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REVIEW

Incidence and Risk Factors for Early Postoperative Arrhythmias in Congenital Heart Disease – Systematic Review

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ABSTRACT

Postoperative arrhythmias are commonly seen in pediatric cardiac intensive care units and are linked to higher rates of both morbidity and mortality in children with congenital heart disease. However, the incidence of early postoperative arrhythmias in the pediatric population is unclear, varying from 7.3% to 48% in the literature. We searched the PubMed, Embase, and Web of Science databases from 2000 to 2025 with the aim to perform a systematic review of the existing literature on the incidence and risk factors of early arrhythmias following heart surgery. A total of 16 cross-sectional observational studies, including 5,563 patients who underwent surgery for congenital heart disease and 901 patients who developed early postoperative arrhythmias, met the inclusion criteria. Patients developing early postoperative dysrhythmias were younger, with a lower body weight, and the duration of cardiopulmonary bypass was significantly longer. Other incriminated risk factors for the occurrence of early postoperative arrhythmias were hemodynamic instability, complexity of the surgical procedure, and higher vasoactive-inotropic scores. Pediatric patients with congenital heart disease who undergo cardiac surgery face increased morbidity and mortality due to the risk associated with the multifactorial complication of early postoperative arrhythmias. The reported incidence of these arrhythmias varies greatly among different studies and a better understanding of risk factors and pathophysiological mechanisms would improve postoperative outcomes for this notably exposed population.

Keywords: early postoperative arrhythmias, congenital heart disease, pediatric, cardiac surgery

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INTRODUCTION

Nearly one-third of all major congenital anomalies involve cardiac defects, affecting roughly 9 per 1,000 births worldwide.[1,2] Cardiac arrhythmias are disorders of the heart's electrical system that result in abnormal heart rhythms.[3] These irregularities may manifest as anomalies in heart rate, rhythm, or conduction pathways. Broadly, arrhythmias are divided into two major categories: tachyarrhythmias, which are characterized by a heart rate exceeding 100 beats per minute, and bradyarrhythmias, in which the heart rate is below 60 beats per minute. Furthermore, arrhythmias are classified according to their site of origin, with supraventricular arrhythmias arising above the ventricles and ventricular arrhythmias originating within the ventricles. Common examples include atrial fibrillation, atrial flutter, supraventricular tachycardia (SVT), ventricular tachycardia (VT), and ventricular fibrillation (VF).[4–9]

Postoperative arrhythmias are frequently observed in pediatric cardiac intensive care units (ICUs)[10] and are associated with significant hemodynamic deterioration,[11–14] which in turn increases both morbidity and mortality.[14] Although these arrhythmias are generally temporary and often resolve with medical treatment, their presence in the ICU can substantially worsen a patient's condition.[6,15–18]

Postoperative arrhythmias in pediatric patients with congenital heart defects (CHDs) are highly diverse, presenting as either tachyarrhythmias or bradyarrhythmias. Bradyarrhythmias typically manifest as sinus bradycardia and atrioventricular blocks (AVB), whereas tachyarrhythmias encompass atrial tachycardia (including intra-atrial reentrant tachycardia and atrial flutter), junctional ectopic tachycardia (JET), and various forms of ventricular tachycardia (such as premature ventricular complexes, monomorphic VT, Torsades de Pointes, and ventricular fibrillation).[8]

The underlying causes of early postoperative arrhythmias include direct injury to the cardiac conduction system during surgery, as well as edema and inflammation in the myocardial tissue adjacent to the conduction pathways. In addition, factors such as ischemia–reperfusion injury associated with cardiopulmonary bypass (CPB), electrolyte imbalances, acid–base disturbances, the patient's age at the time of surgery, hemodynamic instability, pain, sedation, and the use and dosage of inotropic drugs may all contribute to both the development and frequency of early postoperative arrhythmias.[11–15,20–24] In pediatric patients, these arrhythmias can be life-threatening; therefore, prompt and accurate diagnosis coupled with appropriate management is essential to prevent further complications. Although many arrhythmias resolve spontaneously or with pharmacological treatment, some cases require additional interventions, such as defibrillation or the implantation of a permanent pacemaker.[5,11,12,16,17,25–27]

Given that the reported incidence of postoperative arrhythmias varies widely, from 6.78% to 48%, obtaining a more precise estimate is crucial. Improved accuracy in incidence data could allow clinicians to better tailor monitoring and treatment strategies.[6,18–20]

Due to the lack of a clear picture of the incidence of early postoperative arrhythmias in pediatric patients with CHD, we performed a systematic review of the literature. Our aim was to assess the occurrence of dysrhythmias in the pediatric CHD population and to identify the risk factors that contribute to their development.

