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Alleviating central venous oxygen saturation ($S_{cv}O_2$): a new approach of kidney protection after cardiac surgery?

Patrick M. Honore*, Rita Jacobs, Inne Hendrickx, Elisabeth De Waele, Duc Nam Nguyen and Herbert D. Spapen

See related research by Balzer et al., http://www.ccforum.com/content/19/1/168

In a recent article, Balzer et al. [1] reported an unexpected relationship between an initial central venous oxygen saturation ($S_{cv}O_2$) >80 % on ICU admission and increased morbidity and mortality in cardiac surgery patients. Interestingly, as compared with low (<60 %) or normal (60–80 %) $S_{cv}O_2$ levels, values above 80 % were associated with more acute kidney injury (AKI) and a higher hemodialysis need. This goes against common belief that a higher $S_{cv}O_2$ is a prerequisite for more optimal organ, and in particular kidney, protection.

Transient brief episodes of upper arm ischemia after induction of anesthesia reduce the rate of AKI and the

need for renal replacement therapy among high-risk cardiac surgery patients. This so-called "remote ischemic preconditioning" is thought to engage adaptive natural defense mechanisms that protect the kidneys against the subsequent significant inflammatory and ischemia/reperfusion stress caused by cardiac surgery [2]. Theoretically, keeping $S_{\rm cv}O_2$ levels within a "low–normal" range in the early post-cardiac surgery period could act as a surrogate for ischemic preconditioning. In contrast, "boosting" $S_{\rm cv}O_2$ by excessive transfusion, inotropic support or oxygenation might abrogate this protective mechanism [3]. The provocative findings of Balzer et al. highly encourage further research in this area.

Authors' response

Felix Balzer, Sascha Treskatsch and Michael Sander

Professor Honore and colleagues added a very interesting aspect to the discussion of initial ScvO2 assessment after a patient's postoperative admission to the ICU. As published by Zarbock et al. [2], remote ischemic preconditioning is associated with reduced incidence of AKI and use of renal replacement therapy. This remarkable finding requires further investigation. The design of our approach, however, does not allow us to draw conclusions related to this aspect. In our study, patients with a presumably "safe" initial ScvO₂ above 80 % showed a higher incidence of renal dysfunction including an increased rate of renal replacement therapy. Also, these patients had the highest inhospital mortality and the highest mortality during 3-year follow-up. Since pre-existing chronic hemodialysis with arterial-venous shunts is known to lead to increased venous saturation, we excluded such patients from a subanalysis in order to account for this bias, but results remained substantially unchanged.

In the group of patients with high ScvO₂, we found a significantly higher occurrence of epinephrine application. Consequently, it cannot be ruled out that the use of epinephrine aggravates the shunting effects in postoperative cardiac surgery patients. Nonetheless, the lower oxygen extraction in the high ScvO₂ group suggests that tissue hypoperfusion may be another important component explaining the significantly increased rate of organ dysfunction and the higher mortality.

At our hospital, we have developed an algorithm to guide treatment by advanced hemodynamic monitoring in patients that present with an oxygen extraction dysfunction as reflected by a high $ScvO_2$ in the context of a postoperative "sepsis-like" syndrome. Currently we are evaluating the effect of these measures on patient outcome.

^{*} Correspondence: Patrick.Honore@az.vub.ac.be ICU Department, Universitair Ziekenhuis Brussel—Vrije Universiteit Brussel, 101 Laarbeeklaan, 1090 Jette, Brussels, Belgium



Abbreviations

AKI: Acute kidney injury; S_{cv}O₂: Central venous oxygen saturation.

Competing interests

The authors declare that they have no competing interests.

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