

Effect of Postoperative Arrhythmia in Children Less Than 18 Years of Age Following Congenital Cardiac Surgery: A Prospective Study

World Journal for Pediatric and

Congenital Heart Surgery

1-10

© The Author(s) 2026

Article reuse guidelines:

sagepub.com/journals-permissions

DOI: 10.1177/21501351261472478

journals.sagepub.com/home/pch



Raheja D, MD, DM¹ , Sahu MK, MD, DNB¹ , Khan MS, DNB, DM¹ , Singh SP, MD, DM¹ , Talwar S, MS, MCh¹ , and Devagourou V, MS, MCh¹ 

Abstract

Background: Postoperative arrhythmias are common among children after congenital cardiac surgery, potentially compromising hemodynamic stability and postoperative recovery. This study aimed to assess the impact of arrhythmias on hemodynamics and clinical outcomes.

Material and Methods: Three hundred and twenty-nine children under 18 years of age undergoing congenital cardiac surgery were studied prospectively. In addition to standard postoperative hemodynamic monitoring, electrical cardiometry (ICON[®]) was used to assess hemodynamic parameters across time points (prearrhythmia, during arrhythmia, and postarrhythmia). Repeated measures were analyzed using Friedman test followed by Wilcoxon signed-rank test with Bonferroni correction for pairwise comparisons. Poisson regression with robust variance estimation was used to identify independent predictors and calculate incidence rate ratios (IRR).

Results: Postoperative arrhythmias occurred in 53/329 patients (16.1%). Junctional ectopic tachycardia (34%, [18/53]) and supraventricular tachycardia (30.2%, [16/53]) were the most common. Arrhythmias were associated with low cardiac output (IRR 3.43; 95% confidence interval [CI] 2.27-5.17; $P < .001$), ventricular dysfunction (IRR 2.55; $P < .001$), acute kidney injury (IRR 2.01; $P = .006$), and liver dysfunction (IRR 4.56; $P = .002$) on unadjusted analysis. However, these associations were attenuated after adjustment for prearrhythmia hemodynamic variables like central venous pressure (CVP) and vasoactive-inotropic score (VIS) on multivariable analysis. Elevated CVP, VIS, and hypokalemia were predictors of arrhythmia.

Conclusions: Arrhythmias occurred in approximately one-sixth of children following congenital cardiac surgery and were associated with adverse hemodynamic changes, organ function, and recovery. However, these associations were attenuated after adjustment for prearrhythmia hemodynamic variables (CVP and VIS), suggesting that arrhythmia may reflect underlying instability rather than a primary causal factor.

Keywords

postoperative arrhythmia, pediatric cardiac surgery, noninvasive cardiac output monitoring, hemodynamic changes

Received: March 9, 2026; accepted: July 2, 2026

Introduction

Postoperative arrhythmias remain a frequent complication following congenital cardiac surgery. The reported incidence ranges from 7.3% to 48% encompassing a broad spectrum of arrhythmias, including junctional ectopic tachycardia (JET), supraventricular tachycardia (SVT), complete heart block, ventricular tachycardia (VT), ventricular fibrillation (VF), and other conduction abnormalities.¹

In cardiac surgery, apart from the effects of surgery and anesthesia, children are exposed to extracorporeal circulation, haemodilution, anticoagulation, cardioplegia, temperature alterations, myocardial manipulation, and ischemia-reperfusion injury. Along with the underlying disease state and intraoperative altered

homeostasis, these factors predispose the children to various postoperative complications including arrhythmias. Arrhythmias represent a deviation from a normal heart's electrical activity, which subsequently translates into mechanical dysfunction. This mechanical dysfunction further translates to

¹Cardiac Surgical Intensive Care, Department of CTVS, CT Centre, All India Institute of Medical Sciences, New Delhi, India

Corresponding Author:

Sahu MK, CT Centre, All India Institute of Medical Sciences, New Delhi 110029, India.

Email: drmanojssahu@gmail.com

reduced stroke volume which precipitates low cardiac output, thereby contributing to organ dysfunction and prolonged recovery.

Although the incidence and spectrum of arrhythmias have been described, less is known about their dynamic hemodynamic effects when assessed using continuous cardiac output monitoring. This study was conducted to (1) assess the impact of postoperative arrhythmias on clinical outcomes; (2) characterize associated hemodynamic changes during the periarrhythmia period; (3) determine the incidence, spectrum, timing, and duration of postoperative arrhythmias; and (4) identify independent predictors of arrhythmia development.

Methods

Study Design and Population

This prospective observational study was conducted in a high-volume cardiac surgery center at a tertiary care teaching hospital between July 2023 and December 2025. Ethics approval was obtained (Ref. No. AIIMSA00525 / 18.01.2024) and written informed consent was obtained from the parents/guardians of the patients. All consecutive pediatric patients younger than 18 years of age who underwent elective congenital cardiac surgery were included in the study. All surgeries were performed by multiple experienced congenital cardiac surgeons within a dedicated team, following standardized institutional surgical and perioperative protocols to minimize interoperator variability.

Surgical risk adjustment was performed using the Risk Adjustment for Congenital Heart Surgery 1 (RACHS-1) category.² Children with a history of arrhythmia, inotropic support, sepsis, mechanical ventilation, severe renal, or hepatic dysfunction were excluded from the study. Additionally, patients who experienced significant intraoperative adverse events or had a complicated intraoperative course requiring extracorporeal membrane oxygenation were also excluded. These patients were excluded to maintain cohort homogeneity and to minimize confounding from severe intraoperative hemodynamic instability unrelated to postoperative arrhythmia. These cases constituted a small proportion of the total surgical cohort.

