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Mean systemic filling pressure: we can now estimate it, but for what? Response to comment by Parkin

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Dear Editor,

I read with interest the letter by Dr. Parkin [1], who raised several points that are in full agreement with what I wrote in my editorial [2]. Dr. Parkin must be respected for having, in the past, elegantly proposed an estimation of the mean systemic pressure (P_{ms}) from an equation that includes mean arterial pressure (MAP), central venous pressure (CVP), and cardiac output (CO) according to a Guytonian model of the circulation. I agree with him that such estimation seems to be valid. However, in his letter Dr. Parkin seems a little confused about the definition and clinical application of the concept of volume responsiveness or unresponsiveness. The following sentences are aimed to clarify these important points. For a clinician, there are two different issues to deal with: one is prediction of volume responsiveness/unresponsiveness, and one is assessment of the response to fluid once it has been infused. Predicting volume responsiveness/unresponsiveness is of major importance, since we know that volume overload is deleterious for critically ill patients [3]. In this respect, identifying in advance patients who would not benefit (no significant increase in CO) from volume infusion would avoid overloading them. In the study to which the editorial [2] referred, Cecconi et al. [4] showed that the value of P_{ms} before any fluid infusion cannot predict volume responsiveness at all. This result was not so surprising since, at best, P_{ms} is a static measure of effective blood volume and thus should share with other static hemodynamic variables the disadvantage of being unable to predict volume responsiveness/unresponsiveness [5]. A totally different issue is to assess the actual hemodynamic response to fluid administration, once it has been done. In this situation, the clinician wants to know how much was the actual benefit of fluid administration in terms of increase in CO. In order to do this, nothing is better than a direct measure of systemic blood flow, i.e., CO. By definition, calculation of P_{ms} using the formula developed by Dr. Parkin needs real-time CO measurements. In this situation, where a real-time CO monitor is used, the value of CO is under the eyes of the clinician. Therefore, what could be the interest of looking at the changes in P_{ms} (so-called ΔP_{ms}) after fluid administration rather than looking at the changes in CO, which provide a direct and relevant quantification of the response to fluid administration? In addition, because P_{ms} is calculated from MAP, CVP, and CO, it must cumulate the potential errors of measurements of each of these hemodynamic variables. So, why complicate what one can do simply? This is the reason why, in terms of clinical practice, the "so what?" question that I mentioned in the title of my editorial deserves to be asked without any kind of provocation.

Conflicts of interest The author has no conflict of interest related to this manuscript.

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