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Association of Intraoperative Mean Perfusion Pressure with Acute Kidney Injury in Valve Replacement Surgeries: A Single-Center Analytical Cross-Sectional Study

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Abstract

Background: Acute kidney injury (AKI) is a frequent and serious complication following cardiac surgery involving cardiopulmonary bypass (CPB). Intraoperative perfusion pressure, particularly mean arterial pressure (MAP) during CPB, has been suggested as a potentially modifiable risk factor influencing postoperative renal outcomes. However, evidence regarding its association with AKI in valve replacement surgery remains limited. **Aim and Objectives:** To evaluate the association between intraoperative mean perfusion pressure and postoperative acute kidney injury in Valve Replacement patients. To explore the changes in value of mean arterial pressure (ΔMAP) from preoperative to intraoperative phases. To determine whether intraoperative MPP can serve as a predictor of postoperative acute kidney injury. **Materials and Methodology:** This Analytical Cross-Sectional Study included 56 adult patients who underwent valve replacement surgery with CPB between October 2025 and December 2025. Preoperative,

intraoperative, and postoperative data were collected from medical records. Intraoperative perfusion variables, including mean and lowest MAP during CPB, were recorded. Postoperative AKI was defined and staged according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria. Descriptive statistics were used to summarize patient characteristics. A p-value <0.05 was considered statistically significant. **Results:** A total of 56 patients who underwent valve replacement surgery with cardiopulmonary bypass and met the eligibility criteria were included in the study. Postoperative AKI occurred in 29(51.8%). According to KDIGO staging, 24(42.9%) developed stage 1 AKI, 2(3.6%) developed stage 2 AKI, and 3(5.4%) developed stage 3 AKI. Lower intraoperative perfusion pressure was associated with an increased incidence of postoperative AKI. On univariate logistic regression analysis, lowest intraoperative MPP during CPB showed significant results as a predictor of AKI (odds ratio [OR] = 0.914; 95% CI: 0.841–0.992; p = 0.032), indicating a higher risk of AKI with decreasing MPP values. Delta MAP did not show a statistically significant association with AKI. **Conclusion:** Lower mean arterial pressure during cardiopulmonary bypass was associated with a higher incidence of postoperative acute kidney injury in patients undergoing valve replacement surgery. Duration of MAP below 60 mmHg during CPB demonstrated a borderline association. Although increasing age and reduced postoperative urine output are also independent predictors of AKI. These findings highlight the importance of maintaining adequate perfusion pressure during CPB and closely monitoring high-risk patients to potentially reduce the risk of postoperative renal dysfunction.

Keywords: Acute Kidney Injury, Cardiopulmonary Bypass, Central Venous Pressure, Delta MAP, Mean Arterial Pressure , Mean Perfusion Pressure, Venous Congestion, Valve Replacement Surgeries

Introduction

Acute kidney injury (AKI) is one of the most common and serious complications following cardiac surgery, particularly in patients undergoing valve replacement procedures. Cardiac surgery-associated acute kidney injury (CSA-AKI) is linked with prolonged intensive care unit stay, increased healthcare costs, greater morbidity, and higher postoperative mortality (Hobson et al., 2009). Even mild postoperative renal dysfunction has been associated with poor short-term and long-term outcomes (Chertow et al., 2005). The incidence of AKI after cardiac surgery ranges from 20% to 40%, depending upon patient-related risk factors, surgical procedures, and diagnostic criteria used (Rosner & Okusa, 2006).

Valve replacement surgeries commonly require cardiopulmonary bypass (CPB), during which renal perfusion may be adversely affected because of non-pulsatile blood flow, hemodilution, inflammatory responses, oxidative stress, and ischemia-reperfusion injury (Lassnigg et al., 2004). Intraoperative hypotension and inadequate organ perfusion during CPB are considered important contributors to postoperative renal dysfunction. Among various hemodynamic parameters, mean perfusion pressure (MPP), defined as the difference between mean arterial pressure (MAP) and central venous pressure (CVP), is increasingly recognized as a better indicator of effective renal perfusion than MAP alone (Saito et al., 2016).

