## 1126-115 Estimation of tau From the Analysis of Early Diastolic Intraventricular Flow Velocities: Results from a Modeled Human Left Ventricle In Vitro

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The rate of LV relaxation is an important determinant of its early diastolic performance, but is difficult to quantify by noninvasive means. Relaxation being an early diastolic process, we hypothesized that it would govern early diastolic flow events inside the LV and conversely, can be estimated by a combination of early diastolic events measurable by non invasive means. This hypothesis was investigated in a pulsatile LV model with geometric and functional similarities to human LV and biphasic mitral inflow pattern. Thirty-five sets of experiments were conducted at a heart rate of 60 bpm, a wide range of left atrial pressures (8–44 mmHg) and varying rates of LV relaxation. Flow velocities were recorded using pulsed wave Doppler technique at the mitral leaflet tips, mid LV inflow and the apex. LV pressure was recorded using Millar catheters. Amplitude and acceleration time of the E wave at all three locations and amplitude and duration of the LV isovolumic relaxation flow were measured. The mitral E wave transit time to the LV apex, $T_E$, was measured from the onset of the E wave at mitral valve to the onset of the transmitted E wave at the LV apex. Peak negative dP/dt and Tau were computed from the high fidelity LV pressure tracing.

*Results:* Tau had a significant correlation with $T_E$ ($r = 0.40$, $p = 0.0038$), mitral E wave amplitude ($r = -0.35$, $p = 0.041$), mitral E wave acceleration time ($r = 0.35$, $p = 0.043$), E wave acceleration time at mid inflow ($r = -0.50$, $p = 0.0038$) and change in acceleration time between mitral valve and mid inflow ($r = 0.46$, $p = 0.0092$). A multiple linear regression model using the above variables, absolute changes in the E wave amplitudes at the 3 LV locations and amplitude and duration of the LV isovolumic relaxation flow predicted Tau with a high degree of accuracy (cumulative $R = 0.76$). Addition of left atrial pressure to the regression equation increased the R value to 0.89 ($R^2 = 0.78$).

*Conclusions:* 1) This in vitro study indicates that the rate of LV relaxation impacts the amplitude and acceleration of the mitral E wave, the rate of E wave propagation to the LV apex and the profile of the E wave as it is transmitted to the LV apex. 2) It may be possible to derive Tau from a combination of multiple early diastolic flow derivatives obtained at different locations along the LV inflow as detailed above, but this needs to be confirmed in a human setting.

## 1126-116 A New Method for Estimating Negative dP/dt in Patients Without Valvular Regurgitation

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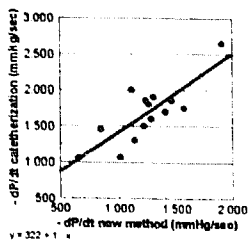
Negative dP/dt of the LV can be calculated noninvasively only from mitral or aortic regurgitant jet registered with continuous-wave Doppler.

*Purpose:* To describe a new method for estimating negative dP/dt in patients without valvular regurgitation.

*Methods:* Fifteen patients, 10 male and 5 female (age 59 ± 10 years) with isquemic heart disease who underwent cardiac catetherizationwere included. We performed an hour before the catetherization, Doppler of the transmittal and pulmonary venous (PV) flow, phonocardiogram, carotid pulse tracing calibrated with the arterial pressure measured with sphygmomanometer, and peak negative dP/dt during catetherization. Mean negative dP/dt was calculated as the slope of pressure fall from end-systolic to the mitral valve opening. End systolic pressure was calculated from calibrated carotid pulse tracing. Left atrial pressure (LAP) was assumed as: 10 mmHg if S wave of the PV was greater than D wave, 15 mmHg if S = D and 20 mmHg if D wave was greater than S wave. Isovolumic relaxation time (IRT) was measured from aortic component of second sound to beginning of transmitral flow. In this way: Mean negative dP/dt= (ESP-LAP) / IRT.

*Results:* Peak negative dP/dt measured at catetherization was correlated with the new method ($r = 0.81$, $p < 0.001$).




*Conclusion:* Mean negative dP/dt can be estimated by non-invasive techniques in patients without valvular regurgitation with Doppler transmitral and pulmonary venous flow combined with calibrated carotid pulse tracing.

## 1126-117 Right and Left Ventricular Diastolic Dysfunction in Patients With Myocardial Infarction: A Color M-Mode Analysis

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*Background:* Flow propagation velocity (FPV) in the LV measured on Color M-mode Doppler echocardiography (CTM) is highly dependent on the LV relaxation rate. As previously described, RV FPV might also be a valuable marker of RV diastolic function.

