Authors have nothing to disclose with regard to commercial support. Support was provided solely from institutional or departmental sources.

SHOULD KIDNEY OXYGEN SATURATION BE MONITORED TO PREVENT ACUTE KIDNEY INJURY AFTER ADULT CARDIAC SURGERY?

To the Editor:

We are pleased to read the commentary by Ferraris¹ entitled “Should We Rely on Receiver Operating Characteristic Curves? From Submarines to Medical Tests, the Answer Is a Definite Maybe!” regarding our recently published article.² We would like to clarify some important issues related to our findings. A decrease in kidney oxygen saturation (kSo₂) less than 65% and a decline of greater than 20% from the baseline value during the postoperative stage were identified as independent predictors of cardiac surgery–associated acute kidney injury (CSA-AKI), but not a decline of more than 20% in baseline serum creatinine.

CSA-AKI remains a severe complication and one of the stronger risk factors for death in patients undergoing cardiac surgery. Scientific advancements in developing universal predictor scales or finding the ideal biomarkers have had limited success.³ For this reason, the addition of kSo₂ to biomarkers could significantly allow real-time monitoring and thus improve CSA-AKI detection.

Regarding Ferraris’ queries, there are evidence-based interventions to improve kSo₂ and therefore prevent CSA-AKI, such as the restoration of mean arterial pressure from 60 to 75 mm Hg—an intervention that improves renal oxygen delivery, glomerular filtration rate, and kSo₂. The pressure-dependent kidney perfusion, filtration, and oxygenation at levels of mean arterial pressure below 75 mm Hg reflect a relatively exhausted kidney autoregulatory reserve.⁴ However, there are few data relating to kidney oxygen consumption, renal blood flow, and glomerular filtration rate, and current views on kSo₂ are presumptive and mostly based on experimental studies. Likewise, we

seize this letter to answer all questions raised by Ferraris¹ that other readers might share (Table 1).

All in all, considering that kSo₂ provides information about regional oxygen metabolism along with an estimation of kidney perfusion, in addition to its role as a novel marker for AKI after adult cardiac surgery, kSo₂ should be carefully monitored in older, frailer, high-risk patients undergoing complex cardiac procedures.

Christian Ortega-Loubon, MD, MEd, PhD^a

Inmaculada Fierro, PhD^b

Eduardo Tamayo, MD, PhD^c

^aDepartment of Cardiac Surgery and

^cAnesthesiology and Critical Care

Clinic University Hospital of Valladolid

Valladolid, Spain

^bDepartment of Health Science

Universidad Europea Miguel de Cervantes

Valladolid, Spain

References

1. Ferraris VA. Should we rely on receiver operating characteristic curves? From submarines to medical tests, the answer is a definite maybe! *J Thorac Cardiovasc Surg.* 2019;157:2354-5.
2. Ortega-Loubon C, Fernandez-Molina M, Fierro I, Jorge-Monjas P, Carrascal Y, Gomez-Herreras JJ, et al. Postoperative kidney oxygen saturation as a novel marker for acute kidney injury after adult cardiac surgery. *J Thorac Cardiovasc Surg.* 2019;157:2340-51.
3. Ortega-Loubon C, Fernandez-Molina M, Paneda-Delgado L, Jorge-Monjas P, Carrascal Y. Predictors of postoperative acute kidney injury after coronary artery bypass graft surgery. *Braz J Cardiovasc Surg.* 2018;33:323-9.
4. Ricksten SE, Bragadottir G, Redfors B. Renal oxygenation in clinical acute kidney injury. *Crit Care.* 2013;17:221.
5. Ortega-Loubon C, Fernandez-Molina M, Jorge-Monjas P, Fierro I, Herrera-Calvo G, Tamayo E. The relevance of renal oxygen saturation over other markers in cardiac surgery-associated acute kidney injury. *J Cardiothorac Vasc Anesth.* September 27, 2018 [Epub ahead of print].
6. Deschamps A, Hall R, Grocott H, Mazer CD, Choi PT, Turgeon AF, et al. Cerebral oximetry monitoring to maintain normal cerebral oxygen saturation during high-risk cardiac surgery: a randomized controlled feasibility trial. *Anesthesiology.* 2016;124:826-36.
7. Ortega-Loubon C, Fernandez-Molina M, Carrascal-Hinojal Y, Fulquet-Carreras E. Cardiac surgery-associated acute kidney injury. *Ann Card Anaesth.* 2016;19:687-98.
8. Lacroix S, Gayda M, Gremeaux V, Juneau M, Tardif JC, Nigam A. Reproducibility of near-infrared spectroscopy parameters measured during brachial artery occlusion and reactive hyperemia in healthy men. *J Biomed Opt.* 2012;17:077010.