METHODS

SEARCH STRATEGY

We searched the PubMed, Embase and Web of Science databases using the following queries: "arrythmias, pediatric, congenital heart disease, incidence", "arrhythmias, pediatric, congenital heart disease, risk factors", "arrhythmias, pediatric, congenital heart disease, cardiac surgery", returning the results displayed in Figure 1. Using predefined search filters in several databases for observational studies, a number of 1,597 records were excluded, and identified references were checked for duplicates. Afterwards, titles and abstracts were screened manually for an additional removal of 324, qualifying a total of 43 articles for further evaluation. Another 27 were excluded due to missing data, absence of control group, no separate analysis of arrhythmias, or small cohorts. Finally, 16 studies were included (Table 1).

INCLUSION AND EXCLUSION CRITERIA

Studies were included if they met the following criteria: (1) the investigation focused on the incidence of early postoperative arrhythmias; (2) it was a cross-sectional observational study; (3) the patient population consisted of individuals under 19 years of age; (4) the data pertained to patients undergoing congenital heart surgery; and (5) the study provided case numbers, means, and standard deviations, or sufficient information to calculate them.

Studies were excluded if: (1) they provided data on late postoperative arrhythmias; (2) they involved therapeutic methods other than surgical interventions; (3) the patient population consisted of individuals over 19 years of age;

TABLE 1. Summary of included studies

No.	Author & year	Country	Type of study	Study period	Population size
1	Pfammater *et al.*[13], 2002	Switzerland	Prospective	July 1996 – unspecified	172
2	Valsangiacomo *et al.*[7], 2002	Switzerland	Prospective	November 1998 – January 2000	100
3	Delaney *et al.*[24], 2006	USA	Prospective	September 2000 – May 2003	189
4	Rekawek *et al.*[12], 2007	Poland	Prospective	January – December 2005	402
5	Chaiyarak *et al.*[22], 2008	China	Prospective	January – December 2006	191
6	Yildirim *et al.*[19], 2008	Turkey	Retrospective	May 2001 – December 2002	580
7	Kamel et Sewielam[27], 2009	Egypt	Retrospective	September 2007 – January 2009	95
8	Massin *et al.*[26], 2010	Belgium	Prospective	January 2007 – June 30	141
9	Talwar *et al.*[17], 2015	India	Prospective	September 2013 – July 2014	200
10	Nelson *et al.*[25], 2018	USA	Retrospective	January 2012 – December 2014	196
11	Sahu *et al.*[23], 2018	India	Prospective	November 2015 – December 2016	369
12	Jain *et al.*[5], 2019	India	Retrospective	April – July 2016	536
13	Öztürk *et al.*[11], 2021	Turkey	Retrospective	December 2018 – November 2019	670
14	Alotaibi *et al.*[21], 2022	Saudi Arabia	Retrospective	January 2015 – December 2020	821
15	Ishaque *et al.*[20], 2022	Pakistan	Retrospective	January 2014 – December 2018	812
16	Yasa *et al.*[8], 2024	Indonesia	Retrospective	January 2020 – December 202	92

(4) they were determined to be of low quality; or 5) they were published before 2000.

QUALITY ASSESSMENT

The Newcastle–Ottawa Scale for assessing the quality of cohort studies was used to evaluate the included studies (Table 2). All studies scored a minimum of seven stars (high-quality studies).

RESULTS

All 16 articles included in the review were cross-sectional, observational studies conducted at single centers. The duration of the studies ranged from 1 to 5 years. In one study,[13] initiated in 1999, no specific end date or overall time span was provided; instead, the study period was defined as the entire postoperative hospital stay for each patient, from the time of surgical intervention until discharge. Given that the length of hospital stay varied for

TABLE 2. Quality assessment using the Newcastle–Ottawa scale

No.	Author & year	Selection	Comparability	Outcome	Total
1	Pfammater *et al.*[13], 2002	4	1	3	8
2	Valsangiacomo *et al.*[7], 2002	4	1	3	8
3	Delaney *et al.*[24], 2006	4	1	3	8
4	Rekawek *et al.*[12], 2007	4	1	3	8
5	Chaiyarak *et al.*[22], 2008	4	1	3	8
6	Yildirim *et al.*[19], 2008	3	1	3	7
7	Kamel et Sewielam[27], 2009	3	1	3	7
8	Massin *et al.*[26], 2010	3	1	3	7
9	Talwar *et al.*[17], 2015	4	1	3	8
10	Nelson *et al.*[25], 2018	4	1	3	8
11	Sahu *et al.*[23], 2018	4	1	3	8
12	Jain *et al.*[5], 2019	4	2	3	9
13	Öztürk *et al.*[11], 2021	4	1	3	8
14	Alotaibi *et al.*[21], 2022	4	2	3	9
15	Ishaque *et al.*[20], 2022	4	1	3	8
16	Yasa *et al.*[8], 2024	4	2	3	9

