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ORIGINAL RESEARCH

# Real-time flow–pressure coupling under pulse contour monitoring in kidney transplantation: prospective algorithm and retrospective phenotype assignment

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**Abstract**
To prospectively evaluate a pulse contour–guided algorithm for real-time flow–pressure coupling to direct fluid versus vasoactive therapy during kidney transplantation, and to retrospectively assign circulation phenotypes from intraoperative hemodynamics and assess their association with fluid responsiveness. We conducted a prospective, nonrandomized evaluation of 65 KT recipients (32 deceased donor, 33 living donor) managed with GDHM. Pulse contour monitoring (HemoSphere/Acumen IQ) provided mean arterial pressure (MAP), cardiac index (CI), systemic vascular resistance index (SVRI), arterial dP/dt, and dynamic arterial elastance (EaDyn = PPV/SVV). Standardized fluid challenges were repeated every 45–60 min. Phenotypes were assigned using CI–SVRI patterns with dP/dt as a contractility proxy. Flow-pressure coupling was predefined as $\Delta SV \geq 10\%$ with EaDyn > 1.0. Primary outcomes were circulatory phenotype (initial and averaged) and per-challenge FPC responsiveness modeled as a proportion with trial weights. Recipients received a mean 3.57 fluid challenges; phenotype shifts were infrequent. Most patients showed limited flow-pressure coupling (41.5% had 0 responsive challenges). In binomial generalized linear models (logit), modeling the proportion of positive FPC tests with the number of challenges as trial weights, phenotype predicted per-challenge flow-pressure coupling (Hyperdynamic had lower odds versus Normal); covariates were not associated. Intraoperative volume, vasoactive support and early outcomes were similar; no protocol-related adverse events. Circulatory phenotypes were associated with flow-pressure coupling and supported real-time fluid/inotrope decisions under GDHM during KT. Phenotype-guided GDHM enabled individualized MAP control while limiting unnecessary fluid/vasoactive therapy. Findings establish feasibility and motivate multicenter trials to test clinical impact and cross-platform validation.

**Keywords** Flow-pressure coupling · Goal directed hemodynamic management (GDHM) · Pulse contour monitoring · Circulation phenotype · Kidney transplantation

## 1 Introduction

Kidney transplantation is the most common solid organ transplant in the United States, with over 27,000 procedures performed annually. Delayed graft function (DGF), defined as dialysis within the first postoperative week, occurs in approximately 25–30% of recipients and is associated with prolonged hospitalization and cardiovascular complications. Risk for DGF reflects a combination of donor, recipient, and intraoperative factors, among which hemodynamic management remains a modifiable determinant [1].

Intraoperative MAP is central to renal allograft perfusion. In a 2024 cohort, maintaining peri-reperfusion MAP near 75 mmHg was associated with lower DGF, whereas escalating vasopressors and intravenous fluids beyond target correlated with higher DGF [2]. These observations suggest that both the achieved pressure and the modality of achieving it (fluid vs vasoactive medication) matter.

Existing GDHM protocols often emphasize preload surrogates (e.g., SVV, CVP) to guide fluid vs vasoactive

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medication allocation under MAP targets, with mixed impact on clinical endpoints [3]. One potential problem with this approach is that the focus is placed on the macrocirculation (fluid responsiveness) and does not account for whether changes to the macrocirculation translate to changes to the microcirculation (perfusion pressure).

We designed a GDHM protocol using arterial waveform pulse contour analysis to provide beat-to-beat CI, SVRI, dP/dt, and dynamic arterial elastance (EaDyn = PPV/SVV) to monitor the pressure response to stroke volume changes. EaDyn integrates arterial load and ventricular–arterial coupling, providing a bedside estimate of whether stroke volume increases will translate into a MAP rise. By observing the flow-pressure coupling (FPC), our GDHM protocol emphasizes both the macrocirculation and the microcirculation. By constructing intraoperative Frank–Starling curves through repeated, standardized fluid challenges and reassessing at regular intervals under signal conditions (controlled ventilation, sinus rhythm), we operationalized real-time decisions: administer fluid only when FPC is present; otherwise, support MAP with inotropy/vasopressors. While FPC testing prospectively guided fluid vs inotropy; circulation phenotypes were assigned retrospectively from CI–SVRI patterns and dP/dt to analyze associations with responsiveness; early postoperative outcomes were collected for context.

## 2 Methods

### 2.1 Study design and setting

We conducted a prospective, exploratory analysis of 65 consecutive adult kidney transplant recipients managed under a protocolized goal-directed hemodynamic management (GDHM) algorithm at Cedars–Sinai Medical Center (Los Angeles, CA) between January and December 2025. All recipients in this manuscript were drawn from the GDHM arm of a pragmatic parent study comparing GDHM with conventional care; allocation in the parent study was operationally determined by anesthesiologist availability (scheduled research anesthesiologist = GDHM; otherwise conventional care). This analysis focuses on the GDHM arm to characterize intraoperative monitoring under a standardized algorithm; no between-arm effect was tested. This design limits causal inference; analyses were estimation-focused.

GDHM was applied prospectively and uniformly to all 65 recipients: standardized fluid challenges were delivered per protocol, signal prerequisites were enforced (controlled ventilation, sinus rhythm, stable arterial waveform), and MAP targets were maintained (±20% of baseline; peri-reperfusion ~ 75 mmHg) using predefined decision rules (fluid vs inotrope/vasopressor). Phenotype labels were not used to direct care during the case.

After all GDHM-managed cases were completed, intraoperative monitoring data (MAP, CI, SVRI, dP/dt, EaDyn) recorded around each fluid challenge were analyzed to assign circulation phenotypes. Specifically, phenotypes were derived post hoc from CI–SVRI patterns with dP/dt as a contractility proxy and summarized at two timepoints: initial (post-induction) and averaged (across all evaluable challenges). We then evaluated per-challenge FPC (events/trials), protocol adherence, and early outcomes by phenotype.

No blinding or allocation concealment was used; assignment reflected research anesthesiologist scheduling. Consecutive GDHM cases were included to minimize selection bias. Six transplant surgeons and ten research anesthesiologists participated. The study was approved by the institutional review board under a quality-improvement/low-risk perioperative monitoring framework; consent was not required per IRB approval.

Primary aim: to operationalize and assess an intraoperative monitoring algorithm (GDHM) and, subsequently, to characterize circulation phenotypes from recorded hemodynamics and quantify their association with per-challenge FPC. Postoperative outcomes (e.g., delayed graft function, cardiac events, length of stay) were collected for completeness and were not primary endpoints.

Intraoperative management was unblinded by necessity. Postoperative outcomes were abstracted from the EHR using predefined case report forms. Outcome abstraction was performed by investigators with access to intraoperative records. Given the quality-improvement designation, this exploratory study was not registered. Reporting follows TREND guidelines.

