Effects of pulmonary artery wedge pressure on right ventricular pulsatile loading in pulmonary hypertension: a reappraisal based on pulmonary arterial isobaric stiffness.

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Background. Pulmonary hypertension (PH) is associated with stiffening of pulmonary arteries. Previous studies have suggested that high pulmonary artery wedge pressure (PAWP) in postcapillary PH (Pc-PH) further augments PA stiffness at a given level of pulmonary vascular resistance as compared to pulmonary arterial hypertension (PAH). However, these studies do not take into account differences in distending pressure (mean PA pressure, mPAP), which has an effect on stiffness due to non-linear stress-strain behavior of arteries.

Purpose. To compare total PA stiffness between Pc-PH and idiopathic PAH (iPAH) studied at similar mPAP (isobaric stiffness).

Methods. This was an analysis of right heart catheterization results obtained in 112 Pc-PH and 112 iPAH patients extracted from the French PAH network registry and matched for mPAP (median 38 vs 39 mmHg, P=NS), age (70.5 years each) and sex (64% female each). Total PA stiffness was calculated as the ratio of PA pulse pressure to indexed stroke volume.

Results. Total PA stiffness (n=224) increased with mPAP (Spearman’s rho = 0.66) and decreased with PAWP (rho = -0.17) (each P<0.01). The isobaric stiffness was lower in Pc-PH (median (IQR) = 0.91 (0.64-1.39) mmHg/mL/m²) than in iPAH (1.18 (0.83-1.62) mmHg/mL/m², P<0.01). The patients were then stratified according to their mPAP (25-35 mmHg, n=74 (37/37); 36-43 mmHg, n=75 (34/41); and 44-66 mmHg, n=75 (41/34)). The isobaric stiffness was lower in Pc-PH than iPAH in the 1st mPAP tertile (0.62 vs 0.83 mmHg/mL/m², P=0.06), in the 2nd mPAP tertile (0.76 vs 1.22 mmHg/mL/m², P<0.01) and in the 3rd mPAP tertile (1.41 vs 1.77 mmHg/mL/m², P<0.01). The pulmonary vascular resistance was lower in Pc-PH than iPAH in every mPAP tertile (each P<0.01). Finally, Pc-PH had a higher indexed stroke volume than iPAH (37 (29-48) vs 32 (27-40) mL/m², P<0.01) while systolic PA pressure and PA pulse pressure were similar.

Conclusion. Unexpectedly, the isobaric pulmonary arterial stiffness was lower in Pc-PH than iPAH patients. It is proposed that PAWP attenuates the increase in RV pulsatile loading in PH when the natural high-strain-induced stiffening was accounted for. This may contribute to a less impaired right ventricular-PA coupling leading to higher indexed stroke volume in Pc-PH than iPAH despite similar PA pressure. At every mPAP level, both the lower PA stiffness and lower pulmonary vascular resistance in Pc-PH than in iPAH may contribute to explain differences in the pressure overload-induced right ventricular adaptation between the two diseased groups, a point that deserves to be confirmed by further studies.