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Reducing anesthetic morbidity and mortality in children with heart failure undergoing non cardiac surgery: a physiology and equity focused approach

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Keypoints

Children with heart failure undergoing non-cardiac procedures remain among the most vulnerable populations in pediatric anesthesia. Postoperative vigilance, extended monitoring, and system-level interventions are essential, particularly in low-resource settings where structural inequities exacerbate risk.

Abstract

Children with heart failure increasingly present for non-cardiac procedures, yet they remain at high risk for perioperative morbidity and mortality. Traditional lesion-focused anesthetic approaches fail to capture the physiologic vulnerability that drives adverse outcomes. This perspective synthesizes current evidence on perioperative risk, emphasizing physiology-based determinants—ventricular dysfunction, pulmonary hypertension, cyanosis, and recent decompensation—as stronger predictors than anatomic diagnosis alone. It highlights the critical role of preoperative optimization, multidisciplinary planning, and intraoperative strategies tailored to fragile cardiovascular physiology. Postoperative vigilance, extended monitoring, and system-level interventions are essential, particularly in low-resource settings where structural inequities exacerbate risk. By integrating a physiology-driven, equity-focused approach, anesthesiologists can anticipate instability, reduce preventable complications, and improve survival for this vulnerable population worldwide.

Keywords

Congenital heart disease, Equity, Heart failure, Low-resource settings, Pediatric anesthesia, Perioperative risk

Introduction

Advances in pediatric cardiology, cardiac surgery, and intensive care have significantly improved survival among children with congenital and acquired heart disease. As a result, an increasing number of children with residual lesions, chronic ventricular dysfunction, pulmonary hypertension, or established heart failure (HF) now present for non-cardiac procedures requiring anesthesia [1,2]. This evolving demographic shift has transformed perioperative pediatric practice: anesthesiologists are no longer managing only structurally normal hearts in routine surgery but increasingly fragile cardiovascular physiology in diverse procedural settings. Children with congenital heart disease (CHD) undergoing non-cardiac surgery consistently demonstrate higher perioperative morbidity and mortality compared with their peers without

cardiac disease [3,4]. Risk is particularly elevated in infants, those undergoing emergency procedures, and patients with complex or unrepaired lesions, pulmonary hypertension, cyanosis, or ventricular dysfunction [3,4]. Importantly, contemporary analyses suggest that physiologic severity—rather than anatomical diagnosis alone—is the dominant determinant of adverse outcomes [4]. In this context, heart failure represents a final common pathway of vulnerability, whether arising from uncorrected CHD, residual postoperative lesions, cardiomyopathy, myocarditis, chemotherapy-induced cardiotoxicity, or rheumatic valvular disease. The anesthetic implications of CHD in non-cardiac surgery have long been recognized [5,6], and more recent reviews have refined practical perioperative recommendations [1,7]. However, most literature remains lesion-focused rather than failure-focused. Yet children with impaired ventricular function or pulmonary vascular disease are uniquely susceptible to anesthetic-induced myocardial depression, abrupt changes in systemic or pulmonary vascular resistance, arrhythmias, and impaired oxygen delivery. A recent narrative review on anesthesia-related cardiac arrest in children underscores underlying heart disease as one of the strongest predictors of perioperative cardiac arrest, mediated through hemodynamic instability, hypoxia, and arrhythmogenic mechanisms [8]. Thus, heart failure physiology—not simply the presence of structural disease—should guide perioperative risk assessment and management strategies. The global burden of pediatric cardiac disease further amplifies this concern. Systematic reviews demonstrate a substantial and likely underrecognized prevalence of CHD across Africa [8,9], with limited access to timely surgical correction in many settings. In parallel, rheumatic heart disease remains a major cause of pediatric and adolescent heart failure in low-income countries [10,11]. These epidemiologic realities mean that, worldwide, many children present for non-cardiac procedures with advanced or untreated cardiac pathology. Broader structural inequities in health systems, highlighted in the 2023/2024 United Nations Development Programme Human Development Report [12], directly influence perioperative risk through delayed diagnosis, limited specialist access, and constrained monitoring resources. Despite this growing and globally diverse population, standardized perioperative pathways specifically addressing children with heart failure undergoing non-cardiac procedures remain insufficiently defined.

