Bioreactance for estimating cardiac output and the effects of passive leg raising in critically ill patients

Editor—I read with interest the study of E. Kupersztych-Hagege and colleagues,1 entitled: ‘Bioreactance is not reliable for estimating cardiac output and the effects of passive leg raising in critically ill patients’. However, I believe that this conclusion is flawed for the following reasons.

First, since 83% of the patients of the study had sepsis and ‘most of them’ had acute respiratory distress syndrome, it would be wise to restrict the title and conclusion to these patients.

Secondly, three thermodilution boluses were averaged as reference method and unexpected results were probably removed to ensure an adequate averaging, as generally recommended. In contrast, only one instantaneous value of bioreactance was collected. In a way, this is like comparing the resolution of a carefully taken picture and a freeze video image. In other papers where acceptable concordance was observed, 10 min of bioreactance trend lines were averaged while thermodilution boluses were performed. This method has been recommended for smoothing the impacts of artifacts, differences in time responses and precisions, and comparing really the two technologies.

Thirdly, it has been well shown that the minimum time response of the bioreactance technology was 1 min. In this study, the passive leg raising (PLR) results were assessed after 1 min. The bioreactance changes were therefore necessarily underestimated. This time delay limited to 1 min is surprising since two co-authors of this paper have popularized the PLR test recommending a time frame 30–90 s, especially in septic patients.

Finally, the study showed that the agreement between bioreactance and thermodilution was below that expected from chance alone (43%). This corroborates the area under the ROC curve close to zero for predicting fluid responsiveness. These results only tell us that, in this study, the inappropriate data acquisition seemingly made the value of bioreactance close to that obtained at random.

Subsequently, four references are provided to support the so-called ‘Bioreactance less promising results’. In reality, the paper from Fagnoul and colleagues2 included 11 patients, the paper from Engoren and Barbée3 investigated another technology (bioimpedance), the study of Weisz and colleagues4 was done in neonates where a bioreactance calibration factor has never been calculated. Finally, the paper from Marik and colleagues5 concluded that ‘Monitoring the hemodynamic response to a PLR manoeuvre using Bioreactance provides an accurate method of assessing volume responsiveness in critically ill patients’. I think it is still true.

Declaration of interest

P.S. was a consultant for Cheetah Med from 2005 to 2010.

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Reply from the authors to Dr Squara

Editor—We are thankful to Dr Squara for his interest in our study2 and for his comments. We would like to answer his criticisms point by point.

First concerning the title of the article, we did not specifically demonstrate that the unreliability of the Nicom was related to septic shock or acute respiratory distress syndrome. In the absence of any certitude about this point and to be scientifically rigorous, we chose a title that simply specified the population that was actually included, that is, critically ill patients.

Secondly, no thermodilution curve was rejected from analysis. We previously showed that, with such a method, the precision of transpulmonary thermodilution is 12%.6 Dr Squara suggests that we should have taken the value of cardiac index averaged over 10 min rather than the instantaneous value of cardiac index displayed by the Nicom device. Of course, it is obvious that this would have reduced the influence of artifacts on cardiac index measurements. Nevertheless, the manufacturer clearly insists on the ‘fast responsiveness’ of the technique. What our study simply shows is that it is actually untrue, at least in critically ill patients.

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Thirdly, we are afraid that Dr Squara did not read our methods section carefully. Indeed, we state that we assessed the effects of passive leg raising when they reached their maximum, which occurs ‘within 1 minute’. This does not mean that passive leg raising was strictly limited to 1 min. Again, the study simply demonstrates that the Nicom could not be used to assess the passive leg raising test. We believe that this information is actually useful for clinicians since they may not be aware of the slow time response.

Fourthly, we strongly disagree with Dr Squara regarding the inappropriate data acquisition for the reasons stated above. Finally, we agree with Dr Squara that the study by Marik and colleagues could seem positive. Nevertheless, one should emphasize that the authors did not use any cardiac output reference technique in this study. Moreover, the positivity of that study suggests that the arguments of Dr Squara regarding averaging over 10 min are not pertinent and that our negative results could not be only explained by the slow time response of the Nicom device.

