Authors have nothing to disclose with regard to commercial support.

REPLY: CARDIAC SURGERY—ASSOCIATED ACUTE KIDNEY INJURY—A PROTECTIVE ARMOR RATHER THAN A LETHAL AMMUNITION

Reply to the Editor:

We are genuinely pleased to read the reply by Montisci and Miceli,¹ “Reply: Cardiac Surgery-Associated Acute Kidney Injury—Finding the Gunpowder,” regarding our recently published article.² Certainly, to comment about cardiac surgery-associated acute kidney injury (CSA-AKI) is nothing if not a complex chore. In this regard, we would like to discuss only 3 specific aspects.

1. Arguably, brain oxygen saturation monitoring has some drawbacks, which does not necessarily translate into a safer environment to other vulnerable organs, such as the kidneys. In fact, this was demonstrated in our study,² in which the kidney oxygen saturation but not the brain oxygen saturation was closely related to the development of CSA-AKI during the postoperative stage.
2. Undeniably, CSA-AKI is a complex entity.³ Numerous factors intervene altogether in its pathogenesis, taking into account not only preoperative factors and intraoperative factors related to the surgery but also several elements that adversely influence the patient's condition during the postoperative period. This is why it is so difficult to exert absolute control over CSA-AKI, which still today remains a notable complication after cardiac surgery, with increased postoperative morbidity and mortality regardless of our tremendous efforts.⁴
3. Finally, with respect to modifiable factors, as Montisci and Miceli¹ mentioned, some risk factors frequently cannot be modified, such as age, long cardiopulmonary bypass time, or preexisting chronic kidney disease. In addition to these, and taking into account that the mechanisms modulating the pathogenesis of CSA-AKI still remain largely undetermined and that there are no specific treatments for CSA-AKI other than routine

supportive therapy and renal replacement therapy,⁵ we should divert all our efforts toward its prevention, which has to remain as our principal aim in the treatment of this disease, rather than eagerly looking for unsatisfactory interventions for this complication.⁶

All in all, carrying on the original analogy of Montisci and colleagues,⁷ we are yet faced with a shortage of munitions against CSA-AKI. Providing an effective preventive measure could be a much more worthwhile initiative to tackle this problem successfully.

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"I'VE MISSED MORE THAN 8000 SHOTS IN MY CAREER. I'VE LOST ALMOST 300 GAMES. 26 TIMES, I'VE BEEN TRUSTED TO TAKE THE GAME-WINNING SHOT AND MISSED. I'VE FAILED OVER AND OVER AND OVER AGAIN IN MY LIFE. AND THAT IS WHY I SUCCEED."

REPLY: IS PREVENTION OF ACUTE KIDNEY INJURY AFTER CARDIAC OPERATIONS HOPELESS? MICHAEL JORDAN WOULD SAY NO!

Reply to the Editor:

"I can accept failure. Everyone fails at something, but I can't accept not trying again."

—Michael Jordan

We are continually looking for additional factors that can favorably modify perioperative concerns in patients

The Editor welcomes submissions for possible publication in the Letters to the Editor section that consist of commentary on an article published in the Journal or other relevant issues. Authors should: • Include no more than 500 words of text, three authors, and five references. • Type with double-spacing. • See <http://jtcvs.ctsnetjournals.org/misc/fora.shtml> for detailed submission instructions. • Submit the letter electronically via jtcvs.editorialmanager.com. Letters commenting on an article published in the JTCVS will be considered if they are received within 6 weeks of the time the article was published. Authors of the article being commented on will be given an opportunity to offer a timely response (2 weeks) to the letter. Authors of letters will be notified that the letter has been received. Unpublished letters cannot be returned.

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undergoing cardiopulmonary bypass (CPB). The literature is replete with articles that offer solutions, critiques, and just plain hope for a favorable impact of interventions to minimize cardiac surgery–associated acute kidney injury (CSA-AKI). The litany of articles, editorials, and commentaries published in the *Journal* document these interventions.¹⁻⁴ These articles revolve around a signal that goal-directed perfusion may have a beneficial impact on decreasing CSA-AKI.¹ There is yet another series of articles on this topic in the current issue of the *Journal*.^{5,6} I am sure that we have not heard the last of commentaries and likely new research directions regarding the prevention and treatment of CSA-AKI.

You cannot be from Kentucky and not have some words of wisdom related to basketball. Michael Jordan's quote about failing at life's endeavors seems appropriate for a description of attempts to minimize CSA-AKI after cardiac operations (Figure 1). The series of articles in this issue of the *Journal* can be summarized by saying that CSA-AKI is a complex complication with multifactorial components, and that no single intervention is completely successful in limiting this complication. This fact is something on which most people who write about CSA-AKI agree. The ongoing dialog about CSA-AKI defines the importance of the original article by Ranucci and colleagues¹ and alerts cardiothoracic surgeons to the need for further research and the creation of new interventions to limit AKI. So Michael Jordan was right. Have we failed to cure CSA-AKI? Clearly, the answer is yes. Like Michael Jordan, we can accept failure, but we must keep on trying to succeed (Figure 1).

