preoperative hypoxaemia and malnutrition may have lasting effects on growth potential.[13–15] A postoperative neonatal study evaluated oxandrolone as a growth-promoting adjunct after surgery for complex CHD; this distinct intervention should not be interpreted as evidence for routine endocrine drug treatment.[34]

Discussion

The central finding of this review is that growth failure in cyanotic CHD is best understood as a multifactorial cardiovascular-nutritional syndrome with secondary endocrine and metabolic adaptations (Table 1). The evidence does not support viewing these children as having isolated primary endocrine disease. Instead, chronic hypoxaemia, myocardial stress, altered perfusion, feeding difficulty, recurrent illness, inflammation, and undernutrition appear to interact with anabolic and appetite-regulating pathways (Figure 2).[1–5,12–14,16–20]

Natriuretic peptides occupy an important place in this framework. They reflect myocardial stretch and haemodynamic stress across the lifespan and are clinically useful for risk stratification and perioperative monitoring. Their relationship with adipose tissue metabolism and insulin sensitivity is biologically plausible, but paediatric evidence remains insufficient to assign them a direct causal role in growth failure. For clinical care, their strongest current value remains cardiovascular assessment rather than endocrine decision-making.[7–10,24,26,27,29]

The growth hormone-insulin-like growth factor axis provides a mechanistic bridge between cardiac disease severity and somatic growth. Reduced insulin-like growth factor 1 in children with cyanotic CHD may reflect impaired hepatic production,

https://doi.org/10.1017/S1047951126124500 Published online by Cambridge University Press

