Second, we identified an error in their Table 1.[1] In the "All" column, the sum of the number of patients from all of the age groups should be 468 386, not 466 386.

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In Reply We appreciate the thoughtful letter by Wada et al in response to our study.[1] In Veterans Affairs Surgical Quality Improvement Program (VASQIP)–assessed cases, data for risk assessment and postoperative events are obtained via 1 of 2 methods. The majority of data are collected manually by a surgical quality nurse, including preoperative, intraoperative, and 30-day postoperative outcome data. The manually collected data are augmented by automatic data transmission from other VA information systems, including demographic characteristics, admission dates, and laboratory values. Of the 305 data fields collected in a VASQIP-assessed case, 82 variables are used for risk prediction and outcome measurement. These fields, when missing, are filled using an IVEWare multiple imputation program.[2] Overall, 47 preoperative variables (57.3%) are collected manually, 12 intraoperative variables (14.6%) are collected automatically, and 23 postoperative variables (28.0%) are collected manually.

Prior to imputation, missing data on manually collected variables range from 0.00% to 0.01%, and missing data on automatically collected laboratory values range from 6.3% (serum creatinine) to 29.2% (serum albumin). On examination of the impact of imputed values, automatically collected albumin levels were abnormal in 19.9% of cases, and imputed albumin levels were abnormal in 20.3% of cases. This demonstrates that the imputation procedure does not overpopulate the imputed values with normal laboratory results. Therefore, the majority of data undergoing imputation are a small subset of automatically collected variables (exclusively laboratory values) that have a minimal effect on VASQIP risk adjustment and on this analysis of *Clostridium difficile*.

Thank you for identifying the error in our Table 1. The number (percentage) of patients in the age group of younger than 50 years should be corrected to reflect 79 694 (17.0%), maintaining a total number of 468 386 patients from all of the age groups combined.

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Perioperative Acute Kidney Injury: Prevention Rather Than Cure

To the Editor In a single-center cohort study of 3646 patients who underwent inpatient vascular surgery, 2000-2010, at a tertiary care teaching hospital, Huber et al[1] had demonstrated that perioperative acute kidney injury (AKI) occurred in 1801 patients (49.4%). Furthermore, adjusted cardiovascular mortality estimates at 10 years were 17%, 31%, 30%, and 41%, respectively, for patients with no kidney disease, AKI without chronic kidney disease (CKD), CKD without AKI, and AKI with CKD.[1] Moreover, adjusted hazard ratios (HRs) and 95% CIs for cardiovascular mortality were significantly elevated among patients with AKI without CKD (HR, 2.07 [95% CI, 1.74-2.45]), CKD without AKI (HR, 2.01 [95% CI, 1.46-2.78]), and AKI with CKD (HR, 2.99 [95% CI, 2.37-3.78]) and were higher than those for other risk factors, including increasing age (HR, 1.03 per 1-year increase [95% CI, 1.02-1.04 per 1-year increase]), and emergent surgery (HR, 1.47 [95% CI, 1.27-1.71]).[1] The authors had very appropriately called for a more rigorous pursuit of preoperative and postoperative risk stratification for kidney disease and the implementation of strategies now available to help prevent perioperative AKI.

While agreeing and being in total support of the later recommendations, I posit that intraoperative hypotension (IOH), a clearly preventable clinical condition, remains, most fortuitously, a neglected causative yet avoidable cause of postoperative AKI.[2,3] In the cognate AKI-surgery literature, there is this generally held consensus that IOH following hypotensive anesthesia or controlled hypotension in the operating room has no significant short- or long-term effect on renal function.[4] Nevertheless, Sun et al,[5] in a recent prospective cohort study of the association of IOH with AKI in 5127 patients undergoing noncardiac surgery with invasive mean arterial pressure monitoring and a length of stay of 1 or more days in the hospital, had established that AKI occurred in 324 patients (6.3%) and was associated with a mean arterial pressure of 60 mm Hg

for 11 to 20 minutes and 50 mm Hg for more than 10 minutes in a clearly graded fashion. As a colleague and I have shown in a recent review,[2] IOH is indeed a modifiable yet preventable causative factor in producing perioperative AKI. Thus, while again agreeing with Huber et al[1] on the importance of preoperative risk stratification for patients with kidney disease and a more rigorous follow-up of such patients to reduce postoperative morbidity and mortality, I strongly contend that more efforts to reduce the level of IOH, if not totally eliminate the condition, in the operating room would indeed pay more dividends in a priori preventing perioperative AKI and its consequent sequele.[2,3] Prevention is better than cure.[3]

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In Reply We thank Dr Onuigbo for his comments on our study.[1] We agree that identification—and thus prevention—of intraoperative hypotension is a crucial part of the equation in preventing acute kidney injury in surgical patients, especially for patients with known vascular disease. The risk for any postoperative complication, including acute kidney injury, arises from the complex interactions between preoperative status and a patient's physiologic capacity to withstand the stress of surgery.[2] In the preoperative period, a complete accounting of health status and comorbidities can facilitate a discussion with the patient about the risks and benefits of surgery, and can help health care professionals plan for perioperative management to minimize the chance for adverse outcomes. An accurate risk assessment allows physicians to identify patients who would benefit the most from intraoperative strategies that can offset any increased risk. Some of these strategies, such as invasive hemodynamic monitoring, are not only costly but carry their own risks. Others, such as the avoidance of nephrotoxic medications in patients at risk for acute kidney injury, are easy to implement if the risk is identified. And yet both are often applied without consideration of a patient's preoperative risk or are not applied at all because risk is underestimated.

For many patients, but especially for those with vascular disease, appropriate intraoperative hemodynamic and anesthetic management can help to ameliorate surgery-related stress.[3] Furthermore, the opportunity to initiate immediate postoperative preventive therapies, such as triage to the intensive care unit for ongoing hemodynamic optimization, initiation of a sepsis protocol, and monitoring for intra-abdominal hypertension, among others, may be missed. Novel analytic methods such as machine learning analysis and prediction, to assess the risk for complications in real time using a dynamic synthesis of the large amount of clinical information generated during surgery and throughout the continuum of perioperative care, are urgently needed.[4]

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Management of Acute Appendicitis, Comparative Effectiveness Research, and the Nuances of Study Design

To the Editor I commend Minneci and colleagues[1] for taking an efficacious treatment and studying its effectiveness. They developed a unique study design in which patients were not randomly assigned to a treatment but were prospectively enrolled (if they met inclusion criteria) and then allowed to choose which treatment they preferred. This very likely aligns with the "real-world" nonoperative management of appendicitis, and experts have called for such investigations so that we may begin to understand whether outcomes are meaningfully improved when an antibiotics-first approach is compared with a therapy as safe and definitive as laparoscopic appendectomy.[2]

The difference in the rates of complicated appendicitis between patients who chose nonoperative management and