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# Association between muscular tissue desaturation and acute kidney injury in patients after surgery for acute type A aortic dissection: a single-center retrospective study

Long Zhao<sup>1†</sup>, Ling Peng<sup>2†</sup>, Qianli Huang<sup>2</sup> and Wei Wei<sup>2\*</sup>

## Abstract

**Background** A significant association between muscular tissue oxygen saturation (SmtO<sub>2</sub>), measured by near-infrared spectroscopy (NIRS), and postoperative complications has been observed in patients undergoing major surgery. However, the association between muscular tissue desaturation and acute kidney injury (AKI) has not yet been reported in patients following surgery for acute type A aortic dissection.

**Method** One hundred seventy-four adult patients who underwent total aortic arch replacement (TAAR) under cardiopulmonary bypass (CPB) and deep hypothermic circulatory arrest (DHCA) for acute type A aortic dissection were retrospectively analyzed. Muscular tissue oxygen saturation (SmtO<sub>2</sub>) in the gastrocnemius muscle region and regional cerebral oxygen saturation (rScO<sub>2</sub>) on the bilateral forehead were measured using near-infrared spectroscopy (NIRS). The thresholds defining muscular tissue desaturation were SmtO<sub>2</sub> < 80%, < 85%, and < 90% of baseline (relative changes compared to the baseline) and < 55% and < 50% (absolute values). Cerebral desaturation was defined as rScO<sub>2</sub> < 55%, < 50%, and < 80% baseline, on either the left or right side. The baseline, minimum, and mean values of SmtO<sub>2</sub> and rScO<sub>2</sub> were also extracted for analysis. The primary objective of this study was to investigate the association between muscular tissue desaturation and AKI.

**Result** AKI occurred in 71 (40.80%) of the 174 patients underwent TAAR under CPB and DHCA. SmtO<sub>2</sub> < 80% of baseline was associated with an increased risk of AKI (odds ratio [OR], 1.021; 95% confidence interval [CI], 1.001–1.041; *P* = 0.034). A receiver operating characteristic curve showed that the optimal cutoff for SmtO<sub>2</sub> < 80% baseline duration was 33.5 min in predicting AKI (sensitivity, 70.00%; specificity, 77.80%). The durations of SmtO<sub>2</sub> < 85% baseline (OR, 1.009; 95% CI, 0.996–1.021; *P* = 0.195) and < 90% baseline (OR, 1.007; 95% CI, 0.996–1.018; *P* = 0.208) were not significantly associated with AKI. There were no significant differences in the durations of absolute SmtO<sub>2</sub> values < 55%

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and < 50% or in the minimum SmtO<sub>2</sub> between the two cohorts. Durations of left and right rScO<sub>2</sub> < 55%, < 50%, and < 80% baseline were not associated with AKI. Patients with AKI experienced significantly higher in-hospital mortality and more postoperative complications compared with non-AKI patients.

**Conclusion** Muscular tissue desaturation, defined as SmtO<sub>2</sub> < 80% of baseline monitored on the lower leg, was significantly associated with an increased risk of AKI in patients who underwent TAAR under CPB and DHCA. Cerebral desaturation, defined as absolute rScO<sub>2</sub> < 55% or < 50%, or < 80% baseline was not associated with AKI.

**Keywords** Acute kidney injury, Muscular tissue desaturation, Regional cerebral desaturation, Near-infrared spectroscopy, Acute type A aortic dissection

## Background

Acute kidney injury (AKI) is a common complication after cardiac surgery performed under cardiopulmonary bypass (CPB), with a reported incidence of 16–30% [1–3]. In patients with acute type A aortic dissection undergoing total aortic arch replacement (TAAR) under CPB and deep hypothermic circulatory arrest (DHCA), the incidence of AKI is reportedly as high as 40–70% [4–7]. Among patients who developed AKI, approximately 2 to 5% require dialysis, which carries a mortality of 50–80% [1]. AKI is associated with adverse outcomes, including prolonged mechanical ventilation, extended intensive care unit (ICU) stays and hospitalizations, increased healthcare costs, and higher mortality [8, 9].

Impaired perfusion and oxygenation of renal tissue during surgery may contribute to postoperative AKI [1, 10, 11]. Tissue oxygen saturation, measured using near-infrared spectroscopy (NIRS), can assess the balance between oxygen supply and consumption in the tissue illuminated by near-infrared light [12–15]. Previous studies have reported that tissue oxygen saturation measured percutaneously over the kidney region in infants correlates with renal perfusion and oxygenation [16]. Furthermore, a significant association between renal tissue oxygen saturation and AKI in infants undergoing cardiac surgery has been demonstrated in several studies [17–19]. However, for infants weighing more than 10 kg and for adults, direct monitoring of renal tissue oxygen saturation is not feasible due to depth limit of near-infrared light penetration (4 cm) relative to the distance between the skin and the kidney [18]. Recent observational studies have shown an association between lower intraoperative muscular tissue oxygen saturation (SmtO<sub>2</sub>) measured on the forearm or legs and an increased risk of postoperative AKI in patients undergoing cardiac surgery with CPB, suggesting that SmtO<sub>2</sub> may serve as a surrogate for renal perfusion and oxygenation [3, 20–22]. However, the association between muscular tissue desaturation and postoperative AKI has not been reported in patients undergoing cardiac surgery with both CPB and DHCA.

Therefore, the primary objective of this study was to investigate relationship between muscular tissue desaturation and postoperative AKI in patients undergoing

TAAR under CPB and DHCA for acute type A acute aortic dissection. The secondary objective was to assess the association between cerebral desaturation and AKI.

## Methods

### Study design and population

This retrospective study reviewed adult patients who underwent TAAR under CPB and DHCA for acute type A aortic dissection from January 2019 to January 2022. The study was approved by the Ethics Committee of West China Hospital, Sichuan University (NO. 2023455). Written informed consent was waived due to the retrospective nature of the analysis. All procedures involving human participants were conducted in accordance with the Declaration of Helsinki.

### Anesthetic care

Standard monitoring, including five-lead electrocardiography, pulse oxygen saturation (SpO<sub>2</sub>), invasive blood pressures, nasopharyngeal and rectal temperatures, and capnography, was performed. Invasive blood pressure was monitored via the bilateral radial arteries and the left dorsal pedis artery. General anesthesia was induced with midazolam (0.04–0.1 mg/kg), sufentanil (1–2 µg/kg), and rocuronium (0.5–1.2 mg/kg). Anesthesia was maintained with sevoflurane inhalation (1–2%), propofol infusion (3–5 mg/kg/h), and intermittent administration of sufentanil and cisatracurium besylate, adjusted according to dynamic surgical stimulation and the CPB process to maintain the target plasma concentration. After tracheal intubation, pressure-controlled mechanical ventilation was adjusted to maintain end-tidal carbon dioxide (EtCO<sub>2</sub>) in the normal range. A central venous catheter was inserted in the right jugular vein for central venous pressure monitoring, and transesophageal echocardiography (iE33; Phillips Medical System, Andover, MA, USA) was routinely applied before surgery.