TABLE 1. Summary of included studies

No.	Author & year	Sample size	Arrhythmia cases (n)	Arrhythmia cases (%)	Groups	No. of patients	Age (months)	CBP time (min)	ACC time (min)
1	Pfammater *et al.*[13], 2002	172	59	34.30%	Arrhythmia	59	16.62 ± 4.79	–	–
					Non-arrhythmia	113	33 ± 11.25		
2	Valsangiacomo *et al.*[7], 2002	100	48	48.00%	Arrhythmia	48	25.26 ± 53.25	–	–
					Non-arrhythmia	52			
3	Delaney *et al.*[24], 2006	189	28	14.81%	Arrhythmia	28	22 ± 39.7	189 ± 106	105 ± 73.6
					Non-arrhythmia	161		109 ± 29.6	44 ± 22.2
4	Rekawek *et al.*[12], 2007	402	57	14.18%	Arrhythmia	57	18.74 ± 39.04	169 ± 69.2	82.6 ± 37.1
					Non-arrhythmia	345	31.23 ± 47.7	128.7 ± 134.9	53.6 ± 34.2
5	Chaiyarak *et al.*[22], 2008	580	45	7.76%	Arrhythmia	45	48.03 ± 45.26	96 (30–251)	48 (0–147)
					Non-arrhythmia	146	38.33 ± 47.47	65 (20–240)	36 (0–147)
6	Yildirim *et al.*[19], 2008	191	51	26.70%	Arrhythmia	51	20.4 ± 27.6	105 .4 ± 54.1	56.7 ± 27.2
					Non-arrhythmia	529	–	–	–
7	Kamel et Sewielam[27], 2009	95	30	31.58%	Arrhythmia	30	11 ± 2	105.4 ± 53.1	50 ± 22.5
					Non-arrhythmia	65	16 ±3	80.8 ± 36.3	50 ± 22.5
8	Massin *et al.*[26], 2010	141	25	17.73%	Arrhythmia	25	2.56 ± 3.28	139.64 ± 51.15	76.20 ± 32.99
					Non-arrhythmia	116	2.39 ± 3.40	113.55 ± 37.47	56.26 ± 24.72
9	Talwar *et al.*[17], 2015	200	15	7.50%	Arrhythmia	15	41.9 ± 65.4	108.5 ± 46.7	62.2 ± 21.42
					Non-arrhythmia	185	50.7 ± 62.2	79.99 ± 36.5	50.6 ± 22.43
10	Nelson *et al.*[25], 2018	196	18	9.18%	Arrhythmia	18	5.9 ± 1.8	123 (112–187)	74 (56–128)
					Non-arrhythmia	178	28.8 ± 24.3	97 (70–144)	45 (16–80)
11	Sahu *et al.*[23], 2018	369	25	6.78%	Arrhythmia	25	39.36 ± 42.6	103.6 ± 57.4	60.04 ± 32.8
					Non-arrhythmia	344	37.2 ± 42	90.04 ± 44/7	55.1 ± 26.5
12	Jain *et al.*[5], 2019	536	77	14.37%	Arrhythmia	77	16	–	–
					Non-arrhythmia	459			
13	Öztürk *et al.*[11], 2021	670	84	12.54%	Arrhythmia	84	4 ± 53.99	–	–
					Non-arrhythmia	586			
14	Alotaibi *et al.*[21], 2022	821	140	17.05%	Arrhythmia	140	–	77 (57–99.5)	54 (38–70)
					Non-arrhythmia	681		60 (45–81)	45 (32–62)
15	Ishaque *et al.*[20], 2022	812	185	22.78%	Arrhythmia	185	–	140.4 ± 70.2	55.6 ± 27.4
					Non-arrhythmia	627			
16	Yasa *et al.*[8], 2024	92	14	15.22%	Arrhythmia	14	69.71 ± 65.71	171.23 ± 52.64	115.58 ± 34.38
					Non-arrhythmia	75	88.78 ± 65.28	108.1 ± 48.44	73.59 ± 37.28

each patient, the overall study duration would be defined as the time interval between the admission date of the first patient included in the study and the discharge date of the last patient to be discharged among all participants. Details concerning patient groups analyzed by extracted studies are presented in Table 3.

INCIDENCE AND TYPE OF ARRHYTHMIAS

The incidence reported in the selected studies ranged from 6.78%[24] to 48%[7] (Table 3). Considering that the oldest studies[7,13] reported the highest incidences, it can be argued that advancements in surgical techniques have contributed to the reduction in the incidence of postoperative arrhythmias, enhancing outcomes for pediatric patients undergoing heart surgery. Studies[5,8,1117,20,21,23,25] published in the last 10 years converge on reporting lower incidences between 7.50%[17] and 22.78%.[20] Our literature search yielded a broad spectrum of postoperative arrhythmias among patients with CHD, with considerable variability in reported incidences across studies. The most frequently reported include JET, AVB, and SVT. Additionally, junctional rhythms, VT, VF, and atrial fibrillation are also noted, though less frequently.

