Sevoflurane and cardioprotection

Editor—We read with interest the study by Piriou and colleagues who observed limited cardioprotection after 1 MAC sevoflurane in humans undergoing cardiopulmonary bypass (CPB). Julier obtained similar results by a higher concentration of sevoflurane (2 MAC) given for 10 min through a vaporizer integrated into the CPB machine. Julier but not Piriou observed PKC translocation in the myocardium, an event related to the protection conferred by preconditioning. However, a protective effect of sevoflurane inhalation has been observed by De Hert’s group in a series of studies, in the last of which, different protocols were used, characterized by administration of propofol only throughout the operative period, sevoflurane only before CPB, sevoflurane only after completion of the coronary anastomoses, and sevoflurane throughout. The lowest troponin I values were observed in the group treated with sevoflurane throughout operation and the highest in the propofol group. Sevoflurane throughout the operative procedure showed a cardioprotective effect. Another substantial difference could be observed between the group of patients treated by De Hert and the ones treated by Piriou and Julier; in the study group of the former, the mean aortic cross-clamp time was shorter (31 ± 15 min compared with 53 and 54 min in the two groups studied by Piriou and 60 ± 24 min and 66 ± 22 min in Julier’s study). There was, however, no significant difference in the mean CPB time. Since it is well known that the cross-clamp time is related to the extent of the ischaemia–reperfusion injury and troponin I release, it could be possible that the lack of cardioprotection in the studies by Piriou and Julier was determined by a more prolonged mean cross-clamp time. The absence of PKC induction in the study by Piriou could be linked to the lower dose of sevoflurane used compared with that used in the study by Julier. Cardioprotection by continuous administration of sevoflurane, as stated by Piriou, may be responsible for the superior protection observed by De Hert, supported by the haemodynamic effects of volatile anaesthetics and, perhaps (De Hert did not study PKC translocation) also by preconditioning, whose induction could be dose and time dependent.

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Editor—We wish to thank Drs Siracusano and Girasole for their interest in our study. As you highlighted in your letter, Julier and colleagues found similar results. The common point of the studies was that they were multicentre studies, in contrast to studies showing a protective effect. From our results, we cannot rule out that volatile agents exert a protective effect by preconditioning, as shown by animal experimental data, in selected patients. However, in experimental studies, all variables can be carefully controlled. It is not the case in clinical studies, where many factors might interfere, for example, surgical skills, patient anatomy, evolution of the coronary disease, duration of aortic cross-clamping, concomitant treatment, cardioprotective strategy, and CPB. By performing multicentre studies, we increase the number of confounding factors, which is one explanation of our results and those of Julier and colleagues. Myocardial protection is multimodal and the ‘magic bullet’ has still not been discovered, with cardioplegia being the major cardioprotective factor. As in non-cardiac surgery, to improve the chance of showing a cardioprotective effect, we have to select carefully the population of patients who will benefit the most from the protective effect. By selecting a very high-risk population of patients, Poldermans and colleagues found a protective effect for beta-blockers, although three recent studies which included more, but less selected patients (i.e. lower risk patients), showed no effect. We think that in cardiac surgery, this may apply; cardioplegia is efficacious in most patients, but additional cardioprotection, such as this one afforded by volatile anaesthetics, is needed for high-risk patients. As it is very difficult to predict a priori which patients will have a myocardial infarction, and as there is no harm in using volatile anaesthetics for all patients, we believe that although our study, and the Julier’s one, did not show significant results, we have to consider the systematic use of volatile anaesthetics for all coronary surgery. We do not have a clear explanation for the conflicting results of PKC activation, and, as you hypothesize, these results could be linked to the sevoflurane administration protocol (dose, duration, sequences, and time of administration). Further clinical studies are needed to clarify this point.

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Contrast-induced nephropathy and endovascular aortic aneurysm repairs

Editor—We read with interest the review of contrast-induced nephropathy (CIN) by Wong and Irwin.1 The authors should be commended for an excellent article, but we would like to comment upon CIN and endovascular aortic aneurysm repairs (EVARs). The authors suggest that performing an EVAR under local anaesthesia (LA) may be of some benefit in preventing CIN. The Eurostar data from which this conclusion is drawn cannot support this assertion.2 This demonstrated that there was no significant difference between the incidence of renal complications (up to 30 days after operation) in patients having an EVAR with general anaesthesia (GA), regional anaesthesia (RA), or LA. In addition, EVARs performed under LA accounted for only 6% of total case numbers and the database does not provide any data about conversions from LA to either RA or GA.

Ruptured aortic aneurysms may now be repaired using an endovascular technique (REVAR), which was first described at our institution. The incidence of renal complications in this population is high when compared with a traditional open technique,3 and this is most probably due to the additional renal damage induced by CIN. It is our practice to hydrate aggressively these patients from the time of their admission to the emergency department in order to try to minimize the nephrotoxic effects of the contrast media.

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Editor—We wish to thank Drs Wiles and Brown for their comments regarding our recent review article. We actually meant to imply the potential overall benefits of LA but also inadvertently quoted the wrong reference in relation to the Eurostar data.4 These data demonstrated distinctly the lower incidence of systemic complications (especially cardiac and pulmonary) with the use of LA or RA compared with GA, with high-risk patients particularly benefitting from loco-regional anaesthesia. High-risk patients with LA also benefited in terms of overall complications (P=0.0017) and cardiac complications (P=0.0281) compared with GA. With specific regard to renal complications, there was a benefit of RA over GA, but there was no significant difference between GA and LA. There was, however, a trend towards a reduction in the small number of patients receiving LA would have led to a low statistical power in this regard. We apologize for not clarifying the difference between LA and RA in the context of this reference.

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