Passive leg raising test in patients with intra-abdominal hypertension: do not throw it

Alexandra Beurton[1,2], Jean-Louis Teboul[3,4], Xavier Monnet[3,4]

[1]Department of Critical Care Medicine, Pitié Salpêtrière Hospital, Sorbonne University, Assistance publique - Hôpitaux de Paris, Paris, France; [2]Inserm UMR S_1158, Sorbonne University, Paris, France; [3]Department of Critical Care Medicine, Bicêtre Hospital, Paris-Saclay University, Assistance publique - Hôpitaux de Paris, Le Kremlin-Bicêtre, France; [4]Inserm UMR S_999, Paris-Saclay University, Le Kremlin-Bicêtre, France

Correspondence to: Alexandra Beurton, MD, PhD. Service de pneumologie médecine intensive réanimation, Hôpital de la Pitié Salpêtrière, 47-83 Bd de l'Hôpital, 75651 Paris Cedex 13, France. Email: alexandra.beurton@aphp.fr.

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We thank Minini, Abraham and Malbrain for their constructive editorial commentary about our recent publication "*Intra-Abdominal Hypertension Is Responsible for False Negatives to the Passive Leg Raising Test*" (1).

In this study, while the infusion of a fluid bolus led to a similar increase in cardiac index in patients with and without intra-abdominal hypertension (IAH), the increase in cardiac index induced by a passive leg raising (PLR) test was 60% less in patients with IAH than in patients without. This logically induced some false negatives to the PLR test for detecting preload responsiveness (1).

For explaining the reduced effect of PLR on cardiac preload and cardiac index in patients with IAH, we made two hypotheses. The first is a potential reduction of the blood volume contained in the large splanchnic venous reservoir due to the increase in its extramural pressure. As this splanchnic blood likely represents a large part of the venous blood transferred to the cardiac cavities (2), this explains the reduction of the PLR-induced increase in cardiac preload. The second hypothesis is an increase of the transmural pressure of the inferior vena cava, which might increase the resistance to venous return and reduce the transfer of blood to the cardiac chambers (3). As a third hypothesis, Minini *et al.* suggest that the capillary leak provoked by IAH could reduce the volume of blood being autotransfused during PLR. However, the 1-min PLR test is likely too short for allowing a capillary leakage of significant volume, as suggested by the delay over which it occurred in the animal study describing this IAH-induced capillary leak (4).

We agree with Minini and colleagues, that these are only hypotheses, and that other investigations estimations of the venous return determinants are actually needed to confirm them.

As pointed out by Minini *et al.*, an interesting finding of our study was that, during the PLR test, intra-abdominal pressure (IAP) significantly decreased in patients with IAH by 29%±11%. This might be due to the cephalic displacement of the diaphragm during PLR, increasing the abdominal compliance. Also, the relief of the weight of the diaphragm on the abdominal cavity might contribute to the decrease of IAP during the PLR test. Minini *et al.* also suspected that the IAP was subject to errors in measurements in our study, due to the change in height of the pressure sensor. This cannot be excluded, since there is no reference for measuring IAP. Nevertheless, we carefully paid attention to our method of measurement for ensuring that the position of the pressure sensor remained stable.

What are the practical consequences of our study? First, one should remind that the reliability of the PLR test has been demonstrated in many studies. With pulse pressure variation, it is the most reliable way of assessing fluid responsiveness, with a much higher number of conditions of validity (5). We agree with Minini *et al.* that the IAP should be checked in patients in whom it is suspected to be elevated. By contrast, we do not agree with the recommendation to check the presence of high positive end-expiratory pressure (PEEP) and auto-PEEP, as there is no clear reason why they may affect the PLR test reliability.

Moreover, many of the studies demonstrating the PLR reliability included patients with acute respiratory distress syndrome receiving PEEP. We also disagree with their recommendation to consider a lower threshold of cardiac output changes to define the test positivity. In our study, the statistical analysis could not identify any threshold providing acceptable sensitivity and specificity in patients with IAH (1).

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Footnote

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