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# Timing of venoarterial extracorporeal membrane oxygenation in infant cardiac surgery: a single-centre retrospective study of clinical outcomes

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## Abstract

**Background** Venoarterial extracorporeal membrane oxygenation (VA ECMO) is a critical therapeutic intervention that is commonly used in the management of paediatric patients with congenital heart disease (CHD). This procedure can be initiated either intraoperatively or postoperatively. However, few studies have reported data on the comparative clinical outcomes associated with different timings of VA ECMO initiation. In this study, patient characteristics and clinical outcomes between intraoperative and postoperative VA ECMO in infants undergoing cardiac surgery were compared and predictors of ECMO initiation were determined, which may improve clinical outcomes.

**Methods** A total of 47 infants who received postcardiotomy VA ECMO support from September 2019 to December 2023 were included in this retrospective, single-centre observational study. Patients who received VA ECMO support in the operating room (intraoperative,  $n = 27$ ) were compared with those who received it in the intensive care unit (postoperative,  $n = 20$ ). Kaplan–Meier curves were further analysed for survival and perioperative factors were evaluated to predict VA ECMO initiation.

**Results** Survival rates were greater in the intraoperative group (70.37% vs. 25%;  $P = 0.002$ ), with a reduced risk of mortality (HR: 2.84; 95% CI: 1.23–6.55). The intraoperative group also had a higher ECMO weaning rate (88.89% vs. 45%,  $P < 0.001$ ) and shorter VA ECMO duration ( $5.00 \pm 1.80$  days vs.  $7.50 \pm 2.76$  days;  $P < 0.001$ ). Continuous renal replacement therapy (CRRT) was needed in 100% of postoperative patients versus 70.40% of intraoperative patients ( $P = 0.014$ ). The combination of preoperative lactate  $\geq 6.495$  mmol/L and cardiopulmonary bypass (CPB) time  $\geq 138$  min predicted the need for intraoperative VA ECMO [AUC (area under the curve): 0.893 (95% CI: 0.805–0.980,  $P < 0.001$ )].

**Conclusions** Compared with postoperative VA ECMO, the use of Intraoperative VA ECMO might improve clinical outcomes in infants undergoing cardiac surgery, highlighting the potential benefits of early intervention. The significant predictive value of the CPB time and preoperative lactate level may inform future clinical practices regarding the timing of ECMO initiation in paediatric patients postcardiotomy.

**Keywords** Extracorporeal membrane oxygenation, Congenital heart disease, Infant, Initiation timing

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## Introduction

Since the 1990s, venoarterial extracorporeal membrane oxygenation (VA ECMO) has been widely applied in the treatment of neonatal and paediatric cardiac diseases, as reported in the ELSO registry [1]. Di Nardo et al. [2] reported that ECMO remains the primary means of mechanical circulatory support for children with complex cardiac anatomy, particularly those who require rapid resuscitation and those with a single functional ventricle. According to data from the Society of Thoracic Surgeons Congenital Heart Surgery Database, out of 96,596 paediatric congenital heart disease (CHD) surgeries, 2,287 paediatric patients, accounting for 2.4% of the total patients, received mechanical circulatory support postoperatively; over 95% of these cases involved ECMO [3].

CHD surgery requiring VA ECMO is still associated with significant morbidity and mortality [4]. One of the more important factors that can significantly influence patient outcomes is the timing of VA ECMO initiation [5]. Mariani S et al. [6] conducted a multicentre, retrospective observational study involving 2003 adult patients who required ECMO support due to cardiogenic shock between 2000 and 2020. Their research revealed that the in-hospital mortality rate was significantly greater in the delayed ECMO initiation group than in the early group (64.5% vs. 57.5%,  $P=0.002$ ). Additionally, the delayed ECMO group presented significantly greater incidences of various complications, such as prolonged ICU stay, diffuse nonsurgical bleeding, arrhythmias, septic shock, and multiple organ failure, than did the early ECMO group [6]. VA ECMO is typically applied following CHD surgery at two critical junctures: when patients fail to wean from cardiopulmonary bypass (CPB) in the operating room and when they encounter refractory cardiac dysfunction postoperatively in the intensive care unit (ICU) [7]. A meta-analysis by Wu Y et al. [4] revealed that 12 institutions preferred initiating ECMO postoperatively for congenital heart disease surgery, whereas 9 institutions preferred initiating it intraoperatively. In an optimal scenario, VA ECMO should be implemented before irreversible damage to end organs occurs [7]. However, its invasive nature can cause complications [4, 8]. Therefore, surgeons and intensivists should carefully consider the timing of initiating VA ECMO on the basis of the risk-benefit ratio. At present, the research on the impact of different timings of ECMO initiation on clinical outcomes in paediatric populations remains debated [9–12], and there is a notable scarcity of relevant studies focusing on infants. Accordingly, we conducted a retrospective review of our institution's experience with VA ECMO as a circulatory support measure following congenital heart surgery in infants. This study aimed to compare patient

characteristics and clinical outcomes between intraoperative and postoperative VA ECMO in infants undergoing cardiac surgery and determine predictors of ECMO initiation, which may improve clinical outcomes.

