In conclusion, patients with PAH exhibit markedly diminished RV contractile reserve and pulmonary arterial vascular reserve. Abnormal stress responses occur even in patients with relatively preserved haemodynamics at rest. There is evidence that an impairment of cardiovascular stress response could be correlated with exercise capacity and prognosis. We agree with SHARMA *et al.* [1] that the potential utility of measuring the cardiovascular reserve and the optimal dosing protocol of dobutamine stress requires further validation studies.

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Patients with PAH exhibit markedly diminished RV contractile reserve and pulmonary arterial vascular reserve http://ow.ly/L7or6

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Received: Feb 09 2015 | Accepted: Feb 15 2015

Conflict of interest: None declared.

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Eur Respir J 2015; 45: 1756–1758 | DOI: 10.1183/09031936.00023015 |

From the authors:

We agree with our colleagues J.C. Grignola and E. Domingo on the importance of measurements of both right ventricular (RV) and pulmonary vascular reserve in patients with pulmonary arterial hypertension, and that further studies are needed to define the most relevant variables and optimal methodology [1]. They have a point that isometric-phase measures of RV systolic function, such as isovolumic myocardial acceleration, may be preferable, as they are less load-dependent than more commonly assessed indices, such as the *S′* wave, tricuspid annular plane systolic excursion, fractional area change or ejection fraction. Isometric-phase measurements best correlate with the gold-standard end-systolic elastance in intact experimental animals [2]. The maximum velocity of isovolumic contraction has been shown to be a stronger predictor of outcome than a number of other echocardiographic measurements of RV function in patients with severe pulmonary hypertension [3]. However, what matters in the end is how RV contractility increases in a fashion adapted to afterload [4]. This may be disclosed by simpler surrogate measurements of contractile reserve such as changes in RV peak systolic pressure during exercise [5].

As for pulmonary vascular reserve, this may be defined by a combination of a shallow slope of the pulmonary vascular pressure–flow relationship, exercise-induced changes in pulmonary vascular resistance, pulmonary blood volume (estimated by lung diffusing capacity) and exercise-related pulmonary transit of agitated contrast, all reflecting the distensibility of pulmonary resistive vessels [6–8]. Whether combining assessment of contractile reserve and pulmonary vascular reserve is of clinical relevance (relationship to exercise capacity and or outcome) remains to be seen. Whether dobutamine can be substituted for exercise is an open question too. Against J.C. Grignola and E. Domingo's concern, it seems that low-dose dobutamine decreases rather than increases the slope of multipoint pulmonary artery pressure–flow relationships both in normal subjects [9] and in patients with severe pulmonary hypertension [10], suggesting β-receptor-mediated pulmonary vasodilation rather than α-receptor-mediated pulmonary vasoconstriction. Thus, a low-dose dobutamine infusion, compared to exercise, does not carry a risk of worsening pulmonary hypertension, but may improve more than simply challenge RV–arterial coupling.

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Clinical utility and optimal indices of RV contractile reserve in PAH remains an open question
http://ow.ly/L7XbS

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Received: Feb 15 2015 | Accepted: Feb 16 2015

Conflict of interest: None declared.

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