The spectrum of postoperative arrhythmias is notably broad, and the selected studies have reported substantial disparities in the incidence and predominance of specific arrhythmia types.

While JET and atrio-ventricular block (AVB) incidences show marked differences across studies, with the highest incidences reported by Alotaibi *et al.*[21] and Kamel and Sewielam,[27] and the lowest by Yildirim *et al.*,[19] the occurrence of other arrhythmias, such as junctional rhythm, VT, VF, and atrial fibrillation, is generally much lower.

Notably, the highest overall incidence of JET (25.6%) was reported by Alotaibi *et al.*[21] and Kamel and Sewielam.[27] In stark contrast, Yildirim *et al.*[19] documented the lowest incidence at only 2.06%. Intermediate rates were observed in several other studies.[5,11,13,22,24] Surgical manipulation near the crux of the heart, particularly during repairs of ventricular septal defects (VSDs), atrioventricular septal defects (AVSDs), and tetralogy of Fallot (TOF), can result in mechanical stretching, edema, or even direct injury to the atrioventricular (AV) node. This trauma may induce enhanced automaticity in the junctional tissue, leading to the high incidence of JET observed in multiple studies.[21,27]

Similarly, there is considerable variability among studies reporting the incidence of postoperative AVB. Alotaibi *et al.*[21] reported the highest overall incidence at 14.6%, whereas Yildirim *et al.*[19] found the lowest at only 1.2%; other studies[5,11,13,20,27] have reported intermediate values. This variability likely reflects differences in surgical technique and the degree of exposure of the AV node during repair, as inadvertent injury to this specialized conduction tissue, especially when operating in close proximity to the AV node, substantially increases the risk for AVB.[28]

SVT exhibits notable variability in incidence among patients with postoperative arrhythmia. Kamel and Sewielam[27] reported the highest incidence at 35.7%, whereas Alotaibi *et al.*[21] and Valsangiacomo *et al.*[7] observed considerably lower rates of 10% and 9%, respectively. This disparity may be partly explained by the systemic inflammatory response induced by CPB, which can lead to myocardial edema and alter the electrophysiological properties of cardiac tissue. Additionally, elevated catecholamine levels in the postoperative period further increase myocardial excitability, creating a setting in which reentrant circuits and abnormal automaticity are more easily triggered, thereby predisposing patients to SVT.[28,29] Electrolyte disturbances, which are common after extensive surgery, may further modify the conduction properties of the myocardium, contributing to the genesis of JET, AVB, and SVT. Additionally, transient ischemia during surgery can alter refractoriness and conduction velocity, facilitating the development of these arrhythmias.[30]

By contrast, arrhythmias such as VT, VF, and atrial fibrillation typically require a more specific arrhythmogenic substrate, such as significant scarring or well-established reentrant circuits, which is less commonly created during congenital cardiac surgeries. This likely accounts for their relatively lower incidence in the postoperative period.

DURATION AND ONSET OF ARRHYTHMIAS

Despite the variability in overall incidence, most studies converge on the observation that most arrhythmias occur within the first 24 h after surgery. Alotaibi *et al.*[21] found that 79.3% of arrhythmias manifested within the first 24 h, a finding corroborated by several other studies[17,23,25,27] in which at least two-thirds of patients developed arrhythmias within this timeframe. Furthermore, Chaiyarak *et al.*[22] provided additional detail, noting that a small subset (4.4%) of arrhythmias occurred during surgical intervention.

Nelson *et al.*[25] added to the perspective by reporting a median arrhythmia duration of 22 h, while Jain *et al.*[5] provided further information on early onset of dysrhythmia, noting that 92% of JET cases and most junctional escape rhythm events (84.6%) were detected within 24 h.

However, Yildirim *et al.*[19] described arrhythmias occurring as late as 72 h post surgery, while Öztürk *et al.*[11] noted that although most JET cases (89.5%) were identified within 24 h, a few were detected 72 h postoperatively. Kamel and Sewielam[27] documented a case that manifested beyond 48 h after surgery. The delayed onset of arrhythmias during the early postoperative period can be attributed to several factors, including the body's inflammatory response, hemodynamic changes, electrolyte imbalances, pain and stress, and the effects of medications administered during and after surgery.[30,31]

RISK FACTORS ASSOCIATED WITH EARLY POSTOPERATIVE ARRHYTHMIAS

Preoperative factors

Congenital heart disease subtype

The variability in conduction system anatomy across different CHDs can be an easily overlooked factor in the development of postoperative arrhythmias. In VSDs, for instance, Milo *et al.*[32] established marked differences in conduction pathways among VSD subtypes. In perimembranous VSDs, conduction tissue typically runs inferior to the defect, but its distance from the VSD crest can vary from as little as 2 mm to over 5 mm, depending on the defect's extension. In contrast, muscular inlet VSDs—with an intact membranous septum—consistently exhibit conduction running superiorly and more than 5 mm away.[32,33] Furthermore, anatomical variations, such as a straddling tricuspid valve, may displace the AV node inferiorly.[34] These differences heighten the risk of unintentional injury during surgical repair. Notably, our review suggests that VSD repairs are associated with a relatively lower incidence of postoperative arrhythmias; however, several selected studies reported a relatively low incidence of postoperative arrhythmias associated with VSD repairs: Alotaibi *et al.*[21] reported that 18.6% of arrhythmia cases occurred after VSD repair, while Chaiyarak *et al.*[22] observed only a 6.5% incidence of AVB; these findings were supported by others as well.[5,17]

