provides a new online bedside monitoring system, which may be of value in such patients.

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The use of nicorandil in cardioplegia solution

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EDITOR:
We would like to comment on the article by Chinnan and co-workers [1] written on myocardial protection by nicorandil during open-heart surgery under cardiopulmonary bypass (CPB).

In relation to cardioplegia, coronary artery spasm (CAS) is frequently underestimated during CPB. Until now, CAS was not detectable with current monitoring techniques during CPB and its impact on cardiac surgery has not been elucidated in previous studies. During CAS the blood flow to the myocardium decreases depending on the degree of coronary vasoconstriction, thereby increasing anaerobic metabolites and myocardial ischaemia. Consequently, CAS could be responsible for right and left ventricular failure after leaving CPB. Therefore, apart from being a potassium channel opening drug preventing intracellular Ca^{2+} loading [2], the fact that nicorandil is a coronary vasodilator might have contributed to favourable results in your study.

The link between vasospastic angina and vulnerability for ventricular tachycardia or ventricular fibrillation outside the setting of cardiac surgery has already been described in several case reports [3–5]. Iida and colleagues [5] reported that ventricular fibrillation improved and disappeared with the start of treatment with nitrates and suggested that CAS should be considered as a differential diagnosis in the presence of ST-segment changes and/or intractable ventricular arrhythmias. Extending this conclusion into CPB surgery, this could imply that rather than giving an anti-arrhythmic drug, a coronary vasodilator should be used to prevent and to treat arrhythmias secondary to ischaemic episodes caused by CAS. As your results show, a link between CAS and ventricular fibrillation seems to be apparent. After cross-clamp removal in coronary artery bypass grafting (CABG) patients, two patients in the nicorandil group developed significant cardiac dysrythmias vs. six patients in the placebo group. Unfortunately, the number of your patients is limited and it would be interesting to pursue your study as a multicentre trial on a larger scale.

The relationship between nicorandil and coronary vasodilatation also explains the enhanced distribution of cardioplegia solution, a faster time until electromechanical arrest in the nicorandil group and a faster return of electromechanical activity after aortic cross clamp removal in patients under nicorandil in the mitral valve replacement group.

Interestingly, although not significantly different, in CABG patients four nicorandil patients compared to two placebo patients had creatine kinase (CK-MB) levels higher than $75\,IU\,L^{-1}$. Within the patients treated with nicorandil in the CABG group, as

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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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