In Reply:

Conflict of interest is managed using a variety of strategies, but they all begin with disclosure. I absolutely agree with Dr. Kempen's insistence that important competing interests must be identified and believe that there could have been more transparency regarding competing interests of the individuals who prepared this editorial which appeared in the January 2014 issue.[1]

The journal style changed with the January issue in two manners relevant to competing interests. First, we now only publish the individual's institution, but not their position in that institution (*e.g.*, fellow, resident, student, assistant professor, and so on). In the submission of this editorial, the authors listed their positions at the American Board of Anesthesiology (ABA) as Director or Chief Assessment Officer, but those were removed in the new style and their institution information printed in the editorial merely states ABA. This statement provides transparency that the authors are associated with the ABA, whether they are paid employees, receive other compensation, or are unpaid. Second, we have consolidated all competing interests into one place, at the end of articles including editorials. Although the authors' affiliation with the ABA was already declared, Dr. Kempen is correct that the competing interest statement should have reiterated what was present on the title page—that the authors are associated with the ABA. This would also apply to the editorial by Dr. Gambill, Chief Learning Officer at the American Society of Anesthesiologists, who describes American Society of Anesthesiologists' efforts to meet the educational needs of its members.[2]

I thank Dr. Kempen for his comments and take responsibility for any confusion there might have been caused between the authors' affiliations and the competing interest statement.

Competing Interests

Dr. Eisenach has received fees for consultation to Aerial Biopharma (Morrisville, North Carolina) and Adynxx (San Francisco, California) on topics unrelated to this letter, and receives salary support from the American Society of Anesthesiologists as Editor-in-Chief of Anesthesiology.

James C. Eisenach, M.D., Wake Forest University School of Medicine, Winston-Salem, North Carolina. editor-in-chief@anesthesiology.org

References

1. Rathmell JP, Lien C, Harman A: Objective structured clinical examination and board certification in anesthesiology. Anesthesiology 2014; 120:4–6
2. Gambill BD: American Society of Anesthesiologists' lifeline to learning. Anesthesiology 2014; 120:7–9

(Accepted for publication April 29, 2014.)

Is Norepinephrine Infusion during Intraoperative Period Justified?

To the Editor:

The study by Wuethrich *et al.*[1] has interesting physiological background and important clinical implications. Veins, particularly splanchnic veins, are much more compliant than arteries.[2,3] Density of α-adrenergic receptors within the veins is much higher than in the arteries.[4] Therefore, veins are more sensitive to sympathetic activation than arteries.[4,5] The reported study has demonstrated that the use of relatively small doses of norepinephrine is not associated with any clinical or biochemical signs of tissue hypoperfusion.[1]

Sympathetic nervous system and α-adrenergic receptors play the main role in maintaining a certain ratio between stressed (Vs) and unstressed (Vu) blood volumes.[2,3] α-Adrenergic agonists induced constriction of compliant veins, resulting in a decrease in Vu and a concomitant increase in Vs.[3,6] General and, particularly, epidural anesthesia decreases the overall sympathetic discharge followed by venodilation, increase in Vu, and decrease in Vs, which could lead to a decrease in venous return and cardiac output. In addition, controlled ventilation with concomitant increase in intrathoracic pressure might decrease venous return and therefore must be associated with an increase in Vs to maintain adequate hemodynamics. The body achieves this by several mechanisms, main and immediate of which is an increase in sympathetic discharge. Such compensation also might be impaired to different degrees by general and epidural anesthesia. This study illustrates that norepinephrine induced reduction in enlarged Vu and restoration of the Vs can maintain adequate hemodynamics. Fluid infusion also can restore the Vs. Thus, an increase in Vs can be achieved by an increase in fluid load (to fill up an increased venous capacity) and/or by restoration of the sympathetic tone that would reduce the increased Vu. The use of norepinephrine in this situation decreases the Vu but not the blood flow to the tissues. The current study demonstrates clinical benefits of such an approach.[1]

Clinical implications of these observations are also important. Use of relatively small doses of vasopressors to restore what has general and epidural anesthesia impaired seems logical. The clinical advantage of such an approach is relatively simple: it allows avoiding additional, not needed infusion of fluid. However, the clinician should realize that such approach might decrease the margin of safety. The main function of the Vu is to serve as a reservoir of blood to be mobilized when needed, for example, during blood loss. However, when the mobilization of blood from Vu to Vs is approximately complete, hemodynamics deteriorate. We all have observed the initial blood loss of 800 to 900 ml not being associated with serious hemodynamic consequences; however, when blood loss