Correspondence on the debate regarding the haemodynamic definition of pulmonary hypertension

To the Editor:

We read with interest the balanced and well-constructed debate on the new haemodynamic definition of pulmonary hypertension (PH) published recently in the *European Respiratory Journal* [1, 2]. The current era of cardiopulmonary medicine is (or at least should be) at its best when defined by evidence-based decision-making. Thus, we agree with Hoeper and Humbert [1], who elegantly chronicle the trajectory of data through time that ultimately caused reconsideration to the mean pulmonary artery pressure (mPAP) threshold associated with clinical risk, now inclusive of >28 000 patients [3–6]. Since the basis of the original PH definition was largely arbitrary, the arc of debate toward a revised understanding on PAP was inevitable. In this regard, it seemed rational to modernise and align the definition of PH with the empirical evidence showing risk at lower mPAP values.

However, as Gibbs and Torbicki [2] emphasise in their position of dissent, the revised definition from the World Symposium on Pulmonary Hypertension charted a course into incompletely tested waters by incorporating pulmonary vascular resistance (PVR) >3.0 Wood units to the pre-capillary and combined pre-/post-capillary PH definitions [7]. In our view, modifying the PH definition by considering combinations of haemodynamic variables was a forward-thinking, innovative, and much-needed advance. Nonetheless, the timing of the assertion was, perhaps, somewhat early owing to limited data on the spectrum of clinical risk relative to PVR, particularly in patients with mildly elevated mPAP. It is not known, for example, to what extent the new PH definition captures end-stage right heart failure rather than early PH, which would be contrary to its intention. Consider the following haemodynamic scenario: mPAP 22 mmHg, pulmonary artery wedge pressure 13 mmHg, and PVR 3.0 Wood units. This profile is within the new PH definition, but must include a cardiac output of 3.0 $L \cdot min^{-1}$, which is well below a cardiac output level associated independently with increased mortality [8].

Sensitive to this point, as emphasised by both sides of the debate in the *European Respiratory Journal*, as well as by Simonneau *et al.* [7] in the original World Symposium on Pulmonary Hypertension report itself, is that the true contribution of PVR to risk in mild PH is not known. Clarifying this issue has, therefore, emerged as a primary goal in the pulmonary vascular field. Preliminary results from our own ongoing analyses suggest that PVR risk emerges well below 3.0 Wood units, and is clinically significant in mild PH. However, these data remain forthcoming. Of course, any further adjustment to the PH definition would not, in and of itself, inform on the appropriateness or efficacy of pharmacotherapy in these patients, nor are lessons from population studies a substitution for the interpretation of individual patient-level information at point of care. Thus, as is the case with most polarising points in medicine, the truth is likely to exist somewhere in between on this debate. Overall, continued international collaborative efforts to address the issue of PH definition seem more timely now than ever, and it is in this spirit that the pulmonary vascular medicine community reinvents itself in a productive way based on clinically relevant terms.

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Additional data are needed to clarify the haemodynamic spectrum of clinical risk in pulmonary hypertension http://bit.ly/2ZXgld8

Cite this article as: Maron BA, Choudhary G, Tedford RJ, *et al.* Correspondence on the debate regarding the haemodynamic definition of pulmonary hypertension. *Eur Respir J* 2019; 53: 1900727 [https://doi.org/10.1183/13993003.00727-2019].

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Received: April 10 2019 | Accepted: April 11 2019

Conflict of interest: None declared.

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