Existing guidance emphasizes careful preoperative assessment, multidisciplinary planning, and tailored anesthetic techniques [1,2,7], yet there is limited synthesis focused explicitly on reducing morbidity and mortality in children with established or impending cardiac failure.

This perspective therefore aims to: (1) synthesize current evidence on perioperative risk in children with heart disease undergoing non-cardiac procedures; (2) reframe risk stratification around heart failure physiology; and (3) propose practical, systems-level approaches to reduce anesthetic morbidity and mortality across both high-resource and resource-constrained environments.

By transitioning from an anatomy-focused to a physiology-oriented and equity-conscious framework, we may more effectively tackle the unequal perioperative susceptibility of this growing pediatric demographic. Determinants of Perioperative

Morbidity and Mortality. Understanding why children with heart failure experience disproportionate perioperative risk requires moving beyond isolated events and examining the interplay between altered cardiovascular physiology and anesthetic stress.

Perioperative deterioration in this population is rarely abrupt or random; it is typically the culmination of fragile compensatory mechanisms destabilized by anesthesia, surgery, and environmental stressors.

Cardiac Arrest and Severe Adverse Events

Recent studies of cardiac arrest during pediatric anesthesia often find that having heart disease is one of the biggest factors that can lead to this problem during surgery[11].

Importantly, cardiac arrest in these children usually reflects progressive hemodynamic compromise, not a

primary electrical catastrophe. Rather, it is most often the end stage of progressive hemodynamic compromise.

Anesthetic agents inherently modulate myocardial contractility, systemic vascular resistance, and autonomic tone. Children with preserved cardiac reserve generally tolerate these changes well. In contrast, children with heart failure depend on heightened sympathetic activity and neurohormonal activation to maintain perfusion. When anesthesia blunts these compensatory mechanisms, myocardial depression and vasodilation may unmask profound circulatory instability. Even modest reductions in contractility can critically impair forward flow and coronary perfusion, particularly in hypertrophied or pressure-loaded ventricles [1,6]. Equally important is the dynamic behavior of the pulmonary circulation. In children with pulmonary hypertension, minor disturbances—hypoxia, hypercarbia, acidosis, and inadequate analgesia—may acutely elevate pulmonary vascular resistance, precipitating right ventricular failure and rapid cardiovascular collapse [1,3]. The right ventricle, often already strained, may be unable to adapt to sudden afterload increases. The resulting reduction in left ventricular preload further compounds systemic hypotension. Arrhythmias represent another frequent pathway to deterioration. Myocardial fibrosis, chamber dilation, electrolyte shifts, and chronic inotropic therapy create an arrhythmogenic substrate. In the setting of limited cardiac reserve, even transient rhythm disturbances may produce profound hemodynamic consequences [11]. Taken together, these mechanisms illustrate a unifying principle: Perioperative cardiac arrest in children with heart failure is usually the terminal expression of cumulative physiologic imbalance rather than isolated anesthetic error.

Prevention therefore requires anticipation, not merely reaction (Figure 1).

