Sildenafil Improves Exercise Hemodynamics in Fontan patients.

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Background: Reduced preload reserve, especially with increased heart rate, is a fundamental limitation of the Fontan circulation. Sildenafil may increase exercise capacity in patients with Fontan physiology, but the underlying mechanism is still unclear. This study tested the hypothesis that sildenafil would improve exercise hemodynamics, especially ventricular filling, in Fontan patients using a novel cardiac magnetic resonance (CMR) methodology.

Methods: Ten Fontan patients (6 male, age 20±4 years) underwent CMR at rest and during supine exercise on a programmable cycle ergometer before (“baseline”) and after a single dose of sildenafil (50 mg oral). Systemic ventricular volumes were obtained at rest and during mild (104±11 bpm), moderate (127±16 bpm) and strenuous (147±15 bpm) exercise. Ventricular filling rate (VFR, ml/msec) was defined as stroke volume corrected for RR-interval. Bi-plane cine images were acquired using an ungated, free-breathing real-time CMR sequence (12-18 contiguous 8mm slices) and analyzed using software developed enabling retrospective gating for cardiac phase and respiratory translation. Endocardial borders were delineated using a bi-plane model. Simultaneously, radial and pulmonary artery pressures were measured.

Results: Under resting conditions as compared with baseline, sildenafil reduced pulmonary artery pressure (9±3 to 8±3 mmHg, P=0.029) and increased cardiac output (6.8 to 8.1 L/min, P=0.006) and VFR (113±27 to 134±35 ml/sec, P=0.006). During exercise sildenafil resulted in improved hemodynamics as compared with baseline. Pulmonary artery pressure decreased (mean difference 1.6±1.0 mmHg, P=0.006), whilst cardiac output (mean difference 1.7±0.8 L/min, P=0.001), ejection fraction (mean difference 4.8±2.8%, P=0.003), stroke volume (mean difference 6.0±5.6 ml, P=0.039) and VFR (mean difference 28±14 ml/sec, P=0.001) all increased.

Conclusion: In patients with Fontan physiology, sildenafil improves cardiac output during exercise despite a reduction in pulmonary artery pressures. This implies that pulmonary vasodilation is a potential physiological target for improving exercise hemodynamics, the clinical significance of which warrants further study.