

Cerebral Oxygenation-Guided Indexed-Flow Normothermic Cardiopulmonary Bypass with Vasopressor-Sparing Pressure Management: A Retrospective Descriptive Cohort Study

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ABSTRACT

Context: Mean arterial pressure targets during cardiopulmonary bypass remain debated; vasopressor-driven pressure augmentation may not improve oxygen delivery.

Aims: The aim of the paper is to describe clinical outcomes and physiologic markers during normothermic cardiopulmonary bypass managed with indexed flow, cerebral oximetry, calculated indexed oxygen delivery (DO_2i), and vasopressor-sparing pressure management.

Settings and Design: Single-center retrospective descriptive cohort study at a cardiac surgery center.

Material and Methods: Consecutive adults undergoing elective or urgent cardiac surgery with normothermic cardiopulmonary bypass from June 2023 to December 2025 were included if complete anesthesia-perfusion, regional cerebral oxygen saturation (rSO_2), circuit-derived venous oxygen saturation (SvO_2), DO_2i , and early outcome data were available. Circulatory arrest was excluded. Indexed pump flow was 2.2–2.4 L/min/m. Mean arterial pressure was interpreted with indexed flow, rSO_2 , SvO_2 , DO_2i , hemoglobin, acid-base status, venous return, and surgical context. Baseline rSO_2 was recorded before ventilation on room air; maximal relative decrease during bypass was recorded.

Statistical Analysis Used: Descriptive statistics and Wilson 95% confidence intervals were used. No comparative testing or causal modeling was performed.

Results: Two hundred patients were analyzed. Operating-room extubation was achieved in 172 patients (86.0%). Stroke occurred in six (3.0%), acute kidney injury in two (1.0%), and venoarterial extracorporeal membrane oxygenation was required in six (3.0%). Baseline right and left rSO_2 were $57.5 \pm 11.2\%$ and $58.5 \pm 11.3\%$; maximal relative rSO_2 decrease was $28.5 \pm 3.4\%$; circuit-derived SvO_2 was $67.4 \pm 5.9\%$; and calculated DO_2i was $611 \pm 74 \text{ mL/min/m}$.

Conclusions: This flow-oriented, cerebral oximetry-guided, oxygen-delivery-aware strategy was accompanied by favorable early recovery. Because the study was retrospective and noncomparative, the findings are hypothesis-generating.

Keywords: Cardiac anesthesia, cardiopulmonary bypass; cerebral oximetry, DO_2i , mean arterial pressure, oxygen delivery, perfusion strategy, vasopressors

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INTRODUCTION

Cardiopulmonary bypass (CPB) remains central to adult cardiac surgery, but optimal mean arterial pressure (MAP)

targets during bypass are still debated. Conventional management often favors empiric pressure thresholds

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supported by vasopressors, although evidence for higher pressure targets is inconsistent.^[1-6] Patient-specific cerebral autoregulation varies substantially, and a universal MAP target may not reflect individual neurologic risk.^[7-13]

Tissue and renal perfusion during CPB also depend on indexed flow, arterial oxygen content, oxygen delivery, vascular resistance, and microcirculatory integrity.^[14-25] Vasopressors can increase MAP by raising systemic vascular resistance, but this mechanism does not necessarily improve regional oxygen balance or microvascular perfusion. Therefore, MAP may be more clinically meaningful when interpreted together with flow, oxygen delivery, cerebral oxygenation, venous oxygen saturation, and acid–base status.

We therefore revised and report a single-center retrospective descriptive cohort of adult cardiac surgical patients managed with a normothermic, indexed-flow, cerebral oxygenation-guided, oxygen-delivery-aware, and vasopressor-sparing institutional CPB strategy. The aim was to describe early clinical outcomes and intraoperative physiologic perfusion markers rather than to establish causal safety or superiority.

