The prevalence of severe symptomatic aortic stenosis (AS) is approximately 5% in patients older than 65 years of age. This condition represents the most common indication for cardiac surgery or transcatheter intervention in Europe and North America, accounting for more than 9000 procedures per year in the United States alone. Recent data on the benefit of aortic valve replacement for severe AS before the development of symptoms are likely to expand the indication for interventional aortic valve replacement for severe AS before the development of symptoms, following both SAVR and TAVR. Several patient- and procedural-related factors are implicated in the pathophysiology of AKI during SAVR and TAVR. Importantly, with the extension of aortic valve replacement indications to earlier stages of the disease, overall comorbidity burden and procedural risk of the patients are expected to decrease, which might carry along a decreased risk of AKI. Chronic kidney disease (CKD) has traditionally played a major role as one of the main risk factors for AKI, in a degree-dependent fashion, following both SAVR and TAVR. Therefore, one would expect that AKI would not be common in patients with normal baseline renal function undergoing such procedures. Scant information has been available thus far on the incidence, mechanisms, and prognostic relevance of AKI in this context.

In the current issue of the *Canadian Journal of Cardiology*, Moriyama and colleagues fill this gap, reporting on the incidence and long-term impact of AKI in a large multicentre registry comprising 4555 subjects with normal renal function undergoing either SAVR or TAVR. The authors report that AKI had an overall incidence of 12.5% in subjects undergoing aortic valve replacement, with significantly higher figures for SAVR compared with TAVR (19.0% vs 5.9%, *P* < 0.001, in the propensity score-matched population). Bleeding complications were consistently associated with AKI in both cohorts. AKI was linked to an increased risk of 5-year all-cause death (adjusted hazard ratio: 2.14, 95% confidence interval: 1.69-2.67, *P* < 0.001), with more severe degrees of AKI associated with higher mortality rates.

The authors are to be congratulated for this well-conducted multicentre registry, with robust data including a SAVR control group, rigorous adjustment by propensity-score matching, and the use of the Kidney Disease-Improving Global Outcomes (KDIGO) definition of AKI, which allows for an easy comparison with previous and future studies. On the other hand, several limitations have also to be acknowledged. First, the authors could not report data on operator or centre volume and numerous key variables including patient hemodynamic conditions, valvular and
vascular computed tomography angiography data, or contrast volume exposure, which limits further speculations on the mechanisms underlying some study findings, such as why AKI rates decreased over time in the TAVR but not SAVR cohort. Second, the study population was identified as patients with an estimated glomerular filtration rate (eGFR) ≥60 mL/min per 1.73 m². The eGFR was estimated with the Modification of Diet in Renal Disease (MDRD) formula. It is interesting that such equation has shown to provide suboptimal prognostic stratification for mortality, AKI, and AKI requiring dialysis compared with the CKD Epidemiology Collaboration (CKD-EPI) equation in patients undergoing percutaneous coronary intervention. Whether these considerations also apply in the context of aortic valve replacement should be evaluated by future studies. Furthermore, an eGFR <60 mL/min per 1.73 m² may not be sensitive enough in identifying subjects with underlying renal disease; indeed, significant renal damage can ensue well before eGFR begins to decrease, and other approaches must be considered to better quantify AKI risk in patients with “normal” renal function (by eGFR). For example, renal functional reserve has been used to measure the capacity of the kidneys to increase GFR under

Figure 1. Factors contributing to acute kidney injury during surgical (SAVR) and transcatheter (TAVR) aortic valve replacement. Note: Atheroembolic showers into the renal arteries can arise either from scraping of the aorta during sheath or catheter manipulation or from valve fragments detached during valvuloplasty or valve implantation. Valve images have been reproduced with permission by Medtronic (Dublin, Ireland).
conditions of physiological stress and was shown to be inversely correlated with the risk of AKI in a small cohort of patients with normal resting eGFR undergoing elective cardiac surgery.\(^{16}\) Finally, the observed association between AKI and long-term mortality in patients undergoing TAVR or SAVR does not imply that AKI holds a causal relationship with mortality in this setting. Indeed, a recent study conducted in patients undergoing coronary angiography, with or without intervention, indicated that AKI does not mediate the association between lower eGFR and higher short-term risk of a composite of all-cause death, new need for dialysis, and persistent impairment in kidney function.\(^{17}\) AKI might therefore be merely a marker of disease, and adopting strategies aimed at decreasing the risk of AKI might not translate into better hard clinical outcomes.

What can we take home from the study by Moriyama et al.\(^{11}\) to improve the outcomes of patients undergoing SAVR or TAVR? The authors clearly showed that AKI also happens frequently in patients with normal resting eGFR undergoing elective cardiac surgery.\(^{16}\) To this extent, it is important to avoid rapid pacing if possible (especially in subjects with severe left ventricular dysfunction) by limiting the use of balloon-expandable valves and balloon aortic valvuloplasty. Efforts should be made to reduce the risk of embolization of atheroma and aortic valve fragments caused by sheath- or catheter-induced scraping of the aortic wall and balloon aortic valvuloplasty, respectively. This can be accomplished using alternative (nontransfemoral) access routes and avoiding valvuloplasty, respectively. As far as SAVR is concerned, cardiopulmonary bypass elicits a proinflammatory reaction that can trigger AKI.\(^{21}\) Therefore, adopting surgical techniques that allow for the reduction of cardiopulmonary bypass time or using kidney-protective perfusion strategies during surgery can mitigate the risk of

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**Figure 2.** Strategies to reduce acute kidney injury during surgical (SAVR) and transcatheter (TAVR) aortic valve replacement. Valve images have been reproduced with permission by Medtronic (Dublin, Ireland).
AKI. Careful postprocedural care aimed at avoiding excessive use of vasodilators, splanchnic vasoconstrictors, and diuretics to guarantee stable renal perfusion may be beneficial after both TAVR and SAVR.\(^{10,22}\)

To conclude, the work by Moriyama and colleagues\(^{11}\) establishes AKI as a relevant prognosticator following SAVR and TAVR, even in the absence of significant previous renal dysfunction, and uncovers new unresolved questions. First, how can we possibly identify in advance non-CKD patients at higher risk of developing AKI? A normal eGFR is clearly not sufficient, and novel renal function parameters including renal functional reserve\(^{16}\) or alternative biomarkers may be considered. Second, further studies are needed to better characterize and address procedure-related AKI risk factors. Finally, we do not know yet whether AKI holds a causal relationship with increased long-term mortality after aortic valve replacement. However, as shown in this and other studies, patients with AKI after TAVR or SAVR do suffer worse long-term outcomes. Therefore, we must be cognizant of this increased risk to be able to target this population with more intensive follow-up and possibly disease-modifying interventions.

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