Thoracic Epidural Anesthesia in Abdominal Aortic Surgery: Use and Advantages

To the Editor:
We read with interest the prospective, randomized, controlled trial by Lindholm et al.1 where they compare the troponin T release after elective major vascular surgery in two groups of patients: one group with fentanyl-sevoflurane anesthesia and the other with propofol–remifentanil anesthesia. These authors concluded that sevoflurane-based anesthesia did not reduce myocardial injury, evaluated by troponin T release, compared with total intravenous anesthesia, suggesting that volatile anesthesia is no more protective than total intravenous anesthesia in elective abdominal aortic surgery. In this study, authors indicate that epidural catheter was introduced at thoracic level T6–T10 and epidural analgesia was started after opening the aortic cross-clamp in the two groups of patients.

In our opinion, the use of thoracic epidural analgesia (TEA) in the two groups of patients included in this trial could be an important issue regarding the obtained results. It has been suggested that intraoperative combination of general and epidural anesthesia with continuing postoperative epidural analgesia could be beneficial in high-risk surgical patients undergoing major noncardiac surgery.2 The effects of TEA are produced by the blockade of cardiac sympathetic efferent nerve fibers that have their origin in segments T1–T5.3 Activation of these fibers results in the stimulation of α- and β-adrenergic receptors, leading to an increased inotropy, chronotropy, vasoconstriction of epicardial coronary arteries, and systemic vasoconstriction, increasing myocardial oxygen demand. Previous studies have shown that combination of TEA and general anesthesia decreases heart rate, myocardial contractility, and systemic vascular resistance, resulting in potential benefits such as an improved balance of myocardial oxygen supply and demand and greater intraoperative hemodynamic stability in patients with coronary artery disease undergoing surgery.4,5 TEA has been reported to improve the status of nonsurgical patients with unstable angina and myocardial ischemia,5 but we must note that studies on cardiac surgery have failed to find significant differences in troponin levels after TEA although this difference could be explained by the varying etiology and pathophysiology of the perioperative ischemia during coronary artery bypass graft surgery.6 To this way, Jacobsen et al.7 found that thoracic epidural anesthesia patients had higher stroke volume index, higher cardiac index, higher venous pressures, and lower systemic vascular resistance index perioperatively and postoperatively in cardiac surgical patients. Therefore, it seems logical to suggest that TEA could have decreased myocardial ischemia in patients included in the two groups of this interesting study and this may have biased the results of this trial. We wonder that what would had happened to the results whether thoracic epidural anesthesia had not been added to the patients included in the two groups of this trial.

References

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Competing Interests
The authors declare no competing interests.

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References
Sevoflurane- Compared with Propofol-based Anesthesia Reduces the Need for Inotropic Support in Patients Undergoing Abdominal Aortic Aneurysm Repair: Evidence of Cardioprotection by Volatile Anesthetics in Noncardiac Surgery

To the Editor:

We read with interest the study by Lindholm et al. comparing cardioprotection by sevoflurane- and propofol-based anesthesia in patients undergoing elective abdominal aortic surgery. The authors chose cardiac troponin T (cTnT) release determined at one single postoperative time point as the primary endpoint of cardioprotection. No difference between the groups was found, and the authors concluded that "potential cardioprotective effects of volatile anesthetics found in cardiac surgery are less obvious in major vascular surgery." 1

We do not agree with this interpretation of the study results. Neither do we think that this study, as designed a priori and ultimately conducted, properly addresses the stated hypothesis. First, the cardioprotective effects of sevoflurane are not "less obvious in major vascular surgery," but indeed very similar to what was reported for volatile anesthetics in previous studies with patients undergoing cardiac surgery in the on-pump or off-pump mode. In fact, Lindholm et al. report a significantly reduced need for inotropic support in the sevoflurane group (P = 0.003), implying improved cardiac function and reflecting a clear advantage of the sevoflurane-based anesthesia. Unfortunately, this important finding is only briefly mentioned in the Results section and completely ignored in the Discussion. No details on the doses of dopamine and noradrenaline or other potentially administered inotropics such as ephedrine and/or phenylephrine are provided. From the currently available eight randomized trials evaluating volatile anesthetic-induced cardioprotection in patients undergoing off-pump coronary artery bypass graft surgery, a type of surgery which is in many aspects comparable with abdominal aortic aneurysm repair, only three of eight (37%) find reductions in cardiac troponin release, whereas four of eight (50%) find improved cardiac function or reductions in inflammatory markers. Although infarct size and the release of cardiac enzymes are the “definitive standard” of cardioprotection, they are by far not the only clinically relevant outcome measures. Cardioprotection in patient care has already reached a high standard, and any additional protection may be unable to further reduce perioperative release of myocardial necrosis markers, specifically so if the majority of patients are already treated with statins, aspirin, β-blockers, and thoracic epidural anesthetics. In support of this, the use of a volatile anesthetic in cardiac surgical patients potentially reduces long-term cardiovascular complications and mortality, as shown by Garcia et al., De Hert et al., and others, despite the clear absence of a reduction in perioperative cardiac troponin release. This notion is compatible with the strong anti-inflammatory and potentially plaque-stabilizing actions of volatile anesthetics during the critical perioperative period. We also think that serial postoperative determinations of cTnT should have been obtained in the study by Lindholm et al. to reliably map postoperative myocardial damage, and if reporting cTnT values of a single postoperative time point, a histogram of the results displaying ranges of cTnT levels and numbers of patients would provide much more information.

Second, we think that the design of the study by Lindholm et al. does not allow to directly answer the hypothesis whether a sevoflurane-based anesthesia as compared with a propofol-based anesthesia is more cardioprotective, because in their sevoflurane group, fentanyl was used as opioid whereas in the propofol group remifentanil was used. Current clinical studies with remifentanil suggest that its cardioprotection may render the protective effects of volatile anesthetics redundant. Studying the interference in cardioprotection by volatile anesthetics, opioids and propofol in a working rat heart model, we recently demonstrated that remifentanil maintains its protection against ischemia–reperfusion injury in combination with propofol, but does not further enhance protection by sevoflurane. Furthermore, in the study by Lindholm et al., patients randomized to propofol-based anesthesia were clearly more aggressively treated with aspirin and β-blockers, making the study groups unbalanced and shifting cardioprotection in favor of the propofol group. Also,