AEPC-YIA Session

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[I-YIA-2]Exercise-Induced Peripheral Venous Hypertension inversely correlates to peak VO2 in Fontan patients

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Introductions: Fontan patients have a reduced exercise capacity due to limitation in the ability to augment cardiac output. Central venous pressure (CVP) rises to increase systemic ventricular preload because of the absence of subpulmonary ventricular in Fontan patients. We performed a prospective trial of cardiopulmonary exercise test (CPET) while monitoring peripheral venous pressure (PVP) which strongly correlates to CVP to investigate the correlation between PVP and exercise capacity.
Methods: Seventeen patients with Fontan circulation (9 males, median age: 12 years) underwent ramp-like progressive exercise test on a treadmill. A 22-gauge venous cannula was inserted into the peripheral vein in the upper extremities before CPET. PVP (mmHg) was monitored at rest and during exercise. We checked correlation between PVP at peak exercise and exercise-related hemodynamic indices including peak VO2 (l/min/kg), HR reserve (bpm) (HRR) and peak oxygen pulse (l/m2) respectively. We also performed catheterization and blood examination to measure CVP (mmHg), ventricular end-diastolic pressure (mmHg), ventricular ejection fraction (%), cardiac index (l/min/m2), pulmonary vascular resistance (unit· m2), pulmonary artery index and brain natriuretic peptide (pg/mL). We also checked correlation between these indices and peak VO2.
Results: PVP rose in concert with exercise intensity in all subjects. PVPs at peak exercise was significantly higher than those at rest (23.5 ± 4.4 vs 12 ± 1.7, $p < 0.01$). Peak PVP inversely correlated to peak VO2, HRR and peak oxygen pulse ($r = -0.66$, $p < 0.01$, $r = -0.6$, $p < 0.05$, $r = -0.5$, $p < 0.05$). No static hemodynamic indices from catheterization or blood examination correlated to Peak VO2.
Conclusions: In Fontan patients, exercise induced peripheral venous hypertension means inefficiency of pulmonary flow augmentation during exercise, causing insufficient ventricular filling which induces not only insufficient cardiac output and insufficient stroke volume but also attenuated HR increase.