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1140-45

Effect of Afterload Reduction on Left Atrial Appendage Tissue Velocities After Percutaneous Mitral Commissurotomy for Mitral Stenosis

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OBJECTIVES: This study sought to investigate the relative load dependency of left atrial appendage (LAA) tissue Doppler velocities in patients (Pts) with mitral stenosis after percutaneous mitral commissurotomy (PCM).

BACKGROUND: Assessment of left atrial function is difficult and depends on loading conditions, transmitral pressure gradient and left atrial intrinsic properties. In mitral stenosis, elevated resistance to blood flow through the stenotic valve is a model of increased left atrial afterload. We sought to determine the influence of the acute decrease in transmitral pressure gradient after successful PMC on LAA velocity measured by tissue Doppler imaging (TDI) and LAA emptying flow velocity, respectively.

METHODS: Transoesophageal echocardiography was performed immediately before and 24 hours after PMC in 34 Pts with pure severe mitral stenosis (55 \pm 11 years, 20 with sinus rhythm, 14 with atrial fibrillation) for measurement of LAA area, LAA emptying flow velocity and LAA wall systolic contraction velocity by pulsed-mode TDI.

RESULTS: After PMC, mitral valve area significantly increased from 1.0 \pm 0.3 cm² to 2.0 \pm 0.5 cm² while left atrial pressure and transmitral pressure gradient decreased from 19 \pm 5 to 11 \pm 8 mmHg and from 9.2 \pm 4.8 to 4.6 \pm 1.6 mmHg, respectively (p < 0.05). LAA area was unchanged (6.0 \pm 0.6 vs. 4.2 \pm 0.3 cm², p=ns) while LAA flow emptying velocity increased from 23 \pm 11 to 34 \pm 13 cm/s (p < 0.05).

LAA tissue velocities consisted of a triphasic velocity profile: V₁ and V₂ occurring during left ventricular contraction and relaxation, respectively and V₃ following the atrial contraction. After PCM, V₂ and V₃ velocities consistently increased (4.8±1.2 to 7.9±2.6 cm.s⁻¹, p < 0.001 and 6.6±2.9 to 8.1±4.2 cm.s⁻¹, p < 0.05, respectively). No correlation was found between transmitral pressure gradient and LAA emptying flow velocity, but a significant correlation was found with LAA systolic contraction velocity (V₃).

CONCLUSIONS: Evaluation of regional left atrial function is feasible by TDI, demonstrating a triphasic pattern in LAA velocity profile. TDI parameters accurately reflect acute changes in left atrial loading conditions whereas LAA flow parameters are poorly influenced.

1140-46 Myocardial Acceleration During Isovolumic Contraction: A Load-Independent Tissue Doppler Marker of Ventricular Contractility

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Background: Myocardial acceleration during isovolumic contraction (IVA) is a tissue Doppler (TDE) marker for contractility. In animal experiments, IVA reportedly was more load-independent and thus a better marker of RV contractility than myocardial velocities. For clinical validation, we studied interventional device-closure of an atrial septal communication (ASD) as a model for acute intra-cardiac volume changes comparing the effect on tissue velocities and IVA. Methods: TDE was performed in 39 pediatric (median 8y; 1.1-19 y) and 12 adult patients (49y; 36-70y) before and right after ASD device closure. Median Qp/Qs-rato was 1.7 with a stretched ASD size of 8 to 30 mm (median 16 mm). 32 Amplatzer and 19 other devices (12-40 mm) were used. TDE cineloops of 3 cycles were stored as digital raw data. Peak systolic (S) longitudinal and radial velocities and IVA were measured in 6 basal segments (Echopac, GE-Vingmed). IVA is the peak isovolumic contraction velocity divided by acceleration time. Multivariate regression analysis was used (p<0.05). Results: Right after ASD closure, S velocities fell in all segments (table). In contrast, IVA did not change. Shortening fraction remained normal (37.7±4.9 vs. 38.1±6.2%; n.s.). The velocity and IVA response was independent of age, shunt or device size. Conclusions: ASD device closure acutely changes systolic myocardial velocities but not IVA or FS. This clinical study supports that IVA is a more load-independent marker of contractility than myocardial velocities.

N=51	S (cm/s)			IVA (m/s2)		
SEGMENT	PRE	POST	ρ	PRE	POST	ρ
ANTERIOR LV	7.8±2.1	5.6±2.0	<0.05	1.5±0.5	1.4±0.5	NS
INFERIOR LV	6.7±1.7	5.3±1.4	<0.05	1.4±0.5	1.4±0.6	NS
LATERAL LV	8.0±2.1	5.8±2.1	<0.05	1.2±0.6	1.2±0.6	NS
SEPTAL LV	5.9±1.6	4.7±1.1	<0.05	1.3±0.5	1.2±0.6	NS
RV	10.3±2.6	8.5±2.2	<0.05	1.8±0.7	1.7±0.7	NS
POSTERIOR LV	4.8±1.4	4.0±1.0	<0.05	0.9±0.5	0.9±0.5	NS

1140-47 Left Ventricle Electro-Mechanical Asynchrony in Patients With Acute Myocardial Infarction Correlates With Infarct Size

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Background: Electrical and mechanical asynchrony of the left ventricle may be part of the remodeling process post myocardial infarction (MI). However, it is not clear to what extent this occurs in the absence of bundle branch block and if it is related to position and size of the infarction. Therefore, we have assessed the degree of LV synchrony of the whole LV post myocardial infarction using tissue Doppier imaging (TDI) and related this to the size and extent of the infarction measured by magnetic resonance imaging (MRI).