In TOF, Dickinson *et al.*[35] reported significant variability in conduction anatomy. Although most TOF specimens display an AV bundle running posteriorly or inferiorly, sufficiently distant from the VSD to minimize risk, approximately 30% of cases with perimembranous extension exhibit conduction tissue positioned on or near the posterior or inferior VSD crest, which increases its susceptibility to surgical trauma.[35] This observation was supported by Nelson *et al.*,[25] who reported a 33% incidence of arrhythmias in TOF, and Kamel and Sewielam,[27] who found 39.2% of arrhythmia cases were associated with TOF.

AVSDs further complicate surgical management. Lev[36] described that in AVSDs the atrioventricular node and bundles are displaced posteroinferiorly, originating near the coronary sinus and coursing along the left aspect of the inferior VSD crest, which requires meticulous suture placement to avoid this particular region. Variations in the location of the His bundle have also been reported.[8] Supporting these findings, Ishaque *et al.*[20] documented a striking 64.3% incidence of arrhythmias (predominantly JET) following AVSD repair, whereas Kamel and Sewielam[27] noted that AVSD repairs in cyanotic patients accounted for 21.4% of arrhythmia cases.

In congenitally corrected transposition of the great arteries (ccTGA), bulboventricular looping significantly influences conduction system orientation. Anderson *et al.*[37–39] found that in L-looped ventricles, conduction typically runs anterosuperiorly relative to the VSD, whereas in D-looped ventricles it is positioned posteroinferiorly. Additional factors, such as septal alignment and the presence of a Monckeberg sling, further modulate the conduction location.[37–40]

In summary, the heterogeneous anatomy of the conduction system in CHDs—particularly in VSDs, TOF, AVCDs, and ccTGA—creates substantial challenges during surgical repair. Although there is some conflicting evidence regarding the risk associated with VSD repairs, our findings align with the literature[32–41] in establishing an obvious association between surgical manipulation near critical conduction tissue and the development of early postoperative arrhythmias, especially in TOF, AVSDs, and ccTGA.

Younger age and lower body weight

Younger age and lower body weight have been repeatedly linked to postoperative arrhythmias, likely due to a more vulnerable myocardium. Several studies[25–27] identified age as a risk factor; both Öztürk *et al.*[11] and Jain *et al.*[5] noted age <1 year as an independent risk factor (OR = 1.96; 95% CI 1.18–3.24; $p = 0.009$), most likely due to an immature cardiac conduction system, whose ion channel expression and autonomic regulation render the heart more susceptible to electrical instability, particularly when exposed to surgical manipulation. By contrast, Yildirim *et al.*[19] observed a nonsignificant trend in the 0–6-month group ($p = 0.351$), while Yasa *et al.*[8] (MW = 0.794; $p = 0.427$) reported no significant impact of age.

A retrospective Egyptian study from 2008[27] found that patients with arrhythmias were both younger (11 ± 2 months vs. 16 ± 3 months, $p < 0.05$) and lighter (8 ± 1.5 kg vs. 11.5 ± 1 kg, $p < 0.05$), a finding supported a decade later by Nelson *et al.*,[25] while Valsangiacomo *et al.*[7] identified only lower weight as independently significant ($p < 0.05$), supporting the hypothesis that lower weight, indicative of reduced myocardial mass, compromises the heart's ability to withstand the hemodynamic fluctuations and inflammatory responses induced by surgery, thereby increasing the risk of arrhythmias. Other studies[17,20–23] found no significant associations between arrhythmias, younger age, and lower weight.

Operative factors

Association of arrhythmias with specific surgical procedures

Arrhythmias following congenital heart surgery show distinct associations with specific repair types. The highest incidence is observed in complex procedures, particularly AVSD and TOF repairs, which inherently involve greater manipulation near the AV node. For example, Ishaque *et al.*[20] reported a 64.3% incidence of arrhythmias (primarily JET) after AVSD repair, while Nelson *et al.*[25] documented rates of 33% in TOF and 28% in AVSD repairs. Kamel and Sewielam[27] further emphasized that in cyanotic patients, TOF and AVSD repairs accounted for 39.2% and 21.4% of arrhythmia cases, respectively.