### Surgical procedure and CPB management

All patients underwent TAAR via median sternotomy in the supine position. A Stockert-5 roller pump (Sorin Group, München, Germany) with a disposable hollow-fiber membrane adult oxygenator (Affinity Pixie;

Medtronic, Minneapolis, MN) was used. Prior to the initiation of CPB, each patient received a bolus of heparin (375 U/kg). CPB was initiated when the activated clotting time reached at least 480 s. Aortic cannulation, right axillary artery cannulation, or femoral artery cannulation was performed for systemic perfusion, and systemic venous return was achieved by vena cava cannulation or transfemoral venous cannulation. Systemic hypothermia was achieved before initiating circulatory arrest, with target temperatures of 24–26 °C in the nasopharynx and 26–28 °C in the rectum. During the cooling phase before circulatory arrest, the pump flow rate was gradually decreased from 3.2 to 2.2 L/min/m<sup>2</sup>, maintaining a mean arterial blood pressure of 50–80 mmHg and a mixed venous oxygen saturation ( $S\bar{v}O_2$ ) above 70%. The target hematocrit was maintained at 25–30% during CPB. Cardiac arrest was induced with cold blood cardioplegia (crystalloid-to-blood ratio of 1:4) at a dose of 30 mL/kg for all patients. Cardioplegia was repeated every 20 to 25 min during surgery at a half the initial dose. After the establishment of DHCA, selective antegrade cerebral perfusion (ACP) was initiated via innominate artery cannulation or axillary artery cannulation. The ACP flow rate was adjusted between 6 and 12 mL/min/kg, with cerebral perfusion pressure maintained between 40 and 50 mmHg. In the clinical practice of our center, if left cerebral oxygen saturation ( $rScO_2$ ) dropped more than 10% lower than right  $rScO_2$  during unilateral ACP, the protocol called for immediate conversion to bilateral ACP to optimize cerebral oxygenation. After DHCA termination, rewarming was conducted at a rate of approximately 0.5 °C per minute. Alpha-stat management was used during the cooling and rewarming phases, while pH-stat was applied during the DHCA and selective ACP phases. All patients were transferred to the ICU after surgery for respiratory and circulatory support.

#### Tissue oxygen saturation monitoring

Two self-adhesive transcutaneous oximetry sensors (EGOS-600 A, Suzhou Engine Bio-medical Electronics, Suzhou, China) were placed bilaterally on the forehead for  $rScO_2$  monitoring, avoiding the midline and ensuring a distance of at least 1 cm above the eyebrows to prevent interference from the frontal sinus. Another sensor was placed on the right lower leg, specifically at the gastrocnemius muscle region, approximately 10 cm (about four finger-widths) below the tibial tuberosity and 5 cm (about two finger-widths) lateral to the anterior border of the tibia, to monitor the  $SmtO_2$  of the right lower leg. Additionally, the sensors were secured using adhesive patches (3 M, Shanghai, China) to ensure consistent contact pressure and avoid movement during the procedure. Real-time  $rScO_2$  and  $SmtO_2$  were recorded at a frequency of 0.5 Hz. Monitoring and data recording routinely began

before anesthesia induction and continued until the end of the surgery.

#### Data collection and definition

The baseline values for  $SmtO_2$  and  $rScO_2$  were averaged over 30 s after anesthesia induction. The minimum  $SmtO_2$  and  $rScO_2$  values represented the lowest measurements taken at any point during surgery. The mean  $SmtO_2$  and  $rScO_2$  values were calculated across the entire monitoring period. The primary objective of our study was to investigate the association between muscular tissue desaturation and AKI in patients undergoing CPB with DHCA. Muscular tissue desaturation was defined as a  $SmtO_2$  value lower than a given threshold during surgery. The thresholds used to define muscular tissue desaturation were <80%, <85%, and <90% baseline, representing relative changes from baseline measurement, and <55%, <50%, representing absolute values. The secondary objective was to analyze the association between cerebral desaturation and AKI. Based on previous studies, cerebral desaturation was defined as left or right  $rScO_2$  lower than 55%, 50%, and 80% baseline during surgery [23–25].

Postoperative complications, including AKI, delirium, stroke, systolic heart failure, and lung infection were documented. AKI was defined according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria as an increase in serum creatinine to more than 1.5 times baseline within 7 days after surgery or a 26.4 mmol/L increase from baseline within 48 h [26]. Postoperative delirium was measured using the Confusion Assessment Method (CAM) or CAM-ICU for intubated patients. Stroke was defined as a global or focal neurological lesion, primarily detected by cerebral computed tomography (CT). Systolic heart failure was defined as an ejection fraction (EF) of less than 50% during postoperative hospitalization.

Demographic and clinical data were collected from patients' electronic medical records, including preoperative comorbidities, cerebral and muscle tissue oxygen saturation data, surgery-related data, and postoperative complications. All medical records were collected by two independent investigators who were blinded to the study's aim, and all researchers were blinded to the study data until the outcomes were generated.

#### Statistical analysis

Data are presented as mean ± standard deviation (SD) for continuous, normally distributed variables and as frequency (percentage or absolute number) for categorical data. The normality of distribution was tested using the Kolmogorov-Smirnov test. Inter-group differences in continuous variables were assessed for significance using Student's t-test, and differences in categorical variables were assessed using  $\chi^2$  or Fisher's exact test. Preoperative

and intraoperative variables were entered into a univariable logistic regression model to assess relationships between each variable and AKI. Covariates with a P-value < 0.10 were manually entered into a multivariable logistic regression model. In cases of intercorrelation, the best single independent variable was selected. For AKI predictors, adequate cutoff values were identified using a receiver operating characteristics (ROC) curve. Statistical analyses were performed using SPSS version 17.0 (IBM, Chicago, IL, USA), GraphPad Prism 7.0 (GraphPad Software, USA), and PASS 15.0 software. A P-value < 0.05 was considered statistically significant.

**Sample size calculation**

The incidence of AKI after mild hypothermic CPB in our institution was approximately 30%. Preliminary analysis indicated that minimum SmtO<sub>2</sub> was 43% in AKI patients and 53% in Non-AKI patients after cardiac surgery. A sample size of 172 was needed to achieve 80% power to detect a 10% difference using a two-side exact test with a significance level of 0.05.