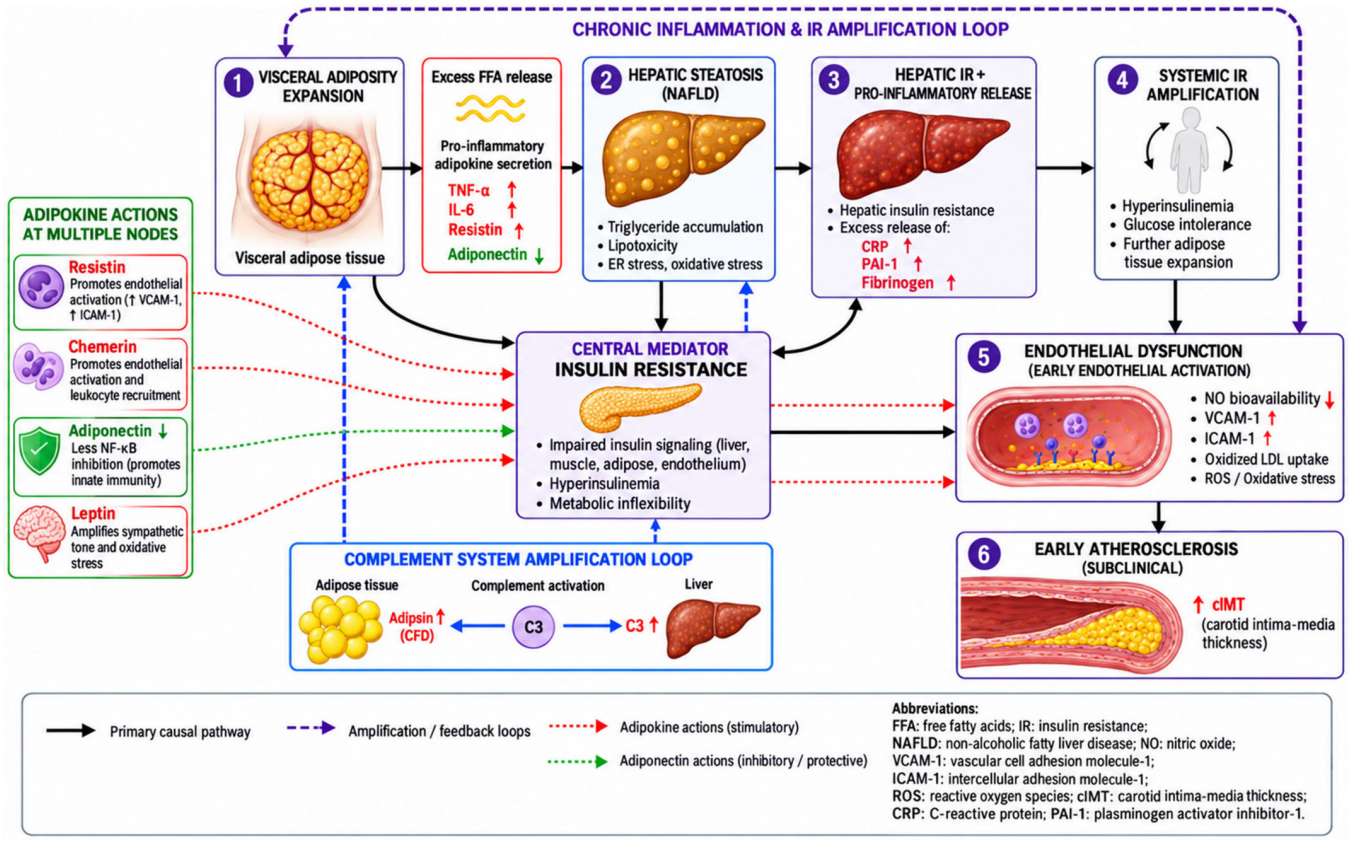


Figure 1. PRISMA-based study selection flow diagram for the structured narrative review. The diagram summarises identification, screening, eligibility assessment, and final inclusion of 36 studies evaluating cardiac biomarkers, hormonal adaptations, growth outcomes, and nutritional status in children with cyanotic CHD.

Table 1. Consolidated evidence map of endocrine, nutritional, and growth pathways in cyanotic CHD

Domain	Representative evidence	Interpretation for paediatric cardiology practice
Cardiac endocrine biomarkers	ANP, BNP, and NT-proBNP are elevated in fetal, paediatric, and adult CHD, especially with ventricular overload, myocardial strain, or cyanosis.[10,24–29]	Use primarily as markers of haemodynamic stress and perioperative risk, not as direct diagnostic tests for endocrine growth failure.
Growth hormone-insulin-like growth factor axis	Several paediatric studies reported lower IGF-1 or IGFBP-3, sometimes despite normal or mildly elevated growth hormone.[11–14,31]	Interpret low IGF-1 in context of hypoxaemia, inflammation, nutrition, hepatic perfusion, and disease severity before diagnosing primary endocrine disease.
Linear growth and weight gain	Stunting, underweight, wasting, and poor weight gain are common, particularly in cyanotic or severe lesions.[1–5]	Measure growth longitudinally and assess height velocity, weight-for-height or body mass index z-score, feeding history, oxygen saturation, and surgical timing.
Appetite and metabolic hormones	Changes in ghrelin, leptin, insulin, cortisol, tumour necrosis factor alpha, and interleukin 6 have been reported.[16–20,33]	These markers are biologically plausible but currently remain research tools rather than routine clinical tests.
Surgical correction and recovery	Longitudinal studies show partial improvement in growth velocity, IGF-1, weight, and nutrition after correction or palliation.[13–15]	Timely surgical management and perioperative nutritional support are the most actionable strategies for improving growth outcomes.

ANP = atrial natriuretic peptide; BNP = B-type natriuretic peptide; IGF-1= insulin-like growth factor 1; IGFBP-3 = insulin-like growth factor binding protein 3; NT-proBNP = N-terminal pro-B-type natriuretic peptide.

inflammation, insufficient substrate availability, and hypoxaemia. Because several studies are cross-sectional and confounding is substantial, growth hormone stimulation testing should be reserved for children with persistent growth failure after cardiac stabilisation and nutritional optimisation, or for those with clinical features suggesting a primary endocrine disorder.[11–14,31,35,36]

Nutritional management should begin early. Routine growth monitoring should include weight, length or height, body mass index, weight-for-height or body mass index z-score, height velocity, feeding history, energy intake, recurrent illness, oxygen saturation, and surgical timing. Dietetic assessment is essential because poor weight gain may be worsened by increased resting energy expenditure and feeding fatigue even when intake appears reasonable. In selected children, targeted nutritional supplementation before and after surgery may reduce postoperative decline and support catch-up growth.[1–5,15]

