# A randomized controlled trial of electrical cardiometry-guided perioperative hemodynamic management in children undergoing cardiac surgery

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## ABSTRACT

**Background and Aims :** **Children undergoing corrective surgery for congenital heart disease (CHD) are at risk of perioperative hemodynamic instability and low cardiac output syndrome (LCOS). Conventional monitoring relies on static parameters that may inadequately reflect real-time cardiac performance. Electrical cardiometry (EC) provides continuous, noninvasive assessment of cardiac output and related indices. This study evaluated whether EC-guided perioperative management improves early postoperative outcomes compared with conventional monitoring in pediatric CHD surgery.**

**Methods :** **In this prospective randomized controlled trial, 60 children (0–15 years) undergoing elective corrective CHD surgery were randomized to EC-guided management (Group 1, *n* = 30) or conventional monitoring (Group 2, *n* = 30). In Group 1, perioperative fluid and vasoactive therapy were guided by EC-derived parameters, including cardiac index (CI), stroke volume, thoracic fluid content, and stroke volume variation. Group 2 was managed using standard clinical and invasive parameters. The primary outcome was the incidence of LCOS within 48 h postoperatively. The secondary outcomes included vasoactive inotropic score (VIS), major adverse cardiac events, and in-hospital mortality.**

**Results :** **Baseline characteristics were comparable. Group 1 demonstrated significantly higher mean arterial pressure (MAP) at 8, 16, and 24 h postoperatively, faster lactate clearance, and lower VIS during the early postoperative period. CI correlated positively with MAP and urine output and inversely with lactate levels. No mortality occurred.**

**Conclusions :** **EC-guided perioperative management aids in achieving early postoperative hemodynamic stability and metabolic recovery in children undergoing corrective CHD surgery and represents a valuable noninvasive adjunct to conventional monitoring.**

**Keywords :** Cardiac index, congenital heart disease, electrical cardiometry, low cardiac output syndrome, pediatric cardiac surgery

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## INTRODUCTION

Congenital heart disease (CHD) is among the most common congenital anomalies worldwide and remains a major contributor to pediatric morbidity and mortality.[1,2] Despite advances in surgical techniques, cardiopulmonary bypass (CPB), and perioperative intensive care, perioperative hemodynamic instability and low cardiac output syndrome (LCOS) continue to be major determinants of adverse outcomes, prolonged intensive care unit (ICU) stay, and increased resource utilization in pediatric cardiac surgery.[3,4] Children undergoing corrective cardiac surgery have limited physiological reserve, immature myocardial compliance, and altered ventricular–vascular coupling, rendering them particularly susceptible to hemodynamic deterioration during induction of anesthesia, separation from CPB, and the early postoperative period.[3,4] Early recognition and timely correction of low cardiac output are, therefore, essential for improving outcomes. Conventional monitoring relies on indirect or static parameters such as heart rate, arterial blood pressure, central venous pressure (CVP), urine output, lactate, and base deficit.[3,5] Commonly used invasive techniques for cardiac output measurement, such as thermodilution, are prone to measurement errors, particularly in the presence of intracardiac shunts and altered flow states.[6]

Electrical cardiometry (EC) is a noninvasive technique that estimates cardiac output and related indices by analyzing changes in electrical thoracic bioimpedance during the cardiac cycle.[7-11] Validation studies and meta-analyses have demonstrated acceptable agreement between EC-derived cardiac output and reference standards, including thermodilution and echocardiography.[7,9-11] EC provides continuous, real-time hemodynamic information without the risks associated with invasive monitoring, making it particularly attractive in pediatric populations.[7,8] Invasive cardiac output monitoring techniques, including pulmonary artery catheter-based methods, have limited applicability in pediatric patients and are associated with procedural risks and interpretative challenges.[12] In addition, broader limitations of invasive hemodynamic monitoring in critically ill patients have been well described.[13]

Despite advances in monitoring technologies, static preload indices such as CVP correlate poorly with preload and fluid responsiveness.[14] Echocardiography, although invaluable for anatomical and functional assessment, is intermittent and operator dependent, limiting its utility for continuous hemodynamic monitoring.[15] LCOS remains a frequent and clinically significant complication following pediatric cardiac surgery, with reported incidence varying depending on diagnostic criteria and timing of assessment.[16-19] LCOS is associated with increased morbidity, prolonged ICU stay, and adverse postoperative outcomes, particularly during the early postoperative period.[18-20] Pediatric-specific validation studies using electrical velocimetry and EC have demonstrated reliable estimation of cardiac output in children with CHD in both intraoperative and postoperative settings.[21-24] However, despite encouraging validation data, robust randomized evidence demonstrating improvement in clinically meaningful outcomes with EC-guided perioperative hemodynamic management in pediatric CHD surgery remains limited.[7] This randomized controlled trial was, therefore, undertaken to evaluate whether EC-guided perioperative hemodynamic management improves early postoperative outcomes compared with conventional monitoring in children undergoing corrective surgery for CHD.