**Results**

**Patient characteristics and perioperative data**

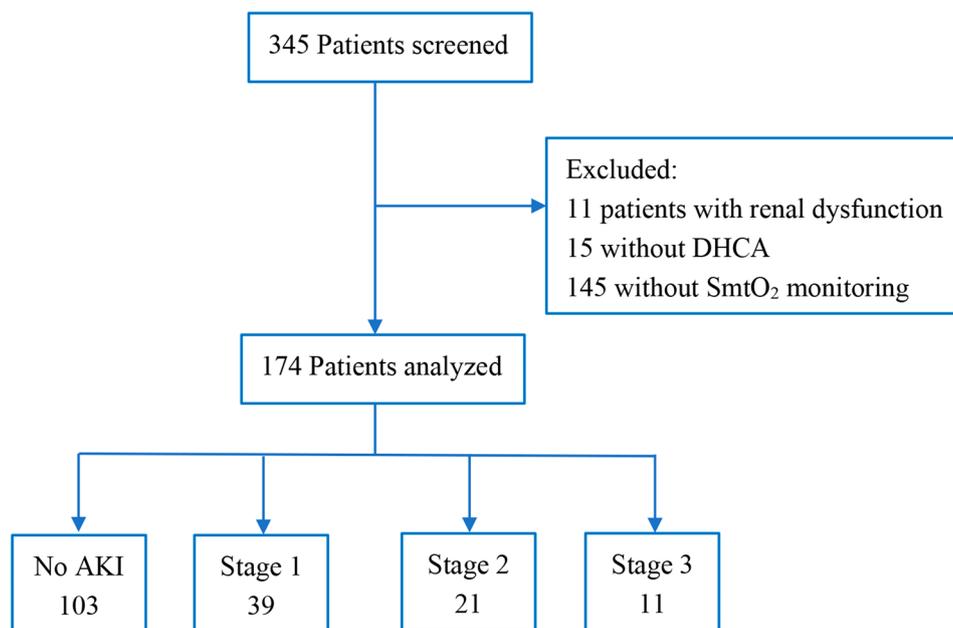
Ultimately, a total of 174 patients were enrolled in this study. Among them, 71 patients (40.80%) developed AKI, with the following distribution according to KDIGO criteria: Stage 1 (54.93%), Stage 2 (29.58%), and Stage 3 (15.49%). The detailed distribution of AKI stages is illustrated in Fig. 1. Additionally, 13 patients required renal replacement therapy (RRT). The preoperative profile

and intraoperative data of the two cohorts are listed in Table 1. There was no significant difference in preoperative status between the two cohorts. Regarding their intraoperative course, patients who developed AKI had longer CPB and DHCA times.

**Association between muscular tissue desaturation and AKI**

The minimum of SmtO<sub>2</sub> values were 48.12 ± 10.83% in patients with AKI and 47.59 ± 7.26% in those without AKI, with no significant difference (Table 2). Additionally, there were no significant differences in the durations of SmtO<sub>2</sub> < 55% and < 50% between the two cohorts. However, patients with AKI had a longer duration of SmtO<sub>2</sub> < 80% baseline (59.80 ± 74.58 min vs. 20.95 ± 34.09 min, P = 0.035). The duration of SmtO<sub>2</sub> < 80% baseline was associated with an increased risk of AKI (odds ratio [OR], 1.021; 95% CI, 1.001–1.041; P = 0.034) (Table 3). The durations of SmtO<sub>2</sub> < 85% baseline (OR, 1.009; 95% CI, 0.996–1.021; P = 0.195) and < 90% baseline (OR, 1.007; 95% CI, 0.996–1.018; P = 0.208) were not significantly associated with AKI.

The risk variables listed in Tables 1 and 2 with an explanatory P < 0.10 in the univariable analysis were tested with multivariable analysis. There was a significant correlation between CPB time and DHCA time. Considering that DHCA can cause severe tissue ischemia and hypoxia, DHCA time was included in the multivariable models. After correction for the other explanatory factors, DHCA time and the duration of SmtO<sub>2</sub> < 80% baseline remained significantly associated with AKI (Table 3; Fig. 2). To explore the capacity of SmtO<sub>2</sub> < 80% baseline



**Fig. 1** Patients with and without AKI according to Kidney Disease: Improving Global Outcomes (KDIGO) criteria. AKI, acute kidney injury; DHCA, deep hypothermic circulatory arrest; SmtO<sub>2</sub>, muscular tissue oxygen saturation

**Table 1** Preoperative and intraoperative characteristics of the patients with and without AKI

Variables	Non-AKI (n = 103)	AKI (n = 71)	P-value
Frequency, %	59.20	40.80	
<b>Baseline characteristics</b>			
Age (years)	45.05 ± 10.82	48.19 ± 11.72	0.299
Female	12 (11.65)	8 (11.27)	0.938
BMI (kg/m <sup>2</sup> )	27.08 ± 17.71	25.17 ± 3.15	0.413
NYHA classification 1–2/3–4	100/3	69/2	0.320
Creatinine (mmol/L)	113.30 ± 96.62	94.83 ± 63.44	0.184
BNP (pg/L)	1276.96 ± 948.43	1094.06 ± 2496.73	0.645
Hemoglobin (g/L)	120.50 ± 25.65	126.71 ± 22.76	0.124
<b>Preoperative medication, n (%)</b>			
β-blockers	33 (32.04)	21 (29.58)	0.730
CCB	37 (35.92)	26 (36.62)	0.925
ACEI	28 (27.18)	19 (26.76)	0.951
Current smoker, n (%)	44(42.72)	38(52.05)	0.161
Hypertension, n (%)	77 (74.76)	47 (66.20)	0.220
Diabetes, n (%)	35 (33.98)	19 (26.76)	0.312
EF < 50%, n (%)	23 (22.33)	15 (21.13)	0.850
<b>Intraoperative data</b>			
CPB time (min)	257.32 ± 63.23	293.41 ± 69.17	0.046*
Cross-clamp time (min)	174.87 ± 44.54	177.68 ± 55.54	0.809
DHCA time (min)	32.67 ± 9.12	37.62 ± 8.44	0.037*
Operation time (h)	7.33 ± 1.16	7.55 ± 1.35	0.447
<b>ACP</b>			
uACP, n (%)	88 (85.44)	62 (87.32)	0.723
biACP, n (%)	15 (14.56)	9 (12.68)	0.723
<b>Systemic perfusion</b>			
Trans-femoral artery, n (%)	70(67.96)	47 (66.20)	0.807
Right-side, n (%)	67(95.72)	46(97.69)	0.938
Left-side, n (%)	3(4.28)	1(2.13)	0.938
Trans-aorta, n (%)	6(5.83)	6 (8.45)	0.502
Trans-axillary artery, n (%)	27(26.21)	18 (25.35)	0.899
Packed RBC transfusion (ml)	142.69 ± 258.88	212.50 ± 333.09	0.147
Plasma transfusion (ml)	414.29 ± 130.73	437.50 ± 69.44	0.648
Platelet transfusion (ml)	366.67 ± 109.13	365.63 ± 83.99	0.741
Lowest nasopharyngeal temperature (°C)	27.05 ± 2.24	26.09 ± 1.95	0.103

**Table 1** (continued)

Variables	Non-AKI (n = 103)	AKI (n = 71)	P-value
Lowest rectal temperature (°C)	28.67 ± 1.91	27.90 ± 1.96	0.161
Lowest hemoglobin (g/L)	80.54 ± 10.96	81.33 ± 10.90	0.792

**Abbreviations:** AKI, acute kidney injury; BMI, body mass index; NYHA, New York Heart Association; BNP, brain natriuretic peptide; CCB, calcium channel blocker; ACEI, angiotensin converting enzyme inhibitor; EF, ejection fraction; CPB, cardiopulmonary bypass; DHCA, deep hypothermic circulatory arrest; uACP, unilateral antegrade cerebral perfusion; biACP, bilateral antegrade cerebral perfusion

in predicting AKI, a ROC curve was applied. The duration of SmtO<sub>2</sub> < 80% baseline had an area under the curve of 0.710 (95% CI, 0.563–0.857) with a cutoff value of 33.5 min (sensitivity, 70.00%; specificity, 77.80%) (Fig. 3).