The kidneys possess autoregulatory mechanisms that maintain renal blood flow within a specific perfusion pressure range; however, prolonged reductions in MPP may impair renal oxygen delivery and lead to ischemic tubular injury (Prowle et al., 2015). Several studies have demonstrated an association between lower intraoperative perfusion pressures and increased incidence of postoperative AKI in cardiac surgery patients (Kanji et al., 2010). Nonetheless, the optimal target for intraoperative MPP during valve replacement surgery remains unclear, and evidence regarding its association with AKI is still limited and inconsistent.

Most previous research has focused predominantly on patients undergoing coronary artery bypass grafting (CABG), whereas fewer studies have specifically evaluated patients undergoing valve replacement surgeries. Moreover, limited evidence is available from single-center studies conducted in developing healthcare settings, where perioperative management practices and patient characteristics may differ substantially from those reported in Western populations.

Identifying modifiable intraoperative risk factors such as mean perfusion pressure is essential for improving renal outcomes in cardiac surgical patients. Better understanding of the relationship between intraoperative MPP and AKI may help clinicians optimize hemodynamic management and develop renal-protective strategies during valve replacement surgeries. Therefore, this single-center analytical cross-sectional study aims to evaluate the association between intraoperative mean perfusion pressure and the development of acute kidney injury in patients undergoing valve replacement surgeries.

Methodology

This analytical cross-sectional study was conducted to evaluate the association between intraoperative mean perfusion pressure (MPP) during cardiopulmonary bypass (CPB) and postoperative acute kidney injury (AKI) in patients undergoing valve replacement surgery. The study was carried out at the Armed Forces Institute of Cardiology /

National Institute of Heart Diseases, a tertiary care cardiac center in Rawalpindi, Pakistan, from October 2025 to December 2025. Data were collected from 56 adult patients undergoing valve replacement surgery.

Adult patients undergoing valve replacement surgery with CPB and fulfilling the eligibility criteria were included after informed consent. Inclusion criteria included patients aged 18 years or above, both male and female, undergoing isolated or combined valve replacement surgery using CPB. Patients with chronic kidney disease stage ⩾3, preoperative serum creatinine >2.0 mg/dL, combined valve replacement with CABG, off-pump cardiac surgery, or preoperative dialysis were excluded from the study. The study population was divided into two groups based on postoperative renal outcomes: patients who developed postoperative AKI and patients who did not develop postoperative AKI. The sample size was calculated using the WHO sample size calculator with 95% confidence level and 80% study power, resulting in 56 patients, with 28 patients in each group. A non-probability consecutive sampling technique was employed.

The primary exposure variable was intraoperative MPP calculated as:

$$MPP = MAP - CVP$$

The primary outcome variable was postoperative AKI diagnosed according to KDIGO criteria. Other variables included age, gender, body mass index, CPB duration, aortic cross-clamp time, hematocrit, urine output, and blood transfusion. Data were collected using a structured proforma including demographic, perioperative, and postoperative variables. Baseline MAP and CVP were recorded, and intraoperative MPP was calculated during CPB. Postoperatively, serum creatinine and urine output were monitored for 72 hours to diagnose AKI. Data were analyzed using IBM SPSS version 23. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, while categorical variables were presented as frequencies and percentages. The association between intraoperative MPP and postoperative AKI was assessed using the Mann–Whitney U test and logistic regression analysis. A p-value <0.05 was considered statistically significant.

Approval for the study was obtained from the Institutional Ethical Review Board of AFIC/NIHD, Rawalpindi. Written informed consent was obtained from all participants, and confidentiality was maintained by assigning coded identification numbers to patients.

RESULTS

A total of 56 patients who underwent valve replacement surgery between October 2025 and December 2025 and fulfilled the eligibility criteria were included in the study.

