

Age At Surgical Repair Reflects Preoperative Severity and Predicts Early Outcomes After Tetralogy of Fallot Repair in A Resource-Limited Setting: A Retrospective Cohort Study

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ABSTRACT

Objective: Children with tetralogy of Fallot (ToF) in resource-limited settings are often repaired years later than the age recommended in high-income countries. We set out to describe the preoperative severity profile of such a cohort and to test whether age at repair predicts low cardiac output syndrome (LCOS) and 30-day mortality independently of hypoxemia, nutritional status, and cardiac anatomy.

Materials and Methods: We conducted a retrospective cohort study of 116 children who underwent ToF repair between 2021 and 2025, comparing preoperative severity between children younger and older than the cohort's median age of 4 years. Logistic regression was used to estimate how age, preoperative oxygen saturation (SpO₂), weight, nutritional status, McGoon ratio, Nakata index, ventricular septal defect (VSD) size, major aortopulmonary collateral arteries (MAPCA), and sex related to LCOS and 30-day mortality, and discrimination was assessed with the area under the receiver operating characteristic curve (AUC).

Results: Median age at repair was 4.0 years (interquartile range 2.0–7.0); no patient was younger than 1 year. LCOS developed in 42 patients (36.2%), and 42 (36.2%) died within 30 days, with 83.3% of deaths occurring in patients who had LCOS. Compared with children 4 years or older (n=66), those younger than 4 years (n=50) had lower preoperative SpO₂ (77.3 ± 7.1% vs 81.6 ± 8.0%, p=0.003) and smaller VSDs (11.1 vs 13.0 mm, p=0.003), while nutritional status, hemoglobin/hematocrit, McGoon ratio, Nakata index, and MAPCA prevalence were similar between the two groups. Younger age was independently associated with both LCOS (OR per year younger 1.23, 95% CI 1.07–1.40) and mortality (OR 1.25, 95% CI 1.09–1.44); this association held after adjustment for VSD size and undernutrition, and again after adjustment for SpO₂ (age <4 years: OR 3.78, 95% CI 1.65–8.64 for mortality; OR 3.24, 95% CI 1.43–7.35 for LCOS). SpO₂, nutritional status, McGoon ratio, Nakata index, and MAPCA showed no independent association with either outcome. Age alone discriminated mortality with an AUC of 0.71 (95% CI 0.61–0.80), which rose to 0.73 once VSD size was added.

Conclusion: Younger age at repair — rather than hypoxemia, undernutrition, or pulmonary/septal anatomy on its own — emerged as the strongest and most consistent preoperative predictor of LCOS and early mortality in this cohort. This likely reflects both a genuine physiological vulnerability in younger, smaller children and a tendency for the most severely cyanotic infants and toddlers to be referred more urgently. Where health-system capacity allows, these findings support prioritizing earlier, elective repair, and where it does not, they support closer perioperative vigilance for young children.

Keywords: Tetralogy of Fallot, age, low cardiac output syndrome, mortality, hypoxemia, malnutrition, developing countries.

MAIN POINTS

- Children in this resource-limited cohort underwent tetralogy of Fallot repair at a median age of 4 years — far later than the 3–12 months recommended where resources allow.
- Compared with older children, those younger than 4 years were more hypoxemic and had smaller ventricular septal defects, despite similar nutritional status and pulmonary artery anatomy.
- Low cardiac output syndrome and 30-day mortality were both independently associated with younger age at repair, and this association held even after adjusting for oxygen saturation, nutritional status, and cardiac anatomy.
- Age at repair discriminated early mortality reasonably well (AUC 0.71), suggesting it could serve as a simple, readily available marker for flagging children who need heightened perioperative vigilance.

