far as it can be concluded from Table 3, the rise in CK-MB mainly occurred between 6 and 24 h post-operatively, which is delayed in respect to the nicorandil patients for mitral valve replacement where the peak is at 6 h postoperatively. You conclude that these patients receiving nicorandil had a lower incidence of significant elevation of postoperative myocardial damage, suggesting that ongoing myocardial injury in the postoperative period might possibly be due to reperfusion injury and that nicorandil may have attenuated this due to its anti-inflammatory property. This could be correct but is in contrast to the finding that four nicorandil CABG patients compared to two placebo CABG patients had significantly elevated CK-MB levels. Theoretically, the incidence of significantly elevated CK-MB levels should be lower in the nicorandil group. Nonetheless, this could mean that there are multifactorial causes for postoperative myocardial infarction in your patients. When did the myocardial infarction actually happen in both groups and could other factors have been responsible such as the quality of coronary vessels? In addition, could postoperative factors rather than perioperative management have influenced the occurrence of postoperative infarction in the cases you mentioned? Unfortunately, the limited number of patients in your study precludes any conclusion on this issue. Ideally, troponin I should also have been measured. Nevertheless, the general outcome in your publication is favourable for the use of nicorandil during cardioplegia.

Just as a remark, because enzymes of the potassium channel openers work more efficiently during normothermia, warm cardioplegia could have improved results due to increased potassium permeability leading to rapid sinus node arrest. In addition, Cohen and colleagues [6] concluded that terminal infusion of warm blood cardioplegia repleted myocardial ATP levels and improved postoperative myocardial function. In general, hypothermic cardioplegia should be avoided because it can trigger CAS, thereby inducing ventricular fibrillation leading to depletion of myocardial ATP. Warm cardioplegia was not applied in your study. On the other hand, CK-MB levels under cold cardioplegia were improved in mitral valve replacement patients with nicorandil, which make your results even more interesting because CAS is more enhanced by hypothermia.

In spite of above remarks, we want to congratulate Chinnan and co-workers for their excellent paper which brings us one step further ahead to optimize coronary blood flow during CPB and thereby decreasing events of myocardial ischaemia.

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Reply

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EDITOR:
We thank Kiss and colleagues for their valuable comments. The faster onset of electromechanical arrest after administration of cardioplegia is very likely due to the coronary vasodilating properties of nicorandil [1]. Coronary vasodilatation could have also resulted in better distribution of the cardioplegic solution and improved myocardial preservation and, hence, fewer arrhythmias while coming off cardiopulmonary bypass (CPB) and less risk of postoperative myocardial infarction. The increased incidence of

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myocardial infarction in the nicorandil group coronary artery bypass grafting (CABG) patients could be multifactorial. However, it is interesting to note that three out of four patients who had a postoperative myocardial infarction in the nicorandil group, CABG patients were diabetics. Our study did not look into coronary artery spasms in the postoperative period. In this context, we would like to refer to a case report where nicorandil infusion was used to manage distal micro-vascular spasm characterized by ST segment elevation in the electrocardiogram, serially elevated cardiac enzymes (creatine kinase and creatine phosphokinase-MB) and new onset regional wall motion abnormalities in the absence of fixed or vasospastic occlusion of coronary vessels on angiography [2]. With regard to reperfusion injuries and arrhythmias, intravenous and intracoronary nicorandil have been shown to be of benefit after percutaneous coronary interventions for acute myocardial infarctions [3,4]. After prolonged cardioplegic arrest (4 h) in an isolated rabbit heart model, nicorandil was found to attenuate ischaemia–reperfusion injury of the myocardium and coronary endothelium, and ameliorate leukocyte activation [5]. These findings by other researchers are in accordance with our observations. We would also like to clarify that we used a terminal infusion of warm blood cardiolplegia (hot shots) before coming off CPB as it is the present day standard of care and apologize for not making that clear in our study protocol. We fully agree with Kiss and colleagues that a larger multicentre randomized controlled trial is required to determine the efficacy and the role of nicorandil in certain subclasses of patients like those developing coronary artery spasms in the postoperative period, and diabetics undergoing cardiac surgery.

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