Cardiac surgery–associated (CSA) acute kidney injury (AKI) is a common and serious complication that contributes to a significantly increased morbidity and mortality. Cardiac output, hemoglobin level, vascular tone, blood pressure, and microcirculatory competence are the physiologic determinants of oxygen delivery to the kidney. During and after cardiac surgery, patients frequently experience hemodynamic instability, manifested by perturbations in cardiac output and blood pressure. This may be due to changes in circulating blood volume, anemia, and systemic vascular resistance, and a reduced cardiac output may be caused by myocardial stunning. The reduced circulating blood volume may be caused by blood loss, fluid shifts, and dilution. Changes in systemic vascular resistance may be a consequence of hypotension and a systemic inflammatory response syndrome triggered by the surgery itself. Short episodes of hypotension in the perioperative period are associated with end-organ injury, including AKI. Reduction in blood pressure of previously normotensive or hypertensive patients may be associated with the subsequent development of AKI. Optimizing hemodynamics and volume status may therefore reduce the occurrence of CSA-AKI. Recently, the PrevAKI trial demonstrated that the implementation of a bundle of supportive measures in high-risk patients, as identified by urinary biomarkers, significantly reduced the rate of CSA-AKI. In this issue of the Journal, Johnston and colleagues present the results of an observational retrospective cohort trial investigating whether a goal-directed resuscitation protocol with fluid and vasoactive agents could reduce the occurrence of CSA-AKI. In this before-and-after design study, Johnston and colleagues used a standardized protocol to achieve quantifiable physiologic goals. In the post-implementation period, a multivariable analysis demonstrated a 37% reduction in the odds of AKI after implementation of a goal-directed resuscitation protocol centered on patient fluid responsiveness. Secondary outcomes, including operative mortality, however, were not different between the groups.

One major limitation is the study design. Before-and-after study designs are inherently susceptible to bias, including selection and assignment, Hawthorne effect, test-retest, and regression to the mean bias, which are subtle but can invalidate conclusions. Another limitation is that the protocol was applied to every patient, and some patients may have received unnecessary fluids that could, in fact, contribute to AKI. Although several trials and an expert group and guidelines support the use of goal-directed resuscitation after cardiac surgery, the use of such protocols should be individually tailored. Combining the protocol with kidney stress biomarkers would allow patients with negative biomarker results to “fast track” without extra fluid. The AKI reduction might have been even more significant had the protocol been used only in patients with significant had the protocol been used only in patients with positive biomarker results. Before widespread adoption, goal-directed volume resuscitation protocols need to be validated in larger, multicenter trials with heterogeneous patient populations.

References