Postoperative Monitoring

All patients were managed in a dedicated cardiac intensive care unit (ICU) with continuous electrocardiography (ECG), monitoring of invasive arterial blood pressure, central venous pressure, pulse oximetry, and electrical cardiometry (ICON®). Hemodynamic parameters, including heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), central venous pressure (CVP), along with the vasoactive inotropic score (VIS) were monitored. This was calculated as follows: [dopamine dose (µg/kg/min) + dobutamine dose (µg/kg/min) + 100 × epinephrine dose (µg/kg/min) + 100 × norepinephrine dose (µg/kg/min) + 10 × milrinone dose (µg/kg/min) + 10,000 × vasopressin dose (U/kg/min)]. ICON-derived parameters were stroke volume (SV), cardiac output (CO), systemic

vascular resistance (SVR) and thoracic fluid content (TFC). Measurements were recorded at 3 predefined time points: (1) pre-arrhythmia period through the first hour of patient transfer to the ICU, (2) during the arrhythmia episode (onset of arrhythmia), and (3) after the arrhythmia episode (post-arrhythmia period—24 h after arrhythmia resolution).

Arrhythmia Diagnosis

Postoperative arrhythmias were initially identified by the on-duty resident based on continuous ECG monitoring, clinical parameters, and a 12-lead ECG recording on ECG paper with long lead II. All cases were subsequently reviewed and confirmed by the consultant intensivist. In situations where the diagnosis remained uncertain, atrial wire ECG recordings were obtained for better rhythm characterization. If ambiguity persisted, a cardiology consultant was involved for final confirmation.

Definitions Used in This Study

Arrhythmia was defined as any nonsinus rhythm lasting >30 s or requiring therapeutic intervention, including supraventricular tachycardia (SVT), JET, atrial flutter/fibrillation, VT/VF and frequent ventricular premature complexes (VPCs).

Junctional ectopic tachycardia was defined as a non-reentrant tachyarrhythmia arising from the atrioventricular (AV) junction, characterized by AV dissociation with a ventricular rate exceeding the atrial rate. QRS morphology may be narrow or wide and was not used as a defining criterion.

Supraventricular tachycardia was defined as a regular tachycardia of supraventricular origin, typically due to AV reentry or enhanced AV nodal conduction, characterized by 1:1 AV association, often with retrograde P waves.

Atrial flutter/fibrillation was defined based on characteristic ECG findings, including organized atrial activity with sawtooth flutter waves (atrial flutter) or an irregular rhythm without distinct P waves (atrial fibrillation), with variable AV conduction.

Ventricular premature complexes were considered significant if occurring at a frequency of >10 per minute.

Other outcomes were defined using standard criteria: ventilator-associated pneumonia (VAP) (>48 h after ventilation), liver dysfunction (abnormal biochemistry/hepatic insufficiency), ventricular dysfunction (echo-defined systolic/diastolic impairment), low cardiac output syndrome (LCOS) (clinical, biochemical, and hemodynamic evidence of hypoperfusion), acute kidney injury (AKI) (creatinine rise or reduced urine output), seizure, stroke, and all-cause ICU mortality. In our institutional terminology, intracardiac repair (ICR) refers specifically to complete intracardiac correction of tetralogy of Fallot (TOF). Other intracardiac procedures, including atrial septal defect/ventricular septal defect (VSD) closures and atrioventricular septal defect (AVSD) repairs were categorized separately.

Statistical Analysis

Statistical analysis was performed using Stata 18.0 (StataCorp, TX, USA). Continuous variables were expressed as median (IQR) and categorical variables as frequency (%). Group comparisons were performed using the Mann-Whitney U test for continuous variables and Chi-square or Fisher's exact test for categorical variables. Hemodynamic changes across time points were analyzed using the Friedman test with Wilcoxon signed-rank test and Bonferroni correction for pairwise comparisons.

Predictors of arrhythmia were assessed using Poisson regression with robust variance estimation. Variables with $P < .20$ in univariable analysis were included in multivariable models using stepwise backward elimination. Adjusted incidence rate ratios (aIRR) with 95% confidence interval (CI) were reported.

To evaluate independent associations between arrhythmia and prearrhythmia hemodynamic variables with clinical outcomes, separate multivariable Poisson regression models with robust variance estimation were constructed for each outcome (LCOS, AKI, ventricular dysfunction, seizure, liver dysfunction and VAP). Arrhythmia was included in all models, a priori based on clinical relevance. Prearrhythmia CVP and VIS were entered as candidate covariates and subjected to backward stepwise selection with a removal threshold of $P \geq .10$. Results were presented as aIRR with 95% confidence intervals.

Outcomes

The primary outcome of this study was to assess the impact of arrhythmias on postoperative hemodynamics. Other secondary outcomes assessed were LCOS, ventricular dysfunction, AKI, liver dysfunction, ICU and hospital length of stay, and mortality.

Results

Patient Characteristics

Among 329 patients, 53 (16.1%) developed postoperative arrhythmia. Demographic variables were comparable between groups. Infants less than 1 year of age comprised 40.1% (132/329) of the cohort and the highest proportion of arrhythmias (41.5%, [22/53]) occurred in this age group. There were no significant differences in weight ($P = .843$), height ($P = .514$), or body surface area ($P = .938$) between arrhythmia and nonarrhythmia groups (Table 1).

Surgical Characteristics of the Patients

Types of Surgical Repair. Out of a total of 329 surgeries, ICR for TOF was the most frequently performed surgical repair (19.8%, $n = 65/329$), followed by VSD closure (15.5%, $n = 51/329$) (Figure 1). Procedures categorized as other congenital cardiac procedures constituted 21.9% ($n = 72/329$). This category included pulmonary artery banding and de-banding, pericardiectomy, combined atrial and ventricular septal defect

closures, septal closure with valve surgery, subaortic membrane resection, right ventricular outflow tract resection, coarctation of aorta repair, double-chambered right ventricle repair, AVSD repair, conduit repair, interrupted aortic arch repair, truncus arteriosus repair, Rastelli and Nikaidoh procedures.

Association Between Arrhythmia and RACHS-I Surgical Risk Category

In RACHS-I category 1, arrhythmias occurred in 5 of 47 patients, constituting 10.64% of the arrhythmia group. In category 2, arrhythmias occurred in 23 of 173 patients, constituting 13.29% of the arrhythmia group. In category 3, the incidence of arrhythmias was highest, with 25 of 92 patients which constituted 27.17% of the arrhythmia group.

