Reply

Post-Conditioning at the Ischemic Region of the Heart to Prevent Acute Kidney Injury in Patients With Non–ST-Segment Elevation Myocardial Infarction

We thank Dr. Zhou and colleagues their interest in our paper (1). However, we would like to note that their arguments pertain to remote ischemic pre-/post-conditioning with the heart as the target organ (all their references are in regard to ischemic conditioning for cardioprotection, not renoprotection). As a result, we cannot really see how their rationale (e.g., regarding times of cardiac ischemia in relation to the conditioning procedure) is of relevance to our study. In any case, we do have the data on times elapsed from the onset of ischemia to the coronary intervention and conditioning procedure. There was no significant difference between the 2 study arms: The median ischemia time was 10 h (interquartile range: 8 to 12 h) in the remote conditioning group versus 9 h (interquartile range: 7 to 13 h) in controls (p = 0.294; Mann-Whitney test).

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http://dx.doi.org/10.1016/j.jacc.2013.04.095

REFERENCE

recent studies by Thuny et al. (3) and Tarantini et al. (11). Thus, readership would benefit more from this missing information, and I am interested to know their use in both the post-conditioning group and the control group.

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Reply

Post-Conditioning the Human Heart: Technical Concerns Beyond the Protocol Algorithm

We thank Drs. Liu and Xu for their letter commenting on our paper (1). However, we are a little confused as to exactly how it relates to our study. All the publications cited by Drs. Liu and Xu and the presented arguments pertain to conditioning for cardioprotection. Ours was a paper on renoprotection, which is something different in almost every respect; for example, we cannot really see how Liu’s argument that “prolongation of delay from 10 to 30 seconds to 60 seconds or 10 minutes has been indicated to result in the failure of cardioprotection by postconditioning in animal studies” relates to the findings of our study regarding renal function. Even if we do change the subject altogether and embark on a discussion regarding postconditioning for cardioprotection (which has nothing to do with the essence of our paper), the bitter truth is that no definitive evidence has been produced to issue a verdict about its efficacy in reducing reperfusion damage, let alone about the best conditioning protocol, in terms of timing, mode, and sequence of the ischemia–reperfusion stimulus. We also do not understand what the purported “missing information” is regarding the description of the protocol applied in our study. We believe that our published paper describes the site, timing, and mode of the conditioning procedure with clarity. We cannot see how a discussion on technical details about how different aspects of the protocol could affect cardioprotection could fit in a paper dedicated to the protection of renal function, especially considering that no outcomes or surrogate markers regarding myocardial damage were evaluated in the context of our study.

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http://dx.doi.org/10.1016/j.jacc.2013.06.038

REFERENCE