### 2.2 Participants

Eligible patients were adult recipients undergoing single-organ KT (living or deceased donors). Exclusion criteria included multi-organ transplants and absence of arterial line monitoring. The analytic cohort included 32 deceased donor (DD) and 33 living donor (LD) recipients. The unit of analysis was the recipient.

### 2.3 Perioperative management

#### 2.3.1 Institutional policy regarding preoperative cardiac workup

All recipients ≥ 50 yrs old undergo myocardial perfusion scans (MPS). Recipients < 50 yrs old receive preoperative cardiac workup at the discretion of the transplant

nephrologist. If repeat testing is performed prior to transplantation and the last MPS was within a year, or if the recipient never had a MPS, transthoracic echocardiography (TTE) is performed. All preoperative cardiac testing that demonstrates perfusion defects is followed up with a left heart catheterization.

#### 2.3.2 Institutional policy regarding pretransplant dialysis

All recipients on hemodialysis or peritoneal dialysis undergo dialysis within 24 h prior to surgery.

#### 2.3.3 Ventilation strategies

Ventilation was controlled with tidal volumes typically 6–8 mL/kg predicted body weight and PEEP per institutional standard. SVV/PPV/EaDyn-based assessments were performed during periods of controlled ventilation and sinus rhythm; if significant arrhythmia, poor arterial waveform or spontaneous breathing occurred, fluid responsiveness assessments were deferred until conditions allowed.

#### 2.3.4 Anesthetic management

Intraoperatively, all recipients had radial arterial catheters placed post-induction and connected to a pulse contour analysis system with Acumen IQ sensors (Edwards Lifesciences, Irvine, California). Continuous parameters included mean arterial pressure (MAP), cardiac index (CI), systemic vascular resistance index (SVRI), stroke volume index (SVI), arterial dP/dt (systolic upslope), stroke volume variation (SVV), pulse pressure variation (PPV), and dynamic arterial elastance (EaDyn = PPV/SVV).

Anesthetic depth was monitored with BIS (target 45–60). The choice of hypnotic, opioid, and neuromuscular blocker was not standardized. Institutional practice targeted MAP within ± 20% of baseline throughout the case and 75 mmHg at reperfusion.

*GDHM Algorithm:* (Fig. 1).

#### 2.3.5 Hemodynamic targets

Peri-reperfusion MAP was targeted at approximately 75 mmHg, within the broader goal of maintaining MAP

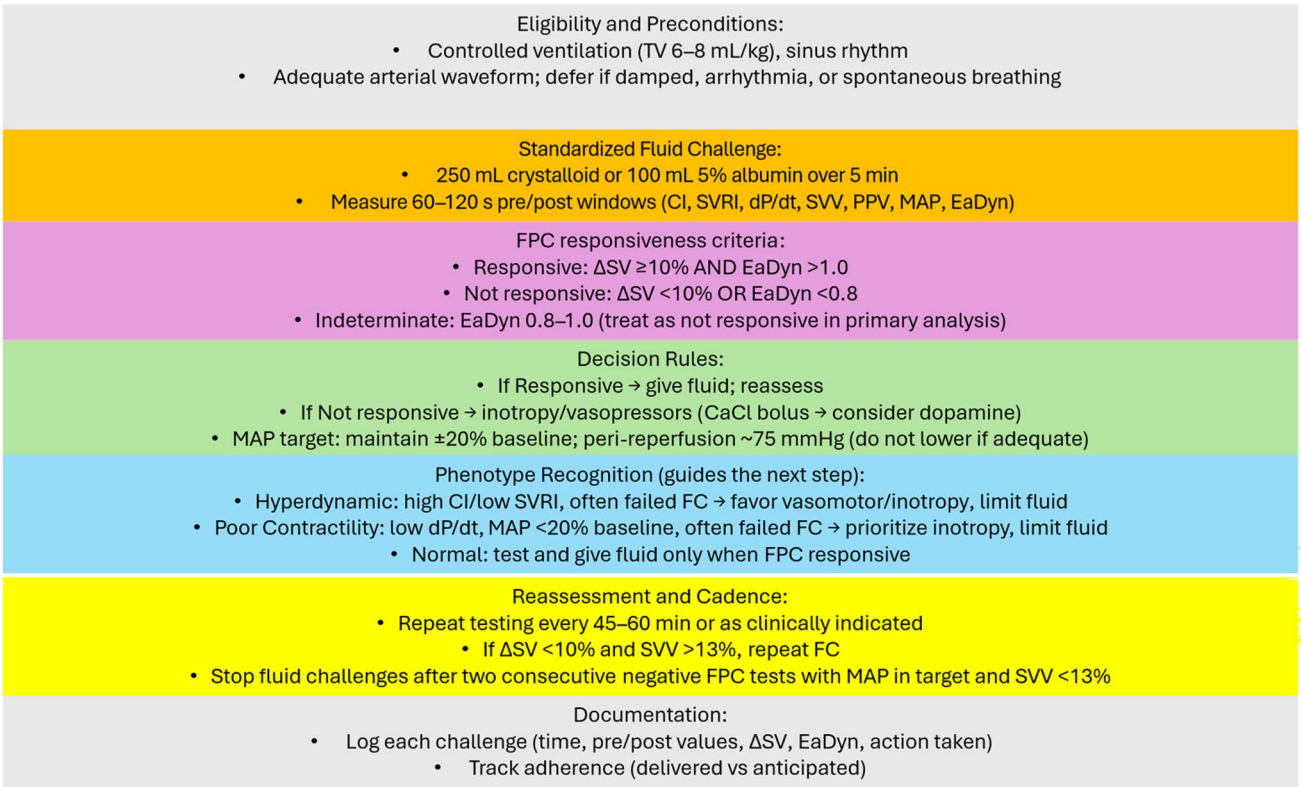


**Fig. 1** Goal directed hemodynamic management algorithm. TV = tidal volume, CI = cardiac index, SVRI = systemic vascular resistance index, dP/dt = change in pressure over time (contractility proxy), SVV = stroke volume variation, PPV = pulse pressure variation, MAP = mean arterial pressure, EaDyn = arterial dynamic elastance, ΔSV = change in stroke volume, CaCl = calcium chloride

within ± 20% of baseline throughout. Peri-reperfusion MAP was not pharmacologically lowered to 75 mmHg if MAP was already ≥ 75 mmHg and remained within ± 20% of baseline without excessive crystalloid or vasoactive support.

