progressive rise in mortality risk was observed in those with pulmonary artery wedge pressure >15 mm Hg, likely observed in the setting of left heart failure. These collective findings reinforce the importance of using the totality of clinical and hemodynamic data to interpret mPAP, and illustrate potential pitfalls that accompany a narrower emphasis on any specific mPAP level for full delineation of normal from pathogenic states.

In conclusion, reduction of the mPAP threshold to >20 mm Hg and inclusion of a new PVR criteria highlights an emerging understanding of clinical risk in PH. However, sufficient data guiding evaluation and treatment of patients with mPAP of 20 to 25 mm Hg and PVR of 2.0 to 3.0 Wood units remain forthcoming. Perhaps a greater challenge to the PH field, however, involves the development of a flexible hemodynamic framework aligned with individual patient profiles, which is likely to require the use of data beyond mPAP of >20 mm Hg alone.

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References

Rebuttal From Dr Kovacs
Gabor Kovacs, MD Graz, Austria

Statements of the World Symposia on Pulmonary Hypertension (WSPH) represent a consensus among experts by summarizing current achievements and starting points for future research in the field of pulmonary vascular science. From this point of view, as discussed earlier, it is understandable that the 6th WSPH suggested the redefinition of the mean pulmonary arterial pressure (mPAP) cutoff for pulmonary hypertension (PH).

A further important role of WSPH is to provide a basis for upcoming PH guidelines, which is probably the main reason that the new mPAP cutoff for PH led to intensive discussions in the scientific community. This is also the cornerstone of the well-considered argumentation of Drs Johnson and Maron in their counterpoint to this question. In addition to previously used arguments against the new suggested mPAP cutoff, such as the lack of data supporting the treatment of patients with mPAP 20 to 25 mm Hg and the skepticism around the capacity for the revised definition to capture more patients, two important new arguments have been proposed: First, there is the risk of analyzing mPAP in isolation, without an appreciation for hemodynamic and clinical context that may lead to false interpretations and preclude the

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I could not agree more with the first argument. It is reassuring, however, that according to current PH guidelines “the interpretation of invasive haemodynamics should be made in the context of the clinical picture”; it is likely that this approach will be kept for future clinical practice guidelines. This means that, instead of focusing on a single hemodynamic variable, all relevant clinical circumstances that include age, comorbidities, and a variety of hemodynamic and clinical parameters should be taken into consideration before diagnostic and treatment choices are made. The final decisions concerning an individual patient must be made by responsible health professionals in consultation with the patient. Nevertheless, in this relation, the prognostic relevance of mPAP >20 mm Hg as a central parameter of the pulmonary hemodynamics becomes even more important and further justifies the redefinition of this hemodynamic cutoff.

The second argument opens a new field of discussions on the clinical relevance of mildly elevated PVR and the optimal threshold of this hemodynamic variable. However, this does not argue against the suggested new mPAP cutoff but rather further extends the discussion on the range of normal pulmonary hemodynamics. Of note, the proceedings of the 6th WSPH recognize that the cutoff value of PVR ≥3 Wood units is arbitrary and that according to recent data PVR >2 Wood units could be also considered abnormal. It will be an important duty of future studies and clinical practice guidelines to provide guidance on the interpretation of PVR 2.0 to 3.0 Wood units.

In conclusion, the redefinition of the mPAP cutoff for PH is an important step towards the right direction. Now, it is time for intense discussions and clinical research to optimize treatment strategies for patients with mild pulmonary hemodynamic abnormalities to help health professionals to make appropriate decisions in their daily practice.

References

Rebuttal From Drs Johnson and Maron

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We agree with many key points raised by Professor Kovacs and expand on his position by suggesting a call-to-action for accumulating evidence that informs the clinical treatment of patients with mean pulmonary artery pressure (mPAP) 21 to 24 mm Hg. Given data supporting the continuum, as opposed to binary risk profile associated with hemodynamic parameters, it may be the case that rather than over emphasizing any single hemodynamic measurement, optimal characterization of pulmonary hypertension (PH) subgroups requires deeper consideration of an integrated assessment of pulmonary hemodynamics.

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