The mean age of the study population was 47.7 ± 14.48 years. Median body weight was 65 kg (58–75), while mean height and body mass index (BMI) were 150 ± 8.38 cm and 1.29 ± 0.19, respectively. The mean cardiopulmonary bypass (CPB) flow rate was 3.53 ± 0.44 L/min.

Table I: *Demographic Profile of Study Population (n=56)*

Variables	Mean ± SD
Age (years)	47.7 ± 14.48
Height (cm)	150 ± 8.38
BMI	1.29 ± 0.19
Flow rate (L/min)	3.53 ± 0.44

Among the participants, 29 (51.8%) were male and 27 (48.2%) were female. Mitral valve replacement (MVR) was the most common procedure performed in 24 (42.9%) patients, followed by aortic valve replacement (AVR) in 15 (26.8%) patients. Double valve replacement (DVR) was performed in 11 (19.6%) patients, while mini MVR, mini AVR, and Bentall procedures accounted for smaller proportions.

Preoperative assessment showed a median baseline serum creatinine of 0.99 mg/dL (0.75–1.10). Mean preoperative MAP was 94.69 ± 14.71 mmHg, while median CVP was 19.50 mmHg (12.25–28.75). Beta blockers 41 (73.2%) and diuretics 47 (83.9%) were the most frequently used medications preoperatively.

Table II: *Preoperative Baseline Assessment of Study Population (n=56)*

Preoperative Medications	Yes n (%)	No n (%)
ACE inhibitors	3 (5.4)	53 (94.6)
ARBs	11 (19.6)	45 (80.4)
Beta blockers	41 (73.2)	15 (26.8)
Antiplatelet medication	5 (8.9)	51 (91.1)
Diuretics	47 (83.9)	9 (16.1)
Anticoagulants	9 (16.1)	47 (83.9)
Inotropes/Vasopressors	1 (1.8)	55 (98.2)
Nitrates	6 (10.7)	50 (89.3)
Cardiac glycosides	10 (17.9)	46 (82.1)
Anti-allergic drugs	4 (7.1)	52 (92.9)
Antibiotics	6 (10.7)	50 (89.3)

Preoperative Medications	Yes n (%)	No n (%)
Lipid lowering drugs	11 (19.6)	45 (80.4)
Proton pump inhibitors	11 (19.6)	45 (80.4)

Variables	Mean ± SD
Lowest hemoglobin during CPB (g/dL)	9.02 ± 1.03
MAP mean during CPB (mmHg)	67.92 ± 5.75
Lowest MAP during CPB (mmHg)	43.96 ± 5.82
Delta MAP	26.76 ± 15.22

Hypertension was the most prevalent comorbidity, observed in 22 (39.3%) patients, followed by diabetes mellitus in 11 (19.6%). Previous general anesthesia exposure was present in 6 (10.7%) patients, while COPD and history of stroke were each present in 1 (1.8%) patient.

Intraoperative assessment demonstrated a median CPB duration of 150.5 minutes (112.0–193.25) and median aortic cross-clamp time of 104 minutes (74.25–140.75). Median mean perfusion pressure (MPP) during CPB was 57.0 mmHg (53.0–62.0), while the lowest MPP recorded was 37.0 mmHg (30.0–41.75). Mean MAP during CPB was 67.92 ± 5.75 mmHg, and the lowest MAP was 43.96 ± 5.82 mmHg. The total duration of MAP below 60 mmHg during CPB was 28.0 minutes (17.25–48.75).

Peak lactate during CPB had a median value of 3.05 mmol/L (2.40–4.22), while the lowest hemoglobin level during CPB was 9.02 ± 1.03 g/dL. Mean delta MAP was 26.76 ± 15.22 mmHg.

