

Effects of Sevofurane versus Propofol Total Intravenous Anaesthesia on Renal Function in Elderly Patients Undergoing Hip Fracture Surgery: A Randomised Controlled Trial [Response to Letter]

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Dear editor

We sincerely thank the authors of the Letter for their positive remarks and thoughtful interpretation of our recent randomised controlled trial comparing propofol-based total intravenous anaesthesia (TIVA) and sevoflurane anaesthesia in elderly patients undergoing hip fracture surgery.¹ Their comments offer valuable perspectives and contribute meaningfully to the continued refinement and understanding of our work.

We fully acknowledge the limitation of serum creatinine (SCr)-based estimated glomerular filtration rate (eGFR) in the assessment of acute kidney injury (AKI), as changes in SCr may lag behind renal injury and are influenced by perioperative hemodilution.^{2,3} In our study, eGFR was selected as the primary outcome because it remains the most widely available and clinically interpretable indicator of renal function in perioperative practice. The early postoperative (1-hour) eGFR measurement was intended to capture rapid and potentially transient functional alterations related to the anaesthetic regimen. To broaden the evaluation beyond a single functional marker, we additionally assessed complementary physiological indices, including urine output, urinary sodium excretion, and plasma renin concentration. We appreciate the suggestion that analyzing intra-individual changes in eGFR may yield greater clinical precision, and we intend to incorporate this analysis in future work.

Regarding the concept of subclinical AKI, we agree that early renal cellular stress may occur even in the absence of overt functional decline. The pattern observed in the sevoflurane group—lower urine output, reduced urinary sodium excretion, and higher plasma renin levels—is consistent with sympathetic activation and transient renal vasoconstriction, as demonstrated in recent experimental and clinical studies.^{4–6} This constellation of findings may therefore reflect a state of early renal cellular stress rather than established injury. We also concur with the Letter's authors that future research should integrate early injury and stress biomarkers such as neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule-1 (KIM-1), and [TIMP-2]·[IGFBP7] to more accurately identify subclinical kidney injury.^{7,8} For patients who develop more severe postoperative complications, longitudinal characterization of renal and systemic clinical trajectories will be particularly informative and is planned for subsequent investigations.

In summary, we appreciate the correspondents' thoughtful perspectives, which underscore the importance of incorporating both functional and molecular assessments in research on anaesthesia-related renal effects. We hope this constructive

exchange will foster continued collaborative efforts to elucidate the mechanisms underlying anaesthetic-associated renal influences and their clinical relevance.

Disclosure

The authors declare that there are no conflicts of interest in this communication.

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