MATERIAL AND METHODS

Study design and setting

This single-center, retrospective, descriptive cohort study used anonymized routine clinical, anesthesia, and perfusion records from adult patients undergoing cardiac surgery with normothermic CPB between June 2023 and December 2025. The study was conducted in accordance with the Declaration of Helsinki. According to institutional policy, retrospective analyses of fully anonymized routine-care data do not require prospective ethics committee review; therefore, no approval number was issued. Consent for participation was not required because the analysis used anonymized retrospective data. Reporting was prepared with reference to the strengthening the reporting of observational studies in epidemiology (STROBE) guidance for observational studies.^[26]

The study was designed to describe an institutional perfusion strategy and was not intended to test superiority, noninferiority, or causal effectiveness.

Participants

Consecutive patients aged 18 years or older were eligible if they underwent elective or urgent adult cardiac surgery requiring CPB and had complete intraoperative anesthesia–perfusion documentation, cerebral oximetry records, circuit-derived venous oxygen saturation, calculated oxygen

delivery data, and available early postoperative outcome data. Patients younger than 18 years, operations performed without CPB, procedures involving circulatory arrest, and cases with incomplete intraoperative documentation or missing key outcome data were excluded.

CPB equipment and conduct

CPB was performed using a Stockert S5 heart-lung machine. Normothermic CPB was conducted according to a unified institutional strategy. The target indexed pump flow was 2.2–2.4 L/min/m². Pump flow was indexed to body surface area and interpreted together with venous return, circuit performance, arterial line pressure, hemoglobin concentration, gas exchange, acid–base status, cerebral oxygenation, circuit-derived venous oxygen saturation, calculated indexed oxygen delivery (DO_{2i}), and surgical context.

Pressure and vasopressor-sparing management

MAP was not managed as an isolated therapeutic endpoint. During CPB, MAP was interpreted in relation to preserved indexed pump flow, bilateral cerebral regional cerebral oxygen saturation (rSO₂), maximal rSO₂ decrease from baseline, circuit-derived venous oxygen saturation, calculated DO_{2i}, hemoglobin concentration, arterial blood gas values, acid–base status, venous return, circuit performance, and surgical conditions.

MAP values within 40–60 mmHg were generally accepted when indexed pump flow was preserved, and there was no evidence of impaired systemic or regional oxygen balance. Routine vasopressor administration during CPB was not part of the institutional strategy. However, vasopressor avoidance was not absolute, and vasoactive therapy remained available if hypotension was persistent, if MAP was considered inadequate in relation to the broader physiologic context, or if there was concern for impaired systemic or regional perfusion.

When MAP exceeded 60 mmHg with preserved indexed flow, nitroglycerin boluses were administered according to local practice to reduce systemic vascular resistance.

Cerebral oximetry and circuit-derived venous oxygen saturation

Bilateral cerebral near-infrared spectroscopy was performed using a Somanetics cerebral oximetry system. Baseline right and left rSO₂ values were recorded before initiation of mechanical ventilation while the patient was breathing room air. During CPB, the maximal relative decrease in rSO₂ from this pre-ventilation room-air baseline was recorded.

Venous oxygen saturation was assessed from the venous sampling line of the CPB circuit and recorded as SvO₂. Because venous oxygen saturation was obtained from the CPB circuit rather than from a pulmonary artery catheter, it was interpreted as a circuit-derived venous oxygenation marker during CPB.

Indexed oxygen delivery assessment

Oxygen delivery was calculated using hemoglobin concentration, arterial oxygen saturation, arterial oxygen tension, and pump flow. The calculated oxygen delivery value was subsequently indexed to body surface area and expressed as DO_{2i}, in mL/min/m².

DO_{2i} was calculated according to the following relationship: $DO_{2i} = 10 \times CI \times CaO_2$, where confidence interval (CI) represents indexed pump flow in L/min/m² and CaO₂ represents arterial oxygen content. Arterial oxygen content was calculated as: $CaO_2 = (Hb \times 1.36 \times SaO_2) + (0.0031 \times PaO_2)$. A perfusion oxygen-delivery calculator was used as a bedside calculation aid, and values were indexed to body surface area when required to obtain DO_{2i}.