## Methods

### Study population

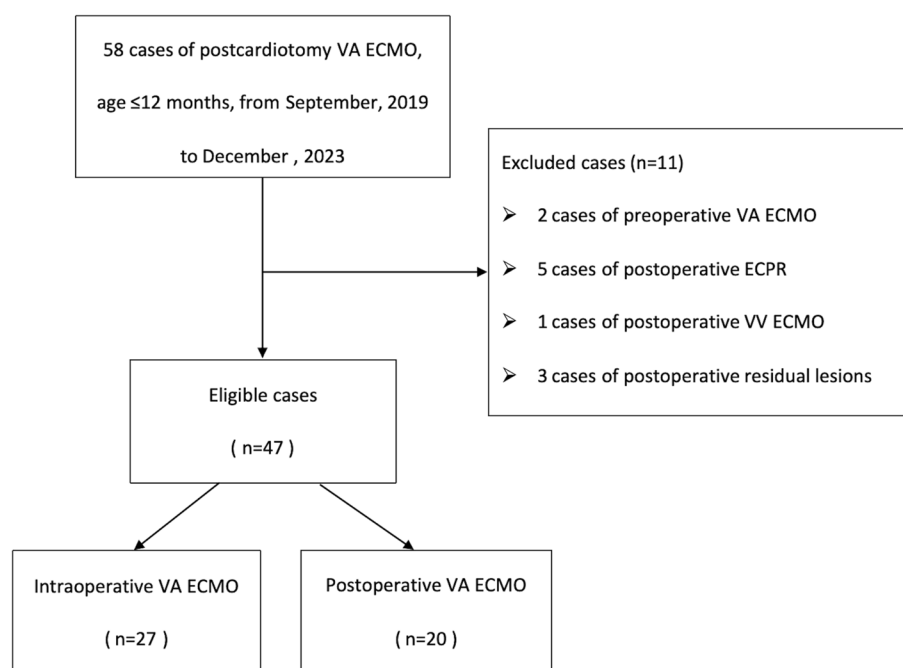
From September 2019 to December 2023, a total of 3670 patients with congenital heart disease (CHD) underwent open-heart surgery at Children's Hospital of Nanjing Medical University, China. Of these, 682 were infants aged  $\leq 12$  months at the time of surgery, and 58 required ECMO support. Patients were excluded on the basis of the following criteria: 1) preoperative ECMO ( $n=2$ ); 2) extracorporeal cardiopulmonary resuscitation (ECPR) ( $n=5$ ); 3) postoperative veno-venous extracorporeal membrane oxygenation ( $n=1$ ); and 4) postoperative surgical residual malformation ( $n=3$ ). After exclusion, the remaining 47 patients were divided into two groups on the basis of the timing of ECMO initiation: the "intraoperative" group ( $n=27$ ) and the "postoperative" group ( $n=20$ ) (Fig. 1).

VA ECMO indications were strictly defined per institutional protocols as follows: (1) intraoperative VA ECMO support stemmed from low cardiac output syndrome (LCOS) precluding successful separation from CPB despite multiple weaning attempts and escalating pharmacologic support; (2) postoperative VA ECMO was initiated for LCOS-induced circulatory compromise refractory to maximal pharmacologic/mechanical ventilation and persisting post-ICU admission, despite successful CPB decannulation. LCOS was defined as a cardiac index  $< 2.2$  L/min/m<sup>2</sup> via echocardiography with signs of end-organ malperfusion in the absence of hypovolaemia [9].

### Clinical data and outcomes

Patient demographics, clinical and laboratory metrics, operative factors and subsequent clinical outcomes were collected. The required doses of inotropes were ascertained for each patient and transformed into a vasoactive inotropic score (VIS) for standardization purposes [13]. The severity of CHD was classified according to the Risk Adjustment in Congenital Heart Surgery-1 (RACHS-1) score [14].

