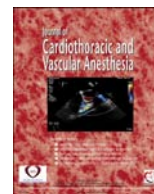




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Original Article

Elevated Pulse Pressure, Intraoperative Hemodynamic Perturbations, and Acute Kidney Injury After Coronary Artery Bypass Grafting Surgery

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Objective: To explore whether baseline pulse pressure (PP) confers an increased risk for acute kidney injury (AKI) independent of intraoperative hypotension or hypertension in patients who undergo coronary artery bypass grafting (CABG) surgery.

Design: Retrospective study.

Setting: Single academic center.

Participants: 5,808 patients who underwent CABG surgery.

Measurements and Main Results: Baseline arterial blood pressure was defined as the mean of the first 5 measurements recorded by the automated record keeping system before anesthesia was induced. Weighted duration of intraoperative hypotension and hypertension were defined as the area (min × mmHg) below a mean arterial pressure of 55 mmHg and above a mean arterial pressure of 100 mmHg. Multivariable logistic and proportional odds regression analyses were performed to determine whether baseline PP and weighted duration of intraoperative hypotension and hypertension were independently associated with postoperative AKI. Of the 5,808 patients, PP was < 40 mmHg in 90 (1.6%), 40-to-80 mmHg in 2,463 (42.4%), and > 80 mmHg in 3,255 (56%) patients. The incidence of AKI was 57.7%, which included 7.4% (249 patients) and 2.8% (93 patients) who experienced stages 2 and 3 AKI, respectively. In the risk-adjusted analyses, baseline PP was associated with higher odds for postoperative AKI (odds ratio for every 20 mmHg increase in PP, 1.15; 95% confidence interval 1.10-1.21; $p < 0.0001$) and a higher severity of postoperative AKI (proportional odds ratio, 1.13; 95% confidence interval 1.03-1.24; $p = 0.0098$). There was no evidence that weighted duration of intraoperative hypotension or hypertension was associated with postoperative AKI or that either interacted with the association of baseline PP with AKI.

Conclusions: Baseline PP was significantly associated with postoperative AKI after CABG surgery, independent of weighted duration of intraoperative hypotension or hypertension.

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Key Words: acute kidney injury; coronary artery bypass grafting surgery; intraoperative hypotension; pulse pressure

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CHRONIC SYSTOLIC AND diastolic hypertension have been associated with varying risks of perioperative cardiovascular events and mortality.¹ Nevertheless, in patients with medically refractory coronary artery disease or valvular heart disease, the primary goal of ambulatory blood pressure (BP) management is to reduce myocardial ischemic burden, stroke

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risk, and symptoms of heart failure. Of importance, no guideline exists for preoperative systolic and diastolic BP control (potentially modifiable risk factors), specifically aimed at reducing surgical morbidity after coronary artery bypass grafting (CABG) surgery.

The influence of elevated preoperative pulse pressure (PP) on major vascular complication after surgery also has been underappreciated and inadequately investigated. In the setting of CABG surgery, it previously was reported that PP²⁻⁴ was a stronger predictor of mortality and cerebral and renal dysfunction/renal failure events compared with systolic or diastolic BP. Both widened PP and isolated systolic hypertension are considered markers of arterial stiffness, which is reflective of macrovascular disease.⁵ The resulting repetitive pulsatile load, coupled with a lower diastolic perfusion pressure and relative intravascular volume depletion, form a pathophysiologic basis for perioperative organ injury, including postoperative acute kidney injury (AKI).^{6,7}

On the other hand, there is some evidence that intraoperative arterial hypertension and hypotension are associated with cardiovascular and neurologic complications and mortality after noncardiac^{8,9} and cardiac surgeries.^{10,11} Moreover, acute perturbations in BP can be “modifiable” and influenced by the anesthesiologist with anesthetic agents and therapies with vasoactive and cardioactive drugs or volume administration. Nonetheless, there is an ongoing dispute regarding what should be “optimal” targets for intraoperative systolic, diastolic, and mean arterial BP management. Indeed, BP is a complex physiologic parameter reflecting distinct information about the macrovasculature, the microvasculature, and cardiac performance that is not readily discernable using conventional BP monitoring. Therefore, the objective of this study was to determine whether elevated preoperative PP was significantly associated with postoperative AKI after CABG surgery, independent of the duration and extent of intraoperative hypotension or hypertension.

Methods

Study Population

The Institutional Review Board for Clinical Investigations at the authors' institution approved this study and waived the requirement for informed consent. Patients in this study were selected from a previously analyzed study of 6,130 patients who underwent CABG surgery with cardiopulmonary bypass (CPB) at a tertiary academic center between January 1, 2001, and July 18, 2015.^{12,13} The objectives of the previous study was to determine whether a decrease in platelet counts after CABG surgery was independently associated with an increased risk for postoperative AKI, mortality, and stroke, adjusted for a set of patient- and procedure-related predictors. Of importance, the association between elevated preoperative PP, independent of the duration and extent of intraoperative hypotension or hypertension, with postoperative AKI after CABG surgery has not been studied.

In brief, the data set for the parent study was constructed from the institution's perioperative electronic database (Innovian Anesthesia; Draeger Medical Inc., Telford, PA); cardiac surgery quality assurance database; databank for cardiovascular diseases; MAESTRO CARE; and an institutional specific platform-based electronic health record (Epic Systems Corporation, Verona, WI) to determine whether perioperative anemia is associated with an increased risk for postoperative AKI and mortality in patients undergoing CABG surgery. Patients in the parent study (1) were 18 years or older, (2) had undergone a CABG surgery with CPB, (3) had no additional procedures during the same study period, and (4) had complete data for preoperative and postoperative hemoglobin concentration.

In the present study, exclusion criteria were as follows: (1) missing information on preinduction systolic and diastolic BP, (2) patients with missing or interrupted intraoperative mean arterial pressure (MAP) monitoring at least 15 minutes or longer, and (3) patients with an unconfirmed intraoperative recording of their vital signs, including their intraoperative MAP monitoring. A total of 5,808 patients were included in the current analyses.

Data Collection and Clinical Risk Factors

For the present study, data on potential determinants of postoperative AKI were collected, including patient characteristics, preoperative and intraoperative cardiovascular medication use, antifibrinolytics, CPB and aortic cross-clamp time, insertion of intraoperative balloon pump, intraoperative and postoperative blood transfusions, preoperative and postoperative serum creatinine values, hemoglobin concentrations and platelet counts, and intraoperative MAP values.

Per institutional protocol, serum creatinine was measured preoperatively in the institution's clinical pathology laboratory and for the first 10 days postoperatively or until discharge, whichever came first, using a method previously described.¹⁴

Baseline BP was defined as the average of the first 5 intraoperative values recorded when the patient was awake and mildly sedated but before anesthesia induction. These values were obtained invasively through an arterial catheter, and, in cases in which this was not available, noninvasively through oscillometric measurements. The mean systolic BP, mean diastolic BP, and PP (mean systolic BP minus mean diastolic BP) were calculated for each patient as described previously.¹⁵

Intraoperative anesthetic, perfusion, and cardioprotective techniques were standardized as described previously.¹⁶ Intraoperative MAP values were recorded at 1-minute intervals. As described previously,¹⁷ MAP values were considered artifactual and were excluded when the recorded value was < 30 mmHg or > 250 mmHg. To better quantify the severity and duration of intraoperative hypotension or hypertension based on MAP, the authors computed the weighted duration of intraoperative hypotension or hypertension, which was defined as the area (minutes × mmHg) below a mean arterial pressure of 55 mmHg or above a mean arterial pressure of 100 mmHg,

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