Table III: *Intraoperative Clinical and Perfusion Parameters (n=56)*

Variables	Median (IQR)
CPB duration (min)	150.5 (112.0–193.25)
X-clamp time (min)	104 (74.25–140.75)
CVP during CPB (mmHg)	9.0 (7.25–12.00)
Mean MPP during CPB (mmHg)	57.0 (53.0–62.0)
Lowest MPP during CPB (mmHg)	37.0 (30.0–41.75)
Total minutes MAP <60 mmHg during CPB	28.0 (17.25–48.75)
Peak lactate on CPB (mmol/L)	3.05 (2.40–4.22)

Postoperative assessment demonstrated a median post-CPB MAP of 76.0 mmHg (68.0–88.0). Median serum creatinine at 24 hours postoperatively was 1.21 mg/dL (0.89–1.59), while postoperative day 2 and day 3 creatinine levels were 1.12 mg/dL (0.86–1.61) and 1.01 mg/dL (0.76–1.56), respectively. Median peak serum creatinine within 7 postoperative days was 1.31 mg/dL (0.92–1.79).

Median urine output before CPB was 200 mL (100–400), increasing to 1550 mL (1100–2300) during CPB. Mean urine output during the first 24 postoperative hours was 4504.80 ± 997.43 mL. Renal replacement therapy was required in only 1 (1.8%) patient.

Table IV: *Postoperative Clinical Outcomes (n=56)*

Variables	Median (IQR)
Post-CPB MAP (first 30 min, mmHg)	76.0 (68.0–88.0)
Urine output pre-CPB (mL)	200 (100–400)
Urine output during CPB (mL)	1550 (1100–2300)
Serum creatinine peak 24h (mg/dL)	1.21 (0.89–1.59)
Creatinine postoperative day 2	1.12 (0.86–1.61)
Creatinine postoperative day 3	1.01 (0.76–1.56)
Serum creatinine peak 7 days (mg/dL)	1.31 (0.92–1.79)
Lactate first 30 min (mmol/L)	4.60 (3.10–7.02)

According to KDIGO staging, 27 (48.2%) patients did not develop AKI, while 24 (42.9%) developed KDIGO stage 1 AKI. KDIGO stage 2 and stage 3 AKI were observed in 2 (3.6%) and 3 (5.4%) patients, respectively. Overall, 29 (51.8%) patients developed postoperative AKI.

Patients who developed postoperative AKI had significantly lower intraoperative mean perfusion pressure compared to those without AKI [56 (50–58) vs 60 (56–62), p=0.00].

Table V: *Comparison of Intraoperative Mean Perfusion Pressure According to AKI Status (n=56)*

Variable	No AKI Median (IQR)	AKI Present Median (IQR)	p-value
Mean intraoperative MPP (mmHg)	60 (56–62)	56 (50–58)	0.00

Univariate and multivariable logistic regression analyses were performed to identify predictors of postoperative AKI. Increasing age was independently associated with postoperative AKI (AOR 1.07, 95% CI 1.01–1.15, p=0.024). Urine output during the first 24 postoperative hours was protective against AKI (AOR 0.99, 95% CI 0.99–1.00,

p=0.043). Total duration of MAP below 60 mmHg during CPB showed a borderline significant association with AKI (AOR 1.06, 95% CI 0.99–1.12, p=0.055).

Table VI: *Independent Predictors of Postoperative AKI (n=56)*

Variable	UOR	95% CI	p-value	AOR	95% CI	p-value
Age	1.069	1.02–1.11	0.00*	1.07	1.01–1.15	0.024
Gender (male)	0.565	0.19–1.62	0.29	—	—	—
Hypertension	0.833	0.28–2.44	0.74	—	—	—
CPB time	1.006	0.99–1.01	0.22	—	—	—
X-clamp time	1.003	0.99–1.01	0.63	—	—	—
Total minutes MAP <60 mmHg	1.065	1.02–1.10	0.00*	1.06	0.99–1.12	0.055
Delta MAP	1.02	0.98–1.05	0.28	—	—	—
Lowest Hb during CPB	0.769	0.45–1.30	0.32	—	—	—
Peak lactate during CPB	1.275	0.88–1.84	0.19	—	—	—
Postoperative lactate	1.506	1.14–1.98	0.00*	—	—	—
Postoperative HCT	0.84	0.70–1.00	0.05*	—	—	—
Baseline serum creatinine	7.291	0.65–80.67	0.10	—	—	—
Urine output first 24 hrs	0.999	0.99–1.00	0.00*	0.99	0.99–1.00	0.043

