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(Accepted for publication April 25, 2013.)

## Cardiac Output Monitoring Is Already Standard

To the Editor:

We congratulate Le Manach *et al.*<sup>1</sup> for their demonstration of the superiority of changes in pulse pressure variation for the assessment of volume expansion–induced changes in cardiac output (CO) as compared with standard measures of arterial blood pressure. The findings clearly demonstrate the fallacy of determining the adequacy of CO based on static measurements of arterial pressure and encourage practitioners to thoughtfully reconsider how we assess patient responses to volume administration.

Certainly, many modalities exist to assess the response to volume infusion. In the current study, thermodilution, pulse contour analysis, esophageal Doppler, and transesophageal echocardiography are specifically mentioned. Although effective, each requires the addition of technology and invasiveness beyond standard monitoring, which is not universally available or necessary. Omitted from both this study and the accompanying editorial is reference to using our standard monitors in the assessment of this response.

Through citation of a 2011 survey of anesthesiologists,<sup>2</sup> the authors state that “most anesthesiologists do not monitor CO during high-risk surgery,” a premise that is a gross oversimplification of standard monitoring. End-tidal carbon dioxide (ETCO<sub>2</sub>) monitoring is a standard anesthetic practice and functions in the determination of both adequate ventilation and circulation. Low-CO states result in increased pulmonary dead-space and altered carbon dioxide elimination. In clinical practice, decreases in ETCO<sub>2</sub> have long been associated with deteriorating cardiovascular status, and the gradient between ETCO<sub>2</sub> and the partial pressure of carbon dioxide in arterial blood has been used as a surrogate for the adequacy of CO.

In 1994, Shibutani *et al.*<sup>3</sup> demonstrated the strong linear correlation between changes in CO and ETCO<sub>2</sub>. Recently, Monge Garcia *et al.*<sup>4</sup> demonstrated that changes in ETCO<sub>2</sub> after passive leg raising had a sensitivity of 90.5% and specificity of 93.7% to predict fluid responsiveness using a methodology similar to many of the pulse pressure variation and stroke volume variation models. We frequently use the arterial-ETCO<sub>2</sub> gradient as part of our assessment of CO and the trend of this gradient to assess the impact of therapeutic interventions, the most common of which being intravascular volume expansion.

We thank Le Manach *et al.* for their work and suggest that it teaches very important lessons in cardiovascular physiology and volume management. Although the authors demonstrated the use of pulse pressure variation for assessing hemodynamic responses to fluid administration, we suggest that our standard monitoring already provides substantial information about our patients’ hemodynamic status. More advanced, invasive, and expensive monitoring should continue to be applied on a case-by-case basis.

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(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<sup>1</sup> 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.

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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.<sup>1</sup>

We agree with Dr. Youngblood<sup>1</sup> 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.<sup>2</sup> In recent years, PPV analysis algorithms have been integrated into different standard monitoring systems,<sup>3</sup> and PPV can be reliably assessed on a noninvasive basis by plethysmography.<sup>4</sup> 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.<sup>1,5</sup> 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).