**Association between cerebral desaturation and AKI**

The minimum left rScO<sub>2</sub> values were 54.79 ± 4.22% and 57.06 ± 5.29% in patients with AKI and those without, respectively. The minimum right rScO<sub>2</sub> values were 55.49 ± 5.59% and 57.05 ± 4.58% in patients with and without AKI, respectively (Table 2). The minimum left rScO<sub>2</sub> (OR, 1.096; 95% CI, 0.982–1.223; P=0.101) and right rScO<sub>2</sub> (OR, 1.066; 95% CI, 0.957–1.187; P=0.243) were not significantly associated with AKI. Moreover, the durations of both left and right rScO<sub>2</sub> < 55%, < 50%, and < 80% baseline, were also not associated with AKI (Table 3).

**Association between AKI and outcomes**

Patients with AKI experienced significantly worse outcomes compared with control cohort, including prolonged ICU stays, hospital stays, and mechanical ventilation times, along with a higher incidence of postoperative complications and in-hospital mortality (Table 4).

**Discussion**

AKI occurred in 40.08% of patients who underwent TAAR under CPB and DHCA. Muscular tissue desaturation, defined as SmtO<sub>2</sub> < 80% baseline monitored on the lower leg for more than 33.5 min, was significantly associated with an increased risk of AKI. Neither the minimum SmtO<sub>2</sub>, nor absolute values of SmtO<sub>2</sub> < 55% or < 50% were associated with postoperative AKI. The results of our study suggest that it may be reasonable to use SmtO<sub>2</sub> < 80% baseline as the threshold for muscular tissue desaturation in patients undergoing CPB and DHCA. Cerebral desaturation, defined as rScO<sub>2</sub> < 55%, < 50%, and < 80% baseline, was not associated with AKI.

Previous studies have indicated that SmtO<sub>2</sub> values below specific thresholds, such as SmtO<sub>2</sub> < 54.5% at the thenar muscle or SmtO<sub>2</sub> < 67% at the thigh in on-pump cardiac surgery patients, and SmtO<sub>2</sub> < 66% at the forearm

**Table 2** Intraoperative data of muscle and cerebral oxygen saturation

Variables	Non-AKI (n=103)	AKI (n=71)	P-value
Left rScO <sub>2</sub> monitoring time (min)	517.96 ± 113.97	568.90 ± 116.30	0.094
Right rScO <sub>2</sub> monitoring time (min)	513.61 ± 124.24	564.55 ± 131.11	0.132
Baseline left rScO <sub>2</sub> (%)	61.79 ± 3.47	61.07 ± 4.16	0.483
Baseline right rScO <sub>2</sub> (%)	61.45 ± 5.16	60.31 ± 4.79	0.377
Minimum left rScO <sub>2</sub> (%)	57.06 ± 5.29	54.79 ± 4.22	0.097
Minimum right rScO <sub>2</sub> (%)	57.05 ± 4.58	55.49 ± 5.59	0.242
Mean left rScO <sub>2</sub> (%)	60.08 ± 4.73	58.15 ± 3.11	0.103
Mean right rScO <sub>2</sub> (%)	60.16 ± 4.48	58.32 ± 4.65	0.136
Left rScO <sub>2</sub> < 55% duration (min)	55.39 ± 80.74	50.33 ± 76.81	0.804
Right rScO <sub>2</sub> < 55% duration (min)	68.74 ± 127.42	43.93 ± 65.38	0.302
Left rScO <sub>2</sub> < 50% duration (min)	3.26 ± 5.19	7.98 ± 22.77	0.333
Right rScO <sub>2</sub> < 50% duration (min)	25.48 ± 69.07	4.45 ± 8.54	0.054
Left rScO <sub>2</sub> < 80% baseline duration (min)	4.30 ± 9.63	3.93 ± 12.09	0.898
Right rScO <sub>2</sub> < 80% baseline duration (min)	4.61 ± 15.17	1.52 ± 2.30	0.208
SmtO <sub>2</sub> monitoring time (min)	408.89 ± 180.22	477.65 ± 197.11	0.215
Baseline SmtO <sub>2</sub> (%)	64.96 ± 7.41	64.45 ± 5.83	0.784
Minimum SmtO <sub>2</sub> (%)	47.59 ± 7.26	48.12 ± 10.83	0.866
Mean SmtO <sub>2</sub> (%)	52.53 ± 7.26	52.15 ± 10.15	0.898
SmtO <sub>2</sub> < 55% duration (min)	23.05 ± 26.17	63.17 ± 94.16	0.076
SmtO <sub>2</sub> < 50% duration (min)	14.11 ± 25.85	40.94 ± 74.94	0.138
< 90% baseline SmtO <sub>2</sub> duration (min)	175.91 ± 45.18	194.31 ± 50.46	0.057
< 85% baseline SmtO <sub>2</sub> duration (min)	84.67 ± 36.22	103.64 ± 49.74	0.239
< 80% baseline SmtO <sub>2</sub> duration (min)	20.95 ± 34.09	59.80 ± 74.58	0.035*

**Abbreviations:** rScO<sub>2</sub>, regional cerebral oxygen saturation; SmtO<sub>2</sub>, muscular tissue oxygen saturation