## METHODS

### Study design and data source

This study was a prospective, single-center, randomized controlled trial conducted in the Department of Cardiac Anaesthesia and Critical Care at AIIMS, New Delhi. The objective was to evaluate whether EC-guided perioperative hemodynamic management improves early postoperative outcomes compared with conventional monitoring in children undergoing corrective surgery for CHDs. The study was conducted after obtaining approval from the Institutional Ethics Committee and in accordance with National Ethical Guidelines. The trial was registered with the Clinical Trials Registry of India (CTRI/2023/11/060316). Written informed consent was obtained from parents or legal guardians before enrollment. Patient recruitment and follow-up were carried out over the study period, with perioperative monitoring extending up to 48 h postoperatively.

### Study population and definitions

The study population consisted of children aged 0–15 years undergoing elective corrective cardiac surgery for CHD with the use of CPB. Inclusion criteria included all pediatric patients within the specified age range scheduled for elective corrective CHD surgery. Exclusion criteria were emergency surgeries, univentricular physiology, requirement for postoperative extracorporeal membrane oxygenation, need for surgical re-exploration after the primary procedure, and refusal of consent. Patients were randomized in a 1:1 ratio using sealed opaque envelopes as follows: Group 1 (EC-guided group) – perioperative management guided by EC-derived hemodynamic parameters and Group 2 (conventional group) – management based on standard clinical and invasive monitoring. Patients were blinded to group allocation, whereas treating clinicians were not. LCOS was defined as the presence of one or

more of the following between ICU admission and 48 h postoperatively: cardiac index (CI), <2.0 $L \cdot min^{-1} \cdot m^{-2}$; serum lactate, >3.0 mmol $L^{-1}$; base deficit, >4.0 mmol $L^{-1}$; urine output, <1.0 $mL \cdot kg^{-1} \cdot h^{-1}$; or a core-to-peripheral temperature difference, >5°C.[4,18-20]

### Perioperative management and monitoring

The EC monitor is based on the principle of electrovelocimetry, a noninvasive method of hemodynamic monitoring that derives cardiac output and related parameters from changes in thoracic bioimpedance.[17] EC measures hemodynamics by analyzing changes in thoracic bioimpedance during the cardiac cycle. When the heart is at rest (diastole), red blood cells are dispersed in random orientations, which creates greater resistance to the flow of a small electrical current. With the onset of systole, ventricular ejection organizes the red blood cells along the axis of blood flow, reducing resistance and permitting greater current conduction. These cyclical impedance fluctuations are captured by the EC monitoring system and processed to derive stroke volume, cardiac output, systemic vascular resistance, and other parameters.[17]

The working principle involves passing a constant, low-amplitude alternating current (about 1.5 mA at 50 kHz) across the thorax using surface electrodes. The device then records the baseline thoracic impedance (Z) and its rate of change over time (dZ/dt). By analyzing the impedance waveform, the EC monitor identifies key time points such as the onset of systole, the maximum slope of impedance change (reflecting peak aortic acceleration), and left ventricular ejection time. These values are processed by a proprietary algorithm to calculate stroke volume, which when multiplied by heart rate provides cardiac output. Additional parameters such as contractility indices and thoracic fluid content (TFC) are also derived.[17] Setting up the EC monitor is relatively simple and can be done at the bedside. Four surface electrodes are required, as shown in Figure 1. The EC monitor requires the placement of four surface electrodes. Two are applied in the neck region (one at the lower neck/clavicular area and another near the sternal notch) to deliver the alternating current. Two additional sensors are positioned on the left lateral chest wall – at the level of the xiphoid process and another slightly inferior to it to detect changes in impedance. In infants, the sensors are placed on the forehead, the second sensor is placed on the left base of the neck, the third sensor is placed on the left thorax at the level of the xiphoid, and the fourth sensor is placed on the left thigh, as shown in Figure 1.[17]

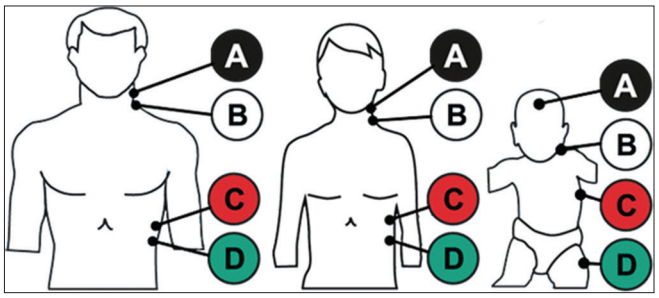


**Figure 1: Sensor placement for electrode setup, Osypka Medical Inc. ICON® Cardiac Output Monitor – User Manual. Berlin: Osypka Medical; 2021**

