Device closure of atrial septal defects acutely changes right ventricular strain in children.

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Background: Strain rate imaging (SRI) is a novel technique to non-invasively quantitate ventricular function and may be particularly useful to study the right ventricle (RV). Device closure of an atrial septal defect leads to rapid volume unloading of the RV and redirection of the pulmonary venous return to the left ventricle (LV). However, a previous study did not find any changes of myocardial velocities in ASD patients after this procedure.

Objective: This study used SRI to quantitate RV function after ASD device closure.

Methods: Color myocardial Doppler imaging including SRI was performed in 11 ASD patients (aged 9.3 ± 4.4 y) and 11 age-matched normal children (10.4 ± 4.5 y) at frame rates >100fps from apical 4 chamber views and stored as digital raw data for off-line post-processing (Echopac, GE Vingmed). All patients had significant left-to-right shunts with a mean Qp/Qs-ratio of 2.0 ± 0.4 and normal RV pressures. Patients were studied PRE, immediately POST and 24h post intervention. Peak systolic and diastolic strain rate (SR) and peak systolic strain were determined in the mid wall of the RV as mean of 3 cardiac cycles. Peak myocardial velocities were measured in the basal third of the same 6 walls. T-test was used for statistics accepting p < 0.05 as significant.

Results: At baseline, ASD patients were similar to control except for a higher diastolic SR (table). Immediately post intervention, strain decreased significantly and was still lower than baseline at the 24h follow up. Systolic and diastolic strain rate and the peak systolic velocity also fell POST but were back to baseline at 24h.

Conclusions: Strain rate imaging is a more sensitive tool to study changes of right ventricular mechanics than myocardial velocities. However, it appears from our study that strain and strain rate like velocities are load-dependent to some degree.

Evaluation of right ventricular diastolic function in patients with chronic obstructive pulmonary disease using pulsed Doppler tissue echocardiography.

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In the recent clinical studies, pulsed doppler tissue echocardiography (DTI) has been a valuable diagnostic tool in the evaluation of diastolic function. In patients with chronic obstructive pulmonary disease (COPD) the pulmonary artery pressure rises and this results in an increase in right ventricular pressure which finally causes diastolic dysfunction of the right ventricle(RV). In this study we compared the diastolic functions of RV in stable COPD patients to normal people using DTI and aimed to detect the most sensitive DTI parameter of RV diastolic dysfunction.

We studied 20 patients with COPD (mean age 61.4 ± 6.8 years, 16 males) who were clinically stable by respiratory function testing (RFT) and arterial blood gasses. Twenty, age and sex matched normal subjects (mean age 57.8 ± 4.0 years, 11 males) were taken as the control group. All the participants underwent conventional 2 dimensional echocardiography. DTI of the RV was performed from the following positions and locations: RV from parasternal long axis view; apical, middle and apical levels of RV from the apical 4 chamber view. The measured DTI parameters are as follows: early diastolic maximal velocity (e wave) and e wave deceleration time (eDT), late diastolic maximal velocity (a wave), systolic maximal velocity (s wave) and duration of s wave, isovolumic relaxation time (IVRT) and isovolumetric contraction time (IVCT).

The conventional echocardiographic measurements of the 2 groups were similar. However, the DTI parameters of stable COPD patients were significantly different from normal controls. Early diastolic maximal velocity and s wave from parasternal long axis view were higher in patients with stable COPD. IVRT measured from apex and parasternal views of RV and eDT measured from RV apex from apical 4 chamber view were significantly longer in stable COPD patients. (parasternal e-wave: in stable COPD patients 13.8±4.6 cm/s, control group 18.7±3.4 cm/s, p<0.01; parasternal s wave duration; 21.5±5.7 ms vs 25.9±3.5 ms, p<0.05; apical e-wave: RV; 19.3±4.8 ms vs 13.6±2.0 ms, p<0.01; apical 4 chamber middle level IVRT; 14.9±5.2 ms vs 9.8±1.6 ms, p<0.05; apical 4 chamber apex IVRT; 17.2±2.1 ms vs 13.3±2.6 ms, p<0.05; apical 4 chamber eDT; 8.6±2.5 ms vs 10.7±2.8 ms, p<0.05).