By contrast, VSD repairs tend to have a lower association with arrhythmias. Alotaibi *et al.*[21] reported that 18.6% of arrhythmia cases occurred after VSD repair, and Chaiyarak *et al.*[22] observed only 6.5% AVB in septal defect repairs. Other studies[5,17] support these findings, linking VSD repair and intraventricular tunnel procedures with a relatively lower, albeit still significant, risk of arrhythmias.

Additional studies[8,11,12,17] further substantiate these trends, highlighting that factors such as longer extracorporeal circulation times and the extent of surgical manipulation contribute to the observed variability. Thus, while AVSD and TOF repairs are most frequently associated with high arrhythmia rates, VSD repairs typically exhibit lower incidences, with intermediate values reported across various studies.

Complex procedures typically require extensive myocardial manipulation and are associated with prolonged CPB and aortic cross-clamp (ACC) times. These factors predispose the myocardium to ischemia, an exaggerated inflammatory response, and electrolyte imbalances, all of which destabilize cardiac electrophysiology. This concept is supported by studies[5,11,20] demonstrating that higher RACHS scores (≥3 or ≥4) and Aristotle levels (≥3) are independent predictors of postoperative arrhythmias. Furthermore, Kamel and Sewielam[27] observed that complex corrective surgeries, such as TOF and AVSD repairs, have a higher incidence of arrhythmias compared to palliative procedures, while Rekawek *et al.*,[12] Sahu *et al.*,[23] and Nelson *et al.*[25] confirmed that increased case complexity, as measured by the Aristotle score, is strongly associated with postoperative arrhythmias. Additionally, redo operations significantly elevate the risk of arrhythmias, as found by a retrospective study[27] in which 14.2% of patients in the arrhythmic group had undergone redo procedures, compared to only 1.7% in the nonarrhythmic group. This disparity is likely attributable to the scar tissue and fibrosis from previous interventions, which predispose the myocardium to conduction abnormalities.

Prolonged CPB and ACC times

The immaturity of the autonomic nervous system in younger patients can result in an exaggerated catecholamine surge during surgical stress. This heightened sympathetic activation further destabilizes the myocardium, increasing its susceptibility to arrhythmias, an effect further amplified by the additional stress of CPB.[28] Consequently, prolonged CPB and ACC times are consistently linked to early postoperative arrhythmias. For example, Alotaibi *et al.*[21] found each extra minute of CPB raised the odds of arrhythmia (OR = 1.014; 95% CI 1.008–1.019; $p = 0.0001$) and a longer ACC time also increased the risk (OR = 1.011; 95% CI 1.003–1.019; $p = 0.009$). Similarly, Kamel and Sewielam[27] noted that patients with arrhythmia had longer CPB times (105.4 ± 53.1 min vs. 80.8 ± 36.3 min, $p < 0.05$), a finding supported by Valsangiacomo *et al.*[7] ($p < 0.05$). Chaiyarak *et al.*[22] demonstrated that CPB times >85 min increased the odds of arrhythmias (OR = 4.87; 95% CI 2.34–10.12; $p < 0.001$; sensitivity 70.2%; specificity 67.4%) and JET (OR = 6.79; 95% CI 2.03–22.68; $p = 0.001$; sensitivity 83.3%; specificity 62.4%). Other studies also reported longer CPB times in the arrhythmia groups. For instance, Delaney *et al.*[24] observed 189 min vs. 109 min ($p < 0.05$), Nelson *et al.*[25] 124 min vs. 97 min ($p < 0.05$), and Pfammater *et al.*[13] found that the incidence of arrhythmia in VSD repairs rose from 7% (<50 min extracorporeal circulation (ECC)) to 39% (>50 min ECC). Ishaque *et al.*20 and Öztürk *et al.*[11] identified CPB cutoffs at >120 min and 140 min, respectively, while Yasa *et al.*[8] reported longer CPB times as significant ($p = 0.034$). By contrast, Jain *et al.*[5] and Rekawek *et al.*[12] did not find CPB time to be an independent factor in multivariate analysis ($p = 0.056$ and $p = 0.9$, respectively), and Sahu *et al.*[23] noted no significant difference ($p < 0.16$).

Regarding ACC time, Pfammater *et al.*[13] reported that in VSD repairs, the incidence of arrhythmia increased from 5% (<30 min) to 47% (30–60 min, $p < 0.01$). Children with arrhythmias had longer ECC (68 ± 17 min vs. 53 ± 14 min; $p < 0.01$) and ACC times (42 ± 15 min vs. 31 ± 9 min; $p < 0.01$). Similar trends were seen in TOF and CAVC repairs. Delaney *et al.*[24] noted ACC times of 105 ± 73.6 min in patients with arrhythmia vs. 44 ± 22.2 min in patients without arrhythmia ($p < 0.05$); these findings are in agreement with those of Massin *et al.*[26] ($p < 0.001$) and Jain *et al.*[5] (ACC >67 min, $p = 0.024$). However, Ishaque *et al.*[20] found that an ACC of >60 min was not significant (OR = 1.97; 95% CI 0.68–2.66; $p = 0.234$). Valsangiacomo *et al.*,[7] Öztürk *et al.*,[11] and Kamel and Sewielam[27] did not evaluate ACC time.