INTRODUCTION

Tetralogy of Fallot (ToF) is treated surgically, and where resources allow, repair is usually performed electively between 3 and 12 months of age.¹ Children in low- and middle-income countries, by contrast, tend to present later, more hypoxemic, and more malnourished, and early mortality after repair in these settings varies considerably — 3.6% in a 20-country quality-improvement collaborative that identified low oxygen saturation

and low weight-for-age as the principal risk factors,² versus 11.6% in an Indonesian series of late repairs.³ Similar factors emerged in a Chinese case-review series, which linked poor pulmonary and ventricular development, along with other markers of chronic disease, to postoperative low cardiac output syndrome (LCOS) and death.⁴ More recent work on predicting LCOS in ToF has instead focused on hemodynamic and oxygenation variables measured during and after surgery.⁵

Chronic hypoxemia in unrepaired ToF also drives growth failure and malnutrition — through increased metabolic demand, poor appetite, and recurrent illness — and malnutrition is, in turn, an independent predictor of poor perioperative outcomes in congenital heart disease.⁶ Age at repair, oxygen saturation, and nutritional status are therefore tightly interwoven in this population, yet studies rarely tease apart their separate contributions to early outcome. A delay in repair could act through worsening hypoxemia, through malnutrition, through anatomical changes as the pulmonary arteries and right ventricular outflow tract evolve over time, or simply through a heightened physiological vulnerability of younger, smaller children to the stress of surgery, regardless of these other factors.

In this study, we characterized the preoperative severity profile of a cohort of children undergoing ToF repair in a resource-limited setting, compared this profile between children operated on before and after the cohort's median age of 4 years, and asked whether age at repair predicts LCOS and 30-day mortality independently of preoperative oxygen saturation, nutritional status, and cardiac anatomy.

MATERIALS AND METHODS

Study design and population

This was a single-center retrospective cohort study, reported in line with the STROBE statement.⁷ It was conducted at the Pediatric Cardiology Clinic, Integrated Cardiac Care Center (Pusat Pelayanan Jantung Terpadu, PPJT), together with the Department of Thoracic, Cardiac and Vascular Surgery, Dr. Soetomo General Academic Hospital, Surabaya, Indonesia. Eligible patients were children aged 1–18 years with ToF confirmed by echocardiography and/or cardiac catheterization who underwent total correction surgery between 1 January 2021 and 31 December 2025, with complete medical-record data available for the preoperative (clinical, echocardiographic, and catheterization), key intraoperative, and early postoperative variables of interest. We excluded patients with another complex congenital heart lesion requiring a different surgical strategy (double-outlet right ventricle, pulmonary atresia with complex-type VSD, atrioventricular septal defect, absent pulmonary valve syndrome, or truncus arteriosus); those who had undergone only a palliative procedure (e.g., a Blalock–Taussig shunt) without subsequent total correction during the study period; those undergoing a redo rather than a first total correction; those with an untraceable 30-day vital status or missing preoperative echocardiographic/catheterization data for the key anatomical parameters (pulmonary valve annulus z-score, McGoon ratio, or Nakata index); those who died before surgery; those referred or transferred to another hospital before their 30-day outcome could be confirmed; and duplicate medical records. Of the 149 children who underwent total correction during the study period, 133 met the inclusion criteria, and after the exclusion criteria were applied, 116 patients remained for analysis; three of them (2.6%) had undergone a previous palliative procedure.

Data collection and quality review

Data came from the study database, which had been compiled from medical records. The variables collected were age at surgery (completed years), sex, weight, height, nutritional status (adequate, moderate undernutrition/stunting, severe malnutrition/stunting, or overweight), preoperative oxygen saturation (SpO₂), hemoglobin, hematocrit, associated lesions (patent ductus arteriosus, atrial septal defect/patent foramen ovale, MAPCA), previous palliation, McGoon ratio, Nakata index,⁸ ventricular septal defect (VSD) size, LV ejection fraction, and RV and LV systolic pressures. We screened these values for internal consistency before analysis and found two isolated problems, both of which were corrected: one patient's operative date was recorded as 1935, an evident data-entry error that was set to missing without affecting that patient's age or any other variable; and one patient's VSD size (1.5 mm) was inconsistent with the value recorded for the same patient in an earlier version of the dataset (15.0 mm) and implausible given that patient's McGoon ratio and Nakata index, so it too was set to missing pending verification against the original record. No other implausible values turned up among the clinical and anthropometric variables used here. Left ventricular echocardiographic dimensions were analyzed separately and are not used or discussed in this report.

Outcomes

The primary outcomes were LCOS and 30-day mortality, defined as death from any cause within 30 days of the operative date, whether during the index admission or after discharge, regardless of how directly it related to the surgical procedure itself. LCOS was ascertained from the clinical diagnosis that the treating cardiology and cardiac surgery team documented in the patient's medical record during the early postoperative period, rather than from a standardized quantitative definition applied by the study team. It was recorded as a discrete postoperative event on the study case-report form, within the broader "major complication" category that also captures ECMO, reoperation, and major postoperative bleeding, following the classification used by Brown et al. (2017).

