It's the beans that count

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Disclosures: Author has nothing to disclose with regard to commercial support.

Received for publication July 8, 2018; accepted for publication July 9, 2018.

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J Thorac Cardiovasc Surg 2018;156:2065-6

0022-5223/\$36.00

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<https://doi.org/10.1016/j.jtcvs.2018.07.022>

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Central Message

In patients undergoing type II thoracoabdominal aortic aneurysm repair, preoperative renal dysfunction is associated with increased postoperative morbidity and renal failure.

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Cardiac surgery–associated acute kidney injury is a frequent postoperative complication,¹ with even a slight elevation of creatinine increasing 30-day mortality 3-fold.² Patients requiring renal-replacement therapy fare significantly worse,³ with preoperative creatinine being one of the strongest predictors of postoperative dysfunction.⁴ Few cardiac procedures rival the complexity and physiologic insult of type II thoracoabdominal aneurysm (TAAA) repair, and increased rates of kidney injury and failure may be expected.

In this issue of the *Journal*, Coselli and colleagues⁵ present a series of more than 1000 type II TAAA repairs stratified by absence or presence of preoperative chronic kidney disease. The authors should be congratulated for these very impressive numbers and meticulous follow-up. In the propensity-matched analysis, patients with chronic kidney disease (CKD) were shown to have a greater incidence of postoperative adverse events and renal failure necessitating hemofiltration, yet operative death rates were comparable. The frequency of postoperative renal failure was 14.1% in the CKD group and 8.7% in patients with normal preoperative renal function. CKD was identified as an independent predictor of adverse events and renal failure but not operative mortality.

The reported occurrence of postoperative renal dysfunction in the current study is intuitive, yet the lack of mortality difference is unexpected. Other investigators⁶ have reported a 6-fold increase in operative mortality in 96 type II TAAA repairs when stratified by preoperative creatinine level of greater than 1.5 mg/dL. However, those patients were not propensity matched and represent a comparatively small sample. The very broad definition of preoperative CKD used in the current study does not permit the identification of incremental risk with progressive renal dysfunction or definition of a critical threshold.

As such, although representing the largest cohort of type II TAAA in the published literature, the study still offers limited guidance for preoperative risk assessment across the clinical spectrum of renal function. To this end, in

addition to renal failure, incremental postoperative renal injury would be a valuable end point that was not evaluated by the authors. Perhaps of greatest interest is the influence of renal protective strategies, such as cold renal perfusion, on the rate of postoperative renal failure. In both healthy patients and those with CKD, cold renal perfusion had surprisingly no effect on the rate of postoperative renal failure, yet it was strongly protective of operative mortality in patients with CKD (9.4% vs 19.7%). These data suggest that cold renal perfusion provides a survival benefit that is independent of the development of postoperative renal failure. Putative mechanisms are difficult to identify but may possibly be mediated through systemic hypothermia or attenuated duration of renal dysfunction. The negative impact of preoperative CKD on distant mortality has been reported for myriad of cardiac procedures, including TAAA.⁶ Similarly, the authors report significant differences in 5- and 10-year survival between patients with and without CKD. However, these survival data were not propensity matched and need to be interpreted with caution, as patients in the CKD cohort were older, sicker, and with greater atherosclerotic burden, which irrespective of renal status may have a powerful influence on long-term survival. With propensity matching, differences in distant survival, as observed with operative mortality, may become smaller or negligible.

Risk stratification of complex and physiologically taxing procedures such as type II TAAA is essential for patient education and optimal outcomes. Preoperative renal dysfunction in cardiac surgery is a powerful predictor of subsequent renal failure, confirmed by the current study in the setting of

extensive TAAA repair. The incomparable data presented by the authors provide an important surgical reference point yet raise questions still unanswered. Whether novel renal protective strategies can attenuate the effect of CKD on postoperative renal function or a nihilistic approach prevails remains to be seen. Irrespective, when predicting adverse events after TAAA repair, it's the "beans" that count.

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