Postoperative factors

Hemodynamic instability

Hemodynamic instability is a critical risk factor for early postoperative arrhythmias. The stress from hemodynamic compromise triggers an exaggerated sympathetic response and increased catecholamine release, which enhances myocardial automaticity and irritability.[42] Alotaibi *et al.*[21] demonstrated this association with an OR of 2.467 in univariate analysis and a persistent significant relationship in multivariate analysis (OR = 1.722; 95% CI 1.104–2.884; $p = 0.018$). Additionally, Kamel and Sewielam[27] observed that patients who require high inotropic support, have hypotension in the ICU, and need prolonged ventilation are more susceptible to arrhythmias. Sahu *et al.*[24] further reported significant links with hypotension and tachycardia, likely because systemic hypoperfusion leads to metabolic acidosis and electrolyte imbalances (particularly of potassium and calcium) that destabilize cardiac electrophysiology.[43] Nelson *et al.*[25] noted that arrhythmic patients had lower early extubation and higher reintubation rates compared to nonarrhythmic patients. Moreover, Yildirim *et al.*[19] documented a high incidence of hemodynamic instability (73.2%) and a 29.4% mortality rate among these patients, although targeted therapy can mitigate these adverse effects, as shown by Valsangiacomo *et al.*[7]

Electrolyte disturbances and serum lactate

Electrolyte imbalances are known to predispose the myocardium to arrhythmias by disturbing normal electrophysiological processes. In the postoperative setting, children with congenital heart disease are especially vulnerable to these disturbances due to factors such as hemodilution during CPB, renal function alterations, and the systemic inflammatory response induced by surgery.[29,30,43] These factors can lead to significant electrolyte shifts, exacerbating myocardial instability and increasing the propensity for arrhythmias, as reported by Alotaibi *et al.*[21] and Yasa *et al.*[8] Although some studies[17,19,23,25,27] have reported conflicting results regarding the impact of specific electrolyte levels, there is evidence that supports the notion that disturbances in electrolyte homeostasis remain a risk factor for postoperative arrhythmias in this high-risk pediatric population.

Inotropic support and related variables

High inotropic support, as reflected by increased vasoactive inotropic scores (VIS), was another incriminated risk factor for arrhythmias. Kamel and Sewielam[27] reported that 32.1% of arrhythmic cases required high inotropic support on leaving the operating room compared to only 5.2% in the nonarrhythmic group ($p < 0.01$). This finding is further supported by Sahu *et al.*,[23] who demonstrated that a higher Werknovsky inotropic score is significantly associated with the occurrence of arrhythmia (arrhythmia group: OR = 20; 95% CI 5–52 vs. non-arrhythmia group: OR = 15; 95% CI 0–59; $p = 0.02$). Additional studies[11,20] identified VIS as a strong independent risk factor, likely due inotropic agents increasing myocardial irritability and automaticity by affecting cardiomyocyte membranes. By contrast, Nelson *et al.*[25] did not find a significant difference in VIS between arrhythmic and nonarrhythmic groups ($p = 0.787$).

DISCUSSION

From a geographical perspective, a higher incidence of postoperative arrhythmias was predominantly reported in developed countries during the early 2000s,[7,13] with a downward trend observed in subsequent years.[25,26] Given that older studies reported the highest incidences, it can be argued that technological progress and the refinement of surgical techniques have contributed to the reduction of the incidence of postoperative arrhythmias, thereby improving outcomes for pediatric patients undergoing cardiac surgery.

On the other hand, while in the first decade of the 21st century studies on the incidence of postoperative arrhythmias are largely absent in certain Asian and African countries, likely due to limited access to advanced cardiac surgical interventions, recent years have seen a notable shift. The economic development of these regions has contributed to significant improvements in medical infrastructure and the capacity to perform complex surgeries for CHD. Consequently, this progress has led not only to an increase in the number of published studies originating from these countries but also to the reporting of relatively high incidences[8,20,21] of postoperative arrhythmias.

However, the discrepancies in reported postoperative arrhythmia incidence mostly stem from variations in definitions, monitoring methods, and patient selection. For instance, while some studies[19,23] define arrhythmias as any non-sinus rhythm lasting over 30 s or requiring intervention, others, such as Delaney *et al.*,[24] include only events that require active treatment. Similarly, inconsistency exists in classifying SVT: some investigators[17,25] group all SVT subtypes under atrial tachycardia; others, such as Pfammater *et al.*,[13] distinguish between atrioventricular reentrant tachycardia and atrial flutter, while others[23,27] define SVT as a narrow complex tachycardia with one-to-one AV conduction. Likewise, criteria for AVB vary, with some studies[7,11,21] differentiating between second- and third-degree AVB and others[22,23] focusing solely on complete AVB.