#### 2.3.6 Standardized fluid challenges and primary FPC criteria

Immediately after induction, standardized fluid challenges (FC) were performed using either 250 mL balanced crystalloid or 100 mL 5% albumin administered over 5 min. Fluid responsiveness was defined as a FC that resulted in a ≥ 10% increase in stroke volume (ΔSV). Dynamic arterial elastance (EaDyn; PPV/SVV) was assessed to interpret the expected FPC response: EaDyn > 1.0 indicated a likely MAP rise with an increase in stroke volume; EaDyn < 0.8 indicated a poor pressure response; values between 0.8 and 1.0 were considered indeterminate. Positive FPC was operationally defined as ΔSV ≥ 10% with EaDyn > 1.0 (responsive), whereas ΔSV < 10% or EaDyn < 0.8 was considered negative FPC. Tests with ΔSV ≥ 10% and EaDyn 0.8–1.0 were classified as indeterminate; for primary analyses, indeterminate tests were treated as negative FPC, and sensitivity analyses treating indeterminate as responsive did not alter the primary conclusions.

#### 2.3.7 Decision rules for support modality

When peri-reperfusion MAP was < 75 mmHg or > 20% below baseline, support modality was determined by standardized testing: positive FPC tests triggered fluid support; negative FPC results triggered inotropic/vasoactive therapy (calcium chloride [CaCl] bolus, dopamine infusion), with pure vasoconstrictors discouraged in order to support contractility and cardiac index.

If ΔSV < 10% and SVV > 13%, a second FC (same volume) was administered to address suspected hypovolemia.

#### 2.3.8 Frequency, adherence, and stop-criteria

Standardized FCs were repeated every 45–60 min or as clinically indicated. Most procedures were approximately 4 h; thus, up to four FCs were anticipated per recipient if they demonstrated positive FPC. Recipients received a mean of 3.57 FCs (≈90% of anticipated opportunities), indicating high adherence. Adherence was monitored prospectively as delivered versus anticipated opportunities; when predefined stop-criteria (two consecutive negative FPC tests with MAP within 20% of baseline and ≥ 75 mmHg during peri-reperfusion and SVV < 13%) were met, subsequent FCs were considered non-opportunities. Deviations from the protocol and reasons were recorded; common deviations included inconsistent arterial waveforms.

#### 2.3.9 Measurement windows and signal conditions

Measurements used 60–120 s pre/post FC windows under controlled ventilation, sinus rhythm and stable arterial waveform to minimize artifact and ensure comparability. FPC was evaluated via EaDyn (PPV/SVV). dP/dt thresholds were used to aid circulation phenotype classification; however, temporal trends and integrated clinical context were prioritized over single thresholds.

#### 2.3.10 Circulation phenotype classification

Hemodynamic analysis following FPC testing was used to determine the circulatory phenotype. Phenotypes were determined using CI, SVRI, and dP/dt in the context of MAP. A Hyperdynamic phenotype was defined as CI > 4 L/min/$m^2$, SVRI < 1970 dyn·s·$cm^5$/$m^2$, dP/dt > 1000 mmHg/s. A Normal phenotype was characterized by CI 2.5–4 L/min/$m^2$, SVRI 1970–2390 dyn·s·$cm^5$/$m^2$, dP/dt 1000–1200 mmHg/s. Poor Contractility was defined as dP/dt < 1000 mmHg/s with MAP < 20% baseline; CI variable; SVRI typically 1970–2390 dyn·s·$cm^5$/$m^2$. In cases without a clear CI–SVRI pattern, dP/dt was used to differentiate hyperdynamic from poor contractility physiology. Mixed physiology was classified as Poor Contractility when dP/dt < 1000 mmHg/s with MAP < 20% below baseline despite high CI/low SVRI.

The defined CI and SVRI thresholds were selected a priori and were consistent with standard physiology references (e.g., Guyton and Hall Textbook of Medical Physiology, 15th ed.)[4, 5] Because arterial dP/dt readings depend on the device, calibration and could be influenced by inotropic/vasopressor medications, absolute thresholds were used for classification support (< 1000 mmHg/s suggesting reduced contractility; > 1000 mmHg/s suggesting increased contractility), but intra-patient trends across sequential FCs were prioritized over absolute readings [6–9]. Primary analyses relied on trend concordance among CI, SVRI, MAP and dP/dt.

Primary outcomes, defined a priori, were FPC responsiveness per the GDHM operational definition (ΔSV ≥ 10% with EaDyn > 1.0) and intraoperative circulatory phenotype (assessed initially post-induction and as the average across all FPC assessments). Secondary outcomes, defined a priori, included delayed graft function (dialysis within 7 postoperative days), postoperative cardiac complications (heart failure, atrial fibrillation, demand ischemia), and hospital length of stay. Exploratory, subgroup, and sensitivity analyses (post hoc) included stratification by donor type

(deceased vs living donor); evaluation of circulatory phenotype shifts versus no shifts with McNemar–Bowker tests by donor type; alternative handling of indeterminate EaDyn values (0.8–1.0); descriptive comparisons of intraoperative vasoactive support intensity and fluid volume across phenotypes; and multivariable models testing associations between recipient factors (age, dialysis vintage, preoperative hemoglobin (Hgb), preoperative ejection fraction (EF), preoperative cardiac abnormality) and both FPC testing and phenotype. Recipient factors were modeled age (per 10 years), preoperative Hgb (per 1 g/dL), dialysis vintage (per year), and EF (per 10%). Preoperative cardiac abnormality was defined as any functional or structural abnormality. Multivariable models that tested recipient factors were based on N = 52 due to lack of preoperative cardiac testing and/or lack of pretransplant dialysis. Because fluid challenges were conducted predominantly before graft implantation, donor type/status were included for completeness but were not expected to materially influence pre-implant responsiveness. No treatment effect between randomized groups was evaluated, and no clinically meaningful treatment effect size was prespecified, consistent with the quality-improvement, exploratory design.

Intraoperative management variables included total fluid volume and vasoactive support intensity, categorized as: none -minimal support (no support-CaCl 0.1–0.5 g); moderate -heavy support (CaCl 0.6–≥1.0 g); maximum support (dopamine infusion). Adjunct use of vasoconstrictors (phenylephrine and/or norepinephrine) occurred in a subset of recipients; these were recorded but not used to reassign category if CaCl/dopamine constituted the primary support. A sensitivity tabulation including adjunct vasoconstrictors did not alter the overall distribution across support categories.

### 2.4 Statistical analysis

The unit of analysis was the recipient. This exploratory study was not powered for postoperative endpoints. Accordingly, analyses were estimation-focused, with results reported as effect estimates and 95% confidence intervals (CIs). No formal efficacy hypothesis testing was prespecified.

Analyses were conducted using jamovi (version 2.7.6; The jamovi project) interfacing with the R statistical engine. Continuous variables were assessed for normality (Shapiro–Wilk) and summarized as mean ± SD or median [IQR]; categorical variables as n (%).