Statistical analysis

We assessed normality with the Shapiro–Wilk test. Continuous variables are presented as mean $\pm$ SD or median (IQR) and were compared with Student's t-test or the Mann–Whitney U test, as appropriate; categorical variables were compared with the chi-square test, switching to Fisher's exact test whenever an expected cell count fell below 5. For descriptive comparison, the cohort was split at its median age (4 years) into younger and older groups. Logistic regression was used to estimate univariable odds ratios (OR) for LCOS and 30-day mortality across age (per year), SpO₂ (per 5% lower), weight (per kg lighter), undernutrition (moderate or severe vs adequate/overweight), McGoon ratio, Nakata index, VSD size, MAPCA, and sex. A multivariable model included age, VSD size, and undernutrition (roughly 14 events per covariate for both mortality and LCOS); weight was left out of this model because of its very strong collinearity with age (Spearman $\rho=0.88$). As a sensitivity analysis, we re-estimated the association between the binary age group (<4 vs ≥ 4 years) and each outcome after adding SpO₂ to the model, to see whether hypoxemia accounted for the age effect. Discrimination was assessed using the AUC, both for age alone and for a combined age + VSD size model, with 95% CIs derived from 2,000 bootstrap resamples. Analyses were complete-case, with VSD size missing in only 1 of 116 patients. We considered two-sided $p<0.05$ statistically significant, though given the number of comparisons made, results should be read with that in mind. All analyses were performed in Python 3 using SciPy, statsmodels, and scikit-learn.

Ethics

The study protocol was approved by the Ethics Committee of Dr. Soetomo General Academic Hospital, Surabaya, Indonesia (approval no. 2345/LOE/301.4.2/IV/2026, approved April 27, 2026), and the study was conducted in accordance with the principles of the Declaration of Helsinki.

RESULTS

Cohort characteristics

The cohort comprised 116 children (70 male, 60.3%) aged 1 to 17 years; median age at repair was 4.0 years (IQR 2.0–7.0), and no child was younger than 1 year (Table 1). Preoperative hypoxemia was common, with a mean SpO₂ of $79.8 \pm 7.9\%$, and 64.7% of children were undernourished (severely so in 50.9%). McGoon ratio (mean 2.3 ± 0.5) and Nakata index (median $281.5 \text{ mm}^2/\text{m}^2$) pointed to broadly preserved pulmonary artery growth in most children, while MAPCA were present in 32.8%. LCOS developed in 42 patients (36.2%), and 42 (36.2%) died within 30 days. The two outcomes were closely linked: 35 of the 42 patients with LCOS died (83.3%), compared with only 7 of 74 without LCOS (9.5%; OR 47.9, 95% CI 15.5–147.3).

Severity profile by age group

Children younger than 4 years ($n=50$) differed from those 4 years or older ($n=66$) in two respects (Table 2). They were more hypoxemic (SpO₂ $77.3 \pm 7.1\%$ vs $81.6 \pm 8.0\%$, $p=0.003$; SpO₂ $<85\%$ in 82.0% vs 57.6%, $p=0.005$) and had smaller VSDs (median 11.1 vs 13.0 mm, $p=0.003$), consistent with more severe right ventricular outflow tract obstruction in the younger group. Hemoglobin, hematocrit, undernutrition (66.0% vs 63.6%), severe malnutrition (50.0% vs 51.5%), McGoon ratio, Nakata index, and MAPCA prevalence (30.0% vs 34.8%), by contrast, did not differ between the two age groups (all $p>0.3$), and previous palliation was uncommon in both.

Univariable associations with LCOS and mortality

Of all the variables examined, age was the one most consistently associated with both outcomes (Table 3): each year younger corresponded to 25% higher odds of mortality (OR 1.25, 95% CI 1.09–1.44) and 23% higher odds of LCOS (OR 1.23, 95% CI 1.07–1.40). Weight showed a similar pattern, unsurprisingly given its collinearity with age, and smaller VSD size was likewise associated with higher odds of both outcomes. SpO₂, undernutrition,

McGoon ratio, Nakata index, MAPCA, and sex were not significantly associated with either outcome in univariable analysis, though undernutrition came close for LCOS (OR 2.31, 95% CI 0.99–5.38; $p=0.053$).

