Predictors of cardio pulmonary resuscitation outcome in postoperative cardiac children

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Background: Outcomes of cardiopulmonary resuscitation (CPR) in children with congenital heart disease have improved and many children have survived after an in-hospital cardiac arrest.

Aim: The purpose of this study is to determine predictors of poor outcome after CPR in critical children undergoing cardiac surgery.

Methods: We conducted a retrospective chart review and data analysis of all CPR records and charts of all postoperative cardiac children who had a cardiac arrest and required resuscitation from 2011 until 2015. Demographic, pre-operative, and postoperative data were reviewed and analyzed.

Results: During the study period, 18 postoperative pediatric cardiac patients had CPR. Nine of them had return of spontaneous circulation and survived (50%). On average CPR was required on the 3rd postoperative day. Univariate analysis demonstrated that poor outcome was associated with higher lactic acid measured 4–6 hours prior to arrest (p = 0.045; p = 0.02) coupled with higher heart rate (p = 0.031), lower O₂ saturation (p = 0.01), and lower core body temperature (p = 0.019) recorded 6 hours before arrest. Nonsurvival required longer resuscitation duration and more epinephrine doses (p < 0.05).

Conclusion: Higher heart rate, lower core body temperature, lower O₂ saturation, and higher lactic acid measured 6 hours before arrest are possible predictors of poorer outcome and mortality following CPR in postoperative cardiac children.

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Introduction

Cardiac arrests occur in 0.7–3% of pediatric hospital admissions and 1.8–5.5% of pediatric intensive care unit admissions [1], representing significant social, familial, and economic burden. Survival rates after in-hospital cardiac arrests improved from 9% in 1986 to 35–40.2% in 2012 and 2013 [2,4]. Children undergoing cardiac surgery are at greater risk of experiencing a cardiac arrest [1]. According to European Resuscitation Council Guidelines for Resuscitation incidence of cardiac arrest after cardiac surgery in children is 4% [3]. Early prediction, prevention, and proper management of cardiac-pulmonary arrest are fundamentally important to avoid cardiopulmonary arrest and to improve outcome after cardiopulmonary resuscitation (CPR). The aim of this retrospective study was to determine influence of different pre-arrest variables on the outcome of CPR in postoperative cardiac children.

Materials and methods

We conducted a retrospective review of the medical charts of all children who needed CPR in the pediatric cardiac surgical intensive care unit in Prince Sultan Cardiac Center, Qassim, Saudi Arabia over a 4-year period extending from April 2011 until April 2015. Institutional approval was obtained for this retrospective study. Patients were divided into two groups. The survivor group includes patients who responded to CPR with return of spontaneous circulation (ROSC) and survived until hospital discharge. The nonsurvivor group includes patients who failed to survive after arrest or had transitional ROSC but subsequently deceased during the same hospital admission and before discharge. Medical charts including patient’s chart, operative reports, and the laboratory database were reviewed. Pre-arrest and resuscitation records were reviewed for the following variables: age of patient, weight, cardiac diagnosis and type of repair, Rasch score for surgical risk category, presence of univentricular or biventricular heart physiology, bypass time, cross clamp time, and highest inotropes score postoperatively. Laboratory results that include serum lactate level, pH, PaO₂, base deficit, HCO₃, white blood cell counts, hemoglobin, platelet count, international normalized ratio, and creatinine at 6 hours, 4 hours, and 2 hours prior to arrest and immediately pre-arrest were also reviewed. Physiological parameters such as heart rate (HR), systolic blood pressure, mean blood pressure, oxygen saturation, central venous

### Table 1. Cardiac diagnosis and surgical repair of patients having a cardio–pulmonary arrest.

| Diagnosis                                      | Procedure                          | Outcome | Survived | Deceased |
|------------------------------------------------|------------------------------------|---------|----------|----------|
| Atrioventricular defect                        | PA banding                         | 2       | –        | –        |
| Atrioventricular defect                        | Total repair                       | 2       | 1        |          |
| Ventricular septal defect                      | VSD closure                        | 1       | –        | –        |
| Ventricular septal defect VSD                  | PA banding                         | 1       | –        | –        |
| VSD, PDA, pulmonary hypertension              | PA Banding and PDA ligation        | –       | 1        |          |
| Double inlet left ventricle                    | Glenn shunt                        | 2       |          |          |
| Coarctation of aorta                           | Coarctation repair                 | 1       |          |          |
| Tetralogy of fallot                            | Tetralogy of Fallot repair         | –       | 4        |          |
| Pulmonary atresia                              | Modified Blalock taussig shunt     | –       | 3        |          |
| Total no. of cases                             |                                    | 18      | 9        | 9        |

PA = pulmonary artery; PDA = patent ductus arteriosus; VSD = ventricular septal defect.