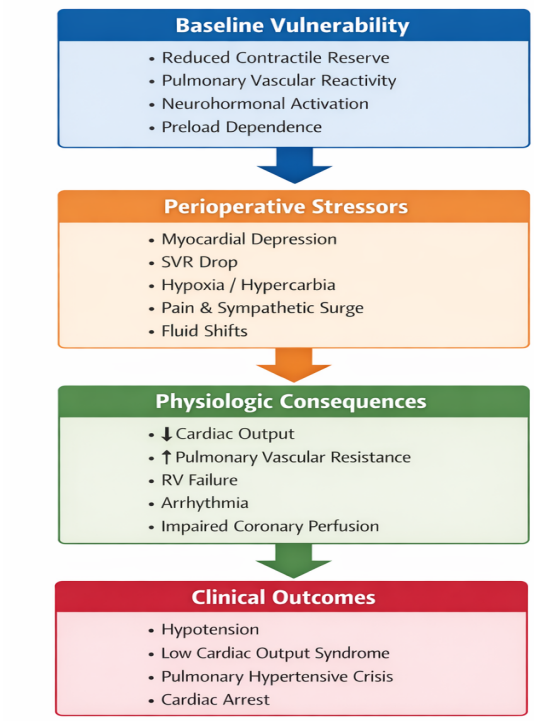

Figure 1. Pathophysiologic Cascade Leading to Perioperative Decompensation in Children With Heart Failure.
This schematic highlights the key stages at which physiologic vulnerability interacts with perioperative stressors to increase risk. It emphasizes the concept of fragile compensatory mechanisms and identifies points where proactive clinical intervention can prevent progression to severe outcomes. The diagram serves as a conceptual framework for understanding perioperative risk and guiding strategy in children with heart failure.

High-Risk Phenotypes: From Anatomy to Physiology

While congenital diagnosis remains clinically relevant, contemporary evidence underscores that physiologic severity more accurately predicts perioperative risk than anatomy alone [4]. Large observational analyses demonstrate that mortality and major morbidity are markedly higher among children with severe ventricular dysfunction, pulmonary hypertension, cyanotic heart disease, complex unrepaired or palliated lesions, and single-ventricle physiology [3,4,7].

Particularly vulnerable are children with Fontan circulation, whose cardiac output is intrinsically preload-dependent and unable to augment effectively under stress. Similarly, patients who have recently undergone decompensation or hospitalization due to heart failure represent a group that has already depleted their physiologic reserves.

These phenotypes converge towards a shared denominator: a diminished ability to withstand changes in preload, afterload, contractility, and oxygen delivery. A child with a relatively "moderate" structural lesion but advanced ventricular dysfunction may therefore carry greater risk than a child with complex anatomy and preserved function. This insight supports a physiology-centered perioperative model that prioritizes ventricular performance, pulmonary vascular status, and recent clinical trajectory over diagnostic labels alone [4]. (Table 1)

Domain	Low Risk	Intermediate Risk	High Risk
Ventricular function	Normal systolic and diastolic function	Mild–moderate dysfunction	Severe LV and/or RV dysfunction
Pulmonary pressure	Normal pulmonary pressure	Mild pulmonary hypertension	Severe pulmonary hypertension or RV failure
Oxygenation status	Normal saturation (>95%)	Mild desaturation	Cyanosis or resting SpO_2 <90%
Clinical trajectory	Stable, no recent changes	Recent medication adjustment	Recent decompensation or hospitalization
Arrhythmia burden	None	Controlled arrhythmia	Recent or unstable arrhythmias
Age	>5 years	1–5 years	<1 year
Surgical context	Minor elective surgery	Intermediate-risk surgery	Emergency or major surgery

Table 1. Physiology-Based Perioperative Risk Stratification in Children With Heart Failure Undergoing Non-Cardiac Surgery. This table summarizes physiologic determinants of perioperative risk in children with heart failure undergoing non-cardiac procedures. Risk increases with impaired ventricular function, pulmonary hypertension, cyanosis, recent decompensation, younger age, and surgical urgency. Stratification should prioritize physiologic severity over anatomical diagnosis alone [3,4,7,11].

Preoperative Optimization: A Functional and Multidisciplinary Approach

If perioperative instability reflects fragile physiology, optimization must begin before anesthesia is induced. Historically, anesthetic planning focused on lesion-specific considerations [5,6]. Contemporary practice, however, emphasizes integrated functional assessment and collaborative planning [1,7]. A structured multidisciplinary evaluation—ideally involving pediatric cardiology, anesthesiology, and, where appropriate, critical care—forms the cornerstone of risk mitigation. Recent echocardiography offers vital details about ventricular systolic and diastolic function, right ventricular performance, and pulmonary pressures. Trends in oxygen saturation, exercise tolerance, arrhythmia burden, and biomarkers offer further context regarding physiologic stability. Equally important is recognition of temporal vulnerability. Elective procedures should be deferred in the presence of active decompensation, escalating diuretic requirements, new arrhythmias, or recent hospitalization for heart failure [1,3]. The timing of surgery relative to acute illness may be decisive, particularly in infants and children with pulmonary hypertension. Medication management also demands thoughtful individualization. Beta-blockers are typically continued to prevent rebound tachycardia and arrhythmias. Chronic heart failure therapies, including diuretics and aldosterone antagonists, are generally maintained with close electrolyte monitoring. Angiotensin-converting enzyme inhibitors require case-by-case consideration due to potential peri-induction hypotension. Critically, pulmonary vasodilators must not be abruptly discontinued, as withdrawal may provoke pulmonary hypertensive crisis [1,6,7]. At this stage, the guiding objective is not merely documentation but stabilization—ensuring that the child enters the operating room in the most compensated physiologic state achievable.