Incidence differences also arise from arrhythmia selection criteria, duration of monitoring, and the patient case mix. For example, Rekawek *et al.*[12] and Delaney *et al.*[24] reported intervention-requiring arrhythmias in 14–15% of patients, whereas Alp *et al.*[44] and Grosse-Wortmann *et al.*[45] documented higher incidences when minor rhythm abnormalities were included, although these rates declined significantly when benign changes were excluded.

Surgical factors further contribute to these disparities. Intraoperative hemodynamic instability, along with prolonged CPB and ACC times, predisposes the myocardium to ischemia, reperfusion injury, and electrolyte shifts, all of which destabilize cardiac electrophysiology.[19,21,24,36] These effects are compounded by an exaggerated catecholamine surge in response to stress[47] and by direct surgical trauma from cannulation, suturing near the conduction system, and rapid intracardiac pressure changes.[7] Moreover, corrective and redo surgeries elevate arrhythmia risk due to additional tissue trauma and inflammatory responses.[27,47]

Specific mechanisms also vary by procedure, according to the literature. For instance, in TOF repairs, high rates of JET and SVT have been attributed to His bundle trauma from right atrial traction.[48] In patients with single ventricle physiology, inherent conduction abnormalities, prolonged CPB, and redo operations further increase the risk of JET. Jacobs *et al.*[49] observed that early arrhythmias were associated with myocardial swelling, unstable hemodynamics, high inotrope doses, and metabolic disturbances, while Nelson *et al.*[25] suggested that para-Hisian injury leading to abnormal automaticity may precipitate JET and subsequent complete heart block. Additionally, Sahu *et al.*[24] linked longer surgical duration, elevated serum lactate, hypotension, and tachycardia to myocardial insults, and other studies[50–53] have associated hypomagnesemia with JET.

Collectively, these findings highlight the complex interplay between methodological differences and pathophysiological factors in determining the incidence of postoperative arrhythmias in children with CHD.

STUDY LIMITATIONS

With a few exceptions that had a medium size cohort, most of the studies had relatively small sample sizes, which may have constrained the statistical power to detect less frequent arrhythmic events and limited the ability to conduct comprehensive subgroup analyses. Furthermore, the study populations were often clinically diverse, including a wide range of CHDs varying in anatomical complexity, surgical approaches, and postoperative management strategies. This clinical diversity may have introduced important confounding factors, including inconsistencies in inclusion criteria, such as differences in age at the time of surgery, type of surgical intervention, use of cardiopulmonary bypass, preoperative clinical status, and postoperative care protocols. This diversity, while representative of real-world pediatric cardiology practice, complicates the identification of specific risk factors for postoperative arrhythmias and makes it more difficult to isolate specific associations. Larger-scale studies with more homogenous subgroups are needed to confirm these findings and better define risk factors for postoperative arrhythmias.

CONCLUSION

The identification of incidence patterns, risk factors, and vulnerable subgroups in children with CHD has important implications for clinical practice, particularly in the prevention and management of postoperative arrhythmias. These insights may support more refined perioperative risk stratification, allowing surgical teams to adapt intraoperative techniques accordingly and enabling clinicians to consider early correction of electrolyte disturbances or the use of prophylactic antiarrhythmic agents in selected cases.

Nevertheless, several important questions remain unanswered. Reported incidence rates vary widely, and the true incidence of specific postoperative arrhythmias within individual CHD subtypes is still difficult to determine, in part due to variations in study design. Additionally, the role of intraoperative factors and preexisting conditions, such as genetic syndromes, requires further research. Addressing these gaps might require further studies based on large, multicenter cohorts that examine individual arrhythmia types rather than combining diverse outcomes. Future research should also explore the utility of intraoperative conduction mapping and its role in minimizing conduction system injury. Combined with genetic and clinical profiling, it could provide valuable insights into arrhythmia mechanisms. Integrating these variables into a comprehensive risk stratification score may facilitate early identification of high-risk patients, improving outcomes in this vulnerable population.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

INFORMED CONSENT

Informed consent was obtained from all subjects involved in the analyzed studies as stated by the respective authors.

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AUTHOR CONTRIBUTIONS

P.C.M., R.O.C. and C.F. conceptualized the review and developed the protocol. G.T., S.I.D. and O.E.F. performed the literature search and screening. P.C.M, O.E.F., and G.T. extracted the data. S.I.D. and L.M conducted the risk of bias assessment. P.C.M., R.O.C., and O.E.F carried out the data synthesis and interpretation. P.C.M., O.E.F., and R.O.C. drafted the original manuscript. C.F. provided supervision. G.T., R.O.C., L.M. and C.F. reviewed and edited the manuscript. All authors have read and agreed to the published version of the manuscript.

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