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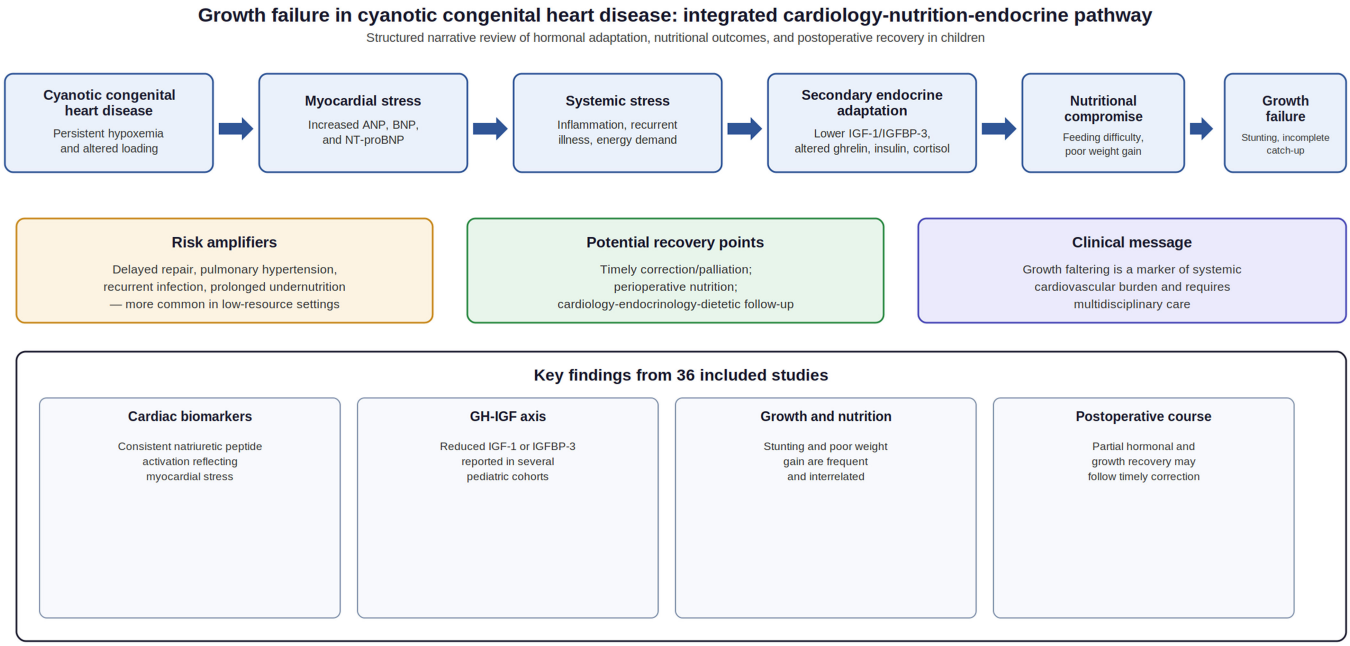


Figure 2. Graphical abstract summarising the proposed pathway by which cyanotic CHD, chronic hypoxaemia, haemodynamic stress, cardiac endocrine biomarker activation, secondary hormonal adaptation, and nutritional compromise interact to impair linear growth and weight gain. Early correction, nutritional optimisation, and multidisciplinary follow-up may partially reverse these abnormalities.