The primary outcome of this study was the in-hospital survival rate. The secondary outcomes included the ECMO weaning rate, the ECMO duration (days from initiation to decannulation or death), the mechanical ventilation (MV) duration (days from initiation to decannulation or death), the total hospital stay (days from ICU admission to discharge or death), the rate of continuous renal replacement therapy (CRRT), and VA



**Fig. 1** Study flowchart. VA ECMO, venoarterial extracorporeal membrane oxygenation; VV ECMO, veno-venous extracorporeal membrane oxygenation; ECPR, extracorporeal cardiopulmonary resuscitation

ECMO-related adverse events, including surgical site bleeding, intracranial haemorrhage, and gastrointestinal bleeding.

CRRT indications in this study were based on the following criteria: a serum creatinine (Scr) level increase of 0.3 mg/L (26.5  $\mu$ mol/L) within a 48-h period; a creatinine level that is 1.5 times higher than the baseline value; and a urine output of less than 0.5 ml/(kg·h) sustained over a 6-h duration.

Successful weaning from VA ECMO was defined as a patient being weaned off ECMO without needing VA ECMO reinsertion or experiencing death within a subsequent 24-h period.

### Statistical analysis

Statistical analyses were conducted using SPSS v26.0. Continuous data are reported as the means  $\pm$  SDs or medians with IQRs, whereas categorical data are expressed as frequencies and percentages. Normality of continuous variables was assessed using the Shapiro–Wilk test and Q–Q plots. The Mann–Whitney U test was used for non-normally distributed variables, while Student’s t-test was used for normally distributed variables. Fisher’s exact test was chosen for categorical data with expected frequencies  $< 5$ , while the  $\chi^2$  test was used for variables with expected frequencies  $\geq 5$ . We used univariate analysis and multivariate logistic regression to assess the independent predictors of intraoperative

VA ECMO initiation. The selection of variables for the model was judicious, considering the number of events and clinical relevance, to maintain the simplicity and interpretability of the model. Multicollinearity among the independent variables was assessed using variance inflation factors (VIFs). Variables with VIF values exceeding 10 were removed from the model. The performance of the variables was assessed via receiver operating characteristic (ROC) curves, with the AUC calculated to quantify the discriminatory ability of the variables. A *P* value of  $< 0.05$  was considered to indicate statistical significance.

## Results

### Baseline characteristics of the patients

The median age at surgery was 1 month (IQR: 0.30–3.16), and the median weight at surgery was 3.6 kg (IQR: 3.1–4.5). The median RACHS-1 score was 3 (IQR: 3–4) (Table 1). The spectrum of congenital heart defects included ventricular septal defects with pulmonary hypertension ( $n=5$ ), truncus arteriosus ( $n=1$ ), coarctation of the aorta/hypoplastic aortic arch with a ventricular septal defect ( $n=13$ ), total anomalous pulmonary venous connection ( $n=11$ ), D-transposition of the great arteries ( $n=5$ ), double outlet right ventricle ( $n=2$ ), complete atrioventricular septal defect ( $n=3$ ), pulmonary atresia with a ventricular septal defect ( $n=2$ ), anomalous origin of the coronary artery ( $n=2$ ), pulmonary artery

