Conflict of interest
None declared.

A. E. Funnell*
J. Griffiths
I. Hodzovic
Gwent, UK
'E-mail: anthony.funnell@gmail.com

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Is preoperative levosimendan indicated to treat normal left ventricular function and left ventricle outflow obstruction?

Editor—we read with great interest the article by Leppikangas and colleagues1 evaluating the haemodynamic effects of preoperative levosimendan in patients undergoing high-risk cardiac surgery. The authors addressed the interesting issue of the high-risk cardiac surgical patient and the optimization of perioperative pharmacological heart support. However, based on our personal experience with levosimendan in cardiac surgery setting and on the evidence from the existing literature, we have several concerns with the study.

First, the study was conducted in patients undergoing combined aortic valve replacement and coronary artery bypass graft as they were considered high risk. All the patients enrolled did not match criteria for inclusion in the study protocol [left ventricular ejection fraction (LVEF) <50% or wall thickness >12 mm] neither for inotropic pretreatment: the preoperative EF was normal. Secondly, all the patients had a serious contraindication to levosimendan; in fact, they suffered from severe aortic valve stenosis with high gradient that naturally increases after an inodilator administration. Moreover, the control group did not receive the standard inotropic treatment in use at the authors’ centre, but only placebo, although considered high risk. Thirdly, the control group had no difference in terms of myocardial injury, mechanical ventilation, and intensive care unit (ICU) stay. Surprisingly, mortality was higher in the levosimendan group.

Fourthly, quite a high-dose bolus was given to coronary artery disease (CAD) patients with ventricular outflow obstruction and myocardial hypertrophy, despite the danger of hypotension due to vasodilatation. At our institutions, we started using levosimendan in 2004. According to our policy, an inotrope is administered only if a need for increased inotropy is demonstrated by echocardiography and clinical evaluation. Therefore, we usually give levosimendan perioperatively in patients with severely depressed ventricular function (EF<30%) starting just after induction of anaesthesia without bolus. When acute ventricular failure occurs at the time of weaning from cardiopulmonary bypass (CPB), we give a bolus of 6–12 μg followed by an infusion of 0.1–0.2 μg kg⁻¹ min⁻¹. However, in severe aortic valve stenosis with severely depressed LV function, we usually give levosimendan in bolus (24 μg) at the initiation of CPB followed by an infusion of 0.1 μg kg⁻¹ min⁻¹ for 24 h. By doing so, we observed a reduced ICU stay and a reduced perioperative intra-aortic balloon pump application over the years. Some considerations need to be added. It is agreed that levosimendan offers haemodynamic support1–4 and myocardial protection5,6 in the perioperative scenario. However, preoperative administration of the drug requires monitoring in a high-dependency setting. Therefore, besides the risks of giving it in aortic stenosis patients, we should take into consideration the logistic and financial implications of such a policy in an era of cost containment. The inodilator itself needs to be used appropriately due to the high cost if compared with other inotropic drugs. Finally, the preoperative use of levosimendan to optimize patients’ cardiovascular function, either in cardiac or non-cardiac surgery, would be appropriate if patients meet some ‘common sense’ criteria, including depressed myocardial function (EF<40%) to increase CI, CAD patients unsuitable for revascularization at risk for ischaemia (cardiac protection, low anaerobic threshold in which it could be worth increasing the DO₂). In conclusion, levosimendan shows relevant advantages in high-risk patients undergoing cardiac surgery in terms of haemodynamic support and cardioprotection, but it is our opinion that an always cautious and cost-effective application should be advocated.

Conflict of interest
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F. Guarracino*
L. Tritapepe
Pisa and Rome, Italy
’E-mail: fguarracino@ao-pisa.toscana.it

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Reply from the authors

Editor—We would like to thank Dr Guarracino and Dr Tritapepe for their interest in and criticism on our article addressing the use of preoperative levosimendan in combined aortic valve and coronary bypass surgery. Currently, levosimendan is indicated for i.v. use in hospitalized patients with acutely decompensated heart failure (ADHF). The mechanism of action and current literature suggest that the therapeutic potential of levosimendan may not be limited only to ADHF patients. We are happy to share the interest in studying levosimendan in new clinical settings with Dr Guarracino and Dr Tritapepe.

Our inclusion criteria were left ventricular (LV) ejection fraction < 50% or LV hypertrophy, as indicated by a wall thickness of > 12 mm. All our study patients met at least one of these criteria. It is possible that by including study patients with lower ejection fractions, we would probably have obtained even better results.

The fact that the patients suffering severe aortic valve stenosis should not receive vasodilators has been questioned earlier. Many studies have shown a positive effect of vasodilators in this patient group. Nitroprusside has been shown to be a safe and effective treatment in patients with decompensated heart failure due to severe LV systolic dysfunction and severe aortic stenosis. We agree that vasodilatation is a potent risk in this patient group, which is why the patients were closely monitored in a cardiac high-dependency setting (including invasive arterial pressure monitoring) during the study drug infusion. The control group received placebo as a preoperative treatment, but during and after the operation, haemodynamics were treated according to the local protocol in both study groups.

This study was underpowered to detect mortality differences between the groups. One patient in the levosimendan group died on the first postoperative morning. The death was not related to the levosimendan infusion.

It is a common practice also in our institution to use perioperative levosimendan in patients with severely depressed LV function. In this study, we wanted to test the hypothesis that the levosimendan metabolite OR-1896 production is better when levosimendan is infused before operation. After the results of this study, we have increased the use of perioperative levosimendan in patients with severely depressed LV function. Levosimendan has been shown to be effective in cardiac surgery patients. Owing to the high costs, future studies are warranted to elucidate the optimal timing and dosing of levosimendan. In our study, we showed that cardiac index (CI) and stroke volume index (SVI) were higher in the levosimendan group for the 4 day postoperative period (P < 0.05). The concentrations of OR-1896 were higher compared with earlier studies using perioperative dosing. We fully agree that caution and cost-effective application should be advocated when levosimendan is used in high-risk patients undergoing cardiac surgery.

Conflict of interest

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H. Leppikangas*
K. Jarvela
Tampere, Finland
E-mail: heli.leppikangas@pshp.fi

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Systemic air embolism during lung biopsy

Editor—Percutaneous biopsy guided by computed tomographic (CT) scan is commonly used for the diagnosis of pulmonary lesions. The occurrence of a systemic air embolism is a rare but potentially lethal complication.

A 57-yr-old man was admitted in our hospital for the investigation of three pulmonary lesions recently found on a chest CT scan. He had a rectal cancer 3 yr previously treated with radiotherapy, neoadjuvant chemotherapy, and an anterior resection. No comorbidity was to be noted.

The patient was undergoing a left lung biopsy. The patient was placed prone and a coaxial biopsy system with a core biopsy needle was used. After local anaesthesia, the percutaneous biopsy was performed with CT scan-fluoroscopic guidance at the left lower lobe of the lung by an experienced radiologist. During the procedure, the patient suddenly presented with haemoptysis, cough, and acute chest pain. Clinical examination retrieved hypotension (systolic arterial pressure < 60 mm Hg) and a severe bradycardia (< 50 beats min⁻¹).

The procedure was immediately stopped. The patient was placed in the Trendelenburg position. He received i.v. saline solution and high-concentration oxygen by a facemask. The ECG showed significant elevation of ST segment at the posterior (II, III, aVF) and anterior (V₁, V₂, V₃) leads. An immediate chest CT scan showed localized parenchymal