All patients received standardized anesthesia, CPB management, and postoperative intensive care according to institutional protocols. In Group 1, EC monitoring was initiated using surface electrodes and the ICON® monitor [Figure 1].[17] Hemodynamic parameters, including cardiac output, CI, stroke volume, stroke volume variation (SVV), TFC, and systemic vascular resistance, were recorded intraoperatively and continued for 48 h postoperatively. Fluid and vasoactive therapies were guided by EC-derived parameters, with SVV > 13% used to predict fluid responsiveness.[6] In Group 2, perioperative management was guided by conventional parameters, including heart rate, invasive arterial blood pressure, CVP, urine output, serum lactate, base deficit, temperature gradients, and overall clinical assessment. EC data were not available to clinicians managing patients in this group.

### Outcome study

The primary outcome was the incidence of LCOS within 48 h following surgery. The secondary outcomes included the requirement of inotropic/vasoactive support, assessed by the Vasoactive Inotropic Score (VIS), occurrence of major adverse cardiac events, and in-hospital mortality during the observation period.

### Statistical analysis

Sample size estimation was based on previously reported LCOS incidence in pediatric CHD surgery[18] using standard methodology for the comparison of proportions. The sample size of 30 in each group and a total of 60 for both the groups was determined to be suitable. Statistical analysis was performed using Stata version 16 Stata version 16.0 (StataCorp LLC, College Station, TX, USA). Continuous variables were expressed as mean ± standard deviation or median as appropriate. Categorical variables were expressed as frequencies and percentages. Intergroup comparisons were performed using appropriate parametric or nonparametric tests. Correlations were assessed using Pearson's analysis. $P$ <0.05 was considered statistically significant.

## RESULTS

All 60 randomized patients completed the study and were included in the final analysis [Figure 2]. Baseline demographic characteristics, anthropometric variables, and types of surgical procedures were

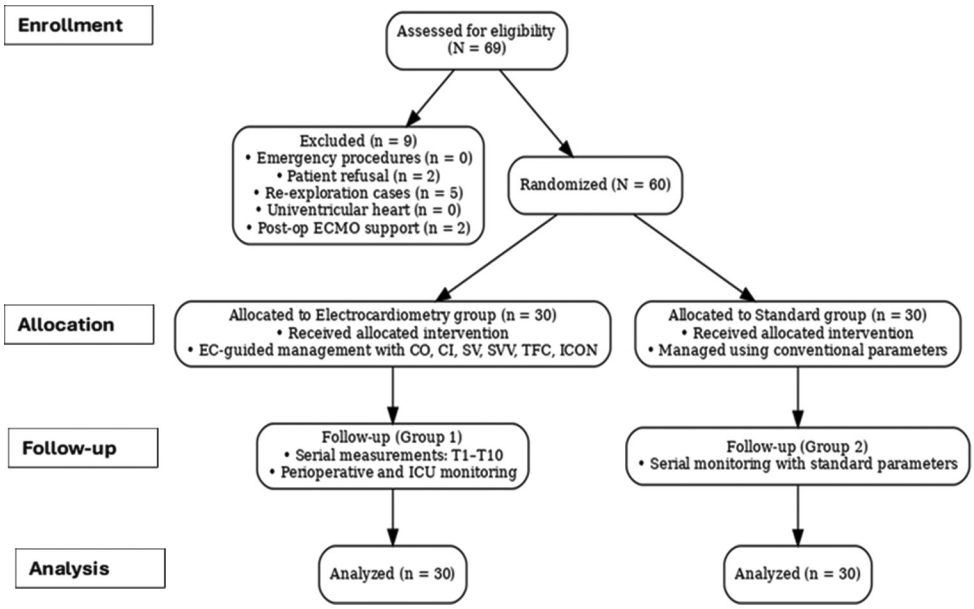


**Figure 2: CONSORT flow diagram**

comparable between the two groups [Table 1]. There were no significant differences in age and weight. Intraoperative mean arterial pressure (MAP) values were similar between the groups at all measured time points ($P > 0.05$). Postoperatively, Group 1 demonstrated significantly higher MAP at 8, 16, and 24 h [Table 2]. At 48 h, MAP values were comparable ($P = 0.08$). Serum lactate levels were comparable intraoperatively and on ICU admission ($P > 0.4$). From postsurgery 8 h onward, Group 1 demonstrated faster lactate clearance at 8, 16, 24, and 48 h and the results were statistically significant ($P < 0.05$) [Table 2]. Base deficit was corrected earlier in Group 1 patients, with a significant difference at 8 h ($P = 0.044$).