Between-phenotype comparisons: Cross-sectional hemodynamics (CI, SVRI, dP/dt, MAP) were compared across phenotypes using Kruskal–Wallis tests, followed by Dwass–Steel–Critchlow–Fligner (DSCF) pairwise comparisons with familywise adjustment. Results are reported as global p-values and DSCF-adjusted pairwise p-values.

Within-subject change: Initial vs average hemodynamics were compared using paired t-tests when differences were approximately normal and Wilcoxon signed-rank otherwise; to control familywise error, p-values were Holm-adjusted within phenotype across CI, SVRI, dP/dt, and MAP.

FPC modeling: Per-challenge responsiveness was modeled with binomial generalized linear models (logit link) using a weighted dependent variable, specified as the proportion of positive FPC tests with the number of fluid challenges entered as trial weights (i.e., events/trials formulation: COUNT_POSITIVE out of NUMBER_FC). Predictors included circulation phenotype (reference = Normal), number of fluid challenges, age (per 10 years), dialysis vintage (per year), preoperative Hgb, preoperative EF (per 10%), preoperative cardiac abnormality (binary), and donor status/type. Results are reported as odds ratios (ORs) with 95% CIs; model performance is summarized with McFadden's $R^2$ and AIC. Dispersion was assessed via deviance/df; no concerning overdispersion was observed. All recipients with ≥1 fluid challenge were included in the binomial GLMs; the number of challenges per recipient was entered as binomial trial weights. Recipients with "not enough tests performed" were retained in descriptive and inferential analyses provided initial and averaged hemodynamics were available.

Group comparisons (context): Intraoperative volume was compared by Welch's ANOVA with Games–Howell post hoc (heteroskedasticity by Levene's test); vasoactive intensity was compared with ordinal logistic regression. The proportional odds assumption was not formally tested and visual diagnostics did not suggest violation; results were robust and consistent with sensitivity analyses.

Multiplicity and power: Two-sided $\alpha = 0.05$. Analyses were estimation-focused; the study was not powered for postoperative outcomes (DGF, cardiac events).

Missing data: three recipients who were on dialysis prior to transplantation did not have a documented dialysis vintage. Two recipients age > 50 did not have documented preoperative cardiac testing in their EHR.

## 3 Results

### 3.1 Study population (Appendix 5.)

Sixty-five KT recipients were analyzed (32 deceased donor, 33 living donor) between January–December 2025. Participant flow: 66 approached, 66 eligible, 66 enrolled, and 65 analyzed; no early follow-up losses. Age, sex, dialysis vintage, preoperative Hgb, preoperative EF and preoperative cardiac abnormalities were similar by donor type. Among recipients with cardiac abnormalities, 6/11 (54.5%) had

perfusion defects. Followup LHC on all 6 revealed nonocclusive coronary artery disease; no further intervention was required. Among deceased donors, 56.2% were DBD.

### 3.2 Protocol adherence and FPC

Protocol adherence and descriptive context (Tables 1A and 1B).

Recipients received a mean 3.57 ± 1.40 standardized FCs. By average phenotype, total FC/case (median [IQR]) were: Hyperdynamic 3 [2.0], Normal 4 [1.75], Poor Contractility 3.5 [1.75]. Most recipients showed limited FPC: 41.5% (27/65) had 0 positive FPC tests; among nonresponders, 48.1% (13/27) were Hyperdynamic. Protocol adherence was high: 81.5% (53/65) met stop-criteria and discontinued testing per protocol, with the primary reason being consecutive negative FPC tests (64.6% of the cohort; 42/65), followed by termination of surgery (16.9%; 11/65). Of the 12 recipients (18.5%) who did not meet stop-criteria, all had too few evaluable tests (e.g., limited opportunities or loss of signal prerequisites). The proportion meeting stop-criteria varied by phenotype: Hyperdynamic 85.7% (18/21), Normal 80.8% (21/26), and Poor Contractility 77.8% (14/18). As predefined, early nonresponders underwent fewer FCs due to cessation after two consecutive nonresponsive tests with MAP in target and SVV < 13.

### 3.3 Predictors of FPC (Table 2)

Using binomial GLMs with the proportion of positive FPC as the dependent variable and the number of challenges as weights, circulation phenotype was associated with FPC probability. All recipients with ≥ 1 fluid challenge were included; the number of challenges was entered as trial weights. In the initial-phenotype model, Hyperdynamic had lower odds of a positive FPC than Normal (OR 0.19, 95% CI 0.08–0.47, $p < 0.001$), while Poor Contractility did not differ. Results were directionally consistent using average phenotype (Hyperdynamic vs Normal OR 0.26, 95% CI 0.10–0.68, $p = 0.006$; Poor Contractility vs Normal not significant). Recipient factors (age, hemoglobin, dialysis vintage, ejection fraction, preoperative cardiac abnormality) and donor type were not associated, and interaction terms were nonsignificant. Model fit was modest (initial AIC 129.5, pseudo-$R^2 \approx 0.10$; average AIC 129.6, pseudo-$R.^2 \approx 0.06$) with no concerning dispersion. Full estimates are shown in Table 2

**Table 1** A. Flow-pressure coupling (FPC) responsiveness by average phenotype

| Phenotype | Total FC/surgery, median [IQR] | Any positive FPC (≥ 1), n (%) | Fully nonresponsive (0 positive FPC) n (%) | Count of positive FPC, median [IQR] |
|---|---|---|---|---|
| Hyperdynamic n = 21 | 3 [2] | 8 (38.1%) | 13 (61.9%) | 0 [2] |
| Normal n = 26 | 4 [1.75] | 18 (69.2%) | 8 (30.8%) | 1 [2] |
| Poor Contractility n = 18 | 3.5 [1.75] | 12 (66.7%) | 6 (33.3%) | 1 [2] |

| Phenotype | Adherence ratio (delivered/anticipated), median [IQR] | Met stop-criteria, n (%) | Stop-criteria n (%): consecutive negative tests | Stop-criteria n (%): termination of surgery | Stop-criteria not met n (%): |
|---|---|---|---|---|---|
| Hyperdynamic n = 21 | 0.75 [0.50] | 18 (85.7%) | 14 (66.7%) | 4 (19.0%) | 3 (14.3%) |
| Normal n = 26 | 1.00 [0.44] | 21 (80.8%) | 16 (61.5%) | 5 (19.2%) | 5 (19.2%) |
| Poor Contractility n = 18 | 0.88 [0.44] | 14 (77.8%) | 12 (66.7%) | 2 (11.1%) | 4 (22.2%) |

FPC definition: Positive FPC was defined as ΔSV ≥ 10% with EaDyn > 1.0. Indeterminate EaDyn (0.8–1.0) were treated as negative FPC for primary analyses. Zero positive FPCs occurred either due to a negative FC (ΔSV ≤ 10%) or a negative FPC (ΔSV ≥ 10% with EaDyn < 0.8).