This study gives an important clue to the fact that DTI is an sensitive diagnostic tool in the evaluation of RV diastolic function where conventional echocardiography fails. In the light of this data IVRT of RV wall should be considered a significant parameter indicating RV diastolic dysfunction.

Right ventricular IVRT measured by tissue Doppler may provide an alternative estimate of right ventricular pressure.

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Background: Measuring the systolic tricuspid valve gradient (TVG) by echocardiographic blood flow Doppler is a well received method for assessing right ventricular (RV) pressure in pulmonary hypertension. In a number of patients, however, this is not feasible due to the lack of tricuspid regurgitation or insufficient Doppler signals in poor image quality. Tissue Doppler is a robust technique for measuring RV wall velocities even in hard to scan patients. This study was performed to investigate how RV pressure is reflected by RV tissue Doppler parameters.

Methods: In a pilot study, 20 patients (age 56±14 years, 11 female) in sinus rhythm, without bundle branch block, with well measurable TVG and not more than mildly reduced left ventricular function (ejection fraction > 40%) were examined using a GE Vingmed Vivid 7 ultrasound scanner. Three heart cycles of both pulsed wave tissue Doppler (TD), color Doppler traces of the basal right atrial wall and of the entire RV were acquired from an apical window and stored digitally. Peak systolic velocities (Vps), time to Vps (Tps), E' and A' wave velocities (Ve, Va) as well as the duration of the biphasic signal between the end of systole and the onset of E'-wave, reflecting RV isovolumic relaxation time (IVRT), were measured in both data sets. Parameters were compared to TVG. In order to avoid subjectivity right atrial pressure was neglected. Patients were divided in a group with lower (up to 25mmHg) and with higher (more than 25mmHg) TVG.

Results: In our study population, TVG ranged from 18 to 61mmHg. There was no or only weak correlation between TVG and Vps, Tps, Ve or Va (all r<0.5). However, TVG correlated well with RV IVRT (r=0.88, p<0.05). Accordingly, patients with lower TVG (n=11, mean TVG 22±2mmHg) and patients with higher TVG (mean 39±11mmHg) differed significantly in RV IVRT (25±17ms vs 70±28ms, resp., p<0.05). A cut-off of 40ms detected patients with higher TVG with a sensitivity and specificity of 88% and 82%, resp.

Conclusion: Due to the low endysystolic pressure difference between right atrium and ventricle, RV IVRT is normally very short. Our results suggest that in disease echocardiographic pressures result in a prolongation of the IVRT, showing systolic lation, may be used as a diagnostic marker. Therefore, we conclude that right ventricular IVRT measured by tissue Doppler may offer an alternative estimate of elevated right ventricular pressure in difficult to scan patients with pulmonary hyper tension.

The value of pressure strain loops in the assessment of right ventricular regional function in patients with pulmonary arterial hypertension.

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Right ventricular (RV) function in pathological conditions remains poorly understood. The contribution of anatomically and morphologically different segments of the RV to global ventricular performance still needs further studies. Ultrasound-based strain analysis allows quantification of regional myocardial deformation. We investigated the feasibility of measuring S during the entire cardiac cycle simultaneously with invasively measured pressure (P) changes in patients with pulmonary arterial hypertension (PAH). Additionally, based on the P and S measurements, P-S loops were derived for the assessment of regional RV dysfunction.

Concomitant right heart catheterisation and echocardiography were performed in 16 pts. For the S analysis, the RV free wall was divided into its 2 morphologically distinct segments: the basal smooth and the apical trabecular part. Digitally stored P traces were matched with simultaneously recorded S profiles. Both traces were averaged over three consecutive heart cycles and plotted as a P-S loop. The area enclosed by P-S loop (PSA) was calculated. the cardiac index (CI) was determined by thermodilution.

The PSA from the basal segment (9.5 mmHg) was significantly higher than that from the apical (5.9 mmHg; p<0.02). In patients with decreased CI (<2.8 l/min/m2), apical PSA was significantly lower (3.2 vs. 8.6, p<0.07) whereas basal PSA remained similar (9.2 vs. 9.9).

Conclusion: P-S loops can be measured in PAH pts. P-S loop area can be interpreted as a surrogate measure of “regional work” performed by the RV in the segment under investigation. In basal patients, decreased RV function, apical PSA was decreased more than basal PSA indicating that the apical RV portion is affected more by PAH and fails earlier than the basal part.