Urine output was higher in Group 1 immediately postinduction (1.49 ± 0.45 vs. 1.01 ± 0.38 mL/kg/h, $P < 0.001$), with no significant differences thereafter [Table 2]. VIS was significantly lower in Group 1 at 8 h (20 ± 5 vs. 25 ± 7, $P = 0.029$), 16 h (25 ± 5 vs. 30 ± 6, $P = 0.032$), and 24 h (25 ± 3 vs. 30 ± 5, $P = 0.038$) [Table 3].

Lactate levels were comparable between the groups until sternotomy, off CPB, and on ICU admission (all $P > 0.4$). Thereafter, Group 1 demonstrated a faster and more sustained decline in serum lactate. The differences were statistically significant at 8 h ($P = 0.018$), 16 h ($P < 0.001$), 24 h ($P < 0.001$), and 48 h ($P < 0.001$), favoring the intervention group. This confirms faster normalization of lactate under ICON-guided management [Table 3].

Postoperative CI values were consistently higher in Group 1 [Table 3]. CI was correlating positively with MAP ($r = 0.42$, $P = 0.007$) and urine output ($r = 0.38$, $P = 0.003$) and inversely with lactate ($r = –0.46$, $P = 0.007$) and base deficit ($r = –0.31$, $P = 0.024$) [Figure 3]. LCOS occurred in 53.3% of patients in the conventional group compared to 33.3% in the EC group, corresponding to an absolute risk reduction of 20% (NNT = 5) [Table 4].

Major adverse cardiac events were also assessed as part of the secondary outcomes. Complete heart block occurred in five patients in Group 1 and four in Group 2 following ventricular septal defect closure. Junctional ectopic tachycardia occurred in three patients in Group 1 and in four patients in Group 2 at ICU admission; all episodes resolved within 8 h. Prompt pacing was instituted in cases of heart block, and ICON monitoring confirmed preserved cardiac output. ICON readings also indicated preserved CI during junctional ectopic tachycardia (JET), supporting a conservative rhythm–management approach. Importantly, none of these conduction disturbances progressed to LCOS in EC group patients, underscoring the effect of optimized preload, afterload, and contractility.

With respect to mortality, no deaths occurred in either group during the 48-h observation period. This absence of mortality demonstrates that both monitoring strategies were safe in the short-term perioperative setting. However, the limited sample size and short duration of follow-up preclude any definitive conclusions about EC-guided management on mortality.

## DISCUSSION

LCOS remains a dreaded complication after pediatric cardiac surgery and is a major determinant of morbidity and mortality.[4,18-20] The present randomized controlled trial demonstrates that EC-guided perioperative hemodynamic management improves early postoperative

hemodynamic stability and metabolic recovery in children undergoing corrective surgery for CHD. The principal findings include higher MAP during the early postoperative period, faster lactate clearance, earlier correction of metabolic acidosis, and reduced vasoactive inotropic requirements in the EC-guided group. Importantly, these benefits were achieved without an increase in adverse events, supporting the safety and clinical utility of noninvasive, flow-based monitoring in pediatric cardiac surgery.

One of the key observations was the significantly higher MAP during the first 24 h postoperatively in the EC-guided group. This finding is clinically relevant, as early postoperative hypotension is a well-recognized marker of inadequate systemic perfusion and is frequently associated with LCOS in children after cardiac surgery.[4,20,25] Notably, the higher MAP in the EC group was accompanied by lower VIS, suggesting that improved perfusion was achieved through optimization of cardiac output, rather than vasoconstrictor-driven increases in systemic vascular resistance.

This distinction is particularly important in pediatric cardiac surgery, where reliance on pressure-based targets may mask inadequate forward flow and expose patients to the adverse effects of excessive vasoactive therapy.[3,4] By enabling continuous assessment of CI and stroke volume, EC facilitated targeted, physiology-driven interventions aimed at restoring effective forward flow, thereby achieving hemodynamic stability with reduced pharmacologic support.

Serum lactate clearance is a robust surrogate marker of global tissue perfusion and oxygen delivery in pediatric cardiac surgical patients.[4,20] In the present study, lactate levels were comparable between groups intraoperatively and on ICU admission, reflecting similar baseline surgical and anesthetic conditions. However, from 8 h postoperatively onward, patients managed with EC demonstrated significantly faster and more sustained lactate clearance.

