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Passive leg raising: easy at last! Reply to Monnet

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Dear Editor,

We thank Drs. Monnet and Teboul [1] for their comments on our work [2]. They underlined that our area under the receiver operating characteristics (ROC) curve (AUC) for PLR-induced changes in cardiac output ($\Delta_{\text{PLR}}\text{CO}$) was lower than reported in other works. First of all, contrary to what they stated, the AUC we reported was 0.89 (0.81–0.94) and not 0.83, i.e., similar (0.89) [3] or not so far from values already reported (0.93–0.96) [4, 5]. The significance of this difference is not obvious and cannot be statistically judged without evaluating individual patient data. Drs. Monnet and Teboul then propose two hypotheses to explain the slightly lower AUC we found:

First, our non-“beat-to-beat” technique of cardiac output (CO) measurement (thermodilution started after 1 min of PLR) may be inappropriate as it may not capture the full magnitude of the transient CO change induced by PLR and therefore risks false negativity of $\Delta_{\text{PLR}}\text{CO}$. Actually, this hypothesis seems unlikely as $\Delta_{\text{PLR}}\text{CO}$ was associated, in our study, with an excellent AUC of 0.98 (0.89–1) if the central venous pressure (CVP) change criterion was fulfilled. Furthermore, when performing a “blind PLR” (without

paying attention to CVP changes), others, using beat-to-beat CO measurement (ultrasonography), reported exactly the same AUC for $\Delta_{\text{PLR}}\text{CO}$ as we did [3].

Second, our PLR technique (trunk initially supine, legs elevated), the only one mentioned in recent international guidelines [6], is deemed to lack sensitivity by comparison with an alternative one (from semi-recumbent to supine posture). A whole part of our discussion section is dedicated to this point. Beyond the fact that the performances of these two techniques were never actually compared (no AUC comparison has been made), using the “semi-recumbent to supine” maneuver, the same poor performance we observed for “blind” PLR-related changes in arterial pulse pressure ($\Delta_{\text{PLR}}\text{PP}$) has been reported [4, 5].

Last, as the measurement of CVP during PLR may be considered cumbersome, one may be tempted to waive it in clinical practice. We believe that CVP is a relatively simple monitoring technique widely used in many intensive care units over the world and that, whatever the chosen PLR technique, measuring CVP is, as recommended [6], mandatory to ensure that the Frank–Starling mechanism of the heart is tested.

As $\Delta_{\text{PLR}}\text{PP}$ was a good predictor of fluid responsiveness in our population with CVP increase ≥ 2 mmHg, and provided that it only requires both an arterial and a central venous line (and no invasive and/or sophisticated device for CO measurement), our opinion is that fluid responsiveness prediction with PLR has never been so easy.

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