Patients who developed AKI had significantly lower intraoperative mean perfusion pressure compared with those without AKI. Increasing age and reduced postoperative urine output were identified as independent predictors of postoperative AKI, while prolonged duration of MAP below 60 mmHg during CPB demonstrated a borderline significant association.

DISCUSSION

Our single-center analysis found that postoperative AKI occurred in 29(51.8%) of valve replacement patients. Crucially, lower intraoperative mean perfusion pressure (MPP) was significantly associated with AKI (p=0.009), and multivariate analysis identified older age and reduced urine output as independent predictors. These findings suggest that hemodynamic management during cardiopulmonary bypass (CPB) strongly influences renal outcomes in this population. In particular, maintaining adequate perfusion pressure appears to be an important, modifiable factor in mitigating AKI risk.

The AKI incidence we observed 29(51.8%) is at the upper end of previously reported ranges (typically 5–30%) (Kanji et al. 2010). Differences in patient mix, AKI definitions, and surgical complexity may explain this disparity. Nevertheless, our

identified risk factors align with the literature. For example, age is a well-known predictor of renal injury – Hu et al. noted that patients older than 65 were more likely to develop postoperative AKI (Hu et al. 2020). Similarly, intraoperative oliguria (low urine output) often reflects poor renal perfusion and has been associated with worse outcomes. These parallels with established risk factors support the validity of our findings and suggest our cohort is comparable to other high-risk groups.

Our finding that lower perfusion pressure correlates with AKI is consistent with prior studies. Hu et al. (2020) reported that prolonged reductions in MPP during bypass were independently associated with AKI. Kanji et al. (2010) likewise found that a large intraoperative MAP drop ($\geqslant$26 mmHg below baseline) nearly tripled AKI odds (OR$\approx$2.8). Similarly, Velho et al. showed that even brief periods of MAP <50 mmHg on CPB significantly increased postoperative AKI risk (Velho et al. 2022). Taken together, these studies underscore that inadequate arterial perfusion – especially relative to a patient's baseline – predisposes to renal injury. Our results reinforce this link by demonstrating a statistically significant association between lower intraoperative MPP and AKI incidence.
Physiologically, the kidneys depend on the pressure gradient between the arterial inflow and venous outflow to drive filtration. Renal perfusion pressure (RPP) is essentially the difference between MAP and renal venous pressure. Under normal conditions, intrinsic autoregulatory mechanisms adjust vascular tone to keep renal blood flow and GFR relatively constant over a wide range of RPP (Panwar et al. 2025). However, during CPB and critical illness, autoregulation can be impaired. If MPP falls below the auto regulatory threshold, renal blood flow and glomerular filtration rate decline, leading to ischemic injury in the renal cortex and medulla (Patel et al. 2015). In practical terms, prolonged or severe hypotension during bypass often drops MPP into a range where the kidney cannot compensate. Compounding this, CPB-related factors such as nonpulsatile flow, hemodilution, hypothermia, and inflammation further reduce oxygen delivery to renal tissue. As noted in one study, episodes of MAP below typical targets (55–60 mmHg) – especially when coupled with anemia or volume shifts – are dangerous because they exceed the limits of auto regulation (Patel et al. 2015). Thus, low intraoperative MPP directly translates into renal hypoperfusion and tubular injury, providing a plausible mechanism for the associations we observed.