This pattern suggests earlier restoration of adequate cardiac output and improved microcirculatory perfusion in the EC-guided group. The concurrent improvement in base deficit further supports enhanced metabolic

**Table 1: Demographic characteristics of patients in Group 1 and Group 2**

| Demographic characteristics | Group 1, *n* (%) | Group 2, *n* (%) |
|---|---|---|
| Gender | | |
| Female | 7 (11.66) | 5 (8.33) |
| Male | 23 (38.33) | 25 (41.33) |
| Age (years) | | |
| ≤1 | 8 (26.7) | 12 (40) |
| >1–<3 | 5 (16.6) | 6 (20) |
| 3–4 | 10 (33.3) | 8 (26.7) |
| >4 | 7 (23.4) | 4 (13.3) |
| Height (cm) | | |
| Mean±SD | 89.91±18.64 | 87.24±17.93 |
| Weight (kg) | | |
| Mean±SD | 12.73±5.40 | 11.48±5.55 |
| Procedure done | | |
| ICR | 17 (56.7) | 18 (60) |
| VSD closure | 13 (43.3) | 12 (40) |

SD: Standard deviation, ICR: Intracardiac repair, VSD: Ventricular septal defect

**Table 2: Mean±standard deviation and *P* values (probability value) for clinical parameters at each time point**

| Clinical parameters | HR | | | MAP | | | Core- peripheral temperature difference | | |
|---|---|---|---|---|---|---|---|---|---|
| | Group 1 | Group 2 | *P* | Group 1 | Group 2 | *P* | Group 1 | Group 2 | *P* |
| T1 | 108.2±29.5 | 106.5±28.9 | 0.68 | 66.1±7.9 | 65.8±8.1 | 0.82 | | | |
| T2 | 111.4±26.0 | 108.1±25.2 | 0.54 | 64.2±6.1 | 63.5±6.0 | 0.65 | | | |
| T3 | 112.8±25.0 | 110.2±24.1 | 0.62 | 65.5±5.9 | 64.8±5.7 | 0.59 | | | |
| T4 | 120.3±23.1 | 118.7±22.6 | 0.71 | 65.0±6.0 | 64.1±6.3 | 0.55 | | | |
| T5 | 113.9±26.3 | 112.3±25.7 | 0.67 | 67.1±5.1 | 65.9±4.9 | 0.28 | | | |
| T6 | 111.8±24.9 | 115.6±26.2 | 0.29 | 68.3±5.4 | 64.8±5.2 | 0.036 | 3.49±1.11 | 3.31±0.68 | 0.454 |
| T7 | 109.2±21.5 | 118.1±22.4 | 0.041 | 69.2±5.3 | 64.9±5.1 | 0.014 | 3.22±0.56 | 3.17±0.52 | 0.703 |
| T8 | 108.5±22.7 | 117.3±23.8 | 0.028 | 70.4±4.8 | 63.7±5.0 | 0.004 | 2.98±0.50 | 2.99±0.59 | 0.957 |
| T9 | 105.2±20.6 | 116.8±21.9 | 0.008 | 71.0±5.0 | 64.2±5.2 | 0.001 | 2.90±0.55 | 2.77±0.46 | 0.344 |
| T10 | 103.7±21.1 | 118.2±22.7 | 0.002 | 66.1±7.9 | 65.8±8.1 | 0.08 | 2.50±0.40 | 2.41±0.37 | 0.355 |

| Clinical parameters | Lactate | | | Base deficit | | | Urine output | | | VIS | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Group 1 | Group 2 | *P* | Group 1 | Group 2 | *P* | Group 1 | Group 2 | *P* | Group 1 | Group 2 | *P* |
| T1 | | | | | | | | | | | | |
| T2 | 1.96±0.57 | 2.02±0.61 | 0.69 | 1.89±1.29 | 3.98±2.02 | <0.01 | 1.49±0.45 | 1.01±0.38 | <0.001 | | | |
| T3 | | | | | | | 1.20±0.22 | 1.10±0.20 | 0.07 | | | |
| T4 | 1.85±0.62 | 1.88±0.64 | 0.81 | 3.16±0.58 | 3.05±0.66 | 0.58 | 1.30±0.28 | 1.25±0.25 | 0.46 | 5±8 | 10±10 | 0.020 |
| T5 | | | | | | | 1.18±0.20 | 1.15±0.18 | 0.54 | 20±7 | 15±9 | 0.024 |
| T6 | 1.88±0.59 | 2.05±0.62 | 0.18 | 3.20±1.48 | 2.66±1.70 | 0.201 | 1.08±0.23 | 1.05±0.22 | 0.60 | 15±6 | 20±8 | 0.026 |
| T7 | 1.62±0.44 | 1.95±0.55 | 0.021 | 3.52±0.99 | 3.17±0.85 | 0.044 | 1.25±0.32 | 1.20±0.30 | 0.53 | 20±5 | 25±7 | 0.029 |
| T8 | 1.48±0.40 | 1.87±0.51 | 0.009 | 3.34±2.17 | 3.65±1.60 | 0.055 | 1.15±0.27 | 1.10±0.26 | 0.46 | 25±5 | 30±6 | 0.032 |
| T9 | 1.30±0.38 | 1.82±0.52 | 0.003 | 2.96±1.16 | 2.57±1.23 | 0.211 | 1.12±0.26 | 1.05±0.25 | 0.29 | 25±3 | 30±5 | 0.038 |
| T10 | 1.05±0.35 | 1.70±0.50 | <0.001 | 2.40±1.05 | 2.19±1.03 | 0.446 | 1.25±0.30 | 1.20±0.28 | 0.50 | 15±2 | 20±4 | 0.041 |