Multivariable analysis and sensitivity to hypoxemia

Age remained independently associated with both mortality (OR per year younger 1.20, 95% CI 1.04–1.39; $p=0.015$) and LCOS (OR 1.19, 95% CI 1.03–1.38; $p=0.017$) in a multivariable model that also included VSD size and undernutrition (Table 4); the latter two were attenuated and no longer statistically significant. Adding SpO₂ to the binary age-group model (<4 vs ≥4 years) barely changed the estimate — for mortality, the crude OR of 3.99 (95% CI 1.79–8.89) became 3.78 (95% CI 1.65–8.64) after adjustment, and for LCOS, 3.39 (95% CI 1.54–7.46) became 3.24 (95% CI 1.43–7.35) — suggesting that differences in preoperative oxygen saturation do not substantially explain the age association.

Discrimination

Age alone discriminated 30-day mortality with an AUC of 0.71 (95% CI 0.61–0.80) and LCOS with an AUC of 0.69 (Figure 3); adding VSD size improved this only modestly, to 0.73 for mortality and 0.71 for LCOS. SpO₂ alone, by comparison, discriminated mortality poorly (AUC 0.58).

DISCUSSION

Children in this cohort underwent ToF repair at a median age of 4 years — several years beyond what is recommended where resources allow¹ — and their preoperative severity profile told a consistent story: younger age at repair was independently associated with LCOS and early mortality, more so than any single measure of hypoxemia, nutrition, or cardiac anatomy on its own. This association held after adjusting for SpO₂, VSD size, and undernutrition, and age alone discriminated mortality with an AUC of 0.71 — a level of discrimination that many single anatomical or laboratory variables in this field fail to reach.

Two explanations, not mutually exclusive, fit these findings. One is physiological: younger and smaller children may simply tolerate the hemodynamic stress of cardiopulmonary bypass and the sudden normalization of pulmonary and systemic blood flow less well, regardless of how hypoxemic or malnourished they are at the time of surgery. The other has to do with selection: age at repair in a resource-limited system is not purely a matter of delay. Children with more severe RVOT obstruction — reflected here in smaller VSDs and lower SpO₂ — tend to become symptomatic earlier and are referred more urgently, whereas children with more balanced pulmonary blood flow (“pink tetralogy”) can remain stable for years without surgery and end up being older by the time they are finally operated on. Our data fit this pattern: younger children had significantly lower SpO₂ and smaller VSDs, yet their nutritional status and pulmonary artery anatomy were no different from those of older children. Age at repair in this setting therefore seems to carry two kinds of information at once — something about how urgently a child was referred, and, independently of that, something about the physiological vulnerability that comes with being young, since the age association survived adjustment for SpO₂. Malnutrition, though strikingly common across the cohort (65% of children, half of them severely affected), did not differ by age group and was not independently associated with LCOS or mortality once other variables were accounted for. This is not at odds with the broader literature linking malnutrition to poor outcomes after congenital heart surgery;⁶ rather, when undernutrition is nearly universal, as it was here, it may simply have little room left to discriminate risk within the cohort, even though it likely still contributes to the overall event rate being higher than in better-resourced settings. Much the same may be true of McGoon ratio and Nakata index, which were relatively well preserved, did not differ by age, and did not discriminate outcome — suggesting that pulmonary artery hypoplasia is not the dominant anatomical driver of risk in this cohort, despite the emphasis it receives in some earlier literature.

Both the overall mortality (36.2%) and LCOS incidence (36.2%) in this cohort are far higher than the 3.6% mortality reported by the international ToF quality-improvement collaborative² or the 11.6% reported in another Indonesian series,³ and LCOS and death were tightly linked in our data — 83% of deaths occurred in patients with LCOS — echoing the emphasis on LCOS as a proximate driver of mortality in recent prediction-model work.⁵ Near-universal hypoxemia, high rates of malnutrition, and delayed repair, together with perioperative and intensive-care resource constraints that this dataset does not capture, likely explain much of this gap; the Chinese series that identified poor pulmonary and ventricular development as LCOS risk factors also involved a cohort with substantial preoperative comorbidity.⁴

Clinical implications

These findings point to two practical responses. Where program capacity allows, prioritizing earlier elective repair — particularly for children on a trajectory toward becoming more severely cyanotic — could keep fewer children from reaching surgery in the highest-risk, youngest-age stratum. Where earlier repair is not achievable because of systemic constraints, age at repair is at least a simple, always-available variable that clinicians could factor into preoperative risk discussions and perioperative planning — for instance, anticipating LCOS and preparing for its management in the youngest children — without waiting on more specialized risk scores.