### Table 2. Summary of demographics data in the survivor group and nonsurvivor group.

| Variable                                    | Survivors | Nonsurvivors | p    |
|---------------------------------------------|-----------|--------------|-----|
| Age (mo)                                     | 11.6 ± 4.2| 9.27 ± 3.01  | 0.55|
| Weight (kg)                                  | 6.19 ± 0.89| 6.09 ± 0.65 | 0.92|
| Presence of associated syndrome             | 4         | 2            | 1   |
| Persistent pulmonary hypertension           | 3         | 2            | 1   |
| Univentricular repair                        | 2         | 3            | 1   |
| Rasch (surgical risk category score)        | 2.4 ± 0.24| 2.6 ± 0.18   | 0.54|
| CPB time                                     | 93 ± 7.04 | 117 ± 12.85  | 0.12|
| Maximum inotropic score                     | 8.6 ± 2.63| 23 ± 8       | 0.2 |

CPB = cardiopulmonary bypass.
pressure, and core body temperature at 6 hours, 4 hours, and 2 hours pre-arrest and immediately before CPR were reviewed and documented based on availability. Primary possible causes of arrest and the time passed between surgery and arrest were documented. Duration of CPR and numbers of adrenalin doses were also recorded. The primary rhythm at the time of arrest was classified as ventricular fibrillation/pulseless ventricular tachycardia (VF/VT) or non-VF/VT. The latter category was subclassified as either bradycardia/asystole or pulseless electrical activity. The number of defibrillations and the total defibrillation energy were recorded for the patients with VF/VT. Data were analyzed using Student t test for continuous variables and Fisher’s exact test or Chi-square test for categorical variables. A p value less than 0.05 was considered statistically significant.

Results

During the 4-year study period, 414 children underwent cardiac surgery and were admitted to the cardiac surgical intensive care unit. Eighteen cases (4.4%) of cardiac arrest were identified and CPR was carried out for all of cases according to Pediatric Advanced Life Support recommendation. Because of logistic and limited resources, rescue extracorporeal membrane oxygenation (ECMO) was not initiated for any cases. Nine patients (50%) survived after resuscitation and remained alive until discharge and were labeled as the survivor group. The remaining patients (9/18) failed to ROSC or subsequently died and were assigned to the nonsurvival group. Diagnosis and surgical repair for all cases that had CPR in both groups are summarized in Table 1. Demographic data between both groups were not statistically different (Table 2). No statistically significant difference in the time of cardiac arrest (2.9 days vs. 4.6 days) between survivors and nonsurvivors was noted. Analyses of possible causes or contributing factors to cardiac arrest demonstrated completely different pre-arrest conditions and causes of arrest between the two groups. The nonsurvivor group had major contributing factors such as pulmonary edema, multi-organ failure, low cardiac output syndrome, or severe infection, while most of the survivor group had acute causes such as pain, agitation, sedation issue, or accidental endotracheal tube obstruction as contributing factors for bradycardia. Pre-arrest physiological parameters such as HR, mean blood pressure, systolic blood pressure, core body temperature, central venous pressure, and O₂ saturation from both groups showed significant differences in HR (p = 0.031, 95% confidence interval: 19–36.9), O₂ saturation (p = 0.013, 95% confidence interval: 9.6–16.3), and core body temp at 6 hours before arrest (p = 0.019, 95% confidence interval: 0.49–0.90) as shown in Table 3.
laboratory results and blood gases showed no significant difference between both groups except for lactic acid which was significantly higher in the nonsurvivor group at 6 hours (5 ± 1.51 hours) and 4 hours (6.7 ± 2.12 hours) before arrest compared with lactic acid in the survivor group at 6 hours (1 ± 0.14 hours) and 4 hours pre-arrest (0.88 ± 0.15 hours) with \( p = 0.045 \) (95% confidence interval: 3.05–5.7) and \( p = 0.021 \) (95% confidence interval: 3.6–7.9), respectively (Table 3). The non-survivor group had a longer duration of CPR compared with the survivors (5 ± 1.33 hours vs. 63.12 ± 13.9 hours) with \( p = 0.0005 \) and they needed more doses of adrenalin during resuscitation. Most of our patients had sinus bradycardia at the time of arrest with only two patients from the nonsurvival group had VF and needed defibrillation. Open chest compression was conducted in two of the nonsurvivor patients with no significant statistical difference in success rate (Table 4).

