To the editor

We read with great interest the article by Veillard Baron et al. about right ventricular (RV) failure in septic shock and its link with fluid responsiveness [1].

The authors defined right ventricular failure as the association of RV dilatation (RV/LVEDA < 0.6) and increased central venous pressure (CVP \( \geq 8 \) mmHg). They showed that this definition of RV failure is associated with lack of fluid responsiveness. Therefore, CVP could be used as an additional measurement to RV dilation to discriminate between patients with and without congestive RV failure. This rationale seems attractive.

However, several limitations may weaken the conclusion of this study.

Parameters used to define RV failure

The echocardiographic pattern chosen by the authors is a limited RV dilation that may be an adaptive response (as shown during high intensity or endurance exercise) without actual RV failure. In two-dimensional echocardiography, the consensus definition of the American Society of Echocardiography and the European Association of Cardiovascular Imaging of RV global systolic dysfunction is still based on RV-fractional area contraction (RV-FAC), whilst global RV function is based on right ventricular index of myocardial performance [2]. These parameters would have been of great interest in this setting.

Measurement of CVP

Several confusing factors could mislead in CVP interpretation in this context, especially because a large number of patients in group 3 has a CVP between 8 and 10 mmHg and several patients of group 1 and 2 has a CVP close to 8 mmHg. First, group 3 patients have a relatively high rate of atrial fibrillation (20%): a factor well known to increase CVP values independently from venous congestion [3]. Second, The CVP threshold of 8 mmHg or greater is questionable knowing that mean systemic filling pressure varies from 7 to 10 cmH2O. Hence, we suggest the use of other markers of venous congestion as hepatic or portal venous Doppler [4].

PLR maneuver and intra-abdominal pressure (IAP)

Regarding the reliability of PLR maneuver to assess fluid responsiveness, it has been shown more than 10 years ago that an IAP over 12 mmHg may induce false negatives [5]. This point has been discussed by
Vieillard-Baron et al. in the limitation part of their study. However, because patients in group 3 have higher values of IAP (median of 11 mmHg and interquartile range of 8–14 mmHg) than other groups of patients, the number of false negatives should not be neglected.

**Authors’ response**

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Dear Dr Abou-Arab,

We acknowledge that definition of right ventricular (RV) failure in our study is far from perfect. The key message is that patients with RV failure could not be fluid-responsive despite significant pulse pressure variations.

Distinction between RV dysfunction and failure is not easy to draw, while crucial in the critically ill patients. Lahm et al. re-emphasized that RV systolic dysfunction indicates structural RV changes, which could cause in the most severe form RV failure [6]. We reported the tricuspid annular systolic excursion (TAPSE) was similar whatever CVP and RV size and did not classify patients in the RV failure group. Such surprising results could be explained because parameters of RV systolic function, as TAPSE and fractional area contraction, did not tightly reflect coupling between the right ventricle and the pulmonary circulation [7].

From a physiological point of view, RV failure is defined as the association of RV dilatation with systemic congestion and may even occur when the cardiac output is still maintained [6, 8]. Moderate or even mild RV dilatation cannot be anymore an adaptation when associated with systemic congestion. Magder’s group has reported the ability of healthy individuals to dramatically increase cardiac output during sustained exercise with only a slight increase in right atrial pressure, while patients who previously received heart transplantation had a much lower increase in cardiac output with a significant elevation in right atrial pressure [9].

We agree that the CVP threshold above which systemic congestion may be still questioned is still questionable. However, the kidney is known to be very sensitive to any slight alteration in CVP. Chen et al. reported in critically ill patients an increased risk of acute kidney injury for each 1 cmH₂O increased CVP when compared to the “normal value” which was ≤ 7 cmH₂O [10]. CVP is also known to be much more associated with worsening of renal function than a low cardiac index, especially when above 8 mmHg. Normal value of mean systemic filling pressure (MSFP) was indeed reported around 7–10 mmHg in animals. How it may be translated to critically-ill patients remains questionable and we reported a MSFP around 13 mmHg in septic patients just after death [11].

Finally, we used passive leg raising to assess fluid-responsiveness, while Mahjoub et al. reported false-negative may be indeed observed in case of elevated intra-abdominal pressure. But what the authors didn’t say is that it was mainly observed in case of pressure ≥ 16 mmHg, a situation rarely observed in our study.

In conclusion, our study evaluated a new definition of RV failure, associating RV size evaluated by echocardiography and CVP. Future works are required to confirm our approach and to improve detection and characterization of RV failure in critically-ill patients.

**Abbreviations**

CVP: Central venous pressure; RV: Right ventricle.

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**Authors’ contributions**

OAA, MDM and CB were responsible for the manuscript draft. YM revised the manuscript. All the authors approved the final version of the manuscript. All authors read and approved the final manuscript.

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**Competing of interests**

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