**Table 1** Baseline characteristics of the study population according to implantation timing

Variable	Overall population (n = 47)	Intraoperative VA ECMO (n = 27)	Postoperative VA ECMO (n = 20)	P value
Age at surgery, months	1 (0.30, 3.16)	0.76 (0.27, 1.56)	1.27 (0.47, 5.62)	0.102
Sex				
Male	24 (51.10)	13 (48.10)	11 (55)	0.642
Female	23 (48.90)	14 (51.90)	9 (45)	
Weight at surgery, kg	3.60 (3.10, 4.50)	3.60 (3, 4.10)	3.65 (3.20, 4.58)	0.249
History of prematurity	10 (21.30)	5 (27)	5 (25)	0.723
neonate	20 (42.55)	13 (48.15)	6 (30)	0.107
RACHS-1 score	3 (3, 4)	4 (3, 4)	3 (3, 4)	0.484
Cyanotic				
Yes	21 (44.70)	15 (55.6)	6 (30)	0.134
No	26 (55.3)	12 (44.4)	14 (70)	
Down's syndrome	3 (6.40)	0	3 (15)	0.070
Preoperative laboratory data				
Leucocyte count, $\times 10^9/L$	9.18 (7.67, 10.27)	9.18 (7.99, 11.02)	8.90 (6.14, 10.25)	0.208
Haemoglobin, g/L	125.28 $\pm$ 18.07	126.48 $\pm$ 16.64	123.65 $\pm$ 20.16	0.601
Platelet count, $\times 10^9/L$	327.98 $\pm$ 99.21	322.04 $\pm$ 122.16	368 $\pm$ 67.13	0.053
CRP, mg/L	6.48 (0.5, 8)	6.48 (0.50, 8)	5.23 (0.53, 8)	0.593
PT, s	13.61 $\pm$ 2.12	13.87 $\pm$ 2.26	13.25 $\pm$ 1.99	0.332
APTT, s	45.70 (36.40, 55.40)	47.70 (38.90, 55.40)	39.95 (35.75, 55.67)	0.263
Scr, $\mu\text{mol/L}$	29 (24, 42)	28 (24, 44)	29.5 (23.50, 39.55)	0.643
ALT, U/L	17 (8, 32)	17 (8, 33)	19 (8.50, 31.25)	0.731
Albumin, g/L	38.06 $\pm$ 5.15	37.11 $\pm$ 5.86	39.34 $\pm$ 3.77	0.121
Peak lactate, mmol/L	6.28 (3.67, 9.03)	7.50 (5.06, 11.31)	3.96 (3.11, 6.22)	<b>&lt; 0.001</b>
Preoperative cardiac ultrasound				
LVEF, %	64 (59, 66)	61.30 (54, 65)	65.55 (62.25, 68.25)	<b>0.005</b>
LVFS, %	32 (29, 35)	31 (26, 34)	33.90 (31.03, 36.95)	<b>0.020</b>
Preoperative intubation	23 (48.90)	14 (51.90)	9 (45)	0.642
Emergency surgery	3 (6.40)	3 (11.10)	0 (0)	0.251
Operative factors				
CPB time, minutes	195 (157, 230)	208 (177, 248)	174.50 (104, 212.25)	<b>0.010</b>
ACC time, minutes	74.54 $\pm$ 35.77	71.63 $\pm$ 35.18	78.48 $\pm$ 37.09	0.522
Diagnosis				
VSD/PAH	5 (10.64)	2 (7.41)	3 (15)	
Truncus arteriosus	1 (2.13)	0	1 (5)	
COA/HAA/VSD	13 (27.66)	9 (33.33)	4 (20)	
TAPVC	11 (23.40)	7 (25.93)	4 (20)	
D-TGA	5 (10.64)	4 (14.81)	1 (5)	
DORV	2 (4.26)	1 (3.70)	1 (5)	
CAVSD	3 (6.38)	1 (3.70)	2 (10)	
PA/VSD	2 (4.26)	0	2 (10)	
Anomalous origin of coronary artery	2 (4.26)	1 (3.70)	1 (5)	
Pulmonary artery sling	1 (2.13)	1 (3.70)	0	
Pulmonary venous stenosis	2 (4.26)	1 (3.70)	1 (5)	

Values are presented as n (%), mean  $\pm$  standard deviation, or median (interquartile range)

RACHS-1 Risk Adjustment in Congenital Heart Surgery-1, CRP C-reactive protein, PT prothrombin time, APTT activated partial thromboplastin time, Scr serum creatinine, ALT alanine transaminase, LVEF left ventricular ejection fraction, LVFS left ventricular fractional shortening, CPB cardiopulmonary bypass, ACC aortic cross-clamping, VSD ventricular septal defect, PAH pulmonary arterial hypertension, COA coarctation of the aorta, HAA hypoplastic aortic arch, TAPVC total anomalous pulmonary venous connection, D-TGA D-transposition of the great artery, DORV double outlet right ventricle, CAVSD complete atrioventricular septal defect, PA pulmonary atresia

sling ( $n=1$ ), and pulmonary venous stenosis ( $n=2$ ) (Supplemental Table S1).

Compared with the postoperative group, the intraoperative group had higher median preoperative lactate levels (7.50 mmol/L vs. 3.96 mmol/L,  $P<0.001$ ) and longer median CPB times (208 min vs. 174.50 min,  $P=0.010$ ). Conversely, the postoperative group had better median LVEF (65.55% vs. 61.30%,  $P=0.005$ ) and LVFS (33.90% vs. 31%,  $P=0.020$ ) values. These differences highlight the distinct preoperative and intraoperative characteristics between the two groups (Table 1).

### Primary outcome

The primary outcome occurred in 19 of 27 patients (70.37%) in the intraoperative group and 5 of 20 patients (25%) in the postoperative group ( $P=0.002$ ) (Table 2). The Kaplan–Meier curves revealed that patients who received postoperative VA ECMO had significantly greater in-hospital mortality than did those who received intraoperative VA ECMO (HR: 2.84, IQR: 1.23–6.55, log-rank  $P=0.01$ ) (Fig. 2). The baseline characteristics and clinical outcomes for both survivors and non-survivors are detailed in Supplementary Tables S2 and S3, respectively.