Intraoperative Management: Protecting Fragile Physiology

Once anesthesia begins, management must remain anchored in physiologic preservation. Across multiple

reviews, several principles consistently emerge [1,2,6,7]: maintenance of preload, avoidance of abrupt reductions in systemic vascular resistance, minimization of myocardial depression, preservation of sinus rhythm, and strict prevention of hypoxia, hypercarbia, and acidosis. Induction is a particularly vulnerable period. Rapid vasodilation or myocardial depression may destabilize already tenuous compensation. Large propofol boluses, for example, may precipitate profound hypotension in severe systolic dysfunction [1,6].

Ketamine may better preserve systemic vascular tone, yet it requires careful ventilation management in pulmonary hypertension [1,2]. No single agent is universally optimal; rather, dosing strategy, titration, and anticipation of physiologic response are paramount. In children with pulmonary hypertension, prevention of crisis must be proactive. Maintaining oxygenation, normocapnia, and adequate anesthetic depth reduces sympathetic surges and pulmonary vasoconstriction.

Where indicated, ready access to inhaled nitric oxide or alternative pulmonary vasodilators provides an additional safety margin [1,3].

Hemodynamic monitoring should reflect the severity of the disease. Invasive arterial blood pressure monitoring allows rapid recognition and correction of hypotension in moderate to severe heart failure [1,7]. Advanced monitoring and point-of-care echocardiography may further refine intraoperative decision-making in high-risk cases.

Fluid management demands particular nuance. Children with diastolic dysfunction or restrictive physiology are exquisitely preload-sensitive. Both hypovolemia and fluid overload may precipitate deterioration [6]. Incremental administration with frequent reassessment is preferable to empiric liberal fluid strategies. When hypotension reflects impaired contractility rather than preload deficiency, vasoactive support may be more physiologically appropriate than additional volume. (Table 2)

Pathophysiologic Vulnerability	Mechanism of Instability	Targeted Perioperative Strategy
Systolic dysfunction	Reduced contractile reserve; impaired coronary perfusion	Avoid myocardial depressants; titrated induction; early vasoactive support
Diastolic dysfunction / Restrictive physiology	Preload dependence; intolerance to tachycardia	Maintain sinus rhythm; cautious fluid boluses; avoid tachycardia
Pulmonary hypertension	Acute ↑ PVR → RV failure → ↓ LV preload	Prevent hypoxia, hypercarbia, acidosis; ensure deep anesthesia; consider pulmonary vasodilators
Cyanotic heart disease	Limited oxygen reserve; hyperviscosity	Optimize oxygen delivery; avoid anemia and dehydration
Fontan physiology	Passive pulmonary blood flow; preload dependence	Maintain adequate preload; avoid increased PVR; cautious positive pressure ventilation
Arrhythmogenic substrate	Fibrosis, dilation, electrolyte imbalance	Correct electrolytes; maintain beta-blockade; rhythm monitoring

Table 2. Pathophysiologic Vulnerabilities in Pediatric Heart Failure and Targeted Perioperative Management Strategies. This table links major physiologic vulnerabilities in pediatric heart failure to specific intraoperative management strategies. Anticipatory, mechanism-based anesthetic planning is essential to prevent perioperative instability [1,2,6,7].