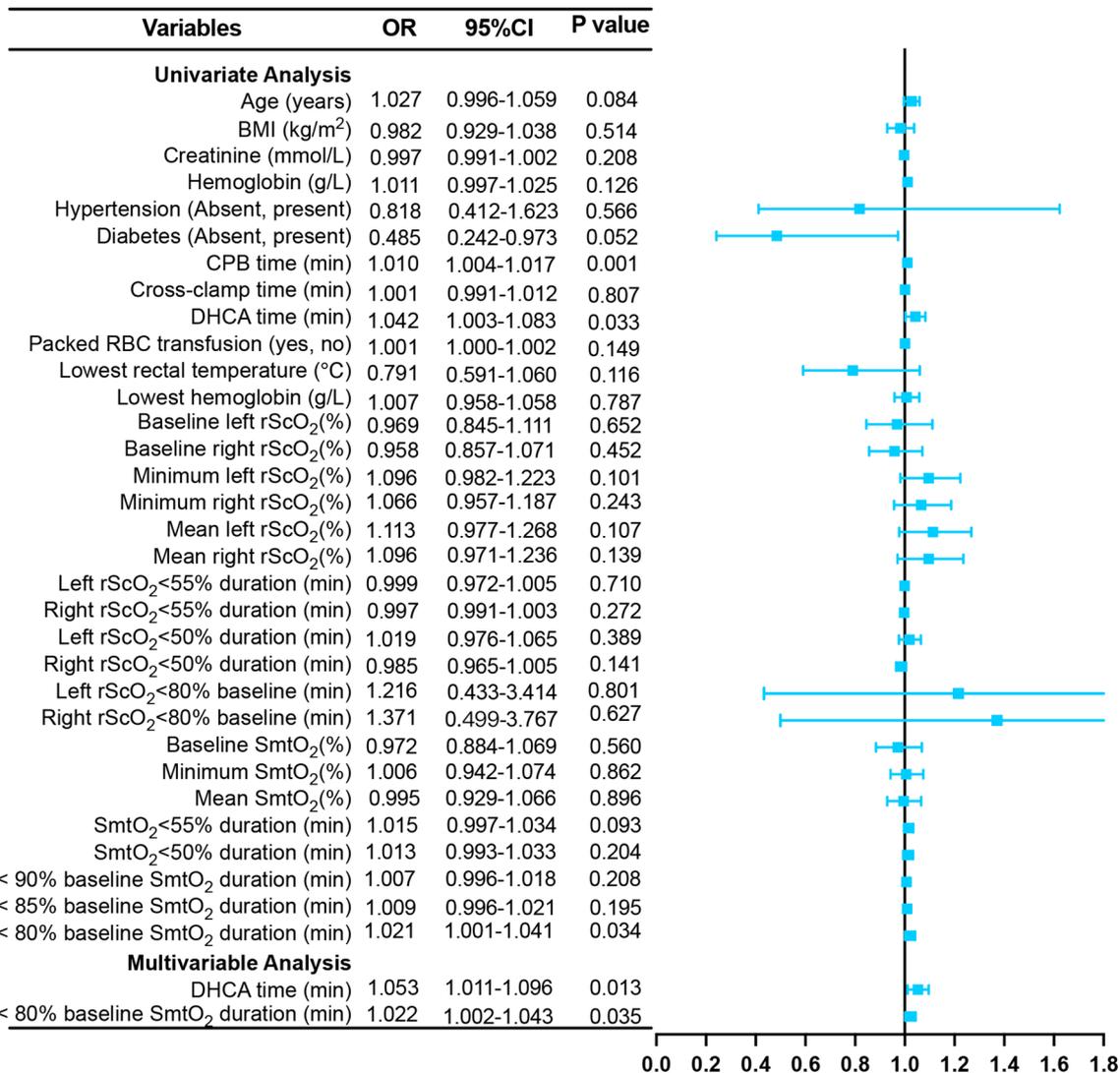
in liver transplantation patients, are associated with the development of postoperative AKI [3, 21, 27]. However, it is widely known that baseline SmtO<sub>2</sub> varies significantly among individuals. Furthermore, different instrument manufacturers may provide different readings even when sensors are placed in the same location, at the same time, and on the same patient, owing to varying designs and algorithms [12]. Therefore, thresholds based on relative changes may be more suitable for defining muscular tissue desaturation than thresholds based on absolute values. Another study suggests that SmtO<sub>2</sub> < 90% baseline monitored at the quadriceps is significantly associated with an increased risk of AKI (OR = 2.84; 95%CI,

**Table 3** Univariable and multivariable analysis for predictors of acute kidney injury

Variables	Odds Ratio	95% Confidence Interval	P value
<b>Univariate Analysis</b>			
Age (years)	1.027	0.996–1.059	0.084
BMI (kg/m <sup>2</sup> )	0.982	0.929–1.038	0.514
NYHA classification > 2	0.804	0.143–4.504	0.804
Creatinine (mmol/L)	0.997	0.991–1.002	0.208
BNP (pg/L)	2.250	0.801–6.321	0.124
Hemoglobin (g/L)	1.011	0.997–1.025	0.126
Hypertension (Absent, present)	0.818	0.412–1.623	0.566
Diabetes (Absent, present)	0.485	0.242–0.973	0.052
EF < 50% (Absent, present)	3.488	0.888–13.711	0.074
CPB time (min)	1.010	1.004–1.017	0.001*
Cross-clamp time (min)	1.001	0.991–1.012	0.807
DHCA time (min)	1.042	1.003–1.083	0.033*
Packed RBC transfusion (yes, no)	1.001	1.000–1.002	0.149
Lowest rectal temperature (°C)	0.791	0.591–1.060	0.116
Lowest hemoglobin (g/L)	1.007	0.958–1.058	0.787
Baseline left rScO <sub>2</sub> (%)	0.969	0.845–1.111	0.652
Baseline right rScO <sub>2</sub> (%)	0.958	0.857–1.071	0.452
Minimum left rScO <sub>2</sub> (%)	1.096	0.982–1.223	0.101
Minimum right rScO <sub>2</sub> (%)	1.066	0.957–1.187	0.243
Mean left rScO <sub>2</sub> (%)	1.113	0.977–1.268	0.107
Mean right rScO <sub>2</sub> (%)	1.096	0.971–1.236	0.139
Left rScO <sub>2</sub> < 55% duration (min)	0.999	0.992–1.005	0.710
Right rScO <sub>2</sub> < 55% duration (min)	0.997	0.991–1.003	0.272
Left rScO <sub>2</sub> < 50% duration (min)	1.019	0.976–1.065	0.389
Right rScO <sub>2</sub> < 50% duration (min)	0.985	0.965–1.005	0.141
Left rScO <sub>2</sub> < 80% baseline duration (min)	1.216	0.433–3.414	0.801
Right rScO <sub>2</sub> < 80% baseline duration (min)	1.371	0.499–3.767	0.627
Baseline SmtO <sub>2</sub> (%)	0.972	0.884–1.069	0.560
Minimum SmtO <sub>2</sub> (%)	1.006	0.942–1.074	0.862
Mean SmtO <sub>2</sub> (%)	0.995	0.929–1.066	0.896
SmtO <sub>2</sub> < 55% duration (min)	1.015	0.997–1.034	0.093
SmtO <sub>2</sub> < 50% duration (min)	1.013	0.993–1.033	0.204
< 90% baseline SmtO <sub>2</sub> duration (min)	1.007	0.996–1.018	0.208
< 85% baseline SmtO <sub>2</sub> duration (min)	1.009	0.996–1.021	0.195
< 80% baseline SmtO <sub>2</sub> duration (min)	1.021	1.001–1.041	0.034*
<b>Multivariable Analysis</b>			
DHCA time (min)	1.053	1.011–1.096	0.013*
< 80% baseline SmtO <sub>2</sub> duration (min)	1.022	1.002–1.043	0.035*

**Abbreviations:** BMI, body mass index; NYHA, New York Heart Association; BNP, brain natriuretic peptide; EF, ejection fraction; CPB, cardiopulmonary bypass; DHCA, deep hypothermic circulatory arrest; RBC, red blood cell; rScO<sub>2</sub>, regional cerebral oxygen saturation; SmtO<sub>2</sub>, muscular tissue oxygen saturation

1.21–6.67; P = 0.016) in major abdominal surgery [20]. Because only one threshold was investigated in this study, the association of other potential thresholds with AKI could not be determined. In comparison, multiple thresholds, including relative changes and absolute values, were explored in our study. Our results also demonstrated that