Stop-criteria definition: Stop-criteria were met when two consecutive negative FC and/or negative FPC tests occurred with MAP within target and SVV < 13%, or when testing ended at case completion per protocol. Protocol adherence was high: 81.5% (53/65) met stop-criteria and discontinued testing per protocol, primarily due to consecutive negative FPC tests (64.6%; 42/65), followed by case completion (16.9%; 11/65). "Not enough tests performed" is counted as "stop-criteria not met"; all 12 recipients (18.5%) in this category had too few evaluable tests (e.g., limited opportunities or loss of signal prerequisites).

Scope of adherence ratio: Adherence ratio uses a fixed anticipated count of 4 FCs per case (protocol cadence). If actual anticipated opportunities differ by case duration, ratios may slightly over-or-underestimate adherence.

Signal prerequisites: FC assessments were performed under controlled ventilation and sinus rhythm; when prerequisites were not met, FCs were deferred, which may contribute to "not enough tests performed."

Timing rationale: Standardized FCs were scheduled every 45–60 min; early cessation in nonresponders reduced delivered FCs.

**Table 2** Predictors of positive FPC (binomial GLM, events/trials via proportion + weights): initial vs average phenotype

| Predictor | Initial phenotype OR (95% CI), p | Average phenotype OR (95% CI), p |
|---|---|---|
| Phenotype: Hyperdynamic vs Normal | 0.13 (0.04–0.41), < 0.001 | 0.26 (0.10–0.68), 0.006 |
| Phenotype: Poor Contractility vs Normal | 0.47 (0.14–1.56), 0.216 | 0.86 (0.37–2.02), 0.729 |
| Age (per 10 years) | 0.91 (0.69–1.19), 0.476 | 0.93 (0.71–1.21), 0.569 |
| Preoperative hemoglobin (per 1 g/dL) | 1.15 (0.91–1.46), 0.239 | 1.16 (0.93–1.44), 0.204 |
| Dialysis vintage (per year) | 1.01 (0.84–1.20), 0.952 | 1.00 (0.84–1.19), 0.968 |
| Ejection fraction (per 10%) | 1.14 (0.52–2.51), 0.746 | 0.93 (0.55–1.57), 0.788 |
| Preoperative cardiac abnormality (yes vs no) | 0.91 (0.21–3.91), 0.899 | 1.34 (0.39–4.64), 0.644 |
| Donor type: LD vs DD | 0.70 (0.27–1.85), 0.476 | 0.81 (0.32–2.05), 0.651 |

Models are binomial GLMs (logit). Dependent variable is the proportion of positive FPC tests; NUMBER_OF_FLUID_CHALLENGES entered as binomial trial weights. All ORs adjusted for listed covariates; Reference categories: phenotype = Normal; donor type = DD; cardiac abnormality = no. All recipients with ≥ 1 fluid challenge were included; the number of challenges was entered as trial weights

Fit: Initial model LR $\chi^2(12) = 21.2$, $p = 0.047$; AIC = 129.5; pseudo-$R^2 \approx 0.10$; deviance/df ≈ 1.07. Average model LR $\chi^2(8) = 13.1$, $p = 0.107$; AIC = 129.6; pseudo-$R^2 \approx 0.06$; deviance/df ≈ 1.37

**Table 3** Intraoperative management signals by circulation phenotype

| Circulation Phenotype | n | Volume mean ± SD (mL) | 95% CI for mean | Vasoactive support (n, %) |
|---|---|---|---|---|
| Hyperdynamic | 21 | 2110 ± 490 | 1887–2332 | None-Minimal: 8 (38.1%); Moderate-Heavy: 11 (52.4%); Maximum: 2 (9.5%) |
| Normal | 26 | 2113 ± 701 | 1830–2396 | None-Minimal: 12 (46.2%); Moderate-Heavy: 14 (53.8%); Maximum: 0 (0.0%) |
| Poor Contractility | 18 | 2508 ± 901 | 2060–2956 | None-Minimal: 3 (16.7%); Moderate-Heavy: 12 (66.7%); Maximum: 3 (16.7%) |

Volume: Welch's ANOVA/Games–Howell; $p = 0.240$. Vasoactive intensity: ordinal logistic regression; Poor Contractility vs Normal OR 4.95 ($p = 0.017$). Vasoactive support defined as: None–Minimal: CaCl 0–0.5 g; Moderate–Heavy: CaCl 0.6–≥1.0 g; Maximum: dopamine infusion

**Table 4** Initial hemodynamics per initial circulation phenotype

| Phenotype | CI, mean ± SD (L/min/$m^2$) | SVRI, median [IQR] (dyn·s·$cm^5$/$m^2$) | dP/dt, median [IQR] (mmHg/s) | MAP, median [IQR] (mmHg) |
|---|---|---|---|---|
| Hyperdynamic n = 26 | 4.60 ± 0.89 | 1457 [306] | 1259 [401] | 87 [20.4] |
| Normal n = 25 | 3.05 ± 0.68 | 2407 [491] | 1000 [443] | 97 [21] |
| Poor Contractility n = 14 | 2.58 ± 0.77 | 1986 [1038] | 535 [170] | 68 [19] |

Summary statistics: mean ± SD shown for approximately normal variables (CI); median [IQR] shown otherwise (SVRI, dP/dt, MAP) based on Shapiro–Wilk diagnostics. Global tests: Kruskal–Wallis across phenotypes for each variable (CI, dP/dt, SVRI, MAP), all $p < 0.001$. Pairwise tests: Dwass–Steel–Critchlow–Fligner comparisons with familywise adjustment; key non-significant pairs were Normal vs Poor Contractility for CI and SVRI, and Normal vs Hyperdynamic for dP/dt and MAP. Abbreviations and units: CI (L/min/$m^2$), SVRI (dyn·s·$cm^5$/$m^2$), dP/dt (mmHg/s), MAP (mmHg)

### 3.4 Intraoperative management signals (Table 3)

Total intraoperative volume did not differ significantly by phenotype (Welch's ANOVA: $F(2, 36.7) = 1.49$, $p = 0.240$), and all Games–Howell pairwise comparisons were likewise nonsignificant. Group means: Hyperdynamic 2110 mL (SD 490), Normal 2113 mL (SD 701), Poor Contractility 2508 mL (SD 901). Poor Contractility exhibited a broader volume distribution (IQR 1300 mL) and higher upper tail (max 4700 mL), but central tendencies overlapped with Normal and Hyperdynamic, and formal comparisons were not significant. Given the ordered categories of vasoactive support (none–minimal, moderate–heavy, maximum), we fit an ordinal logistic model. Poor Contractility was associated with higher odds of greater support intensity versus Normal (OR 4.95, 95% CI 1.40–19.85, $p = 0.017$), whereas Hyperdynamic did not differ from Normal (OR 1.67, 95% CI 0.54–5.37, $p = 0.378$). Model fit was modest (McFadden's $R^2 = 0.055$; AIC = 117). These patterns align with preferential inotropic escalation in Poor Contractility under the protocol.