Clinical safeguards and flow adjustment framework

The institutional strategy was based on the preservation of indexed pump flow and assessment of cerebral and systemic oxygen balance rather than on MAP alone. A disproportionate decrease in cerebral rSO₂ during CPB prompted reassessment of perfusion adequacy and technical factors, including venous drainage, cannula position, pump flow, circuit performance, hemoglobin concentration, oxygenation, acid–base status, and surgical conditions.

When reduced cerebral oxygenation was considered related to flow or oxygen delivery, indexed pump flow was increased proportionally when technically and clinically feasible in order to improve oxygen delivery. Progressive metabolic acidosis was considered an additional warning sign of inadequate systemic perfusion and would have prompted reassessment of pump flow, oxygen delivery, hemoglobin concentration, gas exchange, and acid–base management. In the analyzed cohort, clinically relevant progressive metabolic acidosis requiring abandonment of the institutional strategy was not observed.

Transient low-MAP episodes were not considered target values. Such episodes prompted immediate physiologic and technical reassessment. Corrective measures were individualized and could include optimization of pump flow, correction of venous drainage or volume status, correction of anemia or acid–base disturbance, adjustment

of anesthetic depth, surgical–perfusion reassessment, and vasoactive therapy when clinically indicated.

Urine output and renal interpretation

Urine output during CPB was recorded descriptively but was not used as a primary intra-CPB adjustment trigger. Renal outcomes were interpreted in the broader context of post-CPB cardiac output, pulse pressure, systolic–diastolic pressure difference, vasoactive or inotropic requirement, hemodynamic instability, and cardiogenic shock, rather than attributed solely to intra-CPB MAP.

Outcomes

The primary descriptive outcome was the absence of major early postoperative complications. Secondary descriptive outcomes included operating-room extubation, postoperative stroke, acute kidney injury (AKI), requirement for venoarterial extracorporeal membrane oxygenation (VA-ECMO), and intraoperative physiologic perfusion markers including baseline right and left rSO₂, maximal relative rSO₂ decrease during CPB, circuit-derived SvO₂, and DO_{2i}.

AKI was defined according to kidney disease: Improving Global Outcomes criteria. Stroke was defined as a new focal neurologic deficit confirmed clinically and, when available, by neuroimaging. Operating-room extubation was considered a pragmatic marker of early postoperative recovery and fast-track feasibility, not a direct measure of tissue perfusion.

Statistical analysis

Because the study was retrospective, descriptive, and noncomparative, no formal hypothesis testing was performed. Continuous variables were summarized as mean $\pm$ standard deviation, median with interquartile range, and range, as appropriate. Categorical variables were summarized as counts and percentages. Wilson 95% CIs were calculated for major proportions using Python 3.11. No multivariable modeling was performed because the study did not include a comparator group and was not designed for causal inference.

RESULTS

A total of 200 adult patients undergoing cardiac surgery with normothermic CPB and no circulatory arrest were included in the final analysis [Figure 1]. The cohort was dominated by valve surgery. Aortic valve replacement was performed in 102 patients (51.0%), mitral valve replacement in 62 (31.0%), mitral valve repair in 28 (14.0%), and combined aortic valve replacement plus coronary artery bypass grafting in eight (4.0%). Cohort, procedural,

and intraoperative characteristics are shown in Table 1 and Figure 2.

All patients were managed using the same institutional flow-oriented and oxygen-delivery-aware CPB strategy. Pump flow index was maintained at 2.2–2.4 L/min/m².