Limitations

This is a retrospective, single-center study. LCOS was ascertained from the clinical diagnosis documented in the medical record rather than from a standardized quantitative definition applied uniformly by the study team; because these records were completed by different clinicians over several years, some diagnostic variability cannot be ruled out, and the threshold for documenting LCOS may have shifted somewhat over time or between clinicians. That said, this approach likely captured clinically meaningful episodes that prompted a change in management, and the strength of the association between LCOS and 30-day mortality (83% of deaths occurred in patients with LCOS) argues against the outcome being trivial or arbitrarily assigned. Data on intraoperative variables (cardiopulmonary bypass and cross-clamp time, surgical technique) and on perioperative care processes were not available and may confound or mediate the age association we observed. Nutritional status was recorded as a categorical field rather than a continuous anthropometric z-score, which may have limited its ability to discriminate risk. The multivariable model was necessarily restricted in the number of covariates it could include given the number of events, so residual confounding cannot be excluded, and because age, weight, and VSD size are correlated with one another, their individual contributions should be read with some caution. Follow-up was limited to 30 days, so we cannot say whether the same associations hold for later outcomes.

CONCLUSION

In this cohort of children undergoing tetralogy of Fallot repair in a resource-limited setting, younger age at repair was independently associated with low cardiac output syndrome and 30-day mortality — more consistently than preoperative hypoxemia, nutritional status, or pulmonary/septal anatomy considered on their own — and discriminated mortality reasonably well by itself. These results support efforts to bring elective repair forward where feasible, and suggest that age can still usefully inform perioperative risk anticipation where it cannot be. Prospective, multicenter studies that incorporate intraoperative and postoperative care variables are needed to clarify the mechanisms linking age to early outcome in this population.

DECLARATIONS

Ethics Committee Approval: This study was approved by the Ethics Committee of Dr. Soetomo General Academic Hospital, Surabaya, Indonesia (approval no. 2345/LOE/301.4.2/IV/2026, approved April 27, 2026).

Informed Consent: Because of the retrospective, medical-record-based design and the use of de-identified patient data, the requirement for individual written informed consent was waived by the Health Research Ethics Committee of Dr. Soetomo General Academic Hospital, in accordance with the Declaration of Helsinki and the National Guidelines on Health Research Ethics (Komisi Nasional Etik Penelitian Kesehatan, Kementerian Kesehatan RI).

Data Sharing Statement: The de-identified dataset is available from the corresponding author upon reasonable request.

Author Contributions: DPS – conception and design, data acquisition, statistical analysis, drafting the manuscript; IKAU and MAR – conception and design, supervision, critical revision of the manuscript; TH, HW, and PHN – data acquisition and critical revision of the manuscript; ARH – provision of surgical/intraoperative data and critical revision of the manuscript. All authors approved the final version and agree to be accountable for all aspects of the work.

Conflict of Interest: The authors declare no conflict of interest.

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Table 1. Baseline characteristics of the full cohort (n=116).