Because of the complexity of CSA-AKI, nearly everyone who explores this entity agrees that prevention must be multifactorial and must include prophylactic/preventative interventions as well as intraoperative and postoperative interventions. Is that much different than any other complication that we see in cardiac surgery? The obvious answer is no. Examples of other complex complications related to cardiac operations abound and include interventions to minimize CPB-related bleeding, minimize postoperative pulmonary complications, improve postoperative low cardiac output, and limit complicated wound infections. Solutions to these problems also abound. Are these solutions always successful? No. Do we keep trying to find better alternatives? Yes. It should not be surprising that some skepticism surfaces about the concept of goal-directed perfusion as a solution for CSA-AKI.

Recent articles in the *Journal* describing the attempts and controversies surrounding limiting AKI during CPB sound

"I'VE MISSED MORE THAN 9000 SHOTS IN MY CAREER. I'VE LOST ALMOST 300 GAMES. 26 TIMES, I'VE BEEN TRUSTED TO TAKE THE GAME WINNING SHOT AND MISSED. I'VE FAILED OVER AND OVER AND OVER AGAIN IN MY LIFE. AND THAT IS WHY I SUCCEED."

- MICHAEL JORDAN

FIGURE 1. The power of failure.

a little bit like a war with "gunpowder," "protective armor," "lethal ammunition," and "magic bullets" offered as solutions.^{2,6,7} Is it unfair to say, as some have suggested, that "only supportive therapies, including renal replacement therapy," are available to manage AKI?³ This option does not sit well with most cardiothoracic surgeons. There certainly is a robust literature suggesting the dire prognosis of CSA-AKI, but that does not mean that the situation is hopeless. In the early days of cardiac surgery, perioperative bleeding was thought to have a dire prognosis, but now perioperative bleeding can be managed in most cases with only a modestly increased risk. A similar early dire prognosis was associated with low ejection fraction, concomitant pulmonary disease, severe peripheral vascular disease, and other factors.

A combination of preoperative risk factor modifications coupled with active prophylactic and perioperative interventions has dramatically reduced the perioperative complications compared with the early days of cardiac surgery. This success has encouraged surgeons to manage more and more complex procedures. Consequently, the risk profile for the current cardiac surgical population has increased significantly as surgeons have become more willing and able to treat sicker and sicker patients and manage perioperative complications in this cohort. So why not a similar sequence of events for treatment of CSA-AKI? I suggest that we are getting there, and point out that the work that stimulated this prolonged dialog and that triggered the stream of articles about CSA-AKI management was the article by Ranucci and coauthors¹ that described a randomized trial of the use of goal-directed perfusion to minimize CSA-AKI. In the world of cardiac surgery, this article was ground-breaking. Was this article definitive? Probably not. More than anything, the article proved that CSA-AKI is not a hopeless complication and can be studied in a rigorous manner. There are active interventions that can minimize this complication. Are these interventions always successful? No. Are there other

things that need to be studied that will likely minimize CSA-AKI? Of course. The dialog represented by the *Journal* commentaries related to the article by Ranucci and colleagues reflects the article's importance. Hopefully, this dialog of commentaries will stimulate further rigorous studies to address incompletely resolved issues surrounding CSA-AKI. I look forward to these future studies.

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REPLY FROM THE AUTHORS: ACUTE KIDNEY INJURY: INFINITY TIMES INFINITY Reply to the Editor:

Acute kidney injury (AKI), a complex disease associated with poor outcomes, remains an open issue in cardiac surgery despite much effort. Although our perspectives differ, we are all engaged in improving our results. Whether it is the lack of a magic bullet (our view) or the need of protective armor (your view), the meaning does not change.^{1,2} As specified previously, AKI is a 1 million-piece puzzle that involves many preoperative, intraoperative, and postoperative variables.³ Any potential measure to prevent it is welcome. We can discuss this an infinite number of times. Perhaps we made a mistake. AKI is not a 1 million-piece puzzle—it's much more.

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CEREBROVASCULAR COMPLICATIONS AFTER CARDIAC SURGERY: IT IS TIME TO TRACK AND TREAT LARGE VESSEL OCCLUSION

To the Editor:

We read with a great interest the recent article by Sheriff and colleagues¹ concerning large vessel occlusion (LVO) stroke after cardiothoracic surgery (CTS). Sheriff and colleagues¹ conducted a monocenter study of their own database, with 5947 patients being reviewed between July 2013 and April 2018. Among 148 patients with cerebrovascular complications within 30 days of surgery, 10.1% had LVO. Almost half of these patients were considered candidates for emergency thrombectomy, with encouraging functional outcome at 3 months. This was also suggested by a case-control study on perioperative stroke, which included 68% CTS patients.²

We also collected data on LVO in our center. We reviewed all consecutive adult patients hospitalized in the intensive care unit within 2 days after CTS with cardiopulmonary bypass between August 2016 and March 2018 in our tertiary center. Of 1946 patients, 57 (2.9%)