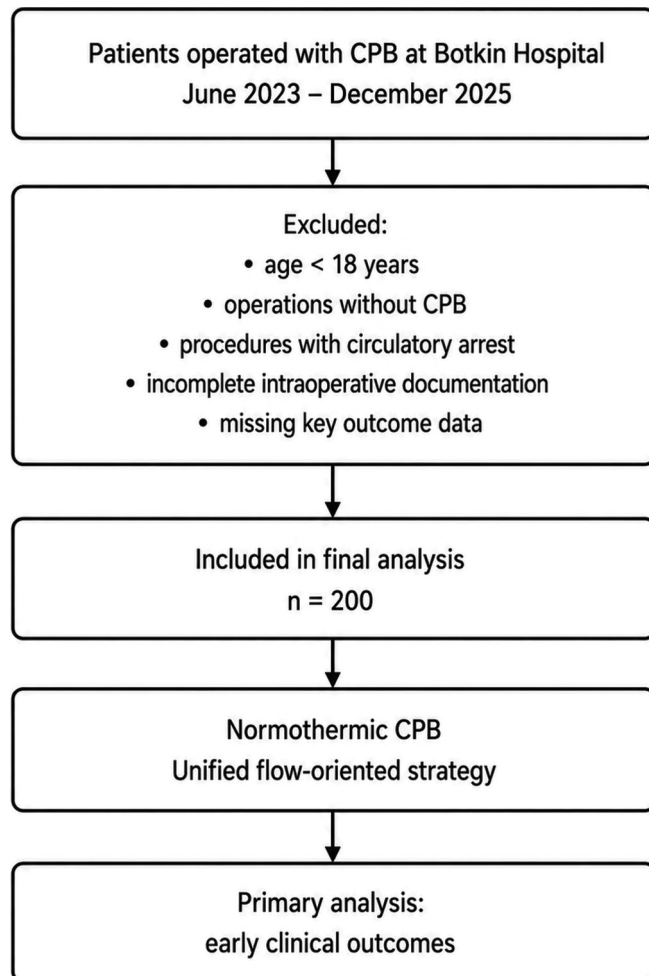


Figure 1: Study flow diagram. CPB: cardiopulmonary bypass

MAP was predominantly within 40–60 mmHg during CPB and was interpreted in relation to indexed pump flow, cerebral oxygenation, circuit-derived venous oxygen saturation, calculated DO_{2i}, hemoglobin concentration, acid–base status, venous return, circuit performance, and surgical context. Routine vasopressors were not used during CPB. Nitroglycerin boluses were administered when MAP exceeded 60 mmHg. The revised strategy is summarized schematically in Figure 3.

Bilateral cerebral oximetry, circuit-derived venous oxygen saturation, and calculated DO_{2i} were available for all 200 patients. Baseline rSO₂ values were obtained before initiation of mechanical ventilation while the patients were breathing room air. During CPB, the maximal relative decrease in rSO₂ from this baseline was recorded.

Baseline right-sided rSO₂ was 57.5 ± 11.2%, with a median of 64% [interquartile range (IQR), 47–67] and a range of 32–73%. Baseline left-sided rSO₂ was 58.5 ± 11.3%, with a median of 65% [IQR, 48–68] and a range of 33–74%.

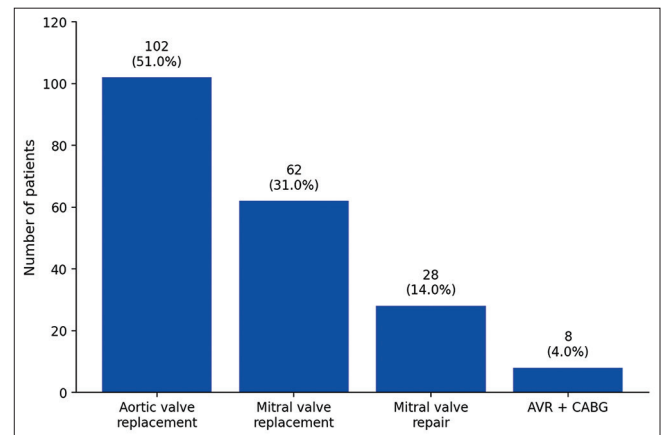


Figure 2: Distribution of surgical procedures in the final cohort. AVR: aortic valve replacement; CABG: coronary artery bypass grafting

Table 1: Cohort, procedural, and intraoperative characteristics.