Characteristic	Value
Age, years, median (IQR)	4.0 (2.0–7.0)
Weight, kg, median (IQR)	12.5 (9.7–17.0)
Height, cm, median (IQR)	95.0 (83.8–114.2)
Preoperative SpO ₂ , %, mean ± SD	79.8 ± 7.9
Hemoglobin, g/dL, mean ± SD	17.2 ± 3.1
Hematocrit, %, median (IQR)	50.5 (44.0–58.7)
McGoon ratio, mean ± SD	2.3 ± 0.5
Nakata index, mm ² /m ² , median (IQR)	281.5 (218.0–365.2)
VSD size, mm, median (IQR)*	12.0 (10.0–14.3)
LV ejection fraction, %, mean ± SD	75.7 ± 8.8
RV systolic pressure, mmHg, median (IQR)	80.0 (68.8–85.0)
LV systolic pressure, mmHg, median (IQR)	80.0 (73.0–87.0)
Male sex, n (%)	70 (60.3%)
Undernutrition (moderate or severe), n (%)	75 (64.7%)
Severe malnutrition/stunting, n (%)	59 (50.9%)
MAPCA, n (%)	38 (32.8%)
Patent ductus arteriosus, n (%)	29 (25.0%)
PFO/ASD, n (%)	51 (44.0%)
Previous palliation, n (%)	3 (2.6%)
30-day mortality, n (%)	42 (36.2%)
Low cardiac output syndrome (LCOS), n (%)	42 (36.2%)
Prolonged ventilation (>96 h), n (%)	40 (34.5%)
Prolonged ICU stay (>3 days), n (%)	61 (52.6%)
Prolonged hospital stay (>7 days), n (%)	74 (63.8%)
Any major complication, n (%)	78 (67.2%)
Reoperation, n (%)	23 (19.8%)
Peritoneal dialysis/CRRT, n (%)	52 (44.8%)

SpO₂, oxygen saturation; MAPCA, major aortopulmonary collateral arteries; LCOS, low cardiac output syndrome; CRRT, continuous renal replacement therapy. This report does not use or discuss left ventricular echocardiographic dimensions. *n=115 (one missing VSD size).

Table 2. Preoperative severity profile by age at repair (<4 vs ≥4 years, the cohort median).

Characteristic	<4 years	≥4 years	p
Age, years, median (IQR)	2.0 (1.2–3.0)	7.0 (5.0–9.0)	<0.001
Weight, kg, median (IQR)	9.5 (8.4–10.2)	16.0 (13.1–23.4)	<0.001
Height, cm, median (IQR)	80.5 (75.0–88.0)	110.5 (99.0–130.0)	<0.001
Preoperative SpO ₂ , %, mean ± SD	77.3 ± 7.1	81.6 ± 8.0	0.003
SpO ₂ <85%, n (%)	41/50 (82.0)	38/66 (57.6)	0.005
SpO ₂ <80%, n (%)	32/50 (64.0)	30/66 (45.5)	0.047
Hemoglobin, g/dL, mean ± SD	17.1 ± 3.0	17.3 ± 3.1	0.773
Hematocrit, %, median (IQR)	50.9 (45.0–58.0)	50.2 (44.0–58.8)	0.861
Undernutrition (moderate or severe), n (%)	33/50 (66.0)	42/66 (63.6)	0.792
Severe malnutrition/stunting, n (%)	25/50 (50.0)	34/66 (51.5)	0.872
McGoon ratio, mean ± SD	2.3 ± 0.5	2.4 ± 0.5	0.312
Nakata index, mm ² /m ² , median (IQR)	273.0 (218.0–348.2)	285.0 (226.0–372.8)	0.512
VSD size, mm, median (IQR)	11.1 (9.5–13.0)	13.0 (10.7–15.6)	0.003
MAPCA, n (%)	15/50 (30.0)	23/66 (34.8)	0.582
Previous palliation, n (%)	2/50 (4.0)	1/66 (1.5)	0.577

Continuous variables were compared with Student's t-test (mean ± SD) or the Mann–Whitney U test (median [IQR]); categorical variables with the chi-square test or Fisher's exact test when an expected count was below 5. MAPCA, major aortopulmonary collateral arteries.

Table 3. Univariable logistic regression: preoperative variables and 30-day mortality/LCOS.

Preoperative variable	OR (95% CI)—mortality	p	OR (95% CI)—LCOS	p
Age, per year younger	1.25 (1.09–1.44)	0.002	1.23 (1.07–1.40)	0.003
Preoperative SpO ₂ , per 5% lower	1.19 (0.93–1.53)	0.166	1.16 (0.91–1.48)	0.233
Weight, per kg lighter	1.10 (1.03–1.18)	0.007	1.10 (1.03–1.19)	0.007
Undernutrition (moderate/severe)	1.92 (0.84–4.41)	0.123	2.31 (0.99–5.38)	0.053
McGoon ratio, per unit	0.89 (0.40–1.99)	0.781	0.79 (0.36–1.78)	0.575
Nakata index, per unit	1.00 (1.00–1.00)	0.272	1.00 (1.00–1.00)	0.526
VSD size, per mm	0.84 (0.74–0.95)	0.006	0.86 (0.76–0.97)	0.013
MAPCA	1.04 (0.47–2.33)	0.921	0.88 (0.39–1.98)	0.755
Male sex	1.52 (0.69–3.36)	0.296	0.95 (0.44–2.05)	0.892

OR, odds ratio; CI, confidence interval; LCOS, low cardiac output syndrome; n=116 for all rows except VSD size (n=115). Weight and age were not combined in the same model given their strong collinearity (p=0.88).