There was a statistically significant association between RACHS-I category and the occurrence of postoperative arrhythmia (Pearson $\chi^2 = 13.65$, $df = 3$, $P = .003$). This association remained significant on Fisher's exact test ($P = .004$), indicating that the distribution of arrhythmias differed significantly across RACHS-I risk categories. Arrhythmia frequency changes monotonically with increasing RACHS-I category.

Association of Cardiopulmonary Bypass Duration and Aortic Cross Clamp Time Among Patients With and Without Arrhythmias

The cardiopulmonary bypass (CPB) time was comparable between patients who developed postoperative arrhythmias and those who did not, with a median duration of 112 min in both groups. Similarly, the aortic cross clamp (ACC) time did not differ between the arrhythmia and nonarrhythmia groups, with a median duration of 68 min in each group.

Incidence and Characteristics of Arrhythmia

Arrhythmia occurred in 16.1% (53/329) of patients, with a median onset at 8 hours (IQR 5.9-11) postoperatively and median duration of 26 hours (IQR 8-36). Junctional ectopic tachycardia at 34% (18/53) and SVT at 30.2% (16/53) were the most common arrhythmias. Amiodarone was the primary therapy used in 50.9% (27/53) of cases followed by inotrope optimization in 20.8% (11/53) cases, and most cases were managed medically (Table 2).

Hemodynamic Changes During Arrhythmia

Heart rate increased significantly during arrhythmia and decreased afterward. Systolic blood pressure declined during arrhythmia and remained lower postresolution, while diastolic blood pressure increased only postarrhythmia. Central venous pressure showed overall variation without significant pairwise differences. Cardiac output showed no significant change, whereas stroke volume decreased during arrhythmia and recovered postresolution. Systemic vascular resistance increased during arrhythmia and

Table 1. Comparison of Demographic Data Between Arrhythmic and Nonarrhythmic Groups.

	Nonarrhythmia, n (%), 276 (83.8%)	Arrhythmia, n (%) 53, (16.2%)	Total 329, (100%)	P Value
Age category (years)				
≤ 1	110 (39.9)	22 (41.5)	132 (40.1)	.987
> 1–6	82 (29.7)	14 (26.4)	96 (29.2)	
> 6–12	53 (19.2)	10 (18.9)	63 (19.1)	
> 12–18	31 (11.2)	7 (13.2)	38 (11.6)	
Gender				
Male	170 (61.6)	33 (62.3)	203 (61.7)	.927
Female	106 (38.4)	20 (37.7)	126 (38.3)	
Anthropometric data	Median (IQR)	Median (IQR)	Total (n = 329)	P Value
Weight (kg)	12.0 (6.47-23.25)	9.0 (6.5-21.0)	329	.843
Height (cm)	90 (71-119)	88 (73-110)	329	.514
Body surface area (m ²)	0.55 (0.35-0.89)	0.47 (0.36-0.82)	329	.938

Categorical variables are presented as number (%), and continuous variables as median with interquartile range (IQR). Comparisons between groups were performed using the chi-square test for categorical variables and the Mann-Whitney U test for continuous variables. A *P* value <.05 was considered statistically significant.

remained elevated, while thoracic fluid content was unchanged. Vasoactive inotropic score increased during arrhythmia and decreased thereafter (Table 3).

Clinical Outcomes

Arrhythmia was associated with greater morbidity, including Low Cardiac Output State (LCOS) (IRR 3.43), ventricular dysfunction (IRR 2.55), liver dysfunction (IRR 4.56), and AKI (IRR 2.01). ICU (6 vs 3 days) and hospital stays (13 vs 6 days) were significantly prolonged (Table 4). Ventilator associated pneumonia (VAP) and mortality were higher but not statistically significant. However, these associations were attenuated after adjustment for pre-arrhythmia hemodynamic variables (CVP and VIS) on multivariable analysis (Table 5).

Predictors of Arrhythmia

Prearrhythmia hemodynamics were similar between the groups except for higher CVP and VIS in the arrhythmia group. Heart rate, BP, and ICON-derived indices did not differ significantly. The arrhythmia group had lower potassium, higher lactate,

higher peak creatinine kinase MB fraction (CK-MB) and troponin I, and lower peak C-reactive protein (CRP). On univariate analysis, higher CVP (IRR 1.62 per mm Hg, *P* < .001), higher

Table 2. Incidence and Clinical Characteristics of Arrhythmia.

Parameters	Category/Description	Values
Incidence and timing	Frequency of postoperative arrhythmia, n (%)	53 (16.1)
	Time to onset of arrhythmia (hours) postsurgery, median (IQR)	8.0 (5.9-11.0)
	Duration of arrhythmia (hours) until arrhythmia resolution, median (IQR)	26.0 (8.0-36.0)
Type of arrhythmia	Junctional ectopic tachycardia, n (%)	18 (34.0)
	Supraventricular tachycardia, n (%)	16 (30.2)
	Complete heart block, n (%)	5 (9.4)
	Ventricular tachycardia, n (%)	3 (5.7)
	Ventricular premature complexes, n (%)	3 (5.7)
	Nonsustained ventricular tachycardia, n (%)	2 (3.8)
	Sinus tachycardia, n (%)	2 (3.8)
	Atrial fibrillation, n (%)	1 (1.9)
	Mixed arrhythmias*, n (%)	3 (5.7)
	General supportive measures, n (%)	3 (5.7)
Treatment modality	Inotrope optimization/moderation, n (%)	11 (20.8)
	Intravenous amiodarone, n (%)	27 (50.9)
	Intravenous digoxin, n (%)	5 (9.4)
	Amiodarone + electrical cardioversion, n (%)	2 (3.8)
	Permanent pacemaker, n (%)	2 (3.8)
	Escalation of inotropes, n (%)	3 (5.7)

*Mixed arrhythmias indicate the occurrence of more than one type of arrhythmia in the same patient. †General supportive measures included adequate analgesia and sedation, correction of hypovolemia, electrolyte abnormalities (potassium, magnesium, and calcium) and temperature correction.