### 3.5 Initial phenotypes and associated hemodynamics (Table 4)

After the first fluid challenge, phenotypes were Hyperdynamic 40.0% (26/65), Normal 38.5% (25/65), and Poor Contractility 21.5% (14/65). Hemodynamics matched definitions: CI was highest in Hyperdynamic and lowest in Poor Contractility (mean 4.60 ± 0.89 vs 2.58 ± 0.77 L/min/$m^2$); SVRI was lowest in Hyperdynamic (median 1457 [306]) and higher in Normal and Poor Contractility; dP/dt was highest in Hyperdynamic and lowest in Poor Contractility (median 1259 [401] vs 535 [170] mmHg/s); and MAP

was highest in Normal. Global tests indicated differences across phenotypes (Kruskal–Wallis, all $p<0.001$). Pairwise DSCF comparisons showed: for CI, Hyperdynamic > Normal and Hyperdynamic > Poor Contractility; for SVRI, Hyperdynamic < Normal and Hyperdynamic < Poor Contractility; for dP/dt, Hyperdynamic > Poor Contractility and Normal > Poor Contractility; and for MAP, Normal > Poor Contractility and Hyperdynamic > Poor Contractility (all DSCF-adjusted $p<0.01$). Normal vs Poor Contractility was not significant for CI and SVRI, and Normal vs Hyperdynamic was not significant for dP/dt and MAP. These initial differences were directionally consistent with post hoc average phenotypes, although most within-phenotype changes were small and not significant after adjustment.

### 3.6 Phenotype stability and within-phenotype hemodynamic change (Appendix 6 and 7)

At the end of surgery, the phenotypes were Hyperdynamic 32.3% (21/65), Normal 40% (26/65), Poor Contractility 27.7% (18/65). Shifts occurred in 21.5% (14/65), most commonly Normal → Poor Contractility (n = 7) and Hyperdynamic → Normal (n = 5). The net distribution moved modestly away from Hyperdynamic and toward Normal and Poor Contractility; the McNemar–Bowker test indicated no significant asymmetry ($\chi^2[3]=7.33$, $p=0.062$). Despite these distributional shifts, within-phenotype hemodynamic changes from initial to average were not significant after Holm's step-down adjustment across CI, SVRI, dP/dt, and MAP.

### 3.7 Postoperative outcomes (Appendix 8)

DGF, postoperative cardiac complications, and LOS did not differ significantly across phenotypes. Given limited power, these comparisons are descriptive and not powered to detect differences in postoperative events. No protocol-related adverse events occurred.

### 3.8 Posthoc exploratory analysis (Appendix 9)

Post hoc analyses examined the distribution of circulation phenotypes and whether recipient factors predicted phenotype. Using multinomial logistic regression (exploratory), recipient factors (age per 10 years, dialysis vintage per year, preoperative hemoglobin, preoperative EF per 10%, preoperative cardiac abnormality) were not significantly associated with average circulation phenotype (reference: Normal; McFadden's $R^2=0.051$, AIC = 130; all $p>0.25$). These analyses were conducted to generate hypotheses about potential determinants of phenotype; they were not prespecified and should be interpreted cautiously.

## 4 Discussion

This study demonstrates that arterial waveform–derived circulation phenotypes, operationalized with CI, SVRI, dP/dt, and dynamic arterial elastance (EaDyn), provide actionable, repeated guidance for intraoperative fluid and inotrope decisions during kidney transplantation, by prospectively testing FPC at standardized intervals under controlled signal conditions. Under a standardized GDHM protocol, phenotype was associated with FPC probability; Hyperdynamic consistently showed lower odds of a positive FPC versus Normal, while Poor Contractility did not differ. Within-phenotype hemodynamic shifts were modest and not significant after adjustment. A key feature was using EaDyn to anticipate whether a positive fluid challenge ($\Delta SV \geq 10\%$) would translate into a clinically meaningful rise in perfusion pressure. Many recipients exhibited fluid responsiveness (positive FC); however, when EaDyn < 1, the expected MAP response was poor. This distinction allowed fluid administration only when FPC was present, reserving inotropy/vasopressors for nonresponsive states, and maintaining MAP within ± 20% of baseline and ~ 75 mmHg peri-reperfusion. Approximately one in five recipients did not meet stop-criteria, largely due to limited evaluable windows or case end; this reflects real-world signal constraints rather than protocol nonadherence, and the binomial weighting mitigates variable exposure in modeling.

Impaired FPC is expected in KT recipients given the hemodynamic hallmarks of ESRD and uremic cardiomyopathy—arterial remodeling, high-output states, and contractile impairment [10–12]. Our monitoring-led analysis categorized recipients into three phenotypes from routine waveform signals, thus correlating the hallmarks of uremic cardiomyopathy with real time FPC. Because absolute dP/dt values can vary across vendors and loading conditions, we prioritized trend direction and cross-signal concordance across CI, SVRI, dP/dt, MAP, EaDyn to support device-agnostic application.

Most recipients (60%) exhibited an abnormal average intraoperative phenotype (Hyperdynamic or Poor Contractility), and 41.5% of all recipients had no responsive FPC challenges. Moreover, only 21.5% phenotype profiles changed after the initial FPC test. The hemodynamic variables defining each phenotype also did not significantly change during the surgery, despite the fact that the phenotypes received varying degrees of intraoperative fluids and vasoactive support. We suspect the lack of within-phenotype hemodynamic change reflects the temporal separation between vasoactive dosing and testing windows; this hypothesis warrants prospective timing analysis.

Importantly, binomial events/trials models (logit link) showed that circulation phenotype, not donor type/status nor routine recipient covariates (age, dialysis vintage, preoperative Hgb, preoperative EF or preoperative cardiac abnormality), predicted per-challenge FPC responsiveness: Hyperdynamic consistently had lower odds relative to Normal, whereas Poor Contractility did not differ from Normal. Given that most testing occurred pre-implant, donor characteristics were not informative for intraoperative decisions; recipient hemodynamic phenotype was.

Contextual signals aligned with the protocol's intent. Although differences in total intraoperative volume did not reach statistical significance, recipients with Poor Contractility ultimately received more intravascular volume (higher central tendency and broader IQR) than Hyperdynamic and even Normal phenotypes—consistent with fluid being reserved for demonstrated FPC and inotropy being used to restore contractility when FPC was absent. Complementing this, the intensity of vasoactive support was significantly higher in Poor Contractility than Normal (ordinal logistic regression), reflecting phenotype-guided inotropic escalation under the protocol. Together, these signals support a monitoring-led strategy: test FPC first, then allocate fluid or inotropy according to real-time flow–pressure coupling rather than preload surrogates alone.

