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Adding an important piece to the pulmonary vascular resistance puzzle in pulmonary arterial hypertension

Bradley A. Maron[1] and Marc Humbert[2]

Affiliations: [1]Division of Cardiovascular Medicine, Brigham and Women's Hospital and Harvard Medical School and Dept of Cardiology, Boston VA Healthcare System, Boston, MA, USA. [2]Université Paris-Saclay, INSERM UMR_S 999, AP-HP, Service de Pneumologie, Hôpital Bicêtre, Le Kremlin-Bicêtre, France.

Correspondence: Bradley A. Maron, 77 Ave Louis Pasteur, NRB Rm 0630-N, Boston, MA 02115, USA. E-mail: bmaron@bwh.harvard.edu

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Clinical outcomes in pulmonary arterial hypertension (PAH) have improved substantially in the modern era, owing to greater clinician awareness, availability of numerous pulmonary vasodilator therapies and, now, more than two decades of sound clinical trial data informing optimal strategies for treating patients [1]. This arc of progress began with therapeutic interventions that aimed to simply delay mortality in patients with end-stage disease, at a time in which PAH was regarded as by-and-large uniformly fatal. The evolution of PAH into a contemporary and treatable disease has been marked by specific sentinel transition points, including proven efficacy of prescription exercise, sequential add-on treatment with different drug classes, and up-front combination therapy in newly diagnosed patients [2]. This has resulted in a collective shift toward greatly enhanced goals for defining treatment success in clinical practice, such as minimal symptom burden, preserved exercise tolerance, and favourable haemodynamic parameters [3].