Table 4. Multivariable logistic regression (age, VSD size, undernutrition).

Preoperative variable	OR (95% CI)—mortality	p	OR (95% CI)—LCOS	p
Age, per year younger	1.20 (1.04–1.39)	0.015	1.19 (1.03–1.38)	0.017
VSD size, per mm	0.90 (0.78–1.03)	0.129	0.93 (0.81–1.06)	0.252
Undernutrition (moderate/severe)	1.65 (0.67–4.08)	0.279	2.07 (0.83–5.15)	0.116

Preoperative variable	OR (95% CI)— mortality	p	OR (95% CI)—LCOS	p
n (events)	115 (42)		115 (42)	

OR, odds ratio; CI, confidence interval; LCOS, low cardiac output syndrome. n=115 (1 missing VSD size).

OR, odds ratio; CI, confidence interval. If the age association were largely explained by hypoxemia, adding SpO₂ would be expected to substantially attenuate the age-group odds ratio; it did not.

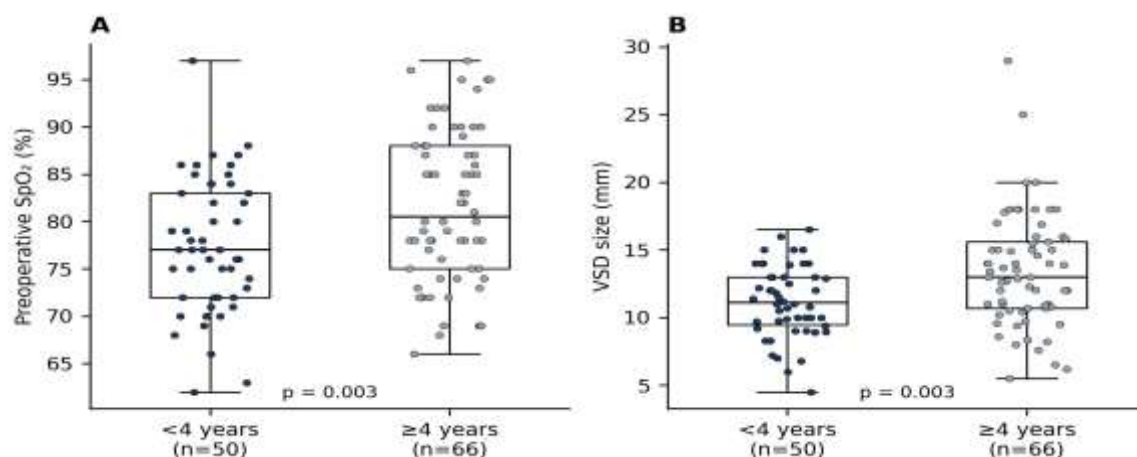


Figure 1. Preoperative (A) oxygen saturation and (B) VSD size by age group. Boxes show median and IQR; points are individual patients.

Table 5. Sensitivity analysis: age group (<4 vs ≥4 years), with and without adjustment for SpO₂.

Model	n (events)	OR (95% CI)	p
Age <4y vs ≥4y, mortality, crude	116 (42)	3.99 (1.79–8.89)	<0.001
Age <4y vs ≥4y, mortality, + SpO ₂	116 (42)	3.78 (1.65–8.64)	0.002
Age <4y vs ≥4y, LCOS, crude	116 (42)	3.39 (1.54–7.46)	0.002
Age <4y vs ≥4y, LCOS, + SpO ₂	116 (42)	3.24 (1.43–7.35)	0.005

