increase in hospital mortality (odds ratio [OR], 1.27; 95% CI, 1.06-1.51; $P = .008$), postoperative infection (OR, 1.21; 95% CI, 1.09-1.35; $P < .001$), stage 3 AKI (OR, 1.34; 95% CI, 1.16-1.55; $P < .001$), prolonged intensive care unit length of stay (OR, 1.14; 95% CI, 1.05-1.24; $P = .002$), and hospital length of stay (OR, 1.11; 95% CI, 1.08-1.13; $P < .001$). The authors conclude that even small changes in creatinine levels before surgery have the potential to cause major adverse events and warrant increased awareness during the perioperative period.

Griffin and colleagues⁴ should be commended for their investigation into this novel predictor, yet questions remain about the accuracy in their determination of creatinine change. No data were available, unfortunately, regarding the circumstances around which the baseline serum creatinine value was drawn. Many factors can influence serum creatinine levels, including volume status, medications, diagnostic testing, and illness acuity. Further, the time frame between measurement of the baseline level and the preoperative creatinine measurement may also have important implications. For example, a patient's documented baseline creatinine level following a cardiac catheterization may represent a different state than his or her true baseline. If a patient were to undergo surgery soon thereafter, the preoperative level may not have normalized. This would make his or her creatinine change relatively minimal and thus confound the association with

postoperative complications. The authors acknowledge this limitation, and without several data points to determine an overall trend or slope of change over time, application of these data clinically is challenging.

Even taking these issues into consideration, there appears to be a signal toward increased morbidity and mortality in patients with an acute change in their creatinine level preoperatively. These findings warrant a future prospective study incorporating consistent methodology for the entire patient journey, including determination of the true baseline creatinine level, as well as subsequent investigation into interventions to identify and mitigate risk and enhance recovery in vulnerable cardiac surgery patients.

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Commentary: Changing the ship's course for a better outcome

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Correcting acute versus chronic physiologic derangements may alter a patient's trajectory.

CENTRAL MESSAGE

Management of preoperative acute kidney injury represents an area worth exploring to further optimize patients before cardiac surgery to improve their outcomes.

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FIGURE 1. Correcting acute versus chronic physiologic derangements may alter a patient's trajectory.

Great surgeons know not just what operation to perform, but most importantly when the operation should be done. Decision algorithms should include options for deferral to correct changes in patient homeostasis. The theory makes a lot of sense; however, in practice we have been disappointed with the futility of reversing the effects of chronic systemic derangements in terms of altering the ship's course. Once we recognized that chronically elevated blood sugars were associated with infection, there was great enthusiasm that delaying surgery and taming sugars would shift the patient to a low-infection risk, but this was all for naught.¹ Preoperative chronic anemia also impacts early outcomes, and yet multiple trials to reverse this with erythropoietin and iron therapy have yielded discouraging results.² The common element of these potentially reversible chronic preoperative diagnoses is that they are specifically that—chronic. The body has bathed in this environment for long time periods, and we are naïve to think that fixing a laboratory value over an accelerated time frame would result in normal functioning.

In this issue of the *Journal*, Griffin and colleagues³ examined whether acute preoperative kidney injury (defined as the increase in creatinine measured on the day of surgery vs the lowest value within 3 months before surgery) leads to worse postoperative outcome. Indeed, acute injury was predictive of worse in-hospital mortality, stage III acute kidney injury, sternal wound infections, and prolonged intensive care unit and hospital stay, independent of baseline estimated glomerular filtration rate of the patient.

This study has highlighted the value of this biochemical marker (ie, acute increase in creatinine) to identify patients at risk. It is not unreasonable to extrapolate that this is an easily modifiable risk factor (unlike chronic changes). Oftentimes, correction of a mild elevation of creatinine of this nature requires minimal surgical deferral. It's unlikely that more drugs are needed, but rather less (diuretics) are called for. And, best of all, the change is acute and not chronic so we may not be as naïve in thinking we have put the patient on a better footing.

Optimizing results is highly dependent on choosing the window that will afford primary success while maintaining the lowest risk of a complication. The authors have enticed us that this may be one of the few instances in which addressing an easily fixable problem with minimal complications could improve postoperative outcome, and we should include this simple step in our assessment algorithm (Figure 1).

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