These data extend prior work by operationalizing an intraoperative Frank–Starling approach augmented with FPC (EaDyn), demonstrating feasibility of phenotype-guided monitoring in KT. Although phenotypes in this cohort were classified after data collection and shifts were infrequent, a practical prospective approach would be to apply the standardized GDHM protocol and, after the first evaluable fluid challenge, use the observed phenotype to prioritize therapy. Specifically, when early testing identifies a Hyperdynamic pattern (high CI/low SVRI with often a failed fluid responsive test), we would avoid non-informative fluid and initiate vasomotor/inotropic support sooner to meet MAP targets. When Poor contractility is observed (low dP/dt with MAP < 20% baseline and often a failed fluid responsive test), earlier inotrope titration to improve contractility is advisable, with fluids reserved for demonstrated FPC responsiveness. Normal phenotypes generally warrant continued testing and fluid administration only when FPC is demonstrated. Decisions should be made under controlled ventilation, sinus rhythm, reliable arterial waveforms with repeated reassessment (every 45–60 min) to detect any change in FPC. Although exploratory, our post hoc analysis suggests that recipient factors are not predictive of phenotype, which would further support performing FPC testing to determine the phenotype, as opposed to estimating based on the recipient's history.

Future multicenter studies should evaluate phenotype-guided algorithms as monitoring interventions using process endpoints (e.g., proportion of informative challenges, time-in-target MAP, fluid/inotrope efficiency indices) and validate phenotype thresholds and dP/dt trends across platforms.

### 4.1 Study limitations

This single-center exploratory study was underpowered to detect modest differences in DGF and cardiac events; findings should be interpreted as hypothesis-generating. Non-randomized design increases risk of confounding; future randomized or stepped-wedge designs could strengthen inference. The EaDyn gray zone (0.8–1.0) introduces uncertainty; we mitigated this via repeated fluid challenges and sensitivity analyses, which did not change conclusions. Results reflect a single-institution practice using specific monitoring technology. Although specific thresholds may vary across monitoring platforms, the FPC concept (stroke volume change linked to pressure response) and the phenotype framework are device-agnostic and can be tested with cross-platform validation. Performing this study in a single center leads to the potential for selection bias, and the nonrandomized design increases the risk of information bias. Phenotype classification was performed post hoc and may be subject to classification bias. External validation in larger, diverse cohorts is warranted.

## Appendix

See Table 5., Table 6., Table 7., Table 8. and Table 9..

**Table 5.** Baseline characteristics of kidney transplant recipients and reference population

| Category | Characteristic | Study cohort (n = 65) | Reference population (N = 319) |
|---|---|---|---|
| Recipient characteristics | Age (years), mean ± SD | 56 ± 14.7 | — |
| | 15–34 y, n (%) | 6 (9.2%) | 33 (10.4%) |
| | 35–49 y, n (%) | 15 (23.1%) | 71 (22.3%) |
| | 50–64 y, n (%) | 26 (40%) | 116 (36.4%) |
| | ≥ 65 y, n (%) | 18 (27.7%) | 98 (30.8%) |
| | Male sex, n (%) | 42/65 (64.6%) | 193 (60.5%) |
| | Preoperative Dialysis | 57/65 (84.6%) | not available |
| | Dialysis Vintage 1–60 months[a] | 41/54 (75.9%) | |
| | Dialysis Vintage > 60 months | 13/54 (24.1%) | |
| | Preoperative Hgb, mean ± SD | 10.4 ± 1.6 | not available |
| | Preoperative EF, mean ± SD [b] | 60.5% ± 8.0 | Not available |
| | Cardiac abnormality [c] | 11/55 (20%) | Not available |
| | Perfusion defect | 6/11 (54.5%) | |
| | Ventricular hypokinesis | 4/11 (36.4%) | |
| | Valvular disease | 1/11 (9%) | |
| Donor characteristics | Donation following Brain Death (DBD), n (%) | 18 (27.7%) | 177 (55.5%) |
| | Donation following Circulatory Death (DCD), n (%) | 14 (21.5%) | 74 (23.2%) |
| | Living donor (LD), n (%) | 33 (50.8%) | 68 (21.3%) |

Analysis: Data are presented as mean ± SD or n (%). Age distribution, dialysis vintage, preoperative Hgb, preoperative EF and preoperative cardiac abnormality are available only for the Study cohort. The Reference population reflects all kidney transplants performed at Cedars–Sinai Medical Center in 2024 derived from the OPTN registry and is provided for contextual comparison only (no inferential testing).

Source: Organ Procurement and Transplantation Network (OPTN) Data Reports, https://optn.transplant.hrsa.gov/data/view-data-reports/. Hgb = Hemoglobin, EF = ejection fraction

DD = deceased donor

LD = living donor.

a Dialysis vintage n = 54. Dialysis vintage status unknown in 3 recipients.

b Preoperative EF n = 55. Preoperative cardiac testing not performed in 10 recipients, 2/10 were age > 50.

c Preoperative cardiac abnormalities n = 55. Preoperative cardiac testing not performed in 10 recipients, 2/10 were age > 50. Of the 6 recipients with perfusion defects, all 6 had followup LHC, no recipients required further intervention following LHC.

**Table 6.** Circulation phenotype distribution and shift analysis.

| Phenotype | Initial n (%) [95% CI] | Average n (%) [95% CI] | Shift directions (Initial → Average) |
|---|---|---|---|
| Section A. Distribution by timepoint | | | |
| Hyperdynamic (HD) | 26 (40.0%) [28.9–51.9] | 21 (32.3%) [22.0–44.4] | From initial HD (n = 26): 21 → HD; 5 → N; 0 → PC |
| Normal (N) | 25 (38.5%) [27.7–50.4] | 26 (40.0%) [28.9–51.9] | From initial N (n = 25): 0 → HD; 18 → N; 7 → PC |
| Poor Contractility (PC) | 14 (21.5%) [13.2–33.1] | 18 (27.7%) [18.2–39.6] | From initial PC (n = 14): 0 → HD; 2 → N; 12 → PC |
| Total | 65 (100%) | 65 (100%) | — |

| Initial \ Average | Hyperdynamic (HD) | Normal (N) | Poor Contractility (PC) | Row total |
|---|---|---|---|---|
| Section B. Shift analysis (initial × average) | | | | |
| Hyperdynamic (HD) | 21 | 5 | 0 | 26 |
| Normal (N) | 0 | 18 | 7 | 25 |
| Poor Contractility (PC) | 0 | 3 | 11 | 14 |
| Column total | 21 | 26 | 18 | 65 |

Section **A**: 95% CIs are Wilson intervals for proportions.

Section **B**: McNemar–Bowker test of marginal homogeneity: $\chi^2(3) = 7.33$, $p = 0.062$.

