surrogates of ventricular–vascular coupling in the pediatric population

Following palliative treatment of congenital heart disease, the cardiovascular functional outcome depends on myocardial, vascular and end-organ factors. Clinical evaluation and research studies have focused on either myocardial or vascular performance, but there is a recent trend towards integrated vascular and ventricular assessment. This is based on the recognition that arterial stiffness is an important determinant of adverse events in patients with cardiovascular risk factors and left ventricle (LV) relaxation could be mediated by changes in arterial function. The mechanism by which arterial function affects myocardial performance is based on arterial wave reflections that normally return during early diastole in compliant arteries, but return during late systole in arteries that are stiffened by atherosclerosis or due to postoperative changes in the setting of congenital heart disease [1]. The increase in late systolic load prolongs systolic interval, resulting in a delayed onset of LV relaxation [2]. On the other hand, decreased distensibility of the arterial wall leads to increased LV afterload and impairment of myocardial blood flow because of decreased coronary perfusion pressure [3]. Elucidation of ventricular–vascular coupling has traditionally involved invasive techniques for measurement of myocardial pressure–volume relationships and arterial pressure. Using echocardiographic (echo) and MR techniques, such as tissue Doppler, speckle tracking, myocardial deformation imaging (strain and strain-rate imaging, torsion), phase-contrast imaging and arterial tonometry, changes in systolic and diastolic function of the myocardium and vessel wall properties can now be studied noninvasively [4]. This group of studies explores whether echo or MR parameters alone can quantify ventricular–arterial coupling on the right and left side of the heart in the pediatric population. It is possible that reliable noninvasive imaging surrogate markers of ventricular–vascular coupling could pave the way for risk stratification of patients with palliated coronary heart disease, pediatric cardiomyopathies or pulmonary hypertension and allow early detection and intervention in patients at increased risk of right ventricle or LV failure.

References


Obese children with lipid abnormalities have reduced systolic and diastolic LV deformation characteristics, early vessel wall changes and increased arterial stiffness. Koopman et al. aimed to characterize the relationship between vascular and myocardial parameters in obese children with lipid abnormalities. Twenty one overweight and obese children (10–18 years old with a BMI >85th percentile) with lipid abnormalities and 27 randomly selected matched, normal-weight controls, were enrolled in the study [1]. Color tissue Doppler and speckle-tracking echocardiography and vascular tonometry were used for cardiac and vascular assessment. When compared with matched controls, obese children had lower LV systolic radial strain values (p = 0.002), lower LV systolic longitudinal strain values (p < 0.001) and lower LV early diastolic strain values (p < 0.001). When subjected to vascular assessment, the obese group had faster pulse-wave velocity, increased carotid intima–media thickness and lower arterial distension coefficients. The authors also noted that the lower arterial elastance to LV end-systolic elastance suggests abnormal ventricular–vascular coupling in the obese group. While the sample size is limited, this study uses established techniques to characterize obesity-related changes in cardiac and vascular parameters.

Reference

Ventricular–vascular coupling in pulmonary hypertension


By comparing cardiac MR with standard right heart catheterization, Sanz et al. aimed to characterize and validate the noninvasive assessment of right ventricle–arterial coupling in patients with pulmonary hypertension [1].

A total of 139 patients with known or suspected pulmonary hypertension who underwent standard right heart catheterization and cardiac MRI were included in this cross-sectional retrospective study. Right ventricle–arterial coupling was defined as the ratio of pulmonary artery effective elastance (Ea) to right ventricular maximal end-systolic elastance (Emax). Their results show that Ea increased linearly with disease progression (p < 0.001), but Emax increased early, plateaued and decreased in the late phase of the disease (p = 0.7). Therefore, Ea/Emax was maintained in earlier stages but increased with late stages of pulmonary hypertension. By using simple cardiac MR-derived functional parameters, such as end-diastolic volume, end-systolic volume, right ventricle ejection fraction and anterograde pulmonary artery stroke volume corrected for body surface area, estimates of Emax and Ea from MRI correlated well with right heart catheterization (p < 0.001). This study demonstrates a simple, noninvasive method for quantification of right ventricle–arterial coupling that can be applied in the clinical setting and that demonstrates the predicted changes in coupling with increasing severity of pulmonary hypertension.

Reference

Effect of sildenafil on ventricular–vascular coupling in Fontan patients


Using noninvasive techniques to assess ventricle–arterial coupling, Shabanian et al. aimed to show sildenafil effects on patients with Fontan physiology [1].

Twenty three patients with extracardiac total cavopulmonary connection (12–31 years old) were enrolled in the study, and echocardiography was performed before and after a 1-week course of sildenafil. Sixteen patients had LV morphology, four patients had right ventricle morphology, and three patients had right and left ventricle morphology. Their results show that sildenafil increased ventricular end-systolic elastance (p = 0.001) and decreased arterial elastance (p < 0.0001), ventricular end-diastolic elastance (p = 0.002) and ventricular–arterial coupling index (p < 0.0001).
There was also a small, but significant, improvement in oxygen saturation with sildenafil without effect on heart rate or blood pressure. While the sample size is limited, the authors conclude that a short-course treatment of sildenafil improved ventricular–arterial coupling in patients with Fontan morphology. The study shows the usefulness of noninvasive techniques to characterize ventricular–vascular coupling in patients with congenitally and surgically altered anatomy.

**Reference**