Characteristic	Value
Study cohort	200 adults undergoing elective or urgent cardiac surgery with normothermic CPB
Procedures with circulatory arrest	Excluded
Isolated valve procedures	192/200 (96.0%)
Aortic valve replacement	102/200 (51.0%)
Mitral valve replacement	62/200 (31.0%)
Mitral valve repair	28/200 (14.0%)
Combined procedure: AVR + CABG	8/200 (4.0%)
CPB equipment	Stockert S5 heart-lung machine
Cerebral oximetry equipment	Somanetics cerebral oximetry system
Target flow index during CPB	2.2–2.4 L/min/m ²
Mean arterial pressure during CPB	Predominantly 40–60 mmHg; interpreted within physiologic context
Routine vasopressor use during CPB	Not used routinely; vasoactive therapy available when clinically indicated
Nitroglycerin administration	Bolus when MAP > 60 mmHg with preserved indexed flow
Hemoglobin transfusion threshold	Approximately 75 g/L, interpreted together with DO _{2i} , rSO ₂ , SvO ₂ , and clinical context

AVR: aortic valve replacement; CABG: coronary artery bypass grafting; CPB: cardiopulmonary bypass; DO_{2i}: indexed oxygen delivery; MAP: mean arterial pressure; rSO₂: regional cerebral oxygen saturation; SvO₂: circuit-derived venous oxygen saturation

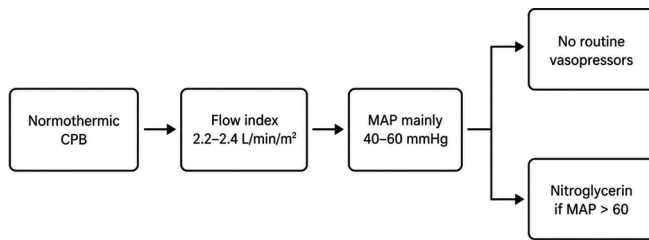


Figure 3: Flow-oriented, cerebral oxygenation-guided, oxygen-delivery-aware normothermic CPB strategy. Schematic representation of the institutional normothermic cardiopulmonary bypass strategy: indexed pump flow 2.2–2.4 L/min/m², mean arterial pressure mainly 40–60 mmHg, no routine vasopressor use, and nitroglycerin administration when MAP exceeded 60 mmHg. CPB: cardiopulmonary bypass; MAP: mean arterial pressure

The maximal relative decrease in rSO₂ during CPB was 28.5 ± 3.4%, with a median of 27% [IQR, 26–32] and a range of 24–38%.

Circuit-derived venous oxygen saturation obtained from the venous sampling line of the CPB circuit was 67.4 ± 5.9%, with a median of 70% [IQR, 62–72] and a range of 54–78%. Calculated DO₂i was 611 ± 74 mL/min/m², with a median of 645 mL/min/m² [IQR, 539–680] and a range of 470–725 mL/min/m² [Table 2]. No case required abandonment of the institutional perfusion strategy because of clinically relevant progressive metabolic acidosis.

Operating-room extubation was achieved in 172 of 200 patients (86.0%; 95% CI, 80.5–90.1). Stroke occurred in six patients (3.0%; 95% CI, 1.4–6.4), AKI in two patients (1.0%; 95% CI, 0.3–3.6), and VA-ECMO was required in six patients (3.0%; 95% CI, 1.4–6.4). Overall, 178 patients (89.0%; 95% CI, 83.9–92.6) had no major early postoperative complications [Table 3]. The composite outcome of no major early postoperative complications was abstracted as a patient-level clinical recovery marker and was not calculated by summing the listed complications.

DISCUSSION

This retrospective descriptive study reports a single-center experience with normothermic CPB managed using a flow-oriented, cerebral oxygenation-guided, oxygen-delivery-aware, and vasopressor-sparing strategy. In this cohort of 200 adult cardiac surgical patients, early clinical recovery was generally favorable, with a high rate of extubation in the operating room and low observed frequencies of stroke, AKI, and VA-ECMO. These findings should be interpreted as an institutional experience rather than evidence that lower MAP targets during CPB are universally safe or superior.

Table 2: Intraoperative physiologic perfusion markers.