Table 2. Proposed clinical surveillance framework and research priorities

Care or research area	Suggested approach	Rationale
Routine growth monitoring	Record weight, length or height, body mass index or weight-for-height z-score, and height velocity at cardiology visits.	Growth faltering may reflect systemic disease burden and should trigger nutritional and endocrine review.[1–5]
Nutrition	Assess feeding fatigue, caloric and protein intake, recurrent illness, and postoperative nutritional recovery.	Increased energy expenditure and feeding difficulty may persist despite apparently adequate intake.[2,4,5,15]
Endocrine evaluation	Consider thyroid function, IGF-1, pubertal status, and formal endocrine testing only when growth failure persists after nutrition and cardiac stabilisation.	Secondary endocrine adaptation is common; overdiagnosis of primary endocrine disease should be avoided.[11–14,31]
Timing of intervention	Coordinate early surgical or catheter-based correction when clinically feasible and optimise perioperative nutrition.	Earlier relief of hypoxaemia and haemodynamic burden may improve catch-up growth potential.[13–15]
Future studies	Use prospective designs with standardised cardiac severity, oxygen saturation, nutrition, endocrine assays, and growth z-scores.	Current evidence is limited by heterogeneity and confounding.

The strongest actionable intervention remains timely correction or palliation of the cyanotic lesion when clinically feasible. Surgical correction improves oxygen delivery and reduces haemodynamic stress, which may permit partial endocrine and nutritional recovery. Nevertheless, catch-up growth may be incomplete after prolonged cyanosis, emphasising the importance of early referral, close surveillance, and coordinated postoperative follow-up.[13–15]

Clinical implications

Children with cyanotic CHD should receive integrated cardiology, nutrition, and endocrine surveillance rather than fragmented assessment. Cardiologists should consider growth faltering as a clinically meaningful marker of systemic disease burden. Endocrinologists should interpret low insulin-like growth factor 1 in the context of hypoxaemia, inflammation, nutrition, and hepatic perfusion. Dietitians should be involved early, particularly before and after surgery, because nutritional recovery may influence both weight gain and linear growth.[1–5,12–15]

A practical approach is to measure growth at every cardiology visit, identify deceleration in height velocity early, document nutritional intake and feeding difficulty, optimise caloric and protein delivery, and re-evaluate endocrine markers after cardiac stabilisation. In practical terms, anthropometric assessment—weight, length or height, and the corresponding z-scores—should be recorded at every cardiology visit, whereas formal endocrine reassessment (including IGF-1 and thyroid function) is generally best deferred until approximately three to six months after cardiac and nutritional stabilisation, or performed sooner if growth failure persists despite adequate caloric and protein intake. Persistent severe short stature, poor height velocity despite adequate nutrition and cardiac repair, delayed puberty, or symptoms suggesting hypothyroidism, adrenal disease, or growth hormone deficiency should prompt formal endocrine evaluation (Table 2).

https://doi.org/10.1017/S1047951126124500 Published online by Cambridge University Press

Limitations

The evidence base is limited by observational designs, small sample sizes, heterogeneous lesions, mixed cyanotic and acyanotic cohorts, variable timing of surgery, and inconsistent hormonal assays. Many studies did not adequately adjust for caloric intake, socioeconomic factors, recurrent infection, pulmonary hypertension, medication exposure, or severity of cyanosis. These limitations prevent strong causal inference and preclude meta-analysis. Future prospective studies should use standardised anthropometric outcomes, serial endocrine and inflammatory measurements, detailed nutritional assessment, and clearly defined surgical time points.[22,23]

Conclusion

Cyanotic CHD has important secondary endocrine, metabolic, and nutritional consequences that contribute to impaired growth. Current evidence supports an association between chronic hypoxaemia, myocardial stress, altered anabolic signalling, poor nutritional status, and incomplete catch-up growth, although causality remains incompletely defined. Early surgical correction, individualised nutritional support, and coordinated cardiology-endocrinology follow-up are the most clinically actionable strategies for improving growth and long-term outcomes. Future prospective, multicenter studies incorporating standardised growth, nutritional, oxygen saturation, and endocrine outcome measures are needed to clarify causal pathways and to optimise surveillance and intervention strategies.[1–5,12–15]

Acknowledgements. None.

Financial support. This research received no specific grant from any funding agency, commercial, or not-for-profit sectors.

Competing interests. The authors declare none.

Ethical standard. Not applicable. This structured narrative review did not involve new human or animal experimentation and therefore did not require institutional review board approval.

Suggested social media synopsis. Growth failure in cyanotic CHD reflects chronic hypoxaemia, nutrition, and secondary endocrine adaptation; early repair and multidisciplinary care are key. #PedsCards.

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