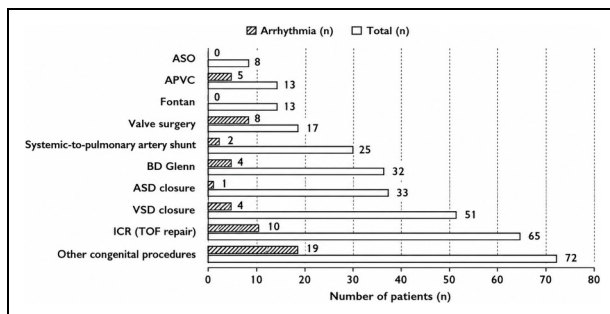


Figure 1. Distribution of arrhythmias among different congenital cardiac surgical procedures. Abbreviations: APVC, anomalous pulmonary venous connection; ASD, atrial septal defect; ASO, arterial switch operation; BD, bidirectional; ICR, intracardiac repair; TOF, Tetralogy of Fallot; VSD, ventricular septal defect.

Table 3. Impact of Arrhythmia on Hemodynamics.

Variables	Prearrhythmia, Median (IQR)	During-Arrhythmia, Median (IQR)	Postarrhythmia, Median (IQR)	Friedman χ^2 (df = 2)	Overall P Value	Adjusted Pairwise P Values†
HR (beats/min)	99 (88-123)	188 (178-199)	112 (89-126)	64.49	<.001	Pre-During <.001; During-Post <.001; Pre-Post 1.000
SBP (mm Hg)	104 (78-112)	42 (38-52)	56 (44-68)	83.15	<.001	Pre-During <.001; During-Post <.001; Pre-Post <.001
DBP (mm Hg)	66 (44-74)	70 (66-78)	88 (78-108)	54.51	<.001	Pre-During .645; During-Post <.001; Pre-Post <.001
CVP (mm Hg) by ICON	15 (12-18)	14 (12-18)	14 (12-18)	12.04	.002	Pre-During .071; During-Post .995; Pre-Post .587
CO (L/min) by ICON	2.4 (1.8-3.8)	2.2 (1.6-3.5)	2.5 (1.9-3.6)	5.88	.053	Pre-During .071; During-Post .995; Pre-Post .587
SV (mL) by ICON	24 (18-36)	20 (15-32)	22 (17-34)	57.61	<.001	Pre-During <.001; During-Post <.001; Pre-Post .177
SVR (dyn·s·cm ⁻⁵) by ICON	987 (876-1245)	1112 (912-1476)	987 (976-1343)	12.04	.002	Pre-During .004; During-Post 1.000; Pre-Post .046
TFC by ICON (kΩ ⁻¹)	32 (28-32)	32 (31-35)	32 (30-33)	5.88	.053	Pre-During .071; During-Post .995; Pre-Post .587
VIS	14 (10-16)	18 (12-18)	12 (5-16)	57.61	<.001	Pre-During <.001; During-Post <.001; Pre-Post .177

Abbreviations: CO, cardiac output; CVP, central venous pressure; DBP, diastolic blood pressure; HR, heart rate; ICON, electro cardiometry noninvasive method of cardiac output monitoring; SBP, systolic blood pressure; SV, stroke volume; SVR, systemic vascular resistance; TFC, thoracic fluid content; VIS, vasoactive inotropic score.

VIS (IRR 1.19 per unit, $P < .001$), lower potassium (IRR 0.22, $P < .001$), and higher lactate (IRR 3.38, $P < .001$) were associated with arrhythmia, whereas demographic and operative variables were not. On multivariable analysis, elevated CVP and VIS remained independent predictors of postoperative arrhythmias and higher potassium levels were protective, indicating that preexisting hemodynamic stress and metabolic instability were the principal determinants of postoperative arrhythmia (Tables 6 and 7).

Differential Impact of Arrhythmia and Prearrhythmia Hemodynamic Variables (Central Venous Pressure & Vasoactive-Inotropic Score) on Clinical Outcomes

On multivariable analysis using Poisson regression with robust variance, arrhythmia was not independently associated with

most adverse clinical outcomes. A significant association was observed only for VAP (aIRR 2.63; 95% CI 1.05-6.58; $P = .040$), while its association with LCOS did not reach statistical significance (aIRR 1.77; 95% CI 0.97-3.24; $P = .065$). In contrast, prearrhythmia CVP emerged as a consistent independent predictor of multiple outcomes, including LCOS (aIRR 1.15; 95% CI 1.06-1.24; $P = .001$), ventricular dysfunction (aIRR 1.16; 95% CI 1.09-1.23; $P < .001$), AKI (aIRR 1.24; 95% CI 1.16-1.33; $P < .001$), and liver dysfunction (aIRR 1.22; 95% CI 1.10-1.36; $P < .001$). Similarly, prearrhythmia VIS was independently associated with LCOS (aIRR 1.07; 95% CI 1.02-1.12; $P = .006$) and ventricular dysfunction (aIRR 1.12; 95% CI 1.06-1.17; $P < .001$), with a borderline association observed for liver dysfunction ($P = .083$). No independent predictors were identified for seizures. All the above findings are presented in Table 5.

Table 4. Impact of Arrhythmia on Clinical Outcomes and Incidence Rate Ratio (IRR) of Different Outcome Variables.

Clinical Outcome	Arrhythmia			P Value	Incidence Rate Ratio (IRR)	95% Confidence Interval	P Value
	Nonarrhythmia (n = 276) n (%)	(n = 53) n (%)	Total (n)				
Ventilator-associated pneumonia	13 (4.8)	6 (11.3)	19	.062†	2.38	0.94–5.98	.066
Low cardiac output syndrome	38 (13.8)	25 (47.2)	63	<.001	3.43	2.27–5.17	<.001
Ventricular dysfunction	45 (16.3)	22 (41.5)	67	<.001	2.55	1.68–3.86	<.001
Acute kidney injury	40 (15.0)	16 (30.2)	56	.008†	2.01	1.22–3.31	.006
Seizures	3 (1.1)	1 (1.9)	4	.627	1.74	0.18–16.43	.631
Liver dysfunction	8 (2.9)	7 (13.2)	15	.001†	4.56	1.72–12.05	.002
ICU length of stay (days) median (IQR)	3 (3-4)	6 (4-9)	-	<.001	—	—	—
Hospital length of stay (days) median (IQR)	6 (5-7)	13 (7-18)	-	<.001	—	—	—
Mortality	14 (5.1)	4 (7.5)	18	.468	—	—	—

IRRs were calculated using Poisson regression with robust variance. An IRR > 1.00 indicates an increased risk of the outcome in the arrhythmia group compared with the nonarrhythmia group. Values are expressed as number (%). Percentages are column percentages. Associations were assessed using the chi-square test; Fisher's exact test was applied where appropriate, due to small cell frequency (†). A P value <.05 was considered statistically significant.