### Secondary outcomes

Compared with the postoperative group, the intraoperative VA ECMO group had a significantly greater weaning success rate (88.89% vs. 45%,  $P=0.001$ ) and shorter VA ECMO duration ( $5.00 \pm 1.80$  days vs.  $7.50 \pm 2.76$  days,  $P<0.001$ ). Although the median MV duration was slightly shorter in the intraoperative

group (11 days, IQR: 10–14) than in the postoperative group (12.5 days, IQR: 9–16.75), this difference was not statistically significant ( $P=0.574$ ). The duration of hospital stay also did not differ significantly between the groups (intraoperative: 38 days, IQR: 32.50–44; postoperative: 43 days, IQR: 40–63;  $P=0.183$ ). The postoperative group had a greater need for CRRT (100% vs. 70.40%,  $P=0.014$ ). Adverse events, such as surgical site bleeding, intracranial haemorrhage, and gastrointestinal bleeding, were similar between the two groups (Table 2).

### Independent predictors of intraoperative VA ECMO initiation

As mentioned above, we found that intraoperative VA ECMO may improve clinical outcomes; thus, we aimed to utilize preoperative variables and surgical factors to predict intraoperative VA ECMO initiation. Predictors with a  $P$ -value less than 0.1, except Down's syndrome, were included in the multivariate logistic regression analysis. As demonstrated in Table 3, both the CPB time and preoperative lactate level emerged as significant independent predictors of this outcome. The overall model performance was evaluated using the Nagelkerke  $R^2$  (0.60), Hosmer–Lemeshow goodness-of-fit test ( $P=0.921$ ).

### Efficacy of the CPB time and preoperative lactate level in predicting intraoperative initiation of VA ECMO

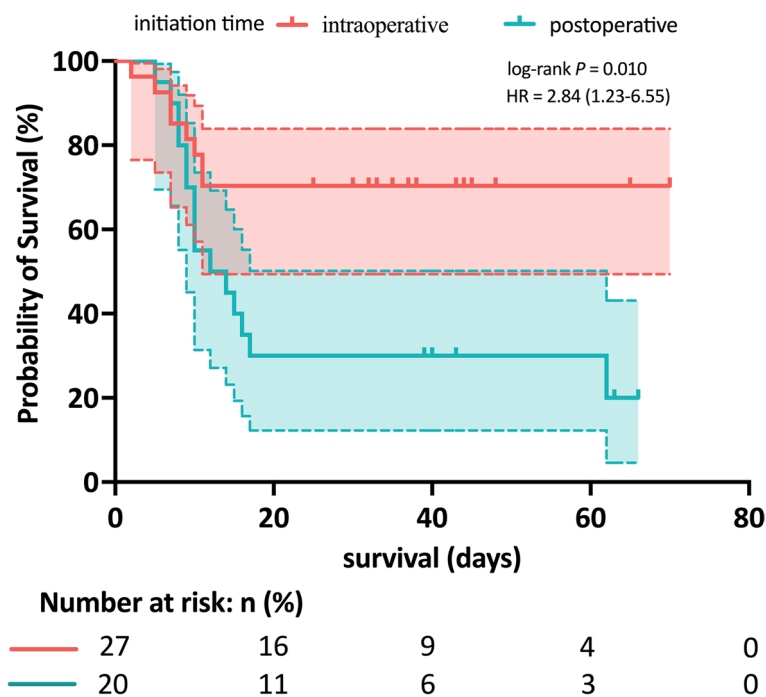
The AUC for the CPB time was 0.722 (95% CI: 0.574–0.871,  $P=0.003$ ). The optimal cut-off value for the CPB time was 138 min. Preoperative peak lactate demonstrated a greater AUC of 0.789 (95% CI: 0.659–0.918,

**Table 2** Comparison of clinical information between two groups

Variable	Overall population ( $n=47$ )	Intraoperative VA ECMO ( $n=27$ )	Postoperative VA ECMO ( $n=20$ )	$P$ value
Primary outcome				
In-hospital survival	24 (51.06)	19 (70.37)	5 (25)	<b>0.002</b>
Secondary outcomes				
ECMO weaning	33 (70.21)	24 (88.89)	9 (45)	<b>0.001</b>
ECMO duration, days	$6.06 \pm 2.56$	$5.00 \pm 1.80$	$7.50 \pm 2.76$	<b>&lt;0.001</b>
MV duration, days	12 (9, 15)	11 (10, 14)	12.5 (9, 16.75)	0.574
Total hospital stay, days	30 (10, 43)	38 (32.50, 44)	43 (40, 63)	0.183
CRRT	39 (83)	19 (70.40)	20 (100)	<b>0.014</b>
Adverse events				
Surgical site bleeding	24 (51.10)	13 (48.10)	11 (55)	0.642
Intracranial haemorrhage	7 (14.90)	3 (11.10)	4 (20)	0.438
Gastrointestinal bleeding	9 (19.15)	6 (22.22)	3 (15)	0.713