Postoperative Vulnerability: The Overlooked Phase

The conclusion of surgery does not mark the end of risk. Observational data indicate that a substantial proportion of adverse outcomes occur postoperatively rather than intraoperatively [3]. Hemodynamic instability may emerge as anesthetic agents dissipate, analgesia wanes, or respiratory compromise develops. Accordingly, children with severe heart failure require extended monitoring and, in many cases, planned admission to high-dependency or intensive care units. Early recognition of low cardiac output—manifested by tachycardia, rising lactate, oliguria, or increasing oxygen requirements—allows timely intervention.

Effective pain control is not merely a comfort measure; it is a hemodynamic intervention. Uncontrolled pain increases systemic and pulmonary vascular resistance, potentially precipitating decompensation. Multimodal analgesia therefore forms an integral component of postoperative cardiovascular protection.

Global Context and Structural Inequities

Beyond individual physiology, broader structural determinants shape perioperative outcomes. In many African and low-income settings, children frequently present with uncorrected congenital heart disease [8,9], advanced heart failure, or severe rheumatic valvular pathology [10]. Limited access to specialist care, invasive monitoring, and pulmonary vasodilators amplifies perioperative vulnerability.

These disparities reflect systemic inequities in health infrastructure and development, as highlighted in the United Nations Development Programme Human Development Report 2023/2024 [12]. Addressing perioperative mortality in children with heart failure therefore requires not only technical refinement but also structural investment—regional referral systems, teleconsultation networks, standardized protocols, and workforce training adapted to resource-constrained environments. (Table 3)

Systems-Level Prevention and Future Directions

Finally, sustainable reduction in morbidity and mortality depends on institutional commitment. Pragmatic system-level interventions include standardized risk stratification pathways, mandatory cardiology consultation for moderate to severe disease, crisis simulation training, and registry-based quality monitoring [1–7, 11]. A structured "cardiac vulnerability checklist" may operationalize physiologic risk assessment and reduce preventable oversights.

Looking ahead, biomarker-guided risk prediction, phenotype-specific anesthetic algorithms, and prospective multicenter registries may further refine care. Equally important is the integration of global health equity into perioperative cardiac frameworks, ensuring that advances benefit children across diverse resource settings.

Domain	High-Resource Settings	Low-Resource Settings
Diagnosis	Early detection; routine echocardiography	Delayed diagnosis; limited imaging access
Corrective cardiac surgery	Widely available	Limited or unavailable
Heart failure stage at presentation	Often compensated or post-repair	Frequently advanced or untreated
Pulmonary hypertension management	Access to inhaled nitric oxide and advanced therapies	Limited access to targeted therapies
Intraoperative monitoring	Invasive arterial lines; advanced hemodynamic tools	Basic monitoring only
Postoperative care	Structured ICU availability	Limited ICU capacity
Specialist expertise	Pediatric cardiac anesthesia teams	Workforce shortages

Table 3. Perioperative Challenges in High- and Low-Resource Settings: Implications for Children With Heart Failure. Structural disparities in diagnosis, therapeutic access, monitoring capacity, and critical care infrastructure significantly influence perioperative outcomes in children with heart failure. Addressing these inequities is essential to reducing global anesthesia-related mortality [8–12].

Conclusion

Children with heart failure undergoing non-cardiac procedures remain among the most vulnerable populations in pediatric anesthesia. Ventricular dysfunction, pulmonary hypertension, cyanosis, infancy, and emergency surgery consistently predict adverse outcomes. Yet these risks are neither random nor inevitable. By embracing a physiology-driven, multidisciplinary, and equity-conscious approach, perioperative care can shift from reactive management to anticipatory protection.

Reducing mortality in this population demands more than technical skill. It requires vigilance, collaboration, system design, and an unwavering commitment to translating cardiovascular insight into safer anesthesia for every child.

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Conflict of Interest

The authors declare that there are no conflicts of interest related to this publication.

Ethical Considerations

As this article is a perspective piece and does not involve human participants, patient data, or primary data collection, formal ethical approval was not required.

Data Availability

This article did not generate or analyze new data. The content is based exclusively on previously published studies and publicly accessible sources**.**

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