

## Letters to the Editor

# Risk Factors for Acute Kidney Injury after Cardiopulmonary Bypass

To the Editor,

With great interest we read the latest feature article by Newland and Baker (1), in which the authors explore whether a relationship exists between oxygen delivery ( $\text{DO}_2$ ) during cardiopulmonary bypass (CPB) and postoperative acute kidney injury (AKI). They found the integral of amount and time below (negative area under the curve,  $\text{AUC-}$ ) or above (positive area under the curve,  $\text{AUC+}$ ) a critical oxygen delivery threshold ( $270 \text{ mL/min/m}^2$ ) to be an independent predictor of postoperative AKI and conclude that their results support that a relationship exists between oxygen delivery during CPB and the incidence of postoperative AKI. However, one may debate the causal effect of low  $\text{DO}_2$  on postoperative AKI in this study.

First of all, the  $\text{AUC}$  groups differ on several important aspects. The  $\text{AUC-}$  group comprises significantly older patients [68 {60–77} years vs. 64 {52–71} years,  $p = .009$ ] and a higher percentage of patients with preoperative diabetes (28 vs. 15%,  $p = .020$ ) and cerebrovascular disease (13 vs. 1.5%,  $p = .009$ ) than the  $\text{AUC+}$  group. Furthermore, although not significant, the  $\text{AUC-}$  group includes a higher percentage of patients with hypertension (57 vs. 49%,  $p = .276$ ) and an almost double percentage of patients with chronic obstructive airway disease (19% vs. 11%,  $p = .126$ ), compared with the  $\text{AUC+}$  group. All these patient characteristics are proven to be independent risk factors for postoperative AKI by enhancing renal failure susceptibility (2).

Intraoperative factors related to the development of AKI are large differences ( $\Delta$ ) in mean arterial pressure and lower CPB flow during cardiac surgery (3). In the study of Newland and Baker, the average CPB flow index was significantly lower in the  $\text{AUC-}$  compared with the  $\text{AUC+}$  group [1.9 {1.8–2.1}  $\text{L/min/m}^2$  vs. 2.0 {1.9–2.1}  $\text{L/min/m}^2$ ,  $p = .005$ , respectively], whereas both groups had an average intraoperative mean arterial blood pressure [ $\text{AUC-}$ : 62 {58–66} mmHg;  $\text{AUC+}$ : 64 {60–68} mmHg] lower than the optimal blood pressure ( $71 \pm 10.3$  mmHg) reported by Hori et al (4). This might explain the increase of postoperative creatinine levels in both  $\text{AUC}$  groups.

Another debatable aspect is the pathophysiological relation between low oxygen delivery ( $\text{DO}_2 < 270 \text{ mL/min/m}^2$ ) and postoperative AKI. Without corresponding oxygen consumption, interpretation of oxygen delivery is rather speculative. As one can assume based on the absence of hyperlactatemia in both groups ( $\text{AUC+}$ : 1.7 mmol/L;  $\text{AUC-}$ : 1.6 mmol/L), the systemic oxygen delivery was adequate.

Taken these considerations into account, the higher observed incidence of AKI in the  $\text{AUC-}$  group of this study (1) is probably not because of the lower  $\text{DO}_2$  but rather because of other AKI risk factors that predispose to renal failure in the  $\text{AUC-}$  group. Therefore, the association between the integral of amount and time below a  $\text{DO}_2$  threshold of  $270 \text{ mL/min/m}^2$  and the incidence of postoperative AKI needs more debate in the light of a “state of the art” extracorporeal circulation.

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