ABSTRACTS - Noninvasive Imaging 441A

Methods: Standard and tissue Doppler echocardiography were performed in 30 healthy volunteers (age 61.7±10.5yrs) and 45 MI patients (age59.5±11.5yrs) within 1~7 days at the apical 4-, 2- chamber and the short axis views. Mid/basal segments of septal, lateral, anterior, inferior and posterior walls were analyzed and their peak systolic and early diastolic velocities and timings were measured. The results were correlated to the site and extent of the infarction on MRI.

Results: All the patients had a normal QRS duration. The MI group had a delayed onset of systole with a prolonged isovolumic contraction time (IVCT) compared to normal (79.72±19.96ms vs 69.80±10.67ms, p=0.007) and an earlier onset of diastole with a shorter systolic duration (SD) (330.30±35.36ms vs 358.97±27.45ms, p<0.0005). The IVCT of the anterior MI. subgroup was significantly prolonged compared to both normal and the inferior MI. subgroup (101.56±28.67ms vs 69.8±10.67ms, p=0.001 and 101.56±28.67ms vs 73.03±10.93ms, p=0.03, respectively). Furthermore, the standard deviation of SD correlated well with the infarct size measured by MRI (r=0.5, p=0.02). **Conclusions:** Myocardial infarction has a significant impact on left ventricular synchronization early after the infarction even in the absence of bundle branch block esp. in the anterior MI patients. The larger the myocardial infracted area, the more severe the LV dyssynchrony.

1140-48 Wringing Motion of Normal Left Ventricle Quantified With Tissue Doppler Echocardiography

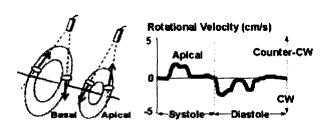
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Background: Release of potential energy stored during systole by left ventricular (LV) twisting improves early filling by promoting LV suction. We quantified this "wringing" motion of LV by a novel echocardiography method.

Methods: 2-D tissue Doppler images (Toshiba SSA-370A) of apical and basal LV short axis were obtained in 6 healthy men. Myocardial velocity profiles were obtained from the lateral and septal wall regions, where the line drawn from each region towards the LV center was perpendicular to the ultrasound beam. The subtraction of these velocities (lateral minus septal velocity) was defined as a rotational velocity.

Results: At the apical level, peak positive velocity in early systole $(1.40 \pm 0.72 \text{ cm/s}, \text{counterclockwise direction, as viewed from apex to base) and peak negative velocity in early diastole <math>(-1.42 \pm 0.64 \text{ cm/s}, \text{clockwise direction}, p = 0.003)$ were observed. At the basal level, systolic rotational velocity was biphasic while early peak diastolic velocity was positive $(2.27 \pm 1.1 \text{ cm/s}, \text{ counterclockwise direction})$. Majority of early systolic twisting and early diastolic untwisting occurred in isovolumic contraction and relaxation periods. R wave to peak twisting time (apical vs basal, 98 ± 30 vs. 89 ± 9 ms) and endo-systole to peak untwisting velocity (apical vs basal, 56 ± 17 vs. 80 ± 14 ms) were similar at both level.

Conclusion: LV "wringing" behavior can be quantified using tissue Doppler echocardiography.





Evaluation of Paradoxical Interventricular Septal Motion After Cardiac Surgery Assessment by Strain Rate Combined With Tissue Doppler Imaging

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Background: Abnormal motion of the interventricular septum (IVS) is frequently observed after open heart surgery without any complication. However it is difficult to evaluate whether IVS motion is really deteriorated or not. Strain rate imaging, recently developed technique in echocardiography, which derived from tissue Doppler imaging, can provide quantitative analysis of regional left ventricular contractile function. The purpose of this study is to evaluate IVS motion after cardiac surgery quantitatively by strain rate imaging. Methods: We examined 20 patients (pts). 10 pts had coronary aftery bypass grafting (CABG) surgery (CABG group). They had no evidence of old myocardial infarction and no significant complication during and after CABG. 10 pts had angina pectoris and treated medically and/or by catheter intervention (non-CABG group). Routine echocardiography and tissue Doppler imaging was performed using strain rate imaging system (Vivid 7, GE medical systems) in all pts. Data were recorded digitally, and tissue Doppler and strain rate was calculated by off-line analysis.

Results: There was no significant difference in end diastolic volume, end systolic volume, and ejection fraction between two groups. There was significant difference in tissue Doppler systolic wave of IVS in the parasternal long axis view which reflected abnormal septal wall motion between CABG group and non-CABG group (1.41±0.83 vs. -2.01±1.78, P<0.00001, respectively). On the other hand, there was no significant difference in peak systolic strain rate of IVS (-1.09±0.27vs. -1.33± 0.41, N.S.) and any other region in the abical views.

Conclusion: These results suggest that IVS motion deteriorates apparently after CABG