Abbreviations: ICU, intensive care unit; IQR, interquartile range; IRR, incidence rate ratios.

Discussion

Postoperative arrhythmias remain a significant challenge after pediatric heart surgery. In our study, the incidence of postoperative arrhythmias was 16.1% (53/329), consistent with most contemporary reports (≈14%–16%), higher than some earlier institutional series, and substantially lower than studies reporting intensive surveillance. Variability across studies likely reflects differences in patient population, surgical complexity, perioperative practices, and monitoring protocols. Institutional differences including surgical team variability, could also have contributed to the variable incidences.^{3–8}

Junctional ectopic tachycardia was the most common arrhythmia (34%, [18/53]), consistent with prior pediatric cardiac surgery reports.^{3–5,9,10} In our cohort, it occurred most frequently after tetralogy of Fallot repair, whereas other studies reported associations with VSD, total anomalous pulmonary venous connection, or transposition of the great artery repairs.^{4,5} These differences reflect the influence of variation in surgical complexity, patient profile, operative technique, and degree of myocardial manipulation, particularly near the AV node on the development of JET.

Management included pharmacological therapy as well as supportive measures such as optimization of sedation, temperature control, and correction of electrolyte abnormalities. Management was primarily pharmacological, with intravenous amiodarone use in 50.9% (27/53) of cases, supplemented by inotrope optimization/moderation (20.8% [11/53] of cases) and supportive measures including sedation, temperature, and electrolyte correction. Few patients required electrical cardioversion or permanent pacing (3.8% [2/53] each), indicating that most postoperative arrhythmias were controlled with medical therapy and hemodynamic optimization, consistent with prior published reports.^{6,8}

After an extensive review of the literature, we were unable to find studies specifically evaluating the effect of arrhythmias on

hemodynamics and clinical outcomes. The majority of available articles primarily focused on risk factors and mortality.

Effect of Arrhythmia on Hemodynamics

Heart rate increased sharply during arrhythmia with subsequent normalization after arrhythmia resolution, indicating that tachycardia was predominantly arrhythmia-driven. Systolic blood pressure showed a decline during arrhythmia and remained significantly low in the postarrhythmia period, suggesting residual myocardial dysfunction or impaired ventricular performance. Diastolic blood pressure showed significant increase following arrhythmia resolution, likely reflecting compensatory systemic vasoconstriction. There was narrowed pulse pressure and impaired tissue perfusion, mimicking a low cardiac output state during and after arrhythmic episodes.

Although CVP demonstrated a statistically significant overall difference, the lack of significant pairwise differences suggested relative preload stability during arrhythmia. This interpretation was reinforced by the absence of significant changes in TFC, indicating that volume overload or depletion was not the primary contributor to arrhythmia associated hemodynamic compromise.

The VIS was found to be high during arrhythmia and low following arrhythmia resolution, suggesting the increased catecholamine levels causing arrhythmia.

ICON-derived hemodynamic parameters demonstrated preserved cardiac output across all phases, suggesting effective compensatory mechanisms. However, preserved cardiac output likely reflected maintenance of macrocirculatory flow through compensatory tachycardia and increased afterload, which may not translate into adequate tissue perfusion.

Stroke volume declined significantly during arrhythmia with subsequent recovery, indicating impaired ventricular filling or contractility. The maintenance of CO despite reduced stroke volume likely reflected rate-dependent compensation.

Table 5. Differential Impact of Arrhythmia and Prearrhythmia Hemodynamic Variables (VIS and CVP) on Clinical Outcomes.

Clinical Outcome	Arrhythmia aIRR (95% CI)	CVP aIRR (95% CI)	VIS aIRR (95% CI)
VAP	2.63 (1.05-6.58), P = .040	—	—
LCOS	1.77 (0.97-3.24), P = .065	1.15 (1.06-1.24), P = .001	1.07 (1.02-1.12), P = .006
Ventricular dysfunction	—	1.16 (1.09-1.23), P < .001	1.12 (1.06-1.17), P < .001
AKI	—	1.24 (1.16-1.33), P < .001	—
Seizure	—	—	—
Liver dysfunction	—	1.22 (1.10-1.36), P < .001	1.10 (0.99-1.21), P = .083

Abbreviations: aIRR, adjusted incidence rate ratio; AKI, acute kidney injury; CI, confidence interval; CVP, central venous pressure; LCOS, low cardiac output syndrome; RACHS-I, Risk Adjustment for Congenital Heart Surgery I; VAP, ventilator associated pneumonia; VIS, vasoactive inotrope score.

Systemic vascular resistance increased significantly during arrhythmia and remained elevated thereafter, which indicated a sustained vasoconstriction that might have contributed to persistent systolic hypotension and delayed hemodynamic recovery. The significant elevation in SVR observed during the arrhythmic period represented a potent, albeit maladaptive, sympathetic response to compromised SV. While the HR increase associated with the arrhythmia might have superficially maintained a baseline CO, the loss of effective CO caused a reflexive increase in vasomotor tone to sustain mean arterial pressure. This sustained vasoconstriction created a state of increased afterload that persisted into the postarrhythmic phase. For a myocardium already stressed by the rhythm disturbance, this increased SVR acted as a mechanical barrier to effective ejection, thereby paradoxically contributing to persistent systolic hypotension and a protracted hemodynamic recovery period.

Overall, these findings demonstrated that postoperative arrhythmia was associated with transient yet clinically significant disturbances in HR, SBP, DBP, SV, and SVR necessitating vigilant monitoring and timely intervention.

