In Reply:

The authors sincerely thank Dr. Roth for his letter regarding their recent article[1] and are happy to respond to his comments and anecdotes.

The problematic of chest wall compliance is of interest and is not new. In 1989, Pizov *et al.*[2] demonstrated in a canine model that reducing chest wall compliance by external chest compression induced an increase in the deltadown component of the systolic pressure variation, suggesting a decrease in the venous return. More recently, Mesquida *et al.*[3] found an increase in pulse pressure variations and stroke volume variations after reduction in chest wall compliance induced by chest and abdominal binders in an experimental study. As suggested by Dr. Roth, decrease in chest wall compliance may be due to truncal rigidity. This situation may be related to a lack of sedation (which contraindicates the use of dynamic indices) or may be provoked by high doses of opioids (an uncommon practice actually even in cardiac surgery). In contrast, a decrease in chest wall compliance secondary to abdominal hypertension is a common situation in an operating room (laparoscopy) or an intensive care unit. As highlighted in the article, abdominal hypertension may lead to an increase in pulse pressure variations in this setting.[1]

To our knowledge, the ability of dynamic index to predict fluid responsiveness in the specific obese population has not yet been tested. The possible impact of obesity should be more complex than stated by Dr. Roth. Indeed, a direct effect of the fat distribution in obese patients is a reduction in the respiratory system compliance due to the reduction in lung and chest wall compliance.[4,5] As mentioned previously, decrease in chest wall compliance will induce an increase in pulse pressure variations. In contrast, during decreased lung compliance, airway pressure transmission is reduced such that the cyclic changes in intrathoracic pressure could be attenuated even in case of marked changes in alveolar pressure.[6] Thus, Monnet *et al.*[7] recently demonstrated that the predictive value of pulse pressure variations is related to lung compliance. The clinical impact of these hypothetic mechanisms, which is probably low, except for morbid obese patients, remains to be demonstrated.

We do not agree with Dr. Roth when he claims that administering neuromuscular blockade may result in a lower volume infusion. This affirmation is not supported by any publication. The impact of neuromuscular blockade administered in anesthetized patients without pathological decrease in chest wall compliance on dynamic index has never been demonstrated. Furthermore, most of the patients included in validation studies of dynamic index in intensive care unit were not paralyzed.[8]

On the contrary, we totally agree with Dr. Roth that stroke volume variations and its surrogates should not be the endpoint of therapy. As clearly mentioned in the article, the dynamic index must be considered as useful tools to predict an increase in stroke volume after volume expansion. But they clearly cannot help to answer the following question: Does my patient need an increase in stroke volume?

Matthieu Biais, M.D.,* Alexandre Ouattara, M.D., Ph.D., Gérard Janvier, M.D., Ph.D., François Sztark, M.D., Ph.D. *Hospital Pellegrin, CHU de Bordeaux, and Université Bordeaux, Adaptation cardiovasculaire à l'ischémie, Bordeaux, France. matthieu.biais@chu-bordeaux

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Pharyngeal Cooling, Brain Temperature Reduction and a Neglect of History

To the Editor:

Takeda *et al.*[1] are to be congratulated for carrying out a series of experiments in monkeys and man in developing and evaluating a minimally invasive technique to selectively lower brain temperature. There are, however, a number of questions concerning the methodology and experimental design, which should be mentioned.

It should be noted that 10 monkeys were involved in this study, five of which were subjected to 30 min of cooling after cardiac arrest and resuscitation, whereas the remaining five served as the controls and arrested, resuscitated but not