Reviewer's report

Title: Difference Between Pre-operative and Cardiopulmonary Bypass Mean Arterial Pressures is Independently Associated with Early Cardiac Surgery-Associated Acute Kidney Injury

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Reviewer: Anthony Delaney

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Difference between pre-operative and cardiopulmonary bypass mean arterial pressure is independently associated with early cardiac surgery associated acute kidney injury

Thank you for the opportunity to review this manuscript. The authors present the results of a prospective cohort study that primarily seeks to investigate the relationship between haemodynamic variables associated with cardiopulmonary bypass and the incidence of acute kidney injury.

The authors are to be commended for performing an interesting study. The methods are generally well described, the writing is of an acceptable standard, and the overall standard of reporting is good. I have only a few comments.

1. In the section describing the study definitions, the authors refer to acute kidney injury (AKI) being defined by the RIFLE criteria. In my copy of the manuscript the "urine output" has been left out of the criteria, I assume this is a minor typographical error. Can the authors please confirm that AKI as defined by the Injury category is what is referred to in this study? Or have the authors defined AKI as patients meeting any of the RIFLE criteria ie, including the Risk category. The authors might consider presenting the data for how many of the cases of AKI met each of the creatinine or urine output criteria for each RIFLE category?

2. With the above point in mind, I found it hard to understand how so many patients were diagnosed as incident cases of AKI, given the results of Table 5. The mean urine output for the first 24 hours in the group with AKI was 0.5ml/kg/hr and the mean creatinine only increased from 100 to 114. It is hard to imagine how these numbers represent meet the injury category of the RIFLE "injury" criteria.

3. There are a couple of additional points that might be worth adding to the statistical methods. The use of an a-priori hypothesised value for the delta MAP that would be associated with a significant increase in AKI would have greatly enhanced the reproducibility of the multivariate model and would have reduced the likelihood for a chance finding. Could the authors justify why an a-priori value for delta MAP was not used in the study? Could the authors also briefly mention why the sample size was chosen for this study?
4. Finally, a minor statistical clarification. My understanding is that for multivariate logistic regression models to have sufficient power to give stable results there needs to be approximately 10 outcome events for every predictor added to the model. It appears that the authors will have included 14 predictors (i.e. those with a p<0.2) in the original multivariate model. Therefore the 65 cases of AKI would be insufficient to produce a stable model and the resulting estimates of the OR would be difficult to reproduce. The validity of the results would be enhanced if the authors followed a pre-specified analysis plan. Can the authors state whether a pre-specified analysis plan was used for this analysis, or was the analysis data driven? Was the modelling performed solely on statistical grounds or were clinical factors also considered? These factors may help readers interpret the results of the study.

Overall the authors are to be commended for performing this interesting study.

**Level of interest:** An article of importance in its field

**Quality of written English:** Acceptable

**Statistical review:** Yes, but I do not feel adequately qualified to assess the statistics.

**Declaration of competing interests:**

My only competing interest is that I have previously performed some work with Dr Bagshaw, one of the authors of the manuscript.