Variable	Mean±SD	Median [IQR]	Range
Baseline right rSO ₂ , %	57.5±11.2	64 [47–67]	32–73
Baseline left rSO ₂ , %	58.5±11.3	65 [48–68]	33–74
Maximal relative rSO ₂ decrease during CPB, %	28.5±3.4	27 [26–32]	24–38
Circuit-derived SvO ₂ , %	67.4±5.9	70 [62–72]	54–78
Calculated DO ₂ i, mL/min/m ²	611±74	645 [539–680]	470–725

CPB: cardiopulmonary bypass; DO₂i: indexed oxygen delivery; IQR: interquartile range; rSO₂: regional cerebral oxygen saturation; SD: standard deviation; SvO₂: circuit-derived venous oxygen saturation. Baseline rSO₂ was recorded before mechanical ventilation while patients were breathing room air

Table 3: Early postoperative outcomes

Outcome	n/N	% (95% CI)
OR extubation	172/200	86.0 (80.5–90.1)
Stroke	6/200	3.0 (1.4–6.4)
Acute kidney injury	2/200	1.0 (0.3–3.6)
VA-ECMO	6/200	3.0 (1.4–6.4)
No major early postoperative complications	178/200	89.0 (83.9–92.6)

AKI: acute kidney injury; CI: confidence interval; OR: operating room; VA-ECMO: venoarterial extracorporeal membrane oxygenation. The composite outcome of no major early postoperative complications was abstracted as a patient-level clinical recovery marker and was not calculated by summing the listed complications

The main conceptual point of the present study is that MAP during CPB should not be interpreted as an isolated macrocirculatory number.^[7–13,16] Organ perfusion during CPB depends on the interaction between indexed pump flow, arterial oxygen content, oxygen delivery, vascular resistance, microcirculatory function, cerebral autoregulation, and patient-specific physiology.^[14–18] In this context, vasopressor-induced MAP elevation may increase systemic vascular resistance without necessarily improving tissue-level oxygen delivery or microvascular perfusion.^[14–16,23–25] Therefore, the institutional strategy prioritized preserved indexed pump flow, oxygen delivery assessment, cerebral oxygenation monitoring, and avoidance of unnecessary vasoconstriction.

The original concern with any vasopressor-sparing or permissive lower-pressure strategy is the potential risk of unrecognized cerebral or systemic hypoperfusion. For this reason, the present revised analysis emphasizes the physiologic safeguards used during CPB. Bilateral cerebral oximetry was used to assess regional cerebral oxygenation, with baseline values recorded before mechanical ventilation while patients were breathing room air. The maximal relative decrease in rSO₂ during CPB was recorded and interpreted together with indexed pump flow, circuit-derived venous oxygen saturation, calculated DO₂i, hemoglobin concentration, acid–base status, venous return, circuit performance, and surgical conditions.

In this cohort, the maximal relative decrease in rSO_2 during CPB was $28.5 \pm 3.4\%$, while circuit-derived venous oxygen saturation and calculated DO_{2i} were recorded as additional markers of systemic oxygen balance. These variables were not used to establish causal safety, but they provide important physiologic context and make the institutional approach more reproducible than a pressure-only description of CPB management.

The role of cerebral oximetry in this strategy was practical rather than purely observational. A disproportionate decrease in cerebral rSO_2 during CPB prompted reassessment of pump flow, venous drainage, cannula position, circuit performance, hemoglobin concentration, oxygenation, acid–base status, and surgical conditions. When reduced cerebral oxygenation was considered related to flow or oxygen delivery, indexed pump flow was increased when technically and clinically feasible. This approach differs from a simple low-MAP strategy because pressure was interpreted within a broader oxygen-delivery framework rather than accepted in isolation.

