anticholinesterase agent. However, because of the condition of the patient and the need to optimize her respiratory effort after operation, we decided to give half the usual dose recommended.

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The preconditioning effects of levosimendan

Editor—We read with interest the article by Tritapepe and colleagues1 on the preconditioning effects of levosimendan and agree with the therapeutic implications of myocardial ischaemic preconditioning. But the question remains: does it actually work? The concept originated from an observation that mortality in ischaemic heart disease was noted to be less in patients who had suffered from anginal episodes in the past, rather than the other subgroup of patients who suffered MI as the first presentation to the hospital. As such, the authors have rightly pointed out the need for a larger number of patients to be recruited for the study. The pilot study demonstrates that pharmacological preconditioning with a short duration infusion of levosimendan in cardiac surgical patients before commencing CPB appears to confer additional myocardial protection beyond that provided by cardioplegia alone, as manifested by a better haemodynamic recovery and lower postoperative TnI concentrations. The authors have also pointed out that, in this setting, the use of cardiac specific markers for diagnosis and quantification of myocardial damage is still debated. As such, we believe that there are a lot of ethical issues involved in new drug trials in a high-surgical risk patient population. We congratulate the authors in conducting the first study, albeit preliminary, that has investigated levosimendan-induced myocardial protection in humans with ischaemic heart disease undergoing a major cardiac and circulatory insult.

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Editor—We thank Drs Kumar and Kumar for their interest in our paper. Over the past 30 years, hundreds of experimental interventions (pharmacological and non-pharmacological) have been reported to protect the ischaemic myocardium in experimental animals. However, with the exception of early reperfusion, none has been translated into clinical practice. The National Heart, Lung, and Blood Institute convened a working group to discuss the reasons for the failure to translate potential therapies for protecting the heart from ischaemia and reperfusion and to recommend new approaches to accomplish this goal. The Working Group concluded that cardioprotection in the setting of acute myocardial infarction, cardiac surgery and cardiac arrest is at a crossroad. The Working Group urged a new focus on translational research that emphasizes efficacy and clinically relevant outcomes, and recommended the establishment of a system for rigorous preclinical testing of promising cardioprotective agents with clinical trial-like approaches (i.e. blinded, randomized, multicentre and adequately powered studies using standardized methods). Our pilot study was designed to provide preliminary data to test the hypothesis that levosimendan has a preconditioning effect in patients. A power analysis performed on the basis of the study suggests that a sample size requirement of 96 patients (48 in each group) would be needed to detect a reduction in median ICU length of stay from 35 h in the control group to 24 h in the protocol group (a=0.05, power 0.9). This further trial is currently underway in our hospital.

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