These findings carry clear clinical implications. They suggest that careful monitoring and optimization of perfusion pressure on CPB could mitigate AKI risk. In practice, this means avoiding sustained hypotension and considering patient-specific targets. For instance, Hu et al. emphasized that maintaining MAP at or near a patient's baseline on bypass is important for kidney protection (Hu et al. 2020). Likewise, recent

perioperative AKI guidelines note that "optimizing renal perfusion via maintenance of systemic blood pressure may decrease the risk" of AKI (Balakrishna et al. 2023). Therefore, perfusion teams should use vasopressors or pump flow adjustments to keep MAP (and thus MPP) within an adequate range during bypass, especially in older or hypertensive patients whose kidneys may demand higher perfusion pressures. Monitoring CVP (when possible) is also useful, since elevated venous pressure markedly reduces MPP. A goal-directed perfusion strategy – for example, titrating blood pressure to ⩾10–20% above baseline or avoiding MAP <60–65 mmHg – might improve renal outcomes. While definitive targets require further research, our data reinforce the concept that "pressure-guided" perfusion management is an actionable approach to prevent CSA-AKI.(Balakrishna et al. 2023; Hu et al. 2020)

Conclusion

Lower intraoperative mean perfusion pressure was linked to the occurrence of postoperative acute kidney injury in patients undergoing valve replacement surgery. Duration of MAP below 60 mmHg during CPB demonstrated a borderline association. Although increasing age and reduced postoperative urine output are also independent predictors of AKI. These findings emphasize the importance of optimizing intraoperative perfusion strategies and closely monitoring high-risk patients to potentially improve renal outcomes following cardiopulmonary bypass.

Limitations

This study has several limitations. The single-center, cross-sectional design limits causal inference and generalizability. With a relatively small sample of valve surgeries, the study may be underpowered to detect some associations and may not reflect outcomes in other cardiac surgery populations (e.g. CABG or combined procedures). Potential confounders – such as pre-existing chronic kidney disease, use of nephrotoxic drugs, or intraoperative transfusions – may not have been fully controlled, which could bias results. The observational methodology also means we can only report associations; we cannot prove that low MPP causes AKI or that intervening on MPP will change outcomes. Finally, urine output and creatinine changes are imperfect and sometimes delayed markers of renal injury. Despite these limitations, the strong association we observed between perfusion pressure and AKI provides a valuable hypothesis-generating insight.

Future Recommendations

Larger multicenter studies should be conducted to validate the association between intraoperative mean perfusion pressure and postoperative acute kidney injury in valve replacement surgeries. Including a larger and more diverse patient population would improve the generalizability of results.

The role of central venous pressure and venous congestion in the development of AKI should be examined in greater detail. Studies focusing on strategies to reduce elevated CVP during CPB, such as optimized fluid management and ultrafiltration, may help improve renal perfusion and outcomes.

Future research should incorporate advanced renal biomarkers such as neutrophil gelatinase-associated lipocalin (NGAL) and cystatin C to detect early or subclinical kidney injury. These biomarkers may help clarify the temporal relationship between perfusion pressure changes and renal damage.

Long-term follow-up studies are recommended to assess the impact of intraoperative perfusion pressure on long-term renal function, hospital length of stay, and mortality, as this study focused primarily on early postoperative outcomes.

