3. Shibutani K, Muraoka M, Shirasaki S, Kubal K, Sanchala VT, Gupte P: Do changes in end-tidal PCO2 quantitatively reflect changes in cardiac output? Anesth Analg 1994; 79:829–33
4. Monge García MI, Gil Cano A, Gracia Romero M, Monterroso Pintado R, Pérez Madueño V, Díaz Monrové JC: Non-invasive assessment of fluid responsiveness by changes in partial end-tidal CO2 pressure during a passive leg-raising maneuver. Ann Intensive Care 2012; 2:9

(Accepted for publication April 25, 2013.)

In Reply:

It is very important to optimize cardiac output and oxygen delivery by optimizing fluid therapy, and pulse pressure variation can help to identify fluid responsiveness. Dr. Sondergaard is right in underlining all the limitations of pulse pressure variation, but anesthesia provides an ideal setting for the use of pulse pressure variation as there is no spontaneous breathing effort during controlled mechanical ventilation and usually no bronchospasm or right heart failure.

Dr. Sondergaard reinforces our provocative statement that we may not have to measure cardiac output during surgery[1] when he writes that "YES, we *have* to measure cardiac output in high-risk surgery to optimize oxygen delivery" without detailing how the measurement can help practically; this sounds to us rather like dogma.

Jean-Louis Vincent, M.D., Ph.D.,* David Fagnoul, M.D.
*Erasme Hospital, Université libre de Bruxelles, Brussels, Belgium. jlvincen@ulb.ac.be

Reference

1. Vincent JL, Fagnoul D: Do we need to monitor cardiac output during major surgery? Anesthesiology 2012; 117:1151–2

(Accepted for publication April 25, 2013.)

In Reply:

We thank Drs. Youngblood and Sondergaard for their letters regarding our recently published article.[1]

We agree with Dr. Youngblood[1] that standard monitoring already provides substantial information about our patients' hemodynamic status. Today, pulse pressure variation (PPV) and its corresponding noninvasive parameters are easily available and used in clinical practice.[2] In recent years, PPV analysis algorithms have been integrated into different standard monitoring systems,[3] and PPV can be reliably assessed on a noninvasive basis by plethysmography.[4] Thus, standard monitoring evolves, and today PPV can be considered a standard hemodynamic parameter for a larger population of patients who require perioperative invasive blood pressure monitoring with an arterial line. When no arterial line is required, pulse oximetry devices, which provide the respective functional hemodynamic parameters, can be applied to assess and predict fluid responsiveness. Clearly, continuous end-tidal carbon dioxide monitoring may further support clinical decision making—but still this requires validation in large perioperative clinical studies. In addition, one has to keep in mind that the changes in end-tidal carbon dioxide, induced by a passive leg raising, cannot always be assessed during surgery.

We read with interest Dr. Sondergaard's acerbic letter regarding our article; we thank him for the querulous interest he has shown in our work. Neither our results nor our discussion demonstrate that the PPV should replace cardiac output (CO) measurements. Dr. Sondergaard's reference of our work when making this claim suggests that he is less familiar with our results.

We do demonstrate that when estimating CO trends, volume expansion-induced changes in PPV is superior to standard measures of arterial pressure.[1,5] PPV is interesting because it identifies patients who will benefit from fluid loading and volume expansion-induced changes in PPV allows for ongoing assessment of fluid-loading efficacy. This provides the clinician with important information which then allows for CO optimization.

Does an increased CO, as a result of fluid loading, result in improved postoperative outcomes? Dr. Sondergaard will be disappointed when we affirm that we do not have the answer to this question. He may be even more disappointed when we affirm that the answer to this question can probably not be framed in his favored "YES/NO" format.

CO is only one of the components of oxygen delivery, and maximizing CO, through fluid administration, does not equate to optimization of oxygen delivery. Fluid loading always causes hemodilution, and hemoglobin concentration plays a key role in oxygen delivery. It is therefore possible that increasing CO, by administering a large fluid load, may actually decrease oxygen delivery. Oxygen delivery is the true parameter to optimize, but it remains difficult to measure and several parameters should be simultaneously optimized to achieve ideal oxygen delivery. PPV and volume expansion-induced changes in PPV are only two of the tools, which may be used to achieve this goal. Using them in isolation may be appropriate in some clinical settings, but it will always remain suboptimal.

Dr. Cannesson is a consultant for Edwards Lifesciences (Irvine, California), Covidien (Boulder, Colorado), Masimo Corp. (Irvine, California), Philips Medical System (Suresnes, France), and Fresenius Kabi (Sèvres, France) and owns equity in Sironis (Newport Beach, California). Dr. Le Manach is a consultant for Air Liquide Santé (Paris, France) and received lectures/travel fees from Masimo Corp. (Irvine, California) and Fresenius Kabi (Sèvres, France). Dr. Tavernier received lectures/travel fees from Masimo Corp. (Irvine, California) and Fresenius Kabi (Sèvres, France). Dr. Hofer is a consultant for Pulsion Medical Systems (Munich, Germany), Edwards LifeSciences (Irvine, California), and CSL Behring (King of Prussia, Pennsylvania). Dr. Vallet received lectures/travel fees from Masimo Corp. (Irvine, California) and Edwards Lifesciences (Irvine, California), Fresenius Kabi (Sèvres, France), and Baxter Corp. (Deerfield, Illinois).