Clinical Impact of Arrhythmia

Patients with arrhythmia were 3.43-times more likely to develop LCOS and 2.55-times more likely to develop ventricular dysfunction. These findings suggested a deleterious feedback loop in which myocardial stress predisposed to arrhythmia, while loss of AV synchrony and rapid ventricular rates further compromised CO, necessitating escalation of inotropic support and perpetuating rhythm instability. Arrhythmia was also associated with a 4.56-times increased risk of liver dysfunction and 2.01-times increased risk of AKI, reflecting their sensitivity to acute hemodynamic deterioration during sustained arrhythmias. While the association with VAP showed an increased trend, it did not reach statistical significance,

suggesting that VAP itself reflected the disease severity rather than being a direct result of arrhythmia. Likewise, seizures were not significantly associated with arrhythmia, indicating that neurological complications were likely multifactorial.

In the present study, patients who developed arrhythmia experienced significantly longer ICU and hospital length of stay compared with those without arrhythmia. This prolongation likely reflected the higher burden of postoperative complications observed in the arrhythmia group, including LCOS, ventricular dysfunction, AKI, and liver dysfunction.

In our study, postoperative arrhythmia was associated with worse clinical outcomes on initial analysis; however, this association did not persist after adjusting for prearrhythmia hemodynamic variables (CVP and VIS). Arrhythmia was not independently associated with adverse clinical outcomes, including LCOS, ventricular dysfunction, AKI, and liver dysfunction, with the exception of a modest association with VAP. In contrast, higher prearrhythmia variables such as CVP and VIS consistently emerged as independent predictors of adverse outcomes. These findings suggested that underlying hemodynamic compromise—reflected by venous congestion and increased inotropic requirement—played a more central role in determining postoperative morbidity. In this context, arrhythmia appears more likely to be a marker of an already unstable cardiovascular state rather than a primary cause. At the same time, the presence of arrhythmia, through tachycardia and loss of AV synchrony, may further contribute to instability. Clinically, this highlights the importance of early recognition and optimization of hemodynamic stress before the onset of arrhythmia, rather than focusing solely on rhythm control.

Risk Factors for Postoperative Arrhythmia

Identifying predictors of postoperative arrhythmia is essential for optimizing recovery. In our analysis, demographic and intraoperative factors were not independent predictors, whereas prearrhythmia hemodynamic and biochemical markers were strongly associated with increased risk.

Analysis of Surgical and Intraoperative Factors

Higher surgical complexity has been linked to increased postoperative arrhythmia risk.⁹ Although RACHS-1 category was associated with arrhythmia on categorical analysis, it was not an independent predictor in regression, suggesting that postoperative hemodynamic factors may be more influential than surgical complexity alone. This suggests that the relationship between surgical complexity and arrhythmia risk may be nonlinear or mediated through other downstream postoperative factors rather than exerting a direct, independent effect.

Interestingly, traditional surgical metrics like CPB duration and ACC time did not emerge as independent risk factors in our study cohort. Our findings differed from those of previous studies that reported an association between prolonged CPB duration and a higher incidence of postoperative arrhythmias.^{5,6,8,10,11}

Table 6. Analysis of Risk Factors for Arrhythmias.

Prearrhythmia Variable	Nonarrhythmia (n = 276)	Arrhythmia (n = 53)	P Value
Prearrhythmia HR (beats/min)	109.5 (88-132)	103.5 (88-123)	.420
Prearrhythmia SBP (mm Hg)	93 (78-112)	103 (78-111)	.454
Prearrhythmia DBP (mm Hg)	54 (44-76)	65 (44-74)	.353
Prearrhythmia CVP (mm Hg)	10 (8-12)	15 (12-18)	<.001
Prearrhythmia VIS	10 (8-12)	14 (10-16)	<.001
Prearrhythmia CO (L/min) by ICON	2.8 (1.8-3.8)	2.45 (1.8-3.8)	.824
Prearrhythmia SV (mL) by ICON	26 (17-36)	24 (18.5-36)	.969
Prearrhythmia SVR (dyn·s·cm ⁻⁵) by ICON	987 (876-1214)	986.5 (876-1183.5)	.569
Prearrhythmia TFC by ICON	31 (28-32)	32 (28-32)	.623
Prearrhythmia serum potassium (mmol/L), median (IQR)	4.5 (3.9-4.8)	3.4 (3.2-4.3)	<.001
Prearrhythmia serum magnesium (mg/dL), median (IQR)	0.8 (0.8-0.9)	0.8 (0.8-0.8)	.291
Prearrhythmia serum lactate (mmol/L), median (IQR)	0.8 (0.8-1.2)	1.2 (0.9-1.9)	<.001
CK-MB Max* (ng/mL)	128.8 (84.7-188.0)	148.9 (112.8-223.8)	.012
Troponin I Max* (pg/mL)	1128.0 (778.5-1612.5)	1438.0 (989.0-2213.0)	.002
CRP Max* (mg/L)	34.3 (29.8-45.9)	30.9 (23.7-41.0)	.016

Abbreviations: CK-MB, creatinine kinase MB; CO, cardiac output; CRP, C-reactive protein; CVP, central venous pressure; DBP, diastolic blood pressure; HR, heart rate; ICON, electro cardiometry based noninvasive cardiac output measurement; IQR, interquartile range; Prearrhythmia all measurement were taken on arrival in ICU; SBP, systolic blood pressure; SV, stroke volume; SVR, systemic vascular resistance; TFC, thoracic fluid content; VIS, vasoactive inotrope score.

Table 7. IRR of Different Variables Studied as Possible Association With Arrhythmia Occurrence.