Discussion

Over the past 3 decades, outcomes for children undergoing congenital heart surgery have improved dramatically due to improvements in diagnostic capabilities, refinement in surgical technique, and advances in peri-operative care and monitoring. Incidence and outcome of CPR postcardiac surgery are limited. In previously published reports, the survival rate post-CPR in pediatric cardiac intensive care units ranged between 37% and 41% [2,4–6]. Identifying a high-risk group of patients before cardiopulmonary arrest may help to improve survival rates and decrease mortality. Few studies tried to find some predictive markers for survival and the majority of these markers were peri-CPR and post-CPR markers. Early in 1999, Rhodes et al [5] described the outcome of cardiac pulmonary arrest in infants after congenital heart surgery and noted that infants with a lower mean arterial blood pressure, higher inotropic support, or lower pH were less likely to survive and prolonged duration of CPR worsened the outcome. Similar results were also reported by Ahmadi and Aarabi [6] in 2013, who issued comparable results. Duke and Butt [8] studied different predictive factors and noted that among them, high lactate level and a rising trend of serum lactate by 0.75 mmol/L per hour with a parallel decrease in central venous oxygen saturation were associated with major adverse events occurring after cardiac surgery and he developed a framework for risk estimation based on demographic and clinical predictors and biochemical markers of inadequate oxygen delivery after cardiac surgery in children. In our patients we observed a development of tachycardia, central hypothermia, and desaturation in nonsurvivors, recognized 6-hours prearrest. These changes in vital signs seem to go hand in hand with a trend in development of lactic acidemia indicating the presence of global ischemia or tissue O\(_2\) debt. Whether the combination of these markers can be used as an early warning for more advanced management such as rescue ECMO (which was proved by many studies to be lifesaving, especially if it is introduced early) [10] remain a subject of debate and a question for future study and research, as failure to interfere at this point may lead to eminent cardiopulmonary catastrophe or arrest. The mortality rate was proven in many studies to be related to the complexity of surgery ranging from 0.64% for atrial septal defect repair to 19.3% for the Norwood procedure [7]. We could not establish a correlation between Rachs score and the development of cardio–pulmonary arrests in our patients. In recently published data from other multi-institutional cohort study of infants undergoing cardiac surgery, the authors found that maximum vasoactive–inotropic score \( \geq 20 \) is significantly associated with morbidity and mortality [9]. We could not establish such a direct link between the inotropic score and mortality rates following CPR in our postoperative cardiac patients. Other investigators have not been able to demonstrate this kind of causal relation between postoperative inotropic score and development of cardio–pulmonary arrest or poor outcome following CPR [5,6]. Limitations of our...
study include the retrospective nature of data collection, the small numbers of patients, and lack of advanced management such as ECMO. A larger study is needed to support our results.

Conclusion

Cardiac arrest following cardiac surgery is not uncommon in the immediate postoperative phase in children with an incidence rate of 4.4–50% survival in our study. Identifying predictive markers precardiopulmonary arrest may help to improve management and increase survival rate. Tachycardia, desaturation, and lower core body temperature particularly in association with a parallel increase in lactate level above 4 mmol/L are alerting signs for significant O₂ debt that may soon progress to cardiopulmonary arrest. Recognizing these changes and initiating immediate management may save a life and improve the outcome postresuscitation.

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