<https://doi.org/10.1016/j.jtcvs.2019.05.012>

TABLE 1. Information about kidney oxygen saturation and acute kidney injury

Question	Relevance	Response
Is kSo ₂ a “predictor” of AKI?	To be a valuable predictor, it is best to know the predictor value before the outcome.	kSo ₂ is indeed an independent predictor of AKI, as shown via the multivariable logistic regression analysis. Considering that the peak of SCr could appear even 7 days after the surgery, which is included in the KDIGO Criteria for CSA-AKI, and that kSo ₂ was only measured during the first 48 h postoperatively, kSo ₂ certainly predicts the possible outcome of CSA-AKI. ²
How frequently should kSo ₂ be measured?	Is it practical to have real-time measurements of kSo ₂ ?	kSo ₂ should be measured especially in older patients and in high-risk surgeries with both long CPB and aortic crossclamp times. What is more, kSo ₂ needs to be monitored not only during the surgery but also during the postoperative span, when there are several factors that still contribute to the appearance of CSA-AKI. It is practical to continuously record kSo ₂ because kSo ₂ real-time measurement predicts CSA-AKI. Unlike renal biomarkers, which represent the most sensitive means of CSA-AKI early detection but cannot be measured continuously, NIRS enables noninvasive, real-time monitoring and serves as a suitable target for the goal-directed therapy to treat impairments in kidney oxygen supply–demand balance. ⁵
Are abnormal values of kSo ₂ actionable?	Is it practical to intervene to improve kSo ₂ (eg, increase oxygen content, transfuse, and increase red cell mass)?	Absolutely. Actions must be taken to enhance kSo ₂ , such as improve MAP, treat abnormal systemic saturation, correct hypoventilation, and consider RBC transfusion depending on an established algorithm to reverse desaturation. ⁶ In fact, it is practical to improve kSo ₂ levels, because a decrease in kSo ₂ <65% threshold value or a decline of >20% from the baseline value is significantly associated with CSA-AKI. As such, avoiding these desaturation levels, CSA-AKI could be prevented.
What factors affect CSA-AKI? Are they different from these affecting non-CSA-AKI?	Validation of CSA-AKI test results is essential.	Risk factors associated with CSA-AKI are multiple. Noncardiac surgery is not an exception. Moreover, cardiac surgery adds renal hypoperfusion, hemodilution, and inflammatory response caused by CPB. It is unlikely that a single etiologic factor will cause AKI. It is rather the consequence of the interaction of multiple factors, not only preoperative but also intraoperative and even postoperative factors. ⁷ Numerous studies have proved the close relationship between intraoperative bSo ₂ and kSo ₂ decline with postoperative cognitive decline and CSA-AKI, respectively. Although validated levels of kSo ₂ were lacking, this study has identified critical kSo ₂ measurements associated with CSA-AKI.
How reproducible are kSo ₂ measurements?	What factors alter kSo ₂ results besides oxygen saturation (eg, skin pigment, obesity)?	NIRS parameters are highly reproducible, which enables them to study the microvascular function. ⁸ kSo ₂ measurements are affected by its depth of penetration, conjugated bilirubin, skin pigment, and external source of light.
Are tools for measurements of kSo ₂ during CPB available?	The authors constructed unique equipment to measure kSo ₂ during CPB. There needs to be some “catch-up” in technology.	Depending on each hospital’s economic resources, NIRS is usually available. In fact, in most cases, it is often used in cardiac surgery for bSo ₂ monitoring. bSo ₂ and kSo ₂ were measured using the same NIRS sensors; no extra or different equipment was required to measure kSo ₂ .
What interventions can improve kSo ₂ during CPB?	How reliable are interventions aimed at improving kSo ₂ (eg, increase oxygen, increase perfusion pressure)?	More investigations should be conducted to develop algorithms to correct kSo ₂ desaturations, in the same way as algorithms are applied to treat bSo ₂ desaturations.

kSo₂, Kidney oxygen saturation; AKI, acute kidney injury; SCr, serum creatinine; KDIGO, Kidney Disease Improving Global Outcomes; CSA-AKI, cardiac surgery–associated acute kidney injury; CPB, cardiopulmonary bypass; MAP, mean arterial pressure; RBC, red blood cell; bSo₂, brain oxygen saturation; NIRS, near-infrared spectroscopy.