Variables	IRR	95% Confidence Interval	P Value	Adjusted IRR	95% Confidence Interval	P Value
Age (years)	0.99	0.94–1.06	.834	—	—	—
RACHS-I category	1.26	0.95–1.68	.107	—	—	—
Cardiopulmonary bypass duration (min)	0.95	0.85–1.5	.625	—	—	—
Aortic cross-clamp time (min)	1.01	0.99–1.02	.554	—	—	—
Prearrhythmia heart rate (bpm)	0.99	0.98–1.01	.342	—	—	—
Prearrhythmia systolic BP (mm Hg)	1.01	0.99–1.02	.560	—	—	—
Prearrhythmia diastolic BP (mm Hg)	1.01	0.99–1.03	.522	—	—	—
Prearrhythmia central venous pressure (mm Hg)	1.62	1.40–1.86	<.001	1.24	1.16–1.33	<.001
Prearrhythmia vasoactive-inotropic score (per unit)	1.19	1.10–1.29	<.001	1.12	1.05–1.19	<.001
Prearrhythmia serum magnesium (mmol/L)	0.28	0.03–2.81	.279	—	—	—
Prearrhythmia serum potassium (mmol/L)	0.22	0.13–0.38	<.001	0.31	0.22–0.46	<.001
Prearrhythmia lactate (mmol/L)	3.38	1.82–6.29	<.001	—	—	—
Prearrhythmia cardiac output (c/min)	0.95	0.78–1.15	.625	—	—	—
Prearrhythmia stroke volume (mL)	1.00	0.97–1.02	.809	—	—	—
Prearrhythmia SVR	1.00	1.00–1.00	.707	—	—	—
Prearrhythmia thoracic fluid content	0.96	0.87–1.07	.483	—	—	—

Univariable and multivariable Poisson regression with robust variance was used to estimate IRR. Variables with $P < .20$ on univariable analysis were entered into the multivariable model using stepwise backward elimination. Adjusted IRRs (aIRR) are shown for variables retained in the final model. A P value $< .05$ was considered statistically significant.

Abbreviations: BP, blood pressure; IRR, incidence rate ratio; SVR, systemic vascular resistance.

The lack of statistical significance in our study cohort might be attributed to several factors: (a) Uniformity of Surgical Complexity: The IQR for both groups was relatively narrow, suggesting a high volume of procedures with moderate complexity (such as VSD and TOF repairs) that fell within a predictable time window. Moreover, we excluded children who had a complicated intraoperative course or experienced any intraoperative adverse events. (b) Effective Myocardial Protection: Modern cardioplegia techniques and consistent delivery could mitigate the arrhythmogenic effects of ischemia, even as ACC time approached the 60 to 90 min mark.

Hemodynamic Predictors—Central Venous Pressure and Vasoactive-Inotropic Score

Elevated CVP independently predicted arrhythmia likely reflecting myocardial stretch from venous congestion or ventricular stress that promotes arrhythmogenesis. This mechanical stretch on the atrial and ventricular myocardium could trigger electrophysiological changes, such as stretch-activated channel signaling, which predispose the heart to reentry or increased automaticity. In the multivariable model, each 1mm Hg increase in CVP was associated

with a 24% increase in the adjusted IRR (aIRR;1.24, 95% CI 1.16-1.33, $P<.001$).

Similarly, the VIS was also an independent predictor (aIRR 1.12, $P<.001$). A higher VIS reflected a greater requirement of catecholamine support to maintain stable hemodynamics, indicating underlying myocardial fragility/ventricular dysfunction or LCOS. The use of exogenous catecholamines, while necessary to support a hypocontractile myocardium, was inherently proarrhythmic as it increased intracellular calcium and enhanced myocardial irritability through beta-adrenergic stimulation. These findings were found to be consistent with prior reports.^{4,6,9}

Therefore, although arrhythmia was associated with adverse outcomes on unadjusted analysis, it was not independently predictive after adjustment for prearrhythmia hemodynamic variables, suggesting that it reflected underlying hemodynamic instability rather than a primary causal factor. However, the temporal interplay between LCOS, inotrope use, and arrhythmia remained complex.

Electrolyte and Metabolic Stability

Potassium is critical for myocardial membrane stability, and even mild hypokalemia can shorten the refractory period and increase susceptibility to triggered activity. Potassium concentrations within the normal range were associated with a lower incidence of arrhythmias following cardiac surgery.¹¹ Consistent with these findings, multivariable analysis showed that higher normal-range serum potassium independently reduced arrhythmia (aIRR 0.31, 95% CI 0.22-0.46; $P<.001$).

Elevated lactate is associated with shock, organ dysfunction, and mortality.¹² Sahu et al⁴ demonstrated that elevated lactate levels are associated with postoperative arrhythmias following cardiac surgery. In our study, although lactate predicted arrhythmia on univariable analysis (IRR 3.38, $P<.001$), it was not an independent risk factor after adjustment. This suggested that while elevated lactate level reflected impaired perfusion, its predictive value for arrhythmia could be largely accounted for by the VIS and CVP, which serve as more direct indicators of the underlying hemodynamic state.

Myocardial Injury, Inflammation, and Risk Factors

Elevated postoperative cardiac biomarkers were associated with adverse clinical outcomes following cardiac surgery. Elevated peak CK-MB independently predicts postoperative mortality,¹³ while increased troponin levels correlate with myocardial dysfunction and LCOS, reflecting significant myocardial injury.¹⁴ Building on this existing evidence, our study further demonstrated that higher peak levels of troponin I and CK-MB were also significantly associated with the occurrence of postoperative arrhythmias, suggesting a potential link between myocardial injury and arrhythmogenesis in this population. In the existing literature, CRP has been associated with infection, organ dysfunction, and mortality.^{15,16} Higher levels of CRP

in the nonarrhythmia group suggested that systemic inflammation alone might not predict postoperative arrhythmia.

Study Limitation

This single-center study may limit generalizability. Hemodynamic assessment relied on electrical cardiometry (ICON[®]), a noninvasive and continuous monitoring tool available at our institution. However, this modality may not be universally available across all congenital cardiac ICUs. Alternative methods such as echocardiography or thermodilution-based techniques are also used to assess CO, SVR, and tissue perfusion in different clinical settings. Therefore, the hemodynamic findings of this study should be interpreted in the context of the monitoring modality used and may not be directly generalizable to all centers. Furthermore, our institutional case mix included relatively few highly complex neonatal procedures. Therefore, the findings may not be directly generalizable to centers performing a larger proportion of complex neonatal operations, such as Norwood palliation or staged single-ventricle palliation. Despite RACHS-1 adjustment, heterogeneity of defects and procedures may have resulted in residual confounding. Multiple comparisons increase the risk of type I error, and the observational design precludes causal inference. Although multivariable analysis was performed, unmeasured confounding and a modest event count may limit estimate stability.

