Critical Care

LETTER

Open Access

Comments on "Right ventricular failure in septic shock: characterization, incidence and impact on fluid responsiveness": which parameter to assess right ventricular failure and venous congestion?

Osama Abou-Arab[1*], Mouhamed D. Moussa[2], Christophe Beyls[1] and Yazine Mahjoub[1]

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 ≥ 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 dilatation 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

This comment refers to the article available online at https://doi.org/10.1186/s13054-020-03345-z.

*Correspondence: osama.abouarab@gmail.com
[1] Department of Anesthesiology and Critical Care Medicine, Amiens Picardie University Hospital, 1 Rue du Professeur Christian Cabrol, 80054 Amiens, France
Full list of author information is available at the end of the article

© The Author(s) 2021. **Open Access** This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit http://creativecommons.org/licenses/by/4.0/. The Creative Commons Public Domain Dedication waiver (http://creativecommons.org/publicdomain/zero/1.0/) applies to the data made available in this article, unless otherwise stated in a credit line to the data.

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

Antoine Vieillard-Baron[3*] and Guillaume Geri[4]

*Correspondence: antoine.vieillard-baron@aphp.fr
[3] Intensive Care Unit, University Hospital Ambroise Paré, Assistance Publique des Hôpitaux de Paris, 92100 Boulogne-Billancourt, France
[4] UFR de Médecine Simone Veil, Université Paris Saclay, 78000 Versailles, France.

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-responsiveness 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 suspected 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 cmH_2O increased CVP when compared to the "normal value" which was ≤ 7 cmH_2O [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.

Acknowledgements
None.

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.

Funding
None.

Availability of data and materials
Not applicable.

Ethics approval and consent to participate
Not applicable.

Consent for publication
Not applicable.

Competing of interests
None.

Author details
[1] Department of Anesthesiology and Critical Care Medicine, Amiens Picardie University Hospital, 1 Rue du Professeur Christian Cabrol, 80054 Amiens, France. [2] Department of Anesthesiology and Critical Care Medicine, Institut, Cœur – Poumon, CHU Lille, 2 Avenue Oscar Lambret, 59037 Lille, France.

Received: 18 December 2020 Accepted: 13 January 2021
Published online: 09 April 2021

References
1. Vieillard-Baron A, Prigent A, Repessé X, et al. Right ventricular failure in septic shock: characterization, incidence and impact on fluid responsiveness. Crit Care. 2020;24:630. https://doi.org/10.1186/s13054-020-03345-z.
2. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28(1–39):e14.

3. Wasmund SL, Li J-M, Page RL, Joglar JA, Kowal RC, Smith ML, et al. Effect of atrial fibrillation and an irregular ventricular response on sympathetic nerve activity in human subjects. Circulation. 2003;107:2011–5.
4. Spiegel R, Teeter W, Sullivan S, et al. The use of venous Doppler to predict adverse kidney events in a general ICU cohort. Crit Care. 2020;24:615. https://doi.org/10.1186/s13054-020-03330-6.
5. Mahjoub Y, Pila C, Friggeri A, Zogheib E, Lobjoie E, Tinturier F, et al. Assessing fluid responsiveness in critically ill patients: False-positive pulse pressure variation is detected by Doppler echocardiographic evaluation of the right ventricle. Crit Care Med. 2009;37:2570–5.
6. Lahm T, Douglas IS, Archer SL, et al. Assessment of right ventricular function in the research setting: knowledge gaps and pathways forward: an official American Thoracic Society research statement. Am J Respir Crit Care Med. 2018;198:e15–43.
7. Sanz J, Sanchez-Quintana D, Bossone E, et al. Anatomy, function, and dysfunction of the right ventricle. *JACC* state-of-the-art review. JACC. 2019;73:1463–82.
8. Vieillard-Baron A, Naeije R, Haddad F, et al. Diagnostic workup, etiologies and management of acute right ventricle failure: a state-of-the-art paper. Care Med. 2018;44:774–90.
9. Notarius CF, Levy RD, Tully A, et al. Cardiac versus noncardiac limits to exercise after heart transplantation. Am Heart J. 1998;135:339–48.
10. Chen KP, Cavender S, Lee J, et al. Peripheral edema, central venous pressure, and risk of AKI in critical illness. Clin J Am Soc Nephrol. 2016;11:602–8.
11. Repessé X, Charron C, Fink J, et al. Value and determinants of the mean systemic filling pressure in critically ill patients. Am J Physiol Heart Circ Physiol. 2015;309:H1003.

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Ready to submit your research? Choose BMC and benefit from:

- fast, convenient online submission
- thorough peer review by experienced researchers in your field
- rapid publication on acceptance
- support for research data, including large and complex data types
- gold Open Access which fosters wider collaboration and increased citations
- maximum visibility for your research: over 100M website views per year

At BMC, research is always in progress.

Learn more biomedcentral.com/submissions