Values are presented as  $n$  (%), mean  $\pm$  standard deviation, or median (interquartile range)

CPB cardiopulmonary bypass, ACC aortic cross-clamping, VIS vasoactive-inotropic score, SBP systolic blood pressure, DBP diastolic blood pressure, MV mechanical ventilation, CRRT continuous renal replacement therapy



**Fig. 2** Kaplan–Meier curve of patients with intraoperative VA ECMO compared to those with postoperative VA ECMO (“death” as event and “postcardiotomy survival to hospital discharge” as censored)

**Table 3** Multivariate logistic regression analysis of independent risk factors affecting intraoperative VA ECMO

Variable	B	S.E	Wald <sup>a</sup>	df	P value	OR	95%CI for OR	
							Lower	Upper
Preoperative LVEF	−0.444	0.253	3.069	1	0.080	0.641	0.390	1.054
Preoperative LVFS	0.565	0.341	2.741	1	0.098	1.759	0.901	3.431
Preoperative peak lactate	0.325	0.159	4.174	1	<b>0.041</b>	1.385	1.013	1.892
CPB time	0.024	0.010	5.295	1	<b>0.021</b>	1.024	1.004	1.045
Platelet count	−0.006	0.005	1.550	1	0.213	0.994	0.984	1.004

<sup>a</sup> The Wald statistic assesses the contribution of each predictor to the model  
CI confidence interval, LVEF left ventricular ejection fraction, LVFS left ventricular fractional shortening, CPB cardiopulmonary bypass

$P < 0.001$ ). The cut-off value for preoperative peak lactate was 6.495 mmol/L. The combination of preoperative lactate and CPB time significantly improved the predictive model, with an AUC of 0.893 (95% CI: 0.805–0.980,  $P < 0.001$ ) (Table 4, Fig. 3).

Discussion

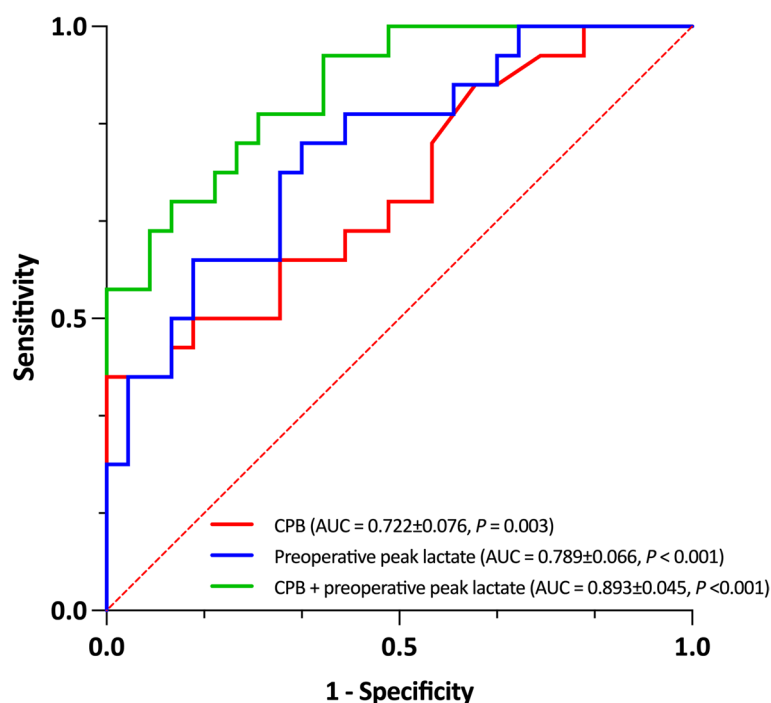
According to historical data, the rates of weaning from postcardiotomy VA ECMO and survival to discharge in children with CHD have varied, with weaning success rates ranging from 60.3% to 78% [4, 15, 16], and survival

rates ranging from 37% to 56.8% [4, 15, 17]. The overall treatment outcomes reported in our study are comparable to those reported for VA ECMO treatment in children with CHD at international paediatric cardiac centres. First, in this study, intraoperative VA ECMO was associated with a reduced risk for in-hospital mortality. However, Deng et al. [9] recently reported no statistically significant difference in survival rates between intraoperative ECMO and postoperative ECMO (intraoperative: 40% vs. postoperative: 37%,  $P = 0.62$ ). In most of these studies, the impact of ECPR and postoperative residual

