Should We Offer the Surgeon a Break?

To the Editor:
Le Manach et al. present a study demonstrating the experimental applicability of changes in pulse pressure variation (dPPV) for the prediction of changes in cardiac output (dCO). The study is accompanied by a panegyric editorial answering the rhetorical question whether we need to monitor CO during surgery with a resounding NO! We now have dPPV!1,2 There is, however, a number of considerations to be made before this opinionist view is accepted—or rejected.

1. None of the studies adduced in support of fluid optimization through CO maximization deal with oxygen delivery; one was a review, one was historical, and remaining four were centered on volume optimization.

2. It is questionable whether transesophageal measurements reliably measures changes in CO of less than 1 l/min,3,4 this may eliminate 152 of 402 patients from the analysis.

3. The evaluation of PPV is strictly restricted to the anesthetized, muscle-relaxed patient being positive pressure ventilated. It is based on the Frank–Starling cardiac function curve to the exclusion of considerations of venous return. The caveats to its use, so far published, run into 10.5–17 The PPV methodology assesses a left-sided response to a right-sided excitation, and the signal has to pass four valves, four chambers, and two vascular systems, and you will never know whether the arrival of the signal causes constructive or destructive interference with the cyclic inflation of the lungs. It is not clear whether the authors checked all of these caveats, or how many patients were excluded from the study for these reasons. It is not clear why optimization should be restricted to the peroperative period.

4. The experimental procedure needs a baseline, an intervention, and a new assessment. The time course is 25–30 min. All peroperative factors are to be kept stable during this period. What is the surgeon supposed to do during the volume expansion? And how often?

5. The factors not accounted for in the PPV methodology are the heart efficiency and vasomotor tone, both significantly affected during anesthesia. Why should they be restored with fluids? The detrimental effects of ignoring these determinants were amply demonstrated by Challand.18

6. Figure 2 in study by Le Manach does not imbue me with any trust: a change in PPV of −5% may indicate a change in CO of −5 to 40% without telling me whether the patient actually needs this for his or her oxygen delivery. What do I tell next-of-kin if the patient suffers a complication from the overload?

The Conclusions Are Obvious

YES, we have to measure CO in high-risk surgery to optimize oxygen delivery.

YES, we have to use validated and calibrated dilution methods with continuous, updated arterial curve–based calculation of CO, and

YES, we have to adopt a cardiovascular model capable of integrating heart efficiency, vasomotor tone, and stressed volume in the assessment of cardiovascular optimization. Guyton’s histocentric model is one suggestion. In one sweep, it updates the Starling-based PPV of 189619 to Guyton-based venous return physiology of 1963.20

And, YES, key opinion holders in the European intensive care community should be very cautious in promoting this PPV dream of cardiovascular control.

The PPV methodology is an instructive reflection of complex cardiopulmonary physiology, and it has not, though, attained the sophistication to be of reliable use clinically.

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


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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, 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 (ETCO₂) 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 ETCO₂ have long been associated with deteriorating cardiovascular status, and the gradient between ETCO₂ 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. demonstrated the strong linear correlation between changes in CO and ETCO₂. Recently, Monge García et al. demonstrated that changes in ETCO₂ 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-ETCO₂ 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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