Units - HR (beatsmin$^{-1}$), MAP (mmHg), lactate (mmolL$^{-1}$), base deficit (mmolL$^{-1}$), urine output (mLkg$^{-1}$h$^{-1}$), VIS (unitless). MAP: Mean arterial pressure, HR: Heart rate, VIS: Vasoactive inotropic score

**Table 3: Mean±standard deviation, *P* value of index of contractility parameters**

| ICON parameters | TFC | | | CO | | | CI | | | SV | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Group 1 | Group 2 | *P* | Group 1 | Group 2 | *P* | Group 1 | Group 2 | *P* | Group 1 | Group 2 | *P* |
| T1 | 13.4±4.9 | 13.6±5.1 | 0.82 | 2.30±1.54 | 2.40±1.49 | 0.096 | 4.0±1.2 | 3.9±1.1 | 0.68 | 17.71±1.57 | 12.51±1.50 | 0.22 |
| T2 | 13.5±4.8 | 13.1±5.0 | 0.73 | 1.97±1.73 | 1.89±1.19 | 0.82 | 3.5±1.1 | 3.2±1.2 | 0.22 | 20.23±1.24 | 20.17±0.91 | 0.522 |
| T3 | 14.5±4.6 | 14.2±4.7 | 0.67 | 2.10±1.11 | 1.91±1.07 | 0.494 | 3.2±1.0 | 2.9±1.1 | 0.18 | 17.47±1.17 | 19.92±0.98 | 0.319 |
| T4 | 16.1±7.0 | 15.9±6.9 | 0.79 | 1.98±1.10 | 1.88±1.01 | 0.711 | 3.1±0.9 | 2.7±1.0 | 0.04 | 16.28±2.62 | 17.54±3.25 | 0.343 |
| T5 | 15.7±6.1 | 15.4±6.2 | 0.71 | 2.11±1.14 | 2.20±1.24 | 0.767 | 3.3±1.0 | 2.8±1.1 | 0.03 | 17.57±1.50 | 16.14±1.34 | 0.153 |
| T6 | 15.6±6.1 | 14.8±6.0 | 0.41 | 2.56±1.21 | 2.34±1.36 | 0.516 | 3.6±0.9 | 3.1±1.0 | 0.02 | 17.89±1.16 | 18.60±1.19 | 0.691 |
| T7 | 15.2±6.0 | 13.5±6.2 | 0.12 | 2.53±1.24 | 2.21±1.22 | 0.313 | 3.7±1.1 | 3.4±1.0 | 0.21 | 23.39±1.11 | 16.25±0.98 | 0.470 |
| T8 | 15.5±6.2 | 12.8±6.1 | 0.048 | 2.38±1.06 | 2.46±1.12 | 0.783 | 3.9±1.0 | 3.6±1.0 | 0.19 | 18.81±1.63 | 14.89±1.58 | 0.983 |
| T9 | 15.7±6.1 | 12.3±5.9 | 0.009 | 2.71±1.33 | 2.37±1.08 | 0.289 | 4.1±1.1 | 3.7±1.0 | 0.11 | 24.12±1.20 | 25.56±1.36 | 0.108 |
| T10 | 16.0±6.3 | 12.5±6.0 | 0.004 | 2.60±1.27 | 2.50±1.35 | 0.767 | 4.3±1.0 | 4.0±1.1 | 0.15 | 27.00±0.69 | 22.50±0.55 | 0.573 |