*Methods:* To assess both RV and LV diastolic function in pts with recent MI, we recorded RV and LV inflow on CTM in pts with normal LV function and no known CAD (gr 1, n = 13, age: 49 ± 14 yrs) and in pts with recent (18 ± 9 days) MI (gr 2, n = 62, age: 58 ± 11 yrs). To calculate LV and RV FPV, the linear slope of the color front wave during early filling was measured between the level of the valvular annulus and a point that was always deeper than 2 cm in the LV and 1.5 cm in the RV. Ratio of maximal velocities of E and A mitral and tricuspid waves (E/A) were also studied by pulsed wave Doppler.

*Results:* End-diastolic and end-systolic LV diameters (mm) were smaller in gr 1 than in gr 2 (LVEDD: 43 ± 3 vs 54 ± 5, $p < 0.0001$; LVESD: 27 ± 3 vs 40 ± 7, $p < 0.0001$). LVEF (%), RV FPV and LV FPV (cm/s) were higher in gr 1 than in gr 2 (LVEF: 66 ± 5 vs 51 ± 14, $p < 0.0002$; RV: 51 ± 11 vs 38 ± 9, $p < 0.0001$; LV: 85 ± 27 vs 57 ± 21, $p < 0001$). Mitral E/A was similar in the two groups (gr 1: 1.3 ± 0.5 vs gr 2: 1.4 ± 0.7, ns) but tricuspid E/A was lower in gr 2 (gr 1: 1.7 ± 0.4 vs gr 2: 1.3 ± 0.5, $p = 0.03$). In gr 2, RV FPV (cm/s) was similar for the two sites of MI (ant: n = 27, 36 ± 8 vs inf/lat: n = 35, 40 ± 10, ns); however, LV FPV (cm/s) was lower in anterior MI (ant: 48 ± 11 vs inf/lat: 65 ± 24, $p < 0.001$).

*Conclusion:* Both RV FPV and LV FPV are decreased in pts with recent MI, suggesting an alteration of both RV and LV diastolic function. Only the degree of LV, but not RV, diastolic dysfunction seems to be related to the site of MI.

## 1126-118 Recovery of Left Atrial Mechanical Function After Chemical Cardioversion of Chronic Atrial Fibrillation

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*Background and Methods:* Recovery of left atrial mechanical function (LAMF) may be delayed for several weeks after successful cardioversion (CV) of chronic atrial fibrillation (AF) to sinus rhythm (SR). It has been suggested that LAM dysfunction relates to the mode of CV and is less severe in patients (pts) undergoing chemical (CCV) than electrical CV (DCC). To evaluate the time course of the recovery of LAMF after CCV, serial transmitral pulsed doppler echocardiographic studies were performed in 12 of 54 pts (22%) who underwent successful CCV from chronic AF to SR (oral loading with propafenone P). We only included pts with AF of 5 ± weeks duration, NYHA class ≤/= II, ejection fraction (EF) ≥50% and without ischemic heart disease. After successful CCV all pts underwent prophylaxis therapy with P 300 mg BID throughout. All pts were in SR throughout the follow up period. Pulsed doppler indices of LAMF were measured within 24 hours, at 2 weeks and 1 month after CV.

*Results:* Over 1 month of follow up, there was significant increase of peak A wave velocity. Similary also integrated late atrial velocities (A-VTI), atrial contribution to total transmitral flow (A VTI/Tot VTI), A wave duration and EF increased significantly.

| Parameters/Time | I | II | III | I/III |
|---|---|---|---|---|
| Peak E wave (cm/sec) | 78 ± 7 | 72 ± 7 | 69 ± 5 | p < 0.001 |
| Peak A wave (cm sec) | 41 ± 5 | 56 ± 8 | 63 ± 7 | p < 0.001 |
| E VTI (cm) | 9.7 ± 1.7 | 9.1 ± 1.5 | 9 ± 1.6 | NS |
| A VTI (cm) | 4.2 ± 1.3 | 5.6 ± 1.4 | 6 ± 1.5 | p < 0.001 |
| E/A | 1.98 ± 0.3 | 1.31 ± 0.27 | 1.11 ± 0.19 | p < 0.001 |
| A VTI/ Tot VTI (%) | 28.8 ± 3.3 | 34 ± 3.5 | 35.8 ± 5 | p < 0.001 |
| A wave d ration (msec) | 110 ± 13.8 | 135 ± 7.5 | 145 ± 7 | p < 0.001 |
| Heart rate (bpm) | 73 ± 10 | 70 ± 7 | 70 ± 7 | p < 025 |
| EF (%) | 62 ± 4 | 66 ± 3.7 | 67 ± 3.2 | p < 0.001 |

*Conclusions:* These data suggest that recovery of LAMF is slow and gradual even in pts subjected to CCV of chronic AF with P. These findings have important implications for assessing the early hemodynamic benefit of successful CV (in terms of EF) and for guiding the duration of anticoagulant therapy after CV.