Conclusions

Postoperative arrhythmias occurred in approximately one-sixth of children following congenital cardiac surgery and were associated with significant hemodynamic changes and adverse clinical outcomes on unadjusted analysis. However, after adjustment for prearrhythmia hemodynamic variables, arrhythmia was not independently associated with most clinical adverse outcomes. This suggests that postoperative arrhythmias represent a marker of underlying hemodynamic instability rather than a primary causal factor. Elevated prearrhythmia CVP and VIS emerged as consistent independent predictors of adverse outcomes, highlighting the central role of preexisting hemodynamic stress. These findings underscore the importance of early identification and optimization of hemodynamic compromise to potentially mitigate postoperative morbidity, while also recognizing that arrhythmia might further exacerbate an already unstable physiological state.

Acknowledgments

The authors would like to express their sincere gratitude to the Biostatistician Dr Sumit for his assistance with statistical analysis of this study.

ORCID iDs

Raheja D  <https://orcid.org/0000-0002-8693-9515>
Sahu MK  <https://orcid.org/0000-0002-3322-5834>
Khan MS  <https://orcid.org/0009-0003-0770-0800>
Singh SP  <https://orcid.org/0000-0001-6899-1670>

Talwar S  <https://orcid.org/0000-0002-9063-7691>
 Devagourou V  <https://orcid.org/0000-0001-8344-4253>

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

References

1. Ganea G, Cinteza EE, Filip C, et al. Postoperative cardiac arrhythmias in pediatric and neonatal patients with congenital heart disease—a narrative review. *Life (Basel)*. 2023;13(12):2278. doi:10.3390/life13122278
2. Jenkins KJ, Gauvreau K, Newburger JW, Spray TL, Moller JH, Iezzoni LI. Consensus-based method for risk adjustment for surgery for congenital heart disease. *J Thorac Cardiovasc Surg*. 2002;123(1):110–118. doi:10.1067/mtc.2002.119064
3. Talwar S, Patel K, Juneja R, Choudhary SK, Airan B. Early postoperative arrhythmias after pediatric cardiac surgery. *Asian Cardiovasc Thorac Ann*. 2015;23(7):795–801. doi:10.1177/0218492315585457
4. Sahu MK, Das A, Siddharth B, et al. Arrhythmias in children in early postoperative period after cardiac surgery. *World J Pediatr Congenit Heart Surg*. 2018;9(1):38–46. doi:10.1177/2150135117737687
5. Delaney JW, Moltedo JM, Dziura JD, Kopf GS, Snyder CS. Early postoperative arrhythmias after pediatric cardiac surgery. *J Thorac Cardiovasc Surg*. 2006;131(6):1296–1300. doi:10.1016/j.jtcvs.2006.02.010
6. Jain A, Alam S, Viralam SK, Sharique T, Kapoor S. Incidence, risk factors, and outcome of cardiac arrhythmia postcardiac surgery in children. *Heart Views*. 2019;20(2):47–52. doi:10.4103/HEARTVIEWS.HEARTVIEWS_88_18
7. Grosse-Wortmann L, Kreitz S, Grabitz RG, et al. Prevalence of and risk factors for perioperative arrhythmias in neonates and children after cardiopulmonary bypass: Continuous holter monitoring before and for three days after surgery. *J Cardiothorac Surg*. 2010;5(1):85. doi:10.1186/1749-8090-5-85
8. Valsangiacomo E, Schmid ER, Schüpbach RW, et al. Early postoperative arrhythmias after cardiac operation in children. *Ann Thorac Surg*. 2002;74(3):792–796. doi:10.1016/S0003-4975(02)03786-4
9. Ozturk E, Kafalı HC, Tanıdır İC, et al. Early postoperative arrhythmias in patients undergoing congenital heart surgery. *Turk J Thorac Cardiovasc Surg*. 2021;29(1):27–35. doi: 10.5606/tgkdc.dergisi.2021.20366
10. Alotaibi RK, Saleem AS, Alsharef FF, et al. Risk factors of early postoperative cardiac arrhythmia after pediatric cardiac surgery: A single-center experience. *Saudi Med J*. 2022;43(10):1111–1119. doi: 10.15537/smj.2022.43.10.20220275
11. Yasa KP, Katritama AA, Harta IK, Sudarma IW. Prevalence and risk factors analysis of early postoperative arrhythmia after congenital heart surgery in pediatric patients. *J Arrhythm*. 2024;40(2):356–362. doi:10.1002/joa3.13011
12. Gulla KM, Sahoo T, Gupta D, Sachdev A. Prediction of early intensive care unit mortality with serial serum lactate levels and its clearance in children with septic shock and multiorgan dysfunction syndrome. *J Pediatr Crit Care*. 2020;7(6):321–326. doi:10.4103/JPCC.JPCC_114_20
13. Wang Z, Ma K, Zhu Y, Li Z, Li S. Predictive value of myocardial markers for early postoperative mortality in children with congenital heart disease. *Pediatr Cardiol* 2025;46(2):324–331. doi:10.1007/s00246-024-03404-0
14. Perez-Navero JL, de la Torre-Aguilar MJ, de la Rosa II, et al. Cardiac biomarkers of low cardiac output syndrome in the postoperative period after congenital heart disease surgery in children. *Rev Esp Cardiol (Engl Ed)*. 2017;70(4):267–274. doi:10.1016/j.rec.2016.09.011
15. Lobo SM, Lobo FR, Bota DP, et al. C reactive protein levels correlate with mortality and organ failure in critically ill patients. *Chest*. 2003;123(6):2043–2049. doi:10.1378/chest.123.6.2043
16. Santonocito C, Sanfilippo F, De Locker I, et al. C reactive protein kinetics after cardiac surgery: A retrospective multicenter study. *Ann Card Anaesth*. 2022;25(4):498–504. doi:10.4103/aca.aca_141_21