| ICON parameters | SI | | | SVV | | | ICON | | |
|---|---|---|---|---|---|---|---|---|---|
| | Group 1 | Group 2 | *P* | Group 1 | Group 2 | *P* | Group 1 | Group 2 | *P* |
| T1 | 25.98±9.64 | 27.44±9.65 | 0.560 | 13.3±1.8 | 12.7±1.6 | 0.411 | 84.73±41.66 | 74.31±38.65 | 0.031 |
| T2 | 24.98±7.85 | 24.74±9.71 | 0.917 | 14.6±1.5 | 13.5±1.4 | 0.048 | 69.99±27.22 | 65.94±28.40 | 0.574 |
| T3 | 27.72±8.73 | 28.13±7.25 | 0.843 | 14.4±1.3 | 14.0±1.1 | 0.289 | 74.16±32.31 | 73.96±38.15 | 0.982 |
| T4 | 24.62±6.91 | 24.12±5.44 | 0.753 | 15.9±3.8 | 14.6±2.9 | 0.223 | 53.49±16.36 | 46.93±19.08 | 0.158 |
| T5 | 28.62±7.08 | 29.73±7.58 | 0.560 | 13.7±1.5 | 13.4±1.4 | 0.612 | 63.29±20.63 | 60.11±31.13 | 0.642 |
| T6 | 33.79±8.87 | 29.58±7.73 | 0.055 | 13.5±1.5 | 13.0±1.2 | 0.327 | 60.67±16.77 | 55.18±16.56 | 0.206 |
| T7 | 32.43±8.27 | 31.02±7.26 | 0.488 | 13.3±1.2 | 12.9±1.3 | 0.356 | 58.91±14.93 | 57.99±18.80 | 0.835 |
| T8 | 31.74±10.40 | 29.39±10.35 | 0.385 | 12.9±1.6 | 12.1±1.4 | 0.058 | 63.10±15.10 | 61.39±18.33 | 0.695 |
| T9 | 34.99±8.70 | 36.35±9.22 | 0.559 | 12.0±1.3 | 11.6±1.2 | 0.387 | 67.28±13.20 | 65.41±15.62 | 0.614 |
| T10 | 30.41±26.90 | 29.86±30.68 | 0.942 | 10.9±0.6 | 10.7±0.7 | 0.445 | 67.61±14.26 | 69.16±11.59 | 0.645 |

SVV: Stroke volume variation, ICON: Index of contractility, SV: Stroke volume, TFC: Thoracic fluid content, CO: Cardiac output, CI: Cardiac index, SI: Stroke Index

**Table 4: The incidence of low cardiac output syndrome in the two study groups**

| Group | LCOS (+) | LCOS (–) | Total |
|---|---|---|---|
| 1 | 10 | 20 | 30 |
| 2 | 16 | 14 | 30 |

LCOS: Low cardiac output syndrome

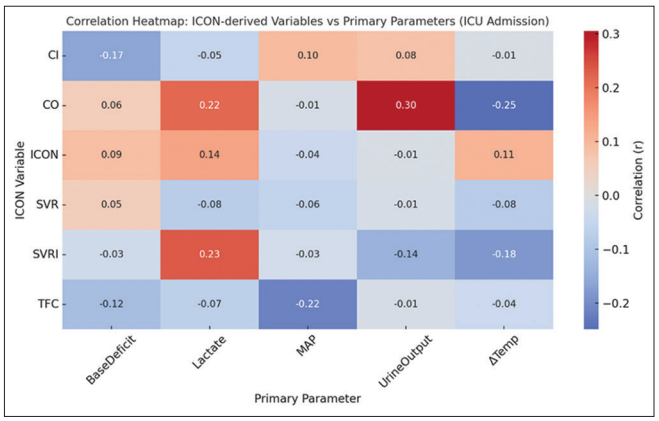


**Figure 3: Correlation heatmap showing the relationship between EC-derived hemodynamic variables (cardiac index, cardiac output, index of contractility, SVR, SVRI, and thoracic fluid content) and primary clinical parameters (base deficit, lactate, MAP, urine output, and ΔTemp) at ICU admission, with colors indicating the strength and direction of correlation (blue = negative, red = positive). CI: Cardiac index, CO: Cardiac output, ICON: Index of contractility, TFC: Thoracic fluid content**

recovery. Importantly, these changes occurred during the early postoperative period, which represents the phase of greatest vulnerability to LCOS. Early normalization of metabolic parameters during this window has been associated with reduced morbidity and improved postoperative recovery in pediatric cardiac surgery.[18,20] The primary outcome of this study was the incidence of LCOS within 48 h postoperatively. LCOS occurred in 53.3% of patients in the conventional group compared to 33.3% in the EC group, corresponding to an absolute risk reduction of 20% (NNT = 5) [Table 4]. Although this difference did not reach statistical significance ($P = 0.192$), the trend suggests that EC-guided monitoring was associated with a numerically lower incidence of LCOS, by facilitating earlier and more precise hemodynamic interventions.

LCOS in children often evolves insidiously, with biochemical and clinical markers lagging behind the initial decline in cardiac performance.[4,25] Conventional monitoring strategies may, therefore, identify LCOS only after the onset of hypotension, oliguria, or rising lactate levels. In contrast, EC provides continuous, real-time assessment of CI and stroke volume, allowing early detection of declining cardiac performance before overt clinical deterioration.