To conclude, we believe that our conclusions are fully supported by the data. Indeed, they showed that the Nicom device was not reliable in our critically ill patients, especially for performing the passive leg raising test, when used in the way that is recommended for current practice. We do not claim that it would be so unreliable for monitoring stable patients over 10 min periods.

Declaration of interest
X.M. and J.-L.T. are members of the Medical Advisory Board of Pulsion Medical Systems.

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3Marik PE, Levitov A, Young A, Andrews L. The use of NICOM (Bioreactance) and Carotid Doppler to determine volume responsiveness and blood flow redistribution following passive leg raising in hemodynamically unstable patients. Chest 2013; 143: 364 – 70

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Bioreactance is not reliable for estimating cardiac output and the effects of passive leg raising in critically ill patients

Editor—We read with interest the study by Kupersztych-Hagege and colleagues,1 ‘Bioreactance is not reliable for estimating cardiac output and the effects of passive leg raising (PLR) in critically ill patients’. Key methods in the study differed from well known and accepted literature and also Cheetah NICOM Instructions for Use (IFU). After a detailed and careful review of the paper, we believe that issues with study execution and the manner in which previous studies are referenced lead to flawed conclusions about NICOM’s capabilities.

1. Cheetah NICOM was not used in accordance with its IFU: Kupersztych-Hagege and colleagues chose a PLR challenge duration of only 1 min, although two of the authors (J.L. Teboul and X. Monnet) have published a paper, which was a clinical review of the literature, where they state the PLR needs to be performed over 30–90 s to provide real-time tracking, especially in septic patients.2 In practice, a patient normally reaches the maximum cardiac index (CI) during the PLR challenge around the 60 s mark. The Cheetah NICOM averages its measurements every 1 min, and the maximum CI resulting from the PLR challenge is normally observed in the second minute of the PLR challenge rather than the first. Kupersztych-Hagege and colleagues performed the Cheetah NICOM measurements outside accepted and even their own group’s suggested PLR protocol. Therefore, the maximum CI value measured in the study’s PLR challenge by the Cheetah NICOM is unlikely to represent the true maximal change. Furthermore, the Cheetah NICOM is equipped with a PLR Wizard that automatically measures CI during a 3 min challenge and calculates the percentage change from baseline. To achieve and measure maximal change in CI due to the PLR, while allowing for a stable baseline, the authors would need to follow the IFU. This would have allowed the maximal change in CI to be determined. For this same reason, when the authors performed the back to baseline validation, the NICOM was still recording the PLR challenge (minutes 2 and 3 from initiation of the PLR challenge). Thus, comparison with the PICCO thermodilution CI value at this point is based on incorrect use of the NICOM and the subsequent measurement is not valid.

2. The manner by which the PLR threshold was selected is vague—Kupersztych-Hagege and colleagues used a threshold of 9% to differentiate responders from non-responders, while co-authors previously have reported a threshold of 10–12% as significant thresholds in reporting fluid responsiveness. We are given no indication as to why they chose the threshold of 9%. In previous PLR studies, the threshold was selected by optimization of the selected device, based on the sensitivity and specificity of that device to detect change; in the form of the Youden index, ROC, and maximum sensitivity and specificity. In the current study, the authors state only one threshold value but fail to inform the reader as to which technology the cut-off (threshold) was applicable, and to which technology the optimization was valid (PICCO or NICOM). As different technologies yield different thresholds for PLR to predict fluid responsiveness, it is imperative to inform the reader of the methodologies in selecting the PLR cut off.2–4 If PLR predictiveness was optimized for PICCO technology, followed by testing with Cheetah NICOM, this would further flaw the results of the study, falsely undermining the reliability of the NICOM device.