Abstract Preview

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Title: From systolic pulmonary arterial pressure to pulmonary vascular reserve: a simplified method for exercise stress echocardiography

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Background: Systolic pulmonary arterial pressure (SPAP) can be noninvasively estimated by Doppler exercise stress echocardiography (ESE) with either tricuspid regurgitant velocity (TRV) or pulmonary flow acceleration time (ACT) when tricuspid regurgitation is missing. Estimated SPAP values are however flow-dependent and this is especially important during ESE, when transpulmonary flow can increase by 2-to-10 times with unpredictable inter-individual variability.

Aim: To assess the feasibility of estimate pulmonary vascular reserve (PVR) during ESE.

Methods: We performed rest TTE and ESE in 81 subjects (age 48±16 yrs, 49 females) recruited by three laboratories from Italy and Poland. 42 patients at risk for pulmonary arterial hypertension (29 systemic sclerosis, 13 with other connective tissue diseases, Group I) and 39 fit healthy normals (evaluated for assessment of exercise capacity, Group II). ESE readers were quality-controlled, upstream to recruitment, with a web-based system. TTE rest and stress assessment included: TRV with continuous-wave Doppler; ACT (from onset of ejection to peak flow velocity) from pulsed-Doppler of systolic pulmonary forward flow in right ventricular outflow tract. Both parameters were measured at rest and peak exercise. SPAP was estimated from the modified Bernoulli equation (4V²+ estimated right atrial pressure, with V=TRV). If TRV recording was not available SPAP was derived from the ACT, on the basis of the correlation: log10 SPAP = 0.004 (ACT) + 2.1. In each subject we calculated an index of pulmonary vascular reserve (PVR, min of exercise/ΔSPAP). Exercise-time was assumed as a proxy of cardiac output. ΔSPAP was defined as the difference between SPAP value at peak minus rest, expressed in tens (for instance: Δ20 mmHg = 2.0; Δ35 mmHg = 3.5). When ΔSPAP was <10 mmHg, it was considered equal to 1.
Results: Average exercise-time was 9.8 ± 3.8 minutes (ranging from 4 to 18 minutes) and workload achieved 122 ± 47 Watts (from 50 to 225 Watts). TRV was interpretable in 49/81 (60%), ACT in 79/81 (98%), and at least one in 61/81 (100%). SPAP increased in Group 1 (rest 24 ± 11 vs stress 41 ± 19 mmHg, p<0.0001) more markedly than in Group 2 (rest 21 ± 4.4 vs stress 28 ± 12.7 mmHg, p=0.002). By TRV and/or ACT criteria, peak stress SPAP was normal (< 43 mmHg) in 24 subjects of Group 1 and 34 of Group 2 (57 vs 87%, p=0.0061). PVR was lower in Group 1 vs Group 2 (4.92 ± 2.82 vs 10.96 ± 4.39, p<0.0001). Representative examples of Group 1 and Group 2 with the same peak SPAP and clearly different PVR are shown in Figure.

Conclusion: PVR can be easily estimated integrating TRV and/or ACT and exercise time in virtually all patients to provide an estimation of pulmonary hemodynamics less dependent from flow than SPAP. Lower PVR values are found in patients at risk for pulmonary arterial hypertension compared to healthy normals.

Figure: PVR values in patient at risk for pulmonary arterial hypertension (red line) and normal subject (green line).