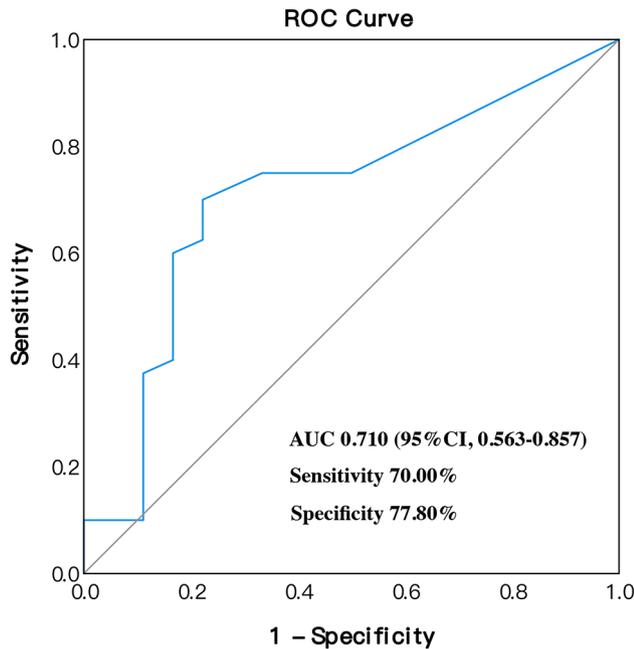


**Fig. 2** Univariable and Multivariable Analysis for association between risk factors and AKI. AKI, acute kidney injury; OR, odds ratio; CI, confidence interval; BMI, body mass index; CPB, cardiopulmonary bypass; DHCA, deep hypothermic circulatory arrest; RBC, red blood cell; rScO<sub>2</sub>, regional cerebral oxygen saturation; SmtO<sub>2</sub>, muscular tissue oxygen saturation

relative change in SmtO<sub>2</sub> was significantly associated with postoperative AKI in patients undergoing CPB and DHCA.

The goal of monitoring is to facilitate clinical decision-making and improve patient outcomes. It is important to understand how monitoring correlates with the outcomes that matter to patients. SmtO<sub>2</sub> monitored on the thenar eminence has been associated with a composite of major complications and mortality after major non-cardiac surgery, as well as failure of weaning from mechanical ventilation [28, 29]. Low SmtO<sub>2</sub> monitored on forearm is independently associated with mortality in patients with community-acquired pneumonia [30]. Care guided by forearm SmtO<sub>2</sub> monitoring has been shown to significantly reduce the incidence of 24-hour postoperative nausea and vomiting in obese patients undergoing

laparoscopic hysterectomy [31]. In our study, prolonged SmtO<sub>2</sub><80% baseline was associated with an increased risk of AKI, with a cutoff value of 33.5 min. Absolute values of low SmtO<sub>2</sub>, such as SmtO<sub>2</sub><55%, SmtO<sub>2</sub><50%, the minimum value, and the averaged value, were not associated with AKI. These findings align with the fact that baseline SmtO<sub>2</sub> and oximeters readings vary among individuals. Additionally, the threshold for muscular desaturation was lower than that in previous studies (<80% baseline vs. <90% baseline), which may be due to the involvement of lower-body circulatory arrest in our patient population. Our results indicate that improving systemic oxygen delivery to maintain SmtO<sub>2</sub>>80% baseline, particularly during DHCA by combining cerebral perfusion with retrograde inferior vena cava perfusion, may reduce the risk of postoperative AKI [32]. However,



**Fig. 3** Receiver operating characteristic curve for the duration of muscular tissue oxygen saturation ( $SmtO_2 < 80\%$  baseline) identified as a predictor for AKI.  $SmtO_2$ , muscular tissue oxygen saturation; AKI, acute kidney injury; AUC, area under the curve; CI, confidence interval

**Table 4** Postoperative outcomes according to the presence of AKI

Variables	Non-AKI (n=103)	AKI (n=71)	P-value
ICU stay, (d)	4.31 ± 2.85	6.00 ± 2.81	0.017*
Hospital stay, (d)	15.38 ± 5.64	21.95 ± 8.68	< 0.001*
Mechanical ventilation > 24 h, n (%)	44 (40.00)	42 (65.63)	0.033*
Systolic heart failure, n (%)	24 (21.82)	28 (43.75)	0.022*
Delirium, n (%)	23 (20.91)	38 (59.38)	< 0.001*
Postoperative stroke			
Ischemic, n (%)	17(16.50)	17 (23.94)	0.224
Hemorrhage, n (%)	4 (3.88)	3 (4.22)	0.910
Lung infection, n (%)	35 (33.98)	37 (52.11)	0.017*
Re-operation for bleeding, n (%)	6 (5.83)	10 (14.08)	0.064
In-hospital death, n (%)	10 (9.71)	15 (21.12)	0.010*

Abbreviations: AKI, acute kidney injury; ICU, intensive care unit; h, hour; d, day

further randomized controlled trials are needed, as our study cannot establish a causal relationship between muscular tissue desaturation and postoperative AKI.

It is well established that  $SmtO_2$  levels can differ across various anatomic sites, primarily due to variations in muscle metabolism and perfusion. The anatomical proximity of renal artery to the iliac artery may lead to a similar distribution of blood flow between the legs and kidneys when cardiac output is insufficient. During DHCA, both the kidneys and legs are in a state of ischemia, while the forearm may receive partial blood via cerebral perfusion cannulation. However, in our cohort,

31.28% of patients underwent femoral artery cannulation, which could potentially affect the perfusion of the ipsilateral lower limb and subsequently influence  $SmtO_2$ , even in the absence of difference in femoral artery cannulation between the two cohorts. This may explain the relatively low OR (OR=1.021; 95% CI, 1.001–1.041) associated with duration of  $SmtO_2 < 80\%$  baseline in our study. Further investigation is warranted to determine whether  $SmtO_2$  measurements obtained from the contralateral lower limb during femoral artery cannulation correlated more closely with the incidence of postoperative AKI.

Total aortic arch replacement under CPB and DHCA is a high-risk surgery with numerous complications. Renal ischemia, reperfusion, inflammation, deep hypothermia, massive hemorrhage, and transfusion of red blood cells, fresh frozen plasma, platelets, and cell saver blood pose serious threats to organ perfusion and tissue oxygen delivery in this type of surgery [7, 33–35]. Although muscular tissue desaturation was a predictor of AKI in our study, its predictive value in different surgical patient populations and procedures remains to be clarified.

Our study did not find an association between cerebral desaturation and postoperative AKI, which is consistent with previous studies [15]. This may be due to the brain's strong capacity for autoregulation compared to kidney and muscles, which resists ischemia and hypoxia [36, 37]. Moreover, as a vital organ, the brain is prioritized during hemodynamic disturbances. Therefore, low  $rScO_2$  may be more associated with neurological deficit [13, 25].