REFRENCES

1. Acute Kidney Injury (AKI) – KDIGO. n.d. Retrieved February 1, 2026. https://kdigo.org/guidelines/acute-kidney-injury/.
2. Azau, A., P. Markowicz, Jj Corbeau, C. Cottineau, X. Moreau, C. Baufreton, and L. Beydon. 2014. "Increasing Mean Arterial Pressure during Cardiac Surgery Does Not Reduce the Rate of Postoperative Acute Kidney Injury." *Perfusion* 29(6):496–504. doi:10.1177/0267659114527331.
3. Balakrishna, Aditi, Jeremy Walco, Frederic T. Billings, and Marcos G. Lopez. 2023. "Perioperative Acute Kidney Injury." *Advances in Anesthesia* 41(1):205–24. doi:10.1016/j.aan.2023.06.005.
4. Bellomo, Rinaldo, John A. Kellum, and Claudio Ronco. 2012. "Acute Kidney Injury." *The Lancet* 380(9843):756–66. doi:10.1016/S0140-6736(11)61454-2.
5. Chertow, Glenn M., Elliott M. Levy, Karl E. Hammermeister, Frederick Grover, and Jennifer Daley. 1998. "Independent Association between Acute Renal Failure and Mortality Following Cardiac Surgery." *The American Journal of Medicine* 104(4):343–48. doi:10.1016/S0002-9343(98)00058-8.
6. Demirjian, Sevag, Faisal Bakaeen, W. H. Wilson Tang, Chase Donaldson, Jon Taliercio, Anne Huml, Crystal A. Gadegbeku, A. Marc Gillinov, and Steven Insler. 2024. "Hemodynamic Determinants of Cardiac Surgery-Associated Acute Kidney Injury." *Critical Care Explorations* 6(4):e1063. doi:10.1097/CCE.0000000000001063.
7. Ge, Yunfen, Tapas Ranjan Behera, Ming Yu, Shuyang Xie, Yue Chen, Hui Mao, Qiong Xu, Yu Zhao, Shuijun Zhang, and Quanquan Shen. 2022. "Higher Mean Arterial Pressure during Cardiopulmonary Bypass May Not Prevent Acute Kidney Injury in Elderly Patients Undergoing Cardiac Surgery" edited by P. Scicchitano. *International Journal of Clinical Practice* 2022(1):7701947. doi:10.1155/2022/7701947.

8. Hobson, Charles E., Sinan Yavas, Mark S. Segal, Jesse D. Schold, Curtis G. Tribble, A. Joseph Layon, and Azra Bihorac. 2009. "Acute Kidney Injury Is Associated With Increased Long-Term Mortality After Cardiothoracic Surgery." *Circulation* 119(18):2444–53. doi:10.1161/CIRCULATIONAHA.108.800011.
9. Hu, Raymond, Yasmean Kalam, Jeremy Broad, Tim Ho, Frank Parker, Matthew Lee, and Rinaldo Bellomo. 2020. "Decreased Mean Perfusion Pressure as an Independent Predictor of Acute Kidney Injury after Cardiac Surgery." *Heart and Vessels* 35(8):1154–63. doi:10.1007/s00380-020-01578-0.
10. Kanji, Hussein D., Costas J. Schulze, Marilou Hervas-Malo, Peter Wang, David B. Ross, Mohamad Zibdawi, and Sean M. Bagshaw. 2010. "Difference between Pre-Operative and Cardiopulmonary Bypass Mean Arterial Pressure Is Independently Associated with Early Cardiac Surgery-Associated Acute Kidney Injury." *Journal of Cardiothoracic Surgery* 5(1):71. doi:10.1186/1749-8090-5-71.
11. Kotani, Yuki, Takuo Yoshida, Junji Kumasawa, Jun Kamei, Akihisa Taguchi, Koji Kido, Naoki Yamaguchi, Takayuki Kariya, Masato Nakasone, Noriko Mikami, Takahiro Koga, Izumi Nakayama, Mami Shibata, Tomonao Yoshida, Hiroshi Nashiki, Shinsuke Karatsu, Kazutaka Nogi, Natsuko Tokuhira, and Junichi Izawa. 2021. "The Impact of Relative Hypotension on Acute Kidney Injury Progression after Cardiac Surgery: A Multicenter Retrospective Cohort Study." *Annals of Intensive Care* 11(1):178. doi:10.1186/s13613-021-00969-4.
12. Mean arterial pressure - Reference.org. n.d. Retrieved February 1, 2026. https://reference.org/facts/mean_arterial_pressure/5q1fu4x9.
13. Najjar, Marc, Michael Salna, and Isaac George. 2015. "Acute Kidney Injury after Aortic Valve Replacement: Incidence, Risk Factors and Outcomes." *Expert Review of Cardiovascular Therapy* 13(3):301–16. doi:10.1586/14779072.2015.1002467.
14. Noe, Khin M., Andrea Don, Andrew D. Cochrane, Michael Z. L. Zhu, Jennifer P. Ngo, Julian A. Smith, Amanda G. Thrift, Johnny Vogiatjis, Andrew Martin, Rinaldo Bellomo, James McMillan, and Roger G. Evans. 2023. "Intraoperative Hemodynamics and Risk of Cardiac Surgery-associated Acute Kidney Injury: An Observation Study and a Feasibility Clinical Trial." *Clinical and Experimental Pharmacology and Physiology* 50(11):878–92. doi:10.1111/1440-1681.13812.
15. Ostermann, Marlies, Anna Hall, and Siobhan Crichton. 2017. "Low Mean Perfusion Pressure Is a Risk Factor for Progression of Acute Kidney Injury in Critically Ill Patients – A Retrospective Analysis." *BMC Nephrology* 18(1):151. doi:10.1186/s12882-017-0568-8.