Renal outcomes should also be interpreted cautiously. In the present cohort, AKI was infrequent, but the retrospective design does not allow attribution of renal outcomes to the CPB pressure strategy. Renal perfusion during and after cardiac surgery is influenced by oxygen delivery, hemoglobin concentration, inflammatory response, venous congestion, post-CPB cardiac output, pulse pressure, vasoactive and inotropic support, and cardiogenic shock. In our institutional practice, urine output during CPB was not used as a primary trigger for pump-flow adjustment. Instead, renal risk was interpreted in the broader context of separation from CPB, restoration of spontaneous cardiac output, post-CPB pulse pressure, hemodynamic stability, and the presence or absence of cardiogenic shock.^[18–25]

The high proportion of patients extubated in the operating room should be interpreted in the broader context of local fast-track cardiac anesthesia practice rather than as a direct consequence of MAP management. Early extubation can be feasible after cardiac surgery when perioperative pathways are coordinated and patient selection is appropriate.^[27,28] In this study, extubation in the operating room is best viewed as a pragmatic marker of early recovery and hemodynamic stability, not as a direct measure of tissue perfusion.

This study has important limitations. First, it was a single-center retrospective descriptive analysis without a comparator group, which precludes causal inference. Second, baseline demographic and risk-score variables were not available in the analyzable aggregate dataset, limiting

risk adjustment and generalizability. Third, the analysis did not include time-resolved MAP exposure, individualized cerebral autoregulation thresholds, or formal modeling of the relationship between MAP, rSO_2 , circuit-derived SvO_2 , DO_{2i} , and postoperative outcomes. Fourth, circuit-derived venous oxygen saturation should not be interpreted as pulmonary artery catheter-derived mixed venous saturation. Fifth, the cohort was dominated by valve procedures, and the findings should not be generalized without caution to isolated coronary surgery, complex aortic surgery, severe carotid disease, profound ventricular dysfunction, high-risk emergency surgery, or procedures requiring circulatory arrest. Sixth, myocardial protection, transfusion practice, anesthetic management, and fast-track recovery pathways were not isolated from the perfusion strategy.

Despite these limitations, this report documents a reproducible institutional alternative to strictly pressure-centered CPB management. The study does not argue that lower MAP should be used routinely in all patients, nor that vasopressors should never be used. Rather, it supports the concept that MAP during CPB should be interpreted within a broader physiologic framework that includes indexed flow, cerebral oxygenation, oxygen delivery, venous oxygen saturation, vascular resistance, microcirculation, and patient-specific autoregulatory capacity. Future prospective studies should include time-resolved MAP exposure, cerebral autoregulation monitoring, cerebral oximetry, DO_{2i} , lactate kinetics, renal biomarkers, and clearly defined comparator strategies.^[18–22]

CONCLUSION

In this single-center retrospective descriptive cohort, normothermic CPB managed with preserved indexed pump flow, cerebral oxygenation-guided flow adjustment, calculated DO_{2i} , and vasopressor-sparing pressure management was accompanied by generally favorable early postoperative recovery. The strategy was not based on MAP alone but on the integrated interpretation of indexed flow, cerebral rSO_2 , maximal rSO_2 decrease, circuit-derived venous oxygen saturation, DO_{2i} , acid–base status, and clinical context. Because the study was retrospective, noncomparative, and not designed for causal inference, the findings should be interpreted as descriptive and hypothesis generating rather than as proof of safety, superiority, or generalizability of lower MAP targets during CPB.

Ethics statement

According to institutional policy, retrospective analyses of fully anonymized routine-care data do not require prospective ethics committee review; therefore, no approval

number was issued. An institutional confirmation or ethics waiver should be supplied if requested by the journal.

Author Contribution Statement

Nikita Chibisov conceived the study, participated in study design, collected and interpreted the data, and drafted the manuscript. Andrey Kornienko contributed to study design, clinical supervision, and critical revision of the manuscript. Anatoly Malyshev contributed to data collection and formal analysis. Evgeny Rodionov contributed to supervision and critical revision of the manuscript. Igor Glushenko contributed to clinical supervision and manuscript revision. Mikhail Shtrymov contributed to data interpretation and manuscript revision. All authors read and approved the final manuscript.

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Conflicts of interest

There are no conflicts of interest.

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