In our study, patients with AKI had an increased risk of prolonged ICU stays, mechanical ventilation > 24 h, systolic heart failure, postoperative delirium, and in-hospital mortality. These findings are consistent with previous studies on AKI after cardiac surgery [38, 39]. Although there was no significant difference in postoperative stroke between patients with and without AKI, this may be due to the fact that cerebral CT examinations were not routinely performed for all patients after surgery. Re-operation due to bleeding is more likely associated with surgical procedure. These findings not only confirmed that AKI carries poor prognosis but may also indicate that AKI is a manifestation of systemic organ injury, underscoring the need for comprehensive monitoring and regulation of systemic oxygen delivery and consumption.

This study has several limitations. First, it is a single-center retrospective observational study with a limited sample size, which restricts the ability to determine the causal effect of muscular tissue desaturation on AKI. Whether there are effective interventions to correct muscular tissue desaturation and whether care guided by  $SmtO_2$  monitoring can decrease the risk of AKI cannot be determined by this study. Second, postoperative AKI

has complex determinants, and it is likely that potential confounding variables were not included in the analysis. As a result, the multivariable analysis model may have overestimated the association between muscular desaturation and AKI in this study. Third, in our study, SmtO<sub>2</sub> was measured in the lower leg, which differs from previous studies that measured SmtO<sub>2</sub> in the forearm or thenar eminence. During DHCA, perfusion of the arm tissue may be affected by ACP, whereas the lower leg is not influenced by this factor. However, it is important to note that right femoral artery cannulation may affect SmtO<sub>2</sub> measurements. Nevertheless, our analysis showed no statistically significant difference in the distribution of right femoral artery cannulation between the non-AKI and AKI groups. Fourth, the baseline measurement was obtained after anesthesia induction, which may reduce cardiac performance, tissue perfusion, and oxygenation. Fifth, our results were obtained from a specific group of surgical patients, so we do not know whether our findings can be generalized to other patient populations. Finally, we do not know whether our findings can be generalized to different tissue oximeters.

## Conclusion

In patients undergoing total aortic arch replacement under CPB and DHCA, 40.08% experienced AKI, which was linked to relatively poorer outcomes. Prolonged SmtO<sub>2</sub> < 80% baseline was associated with an increased risk of AKI, whereas no significant correlation was observed between cerebral desaturation and AKI. Further randomized controlled trials are warranted to explore whether maintaining SmtO<sub>2</sub> above 80% baseline may reduce the risk of postoperative AKI and enhance patient outcomes.

## Abbreviations

AKI	Acute Kidney Injury
CPB	Cardiopulmonary Bypass
TAAR	Total Aortic Arch Replacement
DHCA	Deep Hypothermic Circulatory Arrest
ICU	Intensive Care Unit
NIRS	Near-Infrared Spectroscopy
SmtO <sub>2</sub>	Muscle Tissue Oxygen Saturation
SpO <sub>2</sub>	Pulse Oxygen Saturation
EtCO <sub>2</sub>	End-Tidal Carbon Dioxide
S $\bar{v}$ O <sub>2</sub>	Mixed Venous Oxygen Saturation
ACP	Antegrade Cerebral Perfusion
rScO <sub>2</sub>	regional Cerebral Oxygen Saturation
KDIGO	Kidney Disease Improving Global Outcomes
CAM	Confusion Assessment Method
CT	Computed Tomography
EF	Ejection Fraction
SD	Standard Deviation
ROC	Receiver Operating Characteristics
OR	Odds Ratio
CI	Confidence Interval

## Author contributions

LZ, LP, QH and WW have given substantial contributions to the conception or the design of the manuscript, LZ, LP, and QH to acquisition, analysis and

interpretation of the data. All authors participated to draft the manuscript, LZ, LP, and WW revised it critically. All authors read and approved the manuscript.

## Data availability

Data is provided within the manuscript and available from the corresponding author on reasonable request.

## Declarations

### Ethical approval and consent to participate

The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any of the work are appropriately investigated and resolved. This study was approved by the Ethics Committee of West China Hospital of Sichuan University (NO. 2023455, Chengdu, China). Written informed consent was waived due to the retrospective nature of the analysis. All procedures involving human participants were conducted in accordance with the Declaration of Helsinki.

### Consent for publication

Not applicable.

### Competing interests

The authors declare no competing interests.

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## References

- Lannemyr L, Bragadottir G, Krumbholz V, Redfors B, Sellgren J, Ricksten SE. Effects of cardiopulmonary bypass on renal perfusion, filtration, and oxygenation in patients undergoing cardiac surgery. *Anesthesiology*. 2017;126(2):205–13.
- Zhang Y, Wang B, Zhou XJ, Guo LJ, Zhou RH. Nadir oxygen delivery during pediatric bypass as a predictor of acute kidney injury. *Ann Thorac Surg*. 2022;113(2):647–53.
- Szymanowicz W, Daniłowicz-Szymanowicz L, Karolak W, Kowalik MM, Lango R. Brain and muscle oxygen saturation combined with kidney injury biomarkers predict cardiac surgery related acute kidney injury. *Diagnostics (Basel)*. 2021;11(9):1591.
- Helgason D, Helgadóttir S, Ahlsson A, Gunn J, Hjortdal V, Hansson EC, et al. Acute kidney injury after acute repair of type A aortic dissection. *Ann Thorac Surg*. 2021;111(4):1292–8.
- Li L, Zhou J, Hao X, Zhang W, Yu D, Xie Y, et al. The incidence, risk factors and In-Hospital mortality of acute kidney injury in patients after surgery for acute type A aortic dissection: A Single-Center retrospective analysis of 335 patients. *Front Med (Lausanne)*. 2020;7:557044.
- Sheng W, Xia W, Niu Z, Yang H. Incidence of acute kidney injury and risk factors of prognosis in patients with acute Stanford type A aortic dissection. *Ann Thorac Cardiovasc Surg*. 2023;29(5):249–55.
- Li CN, Ge YP, Liu H, Zhang CH, Zhong YL, Chen SW, et al. Blood transfusion and acute kidney injury after total aortic arch replacement for acute Stanford type A aortic dissection. *Heart Lung Circ*. 2022;31(1):136–43.
- Wang J, Yu W, Zhai G, Liu N, Sun L, Zhu J. Independent risk factors for postoperative AKI and the impact of the AKI on 30-day postoperative outcomes in patients with type A acute aortic dissection: an updated meta-analysis and meta-regression. *J Thorac Dis*. 2018;10(5):2590–8.
- Arnaoutakis GJ, Ogami T, Patel HJ, Pai CW, Woznicki EM, Brinster DR, et al. Acute kidney injury in patients undergoing surgery for type A acute aortic dissection. *Ann Thorac Surg*. 2023;115(4):879–85.
- Bellomo R, Auremma S, Fabbri A, D'Onofrio A, Katz N, McCullough PA, et al. The pathophysiology of cardiac surgery-associated acute kidney injury (CSA-AKI). *Int J Artif Organs*. 2008;31(2):166–78.