Our findings are consistent with those of Pérez-Navero *et al.*, who investigated biomarkers and clinical scores as predictors of LCOS in children following congenital heart surgery. Similar to our study, they identified LCOS as a frequent and clinically significant complication, with an incidence of approximately 25%–30%.[18] This incidence is comparable to that observed in the interventional monitoring arm of our study. However, the overall incidence of LCOS in both the interventional and control groups in our cohort was higher. Pérez-Navero *et al.*

demonstrated that a VIS >15.5 at 2 h following CPB was a strong predictor of subsequent LCOS.[18] In our study, on ICU admission, the conventional monitoring group exhibited a higher VIS (20 ± 8) compared with the EC-guided group (15 ± 6), indicating a greater early vasoactive requirement in our population when compared with the thresholds reported by Pérez-Navero *et al*. While their study highlighted the role of biomarkers and VIS in early identification of LCOS, our findings extend this concept by demonstrating that real-time EC monitoring can not only facilitate early recognition of hemodynamic compromise but also enable proactive, physiology-directed interventions that reduce the overall incidence of LCOS.[18]

The strong correlations observed in our study between CI and clinically relevant endpoints – including MAP, urine output, lactate levels, and base deficit – further underscore the central role of flow-based monitoring in perioperative management. These findings suggest that EC-guided management may mitigate progression from subclinical hypoperfusion to established LCOS by enabling timely intervention based on objective hemodynamic parameters.

Another important observation was the consistently lower VIS in the EC-guided group during the early postoperative period. Excessive vasoactive support is associated with increased myocardial oxygen consumption, arrhythmias, and impaired ventricular recovery.[18,20,26] By facilitating early differentiation between preload deficiency, impaired contractility, and altered afterload, EC may reduce unnecessary escalation of inotropic and vasopressor therapy.

The reduction in vasoactive requirements observed reflects a shift from reactive to proactive hemodynamic management. Rather than responding to late manifestations of low cardiac output, clinicians were able to intervene earlier using objective flow-based parameters, achieving hemodynamic stability with a lower pharmacologic burden.

Fluid management represents another critical component of perioperative hemodynamic optimization. Static preload indices such as CVP have been shown to have poor predictive value for fluid responsiveness, particularly in pediatric cardiac patients.[14] In contrast, dynamic indices such as SVV have demonstrated superior predictive performance.[6] In our study, SVV derived from EC effectively guided fluid optimization, with values >13% predicting fluid responsiveness.[6] These findings are consistent with those reported by Monnet *et al*., supporting the physiological validity of EC-derived dynamic parameters in this setting.[6]

Collectively, these findings have important implications for perioperative care in pediatric cardiac surgery. EC offers a noninvasive, continuous, and readily deployable method for cardiac output assessment in children, a population in whom invasive monitoring options are often limited.[21-24] The observed improvements in hemodynamic stability and metabolic recovery suggest that incorporation of EC into routine perioperative monitoring may enhance early postoperative management, particularly during the critical first 24–48 h after surgery.

From a practical perspective, EC-guided management may assist clinicians in making more informed decisions regarding fluid therapy, initiation or escalation of inotropes, and vasopressor use. By emphasizing flow rather than pressure alone, this approach aligns with contemporary principles of goal-directed hemodynamic therapy and supports more individualized patient care.[3,6]

### Limitations

This was a single-center study with a modest sample size, potentially limiting generalizability. Treating clinicians were not blinded, introducing the possibility of performance bias. The observed risk reduction in our study is statistically underpowered. In addition, outcomes were confined to the early postoperative period, and the impact of EC-guided management on longer-term outcomes – including ICU length of stay, duration of mechanical ventilation, and hospital stay – remains to be determined. The age group was of a wide range from 0 to 15 years. The spectrum of CHD s tackled through surgery introduces difficulties in generalizing the benefits of using EC in varying physiologies in these age groups. Future multicenter studies with larger sample sizes and extended follow-up are required to validate these findings and to determine whether early improvements in hemodynamic and metabolic parameters translate into sustained clinical benefits.

## CONCLUSIONS

The EC-guided management after congenital cardiac surgery in early 48 h reduces the incidence of LCOS compared to the routine standard protocol. EC-guided perioperative hemodynamic management improves early postoperative hemodynamic stability and metabolic recovery and reduces vasoactive support in children undergoing corrective surgery for CHD. Hence, EC represents a safe and noninvasive adjunct to conventional monitoring in pediatric cardiac surgery.

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### Conflicts of interest

There are no conflicts of interest.

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