**Table 4** Predictive value of CPB time, preoperative lactate, and their combination for intraoperative VA ECMO

Variable	AUC	95%CI		P value	Cutoff value	Sensitivity (%)	Specificity (%)
		Lower	Upper				
CPB time (min)	0.722	0.574	0.871	0.003	138	100	40
Preoperative peak lactate (mmol/L)	0.789	0.659	0.918	< 0.001	6.495	66.7	80
Preoperative peak lactate + CPB time	0.893 ± 0.045	0.805	0.980	< 0.001	-	74.1	85

AUC area under the curve, CPB cardiopulmonary bypass

**Fig. 3** Receiver operating characteristic curves for intraoperative VA

lesions, which are currently considered risk factors for ECMO mortality [4, 7, 11, 15, 18], as confounding factors on outcomes has been overlooked; this potentially weakens the statistical power. Therefore, the results of these studies have not yet been unified [9–11, 19, 20]. Consequently, we excluded patients with ECPR and those with postoperative residual lesions to further clarify the impact of initiation timing on the prognosis of VA ECMO in children with CHD undergoing cardiac surgery.

Second, Prior studies, including a multicentre analysis by Gupta et al. [17] and a systematic review by Wu et al. [4], have shown that extended ECMO use beyond seven days and over 144 h, respectively, correlates with increased mortality risk. This study demonstrated that the implementation of intraoperative VA ECMO, as opposed to postoperative initiation, significantly reduced the duration of ECMO support and improved the success rates of weaning. This conclusion is corroborated by

numerous studies. For instance, one investigation demonstrated that the intraoperative application of ECMO as a circulatory support mechanism post-heart transplantation can effectively stabilize hemodynamics and facilitate myocardial recovery, thereby enhancing the success rate of weaning [21]. Additionally, another study explored the determinants affecting the success and failure of VA ECMO withdrawal, indicating that reducing the duration of VA ECMO support may improve the likelihood of successful weaning [22]. The reduced duration of ECMO has significant clinical implications. Studies have demonstrated that prolonged ECMO support is correlated with an increased risk of adverse events, such as intracranial hemorrhage, infection, and multiorgan failure [23, 24]. Additionally, extended ECMO duration is linked to longer ICU stays, thereby imposing greater demands on ICU resource allocation [25, 26]. Although there were no statistically significant differences in the duration

of mechanical ventilation and length of hospital stay between the two groups, the intraoperative ECMO group demonstrated a tendency towards shorter durations in both outcomes. This observation aligns with the findings of previous studies [27, 28]. These results suggest that initiating intraoperative VA ECMO may potentially reduce recovery time; however, further research is necessary to substantiate this hypothesis.

Third, intraoperative VA ECMO initiation was associated with lower preoperative LVEF and LVFS, higher preoperative lactate and prolonged CPB time, reflecting preoperative haemodynamic instability and surgical complexity. Elevated lactate reflects systemic hypoperfusion and anaerobic metabolism, which may worsen postoperative multiorgan dysfunction [29] and increase mortality risk [30, 31]. A prolonged CPB time is associated with an increased need for early ECMO, high-dose inotropic support and a greater risk of multiorgan failure [32–35]. Despite these factors, the in-hospital survival rate of the intraoperative group was significantly greater than that of the postoperative group, with the latter having a 2.84-fold greater risk of death. Several mechanisms may underlie this result: (1) Intraoperative VA ECMO rapidly restores systemic oxygen delivery, crucial for patients with LCOS, by providing external circulatory support and reducing the production and accumulation of lactic acid [36]. (2) VA ECMO can mitigate ischemic organ damage, with a study showing that early ECMO application reduces myocardial infarct size and maintains mitochondrial integrity [37]. Calabrese F et al. [38] demonstrated that intraoperative VA ECMO is a promising strategy for reducing ischemic reperfusion injury in lung tissue. (3) Intraoperative VA ECMO decreases ventricular end-diastolic pressure during CPB separation, reducing the need for high-dose inotropic drugs in damaged ventricles [39].