11. Hallqvist L, Granath F, Hult E, Bell M. Intraoperative hypotension is associated with acute kidney injury in noncardiac surgery: an observational study. *Eur J Anaesthesiol*. 2018;35(4):273–9.
12. Bickler P, Feiner J, Rollins M, Meng L. Tissue oximetry and clinical outcomes. *Anesth Analg*. 2017;124(1):72–82.
13. Cui F, Zhao W, Mu DL, Zhao X, Li XY, Wang DX, et al. Association between cerebral desaturation and postoperative delirium in thoracotomy with One-Lung ventilation: A prospective cohort study. *Anesth Analg*. 2021;133(1):176–86.
14. Zhao X, Xiao H, Cai J, Meng L. Double standards: why is pulse oximetry standard care, whereas tissue oximetry is not? *Curr Opin Anaesthesiol*. 2020;33(5):619–25.
15. Meng L, Xiao J, Gudelunas K, Yu Z, Zhong Z, Hu X. Association of intraoperative cerebral and muscular tissue oxygen saturation with postoperative complications and length of hospital stay after major spine surgery: an observational study. *Br J Anaesth*. 2017;118(4):551–62.
16. Ortmann LA, Fontenet EE, Seib PM, Eble BK, Brown R, Bhutta AT. Use of near-infrared spectroscopy for Estimation of renal oxygenation in children with heart disease. *Pediatr Cardiol*. 2011;32(6):748–53.
17. Zhang D, Ouyang C, Zhao X, Cui B, Dai F, Meng L, et al. Renal tissue desaturation and acute kidney injury in infant cardiac surgery: a prospective propensity score-matched cohort study. *Br J Anaesth*. 2021;127(4):620–8.
18. Owens GE, King K, Gurney JG, Charpie JR. Low renal oximetry correlates with acute kidney injury after infant cardiac surgery. *Pediatr Cardiol*. 2011;32(2):183–8.
19. Ruf B, Bonelli V, Balling G, Hörer J, Nagdyman N, Braun SL, et al. Intraoperative renal near-infrared spectroscopy indicates developing acute kidney injury in infants undergoing cardiac surgery with cardiopulmonary bypass: a case-control study. *Crit Care*. 2015;19(1):27.
20. Yin L, Wang C, Zhao W, Yang X, Guo Y, Mu D, et al. Association between muscular tissue desaturation and acute kidney injury in older patients undergoing major abdominal surgery: a prospective cohort study. *J Anesth*. 2024;38(4):434–44.
21. Sakaki K, Kitamura T, Kohira S, Torii S, Mishima T, Hanayama N, Kobayashi K, et al. Regional thigh tissue oxygen saturation during cardiopulmonary bypass predicts acute kidney injury after cardiac surgery. *J Artif Organs*. 2020;23(4):315–20.
22. Zhang H, Zhang T, Hou L, Zhao J, Fan Q, Wang L, Lu Z, et al. Association of intraoperative cerebral and somatic tissue oxygen saturation with postoperative acute kidney injury in adult patients undergoing multiple valve surgery. *BMC Anesthesiol*. 2023;23(1):319.
23. Ikeda K, MacLeod DB, Grocott HP, Moretti EW, Ames W, Vacchiano C. The accuracy of a near-infrared spectroscopy cerebral oximetry device and its potential value for estimating jugular venous oxygen saturation. *Anesth Analg*. 2014;119(6):1381–92.
24. Peng L, Guo D, Shi Y, Yang J, Wei W. The incidence, risk factors and outcomes of impaired cerebral autoregulation in aortic arch surgery: a single-center, retrospective cohort study. *J Cardiothorac Surg*. 2023;18(1):312.
25. Eertmans W, De Deyne C, Genbrugge C, Marcus B, Bouneb S, Beran M, et al. Association between oxygen desaturation in older patients after cardiac surgery. *Br J Anaesth*. 2020;124(2):146–53.
26. Yu Y, Wu H, Liu C, Zhang C, Song Y, Ma Y, et al. Intraoperative renal desaturation and postoperative acute kidney injury in older patients undergoing liver resection: A prospective cohort study. *J Clin Anesth*. 2023;87:111084.
27. Hong SH, Chae MS. Clinical application of intraoperative somatic tissue oxygen saturation for detecting postoperative early kidney dysfunction patients undergoing living donor liver transplantation: A propensity score matching analysis. *PLoS ONE*. 2022;17(1):e0262847.
28. Abdelmalak BB, Cata JP, Bonilla A, You J, Kopyeva T, Vogel JD, et al. Intraoperative tissue oxygenation and postoperative outcomes after major non-cardiac surgery: an observational study. *Br J Anaesth*. 2013;110(2):241–9.
29. Poriazis M, Kontogiorgi M, Angelopoulos E, Vasileiadis I, Tripodaki ES, Nanou V, et al. Changes in Thenar muscle tissue oxygen saturation assessed by near-infrared spectroscopy during weaning from mechanical ventilation. *Minerva Anesthesiol*. 2014;80(6):666–75.
30. Claverias L, Mari M, Marín-Corral J, Magret M, Trefler S, Bodí M, et al. The prognostic value of muscle regional oxygen saturation index in severe community-acquired pneumonia: a prospective observational study. *J Intensive Care*. 2016;4:7.
31. Li G, Tian DD, Wang X, Feng X, Zhang W, Bao J, et al. Muscular tissue oxygen saturation and posthysterectomy nausea and vomiting: the iMODIPONV randomized controlled trial. *Anesthesiology*. 2020;133(2):318–31.
32. Lin J, Xiong J, Luo M, Tan Z, Wu Z, Guo Y, et al. Combining cerebral perfusion with retrograde inferior Vena caval perfusion for aortic arch surgery. *Ann Thorac Surg*. 2019;107(1):e67–9.
33. Ge YP, Li CN, Li Y, Zhu JM, Liu YM, Zheng J, et al. Relationship between renal function and renal artery involvement in acute debakey type I aortic dissection. *Heart Surg Forum*. 2020;23(4):E465–9.
34. Luo CC, Zhong YL, Qiao ZY, Li CN, Liu YM, Zheng J, et al. Development and validation of a nomogram for postoperative severe acute kidney injury in acute type A aortic dissection. *J Geriatr Cardiol*. 2022;19(10):734–42.
35. Sun L, Qi R, Zhu J, Liu Y, Chang Q, Zheng J. Repair of acute type A dissection: our experiences and results. *Ann Thorac Surg*. 2011;91(4):1147–52.
36. Ono M, Brown C, Lee JK, Gottesman RF, Kraut M, Black J, et al. Cerebral blood flow autoregulation is preserved after hypothermic circulatory arrest. *Ann Thorac Surg*. 2013;96(6):2045.
37. Ono M, Joshi B, Brady K, Easley RB, Zheng Y, Brown C, et al. Risks for impaired cerebral autoregulation during cardiopulmonary bypass and postoperative stroke. *Br J Anaesth*. 2012;109(3):391–8.
38. Lau D, Pannu N, James MT, Hemmelgarn BR, Kieser TM, Meyer SR, et al. Costs and consequences of acute kidney injury after cardiac surgery: A cohort study. *J Thorac Cardiovasc Surg*. 2021;162(3):880–7.
39. Lysak N, Bihorac A, Hobson C. Mortality and cost of acute and chronic kidney disease after cardiac surgery. *Curr Opin Anaesthesiol*. 2017;30(1):113–7.

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