16. Panwar, Rakshit, Bairbre McNicholas, J. Pedro Teixeira, and Amit Kansal. 2025. "Renal Perfusion Pressure: Role and Implications in Critical Illness." *Annals of Intensive Care* 15(1):115. doi:10.1186/s13613-025-01535-y.
17. Patel, Ramesh, Atul Solanki, Hasmukh Patel, Himani Pandya, Jignesh Patel, and Sanjay Patel. 2015. "DOES MEAN PERFUSION PRESSURE DURING CARDIOPULMONARY BYPASS AFFECT RENAL FUNCTION?" *Journal of Evidence Based Medicine and Healthcare* 2(41):6946–53. doi:10.18410/jebmh/2015/948.
18. Rosner, Mitchell H., and Mark D. Okusa. 2006. "Acute Kidney Injury Associated with Cardiac Surgery." *Clinical Journal of the American Society of Nephrology* 1(1):19–32. doi:10.2215/CJN.00240605.
19. Sato, Ryota, Sarah Kyuragi Luthe, and Michitaka Nasu. 2017. "Blood Pressure and Acute Kidney Injury." *Critical Care* 21(1):28. doi:10.1186/s13054-017-1611-7.
20. Shah, Parth, and Martine A. Louis. 2025. "Physiology, Central Venous Pressure." in *StatPearls*. Treasure Island (FL): StatPearls Publishing.
21. Sun, Louise Y., Duminda N. Wijeysundera, Gordon A. Tait, and W. Scott Beattie. 2015. "Association of Intraoperative Hypotension with Acute Kidney Injury after Elective Noncardiac Surgery." *Anesthesiology* 123(3):515–23. doi:10.1097/ALN.0000000000000765.
22. Velho, Tiago R., Rafael M. Pereira, Nuno C. Guerra, Hugo Ferreira, André Sena, Ricardo Ferreira, and Ângelo Nobre. 2022. "Low Mean Arterial Pressure During Cardiopulmonary Bypass and the Risk of Acute Kidney Injury: A Propensity Score Matched Observational Study." *Seminars in Cardiothoracic and Vascular Anesthesia* 26(3):179–86. doi:10.1177/10892532211045765.
23. Wang, Lei, Lanxin Hu, Qiong Yan Dai, HaoYu Qi, ZhenHong Wang, and Xin Chen. 2024. "Intraoperative Central Venous Pressure during Cardiopulmonary Bypass Is an Alternative Indicator for Early Prediction of Acute Kidney Injury in Adult Cardiac Surgery." *Journal of Cardiothoracic Surgery* 19(1):262. doi:10.1186/s13019-024-02734-7.
24. Yang, Xutao, Li Zhu, Hong Pan, and Yi Yang. 2024. "Cardiopulmonary Bypass Associated Acute Kidney Injury: Better Understanding and Better Prevention." *Renal Failure* 46(1):2331062. doi:10.1080/0886022X.2024.2331062.