**Table 7.** Hemodynamic shift within average circulation phenotypes (initial → average) primary inference is based on Holm-adjusted p-values; unadjusted p-values are presented for completeness

| Variable | Mean diff | 95% CI for mean diff | Primary p (test) | Adjusted p (Holm) | Effect size |
|---|---|---|---|---|---|
| 3A. Average Hyperdynamic (n = 21) | | | | | |
| Cardiac index (L/min/m$^2$) | +0.185 | −0.077 to +0.433 | 0.191 (Wilcoxon) | 0.382 | Rank-biserial r = +0.357 |
| SVRI (dyn·s·cm$^5$/m$^2$) | −9.52 | −121.95 to +102.91 | 0.862 (paired t) | 0.862 | Cohen's d = −0.039 |
| dP/dt (mmHg/s) | +112.54 | −14.06 to +239.13 | 0.079 (paired t) | 0.237 | Cohen's d = +0.405 |
| MAP (mmHg) | +2.50 | −4.55 to +9.54 | 0.468 (paired t) | 0.703 | Cohen's d = +0.161 |
| Variable | Mean diff | 95% CI for mean diff | Primary p (test) | Adjusted p (Holm) | Effect size |
| 3B. Average Normal (n = 26) | | | | | |
| Cardiac index (L/min/m$^2$) | −0.104 | −0.278 to +0.070 | 0.229 (paired t) | 0.687 | Cohen's d = −0.242 |
| SVRI (dyn·s·cm$^5$/m$^2$) | +81.18 | −45.59 to +207.94 | 0.199 (paired t) | 0.597 | Cohen's d = +0.259 |
| dP/dt (mmHg/s) | −17.70 | −109.88 to +74.48 | 0.696 (paired t) | 0.696 | Cohen's d = −0.078 |
| MAP (mmHg) | +0.856 | −4.05 to +5.76 | 0.722 (paired t) | 0.722 | Cohen's d = +0.071 |
| Variable | Mean diff | 95% CI for mean diff | Primary p (test) | Adjusted p (Holm) | Effect size |
| 3C. Average Poor Contractility (n = 18) | | | | | |
| Cardiac index (L/min/m$^2$) | −0.217 | −0.434 to −0.001 | 0.049 (paired t) | 0.196 | Cohen's d ≈ small |
| SVRI (dyn·s·cm$^5$/m$^2$) | +176.00 | −40.88 to +392.86 | 0.105 (paired t) | 0.315 | Cohen's d = +0.404 |
| dP/dt (mmHg/s) | +16.75 | −115.33 to +236.88 | 0.705 (Wilcoxon) | 0.705 | Rank-biserial r = +0.111 |
| MAP (mmHg) | −2.19 | −10.66 to +6.29 | 0.593 (paired t) | 0.593 | Cohen's d = −0.128 |

Primary tests: paired t-test when Shapiro–Wilk on differences > 0.05; Wilcoxon signed-rank when ≤ 0.05 or borderline. Multiple testing: Holm's step-down adjustment applied within phenotype across the four paired tests (CI, SVRI, dP/dt, MAP). Statistical interpretation is based on adjusted p-values. None of the within-phenotype shifts remained significant after Holm adjustment in the average-phenotype grouping.

**Table 8.** Secondary outcomes by circulation phenotype

| Outcome | Hyperdynamic | Normal | Poor contractility | Comparison across phenotypes |
|---|---|---|---|---|
| Delayed graft function (DGF) | 12/21 = 57.1% | 11/26 = 42.3% | 10/18 = 55.6% | $X^2$ p = 0.535<br>Cramer's V = 0.139 |
| Postoperative cardiac complications (any) | 1/21 = 4.8% | 4/26 = 15.4% | 4/18 = 22.2% | Fisher's exact p = 0.286; Cramer's V = 0.199 |
| Length of stay (LOS), days (median [IQR]) | 4 [2] | 4.5 [2] | 4.5 [1.75] | Kruskal–Wallis p = 0.869, $\varepsilon^2 \approx 0.005$ |
| Subtype | Hyperdynamic | Normal | Poor Contractility | Total |
| Demand ischemia | 0 | 1 | 0 | 1 |
| Atrial fibrillation | 0 | 0 | 2 | 2 |
| Heart failure | 1 | 2 | 1 | 4 |
| Hypotension | 0 | 1 | 1 | 2 |

Subtypes of postoperative cardiac complications (event counts by phenotype)

Statistical Analysis: Denominators reflect phenotype group sizes. Data are presented as n (%) or median [IQR]; means are reported for completeness. Categorical comparisons use Fisher's exact test due to small expected cell counts (< 5). Effect size for the "any cardiac complications" table is reported as Cramer's V. LOS compared using Kruskal–Wallis; $\varepsilon^2$ reported as a nonparametric effect size. Analyses were exploratory and underpowered, p-values are presented for context. DGF defined as dialysis within 7 postoperative days. Interpretation: DGF and cardiac complication rates are similar across phenotypes. LOS did not differ.

**Table 9.** Multinomial logistic regression: recipient factors associated with average circulation phenotype (n = 52)

| Predictor | OR | 95% CI | p-value |
|---|---|---|---|
| **Hyperdynamic vs Normal** | | | |
| Age (per 10 years) | 1.34 | 0.81–2.20 | 0.251 |
| Preoperative hemoglobin (per 1 g/dL) | 0.82 | 0.52–1.29 | 0.388 |
| Dialysis vintage (per year) | 1.10 | 0.82–1.46 | 0.531 |
| Ejection fraction (per 10%) | 0.59 | 0.21–1.67 | 0.321 |
| Preoperative cardiac abnormality (yes vs no) | 2.63 | 0.26–26.95 | 0.416 |
| **Poor Contractility vs Normal** | | | |
| Age (per 10 years) | 1.53 | 0.86–2.73 | 0.152 |
| Preoperative hemoglobin (per 1 g/dL) | 0.85 | 0.52–1.38 | 0.505 |
| Dialysis vintage (per year) | 1.24 | 0.92–1.67 | 0.163 |
| Ejection fraction (per 10%) | 0.68 | 0.23–1.96 | 0.470 |
| Preoperative cardiac abnormality (yes vs no) | 2.65 | 0.24–29.73 | 0.428 |

Scaling: age per 10 years; dialysis vintage per year (months/12); EF per 10%, preoperative cardiac abnormalities binary (yes vs no). Model fit: McFadden's $R^2 = 0.061$; AIC = 130. No recipient factor was statistically significant in either contrast.

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**Data availability** No datasets were generated or analysed during the current study.

## Declarations

**Ethics approval** The study was approved by the institutional review board; the requirement for written informed consent followed local policy for quality-improvement/low-risk perioperative monitoring research.

**Conflict of interest** The authors declare no competing interests.



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