Fourth, we found that all three children with Down's syndrome received postoperative VA ECMO treatment. Considering the small number of patients and selective bias, we removed Down's syndrome from the multivariate logistic regression. Our predictive model ( $CPB \geq 138$  min and preoperative peak lactate  $\geq 6.495$  mmol/L, AUC: 0.893) offers actionable thresholds for timely intervention. Unlike Kuraim et al.'s findings that high lactate levels and VISs predict the need for ECMO within 48 h postsurgery [24], our study first introduces the combined use of CPB time and preoperative lactate as predictors for intraoperative VA ECMO. The combined use of CPB time and preoperative peak lactate levels can be integrated into real-time decision-making in the operating room. For example, if a patient has both a CPB time exceeding 138 min and a preoperative lactate level above 6.495 mmol/L,

surgeons can consider initiating VA ECMO to improve clinical outcomes. These findings may be generalizable to other centres or patient populations, but further validation in larger, multicenter cohorts is needed.

Fifth, compared with nonsurvivors, survivors demonstrated prolonged CPB times and MV durations — a finding that is contradictory to conventional clinical observations [40, 41]. This apparent paradox may be attributed to two key factors: (1) the high prevalence of intraoperative VA ECMO support in survivors (79%, 19/24), which potentially offsets the mortality risk associated with extended CPB duration; and (2) the truncated clinical course in nonsurvivors due to early mortality, consequently limiting their MV exposure time. Prolonged aortic cross-clamping (ACC) and CPB times have been associated with various postoperative complications, including hemodynamic instability and increased morbidity [42]. In our study, the duration of ACC did not exhibit a statistically significant difference between survivors and nonsurvivors, nor between the intraoperative and postoperative groups. This suggests that ACC duration may not serve as a predictive factor for patient outcomes within this cohort.

### Study limitations

Our findings highlight critical clinical implications but must be interpreted within the study's limitations. First, the single-centre retrospective design inherently introduces selection bias, as surgeon preferences and institutional protocols (e.g., ultrafiltration strategies) may influence VA ECMO timing. While our exclusion of ECPR and residual lesions strengthened internal validity, generalizability to broader populations requires confirmation through multicentre trials. Second, the lack of long-term follow-up data precludes assessment of survival beyond discharge, although prior studies suggest that intraoperative VA ECMO may be correlated with sustained benefits [6, 43]. Third, as an exploratory study, focusing solely on VA ECMO patients limits the specificity of predictors; the inclusion of non-ECMO cohorts in future studies could refine risk stratification. Despite these constraints, our predictive model offers actionable thresholds for infant VA ECMO initiation. Future studies should aim to validate these findings in larger, multicentre cohorts to assess their generalizability.

### Conclusions

Intraoperative VA ECMO was correlated with shorter durations, better weaning rates, and higher survival rates. It may benefit high-risk paediatric patients before CHD surgery. The CPB time and preoperative lactate

level significantly predicted the need for intraoperative VA ECMO. These findings offer insights into the timing of VA ECMO in paediatric cardiac surgery, indicating potential advantages in starting VA ECMO intraoperatively.

#### Abbreviations

ACC	Aortic cross-clamping
ACT	Activated clotting time
APTT	Activated partial thromboplastin time
AUC	Area under the curve
CHD	Congenital heart disease
CPB	Cardiopulmonary bypass
CRRT	Continuous renal replacement therapy
ECPR	Extracorporeal cardiopulmonary resuscitation
HR	Hazard ratio
ICU	Intensive care unit
IQR	Interquartile range
LCOS	Low cardiac output syndrome
LVEF	Left ventricular ejection fraction
LVFS	Left ventricular fractional shortening
MV	Mechanical ventilation
RACHS-1	Risk Adjustment in Congenital Heart Surgery-1
ROC	Receiver operating characteristic
VA ECMO	Venoarterial extracorporeal membrane oxygenation
VIS	Vasoactive inotropic score
VIF	Variance inflation factor

#### Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12872-025-04635-6>.

Supplementary Material 1.  
Supplementary Material 2.  
Supplementary Material 3.

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Not applicable.

#### Clinical trial number

Not applicable.

#### Authors' contributions

YQS and WP designed the study. LZ, DY wrote the manuscript and QFW revised the manuscript. QFW, HL, YSC, YPL and JRQ collected the data and LZ and XMM analyzed the data. All authors read and approved the manuscript.

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#### Data availability

The data are available from the corresponding author upon reasonable request.

#### Declarations

##### Ethics approval and consent to participate

This study, adhering to the Declaration of Helsinki, was approved by the Institutional Ethical Committees of the Children's Hospital of Nanjing Medical University (approval number: 202407004–1). Informed consent was not required due to the retrospective analysis of existing data.

#### Consent for publication

Not applicable.

#### Competing interests

The authors declare no competing interests.

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