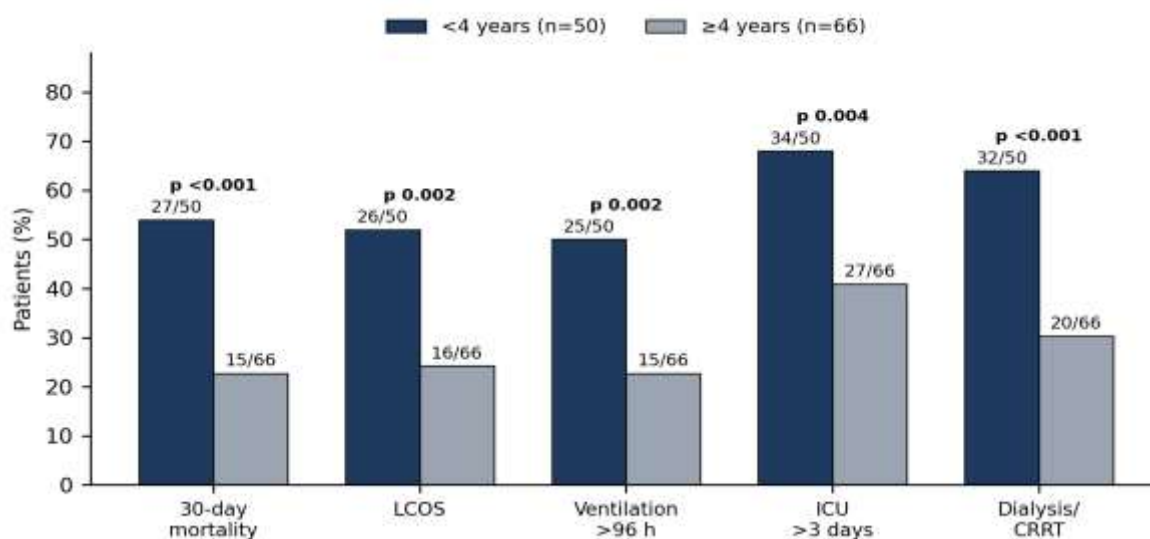


Figure 2. Key postoperative outcomes by age group (events/patients shown above bars).

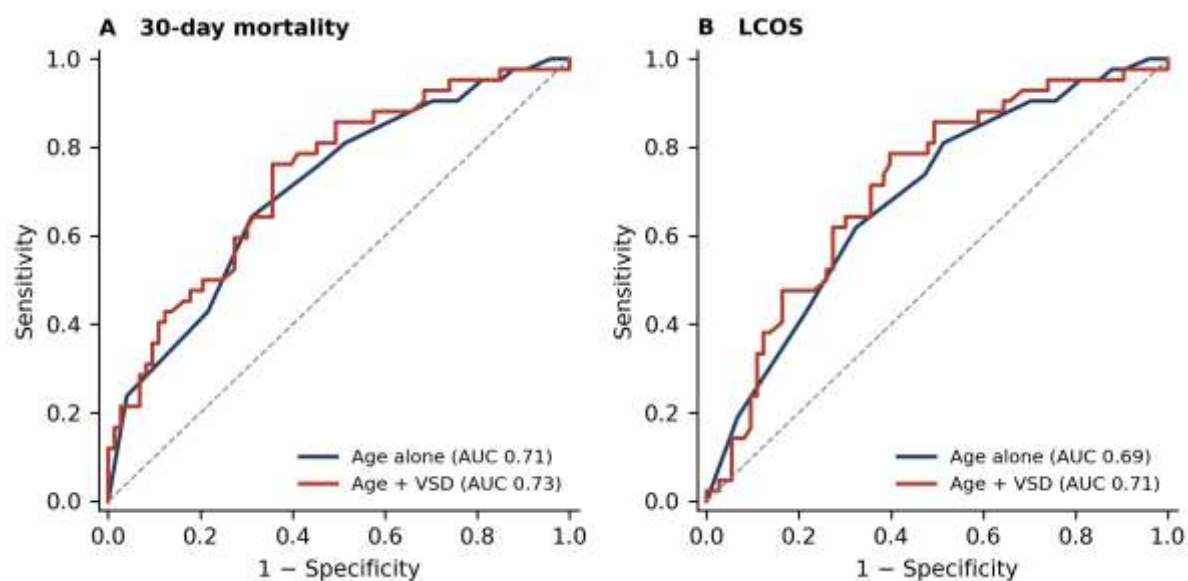


Figure 3. Receiver operating characteristic curves for age alone versus age combined with VSD size, for (A) 30-day mortality and (B) LCOS.