

3226

Vortical Structures in Cardiac Filling Flows: A Universal Descriptor of Diastolic Efficiency. In Vitro and Clinical Studies

Morteza Gharib, Edmond Rambod, Takahiro Shiota, Nancy Dalton, Ray Hershberger, Arnaldo Passafiumi, David J. Sahn. Oregon Hlth Sci Univ, Portland, OR

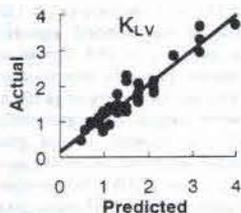
Maximal efficiency of cardiac filling requires that blood entering the LV decelerate and mix evenly before energy is spent. This requires a rapid rollup of the flowing blood into the vortical structures within the left ventricle. The strength and efficiency of vortex rollup can be measured in terms of the maximum amount of the circulation attained by the vortex during the formation process. Formation number, a descriptor of vortex formation, in its non-dimensional form ($f = ut/D$, where u = mean velocity over inflow period, t = inflow period, D = mitral annulus diameter) would be expected to stay within a narrow range. We performed both in vitro and clinical studies to quantify this parameter. In the *in vitro* study, we used a vortex ring generator comprised of a long, cylindrical tube and a piston to create ideal filling and mixing flow patterns in the flow field. Digital particle imaging was utilized to visualize and to map the vorticity field in the pulsatile flow model, showing that the most effective pattern for filling was generated within a formation number between 3 and 4. For the *clinical* study, echo Doppler studies were performed in 97 healthy subjects, ranging in age from 7 days to 84 years, and in 10 patients with severe dilated cardiomyopathy (DCM). Over this range of ages, the mitral inflow formation number was constantly between 3.7 and 6.2 for healthy subjects, whereas all of the DCM patients showed formation numbers between 0.27 and 2.88 (mean 1.89 ± 0.70 , $p > 0.0001$). The *in vitro* determined formation number, indicating the natural stability of the vortex flow, is close to findings in healthy subjects, and fulfills efficiencies of an idealized filling process which are lost when disease is present.

3227

Left Ventricular Chamber Stiffness can be Determined from the Time for Deceleration of Early Filling.

Michiya Ohno, Che-Ping Cheng, Dalane W. Kitzman, James D. Thomas, William C. Little. Bowman Gray School of Medicine, Wake Forest University, Winston-Salem, NC

A non-invasive measure of left ventricular (LV) chamber stiffness (K_{LV}) would be clinically useful. Our theoretical analysis predicts that the time for deceleration of LV early filling (t_{dec}) is given by $t_{dec} = \frac{\pi}{2} \sqrt{\frac{\rho \cdot L}{A} \frac{1}{\sqrt{K_{LV}}}}$, where ρ =viscosity of blood, L =effective mitral length, A =mitral area. This suggests that K_{LV} can be measured from t_{dec} . We evaluated 8 conscious dogs instrumented to measure LV pressure (P) with a micromanometer, and volume (V) from sonomicrometers. K_{LV} was determined as the slope of the late diastolic portion of the LV P-V loop. K_{LV} was varied from 0.99 ± 0.35 to 2.58 ± 0.92 mmHg/ml using 3 graded doses of phenylephrine. We assumed that $\rho=1.0$ and that $L/A=3.4$. Thus, we predicted that $K_{LV}=(0.08/t_{dec})^2$. The LV filling pattern was determined from dV/dt . t_{dec} was measured from peak early filling to the end of early filling. Predicted K_{LV} and actual K_{LV} were closely correlated ($r=0.94$, $SEE=0.06$ mmHg/ml, $p<0.05$). The regression line was close to the line of identity (slope=0.95, intercept=0.13 mmHg/ml). We conclude that LV chamber stiffness (K_{LV}) can be determined from the time for deceleration of LV early filling, which can be measured non-invasively.



3228

Evaluation of Left Ventricular Relaxation by Color-coded M-mode Doppler Echocardiography

Toshiro Aoki, Katsu Takenaka, Keichiro Atarashi, Fumiyoshi Watanabe, Jun-ichi Suzuki, Tsutomu Igarashi, Makoto Sonoda, Mikki Mashita, University of Tokyo, Tokyo, Japan

LV inflow velocity measured by pulsed Doppler ultrasound has been hardly successful in quantitating LV diastolic function. To assess whether color-coded M-mode Doppler echocardiography can be used for quantitating LV relaxation property, velocity of diastolic LV flow propagation and time constant T of LV pressure decay were measured simultaneously in 15 patients (age: 58 ± 10 yrs). Velocity of diastolic LV flow propagation was defined as the slope of the wave front of LV inflow color M-mode Doppler signal and time constant T was calculated by the best fitting method. In addition, velocity of diastolic LV flow propagation was measured in 21 patients with diabetes mellitus and 8 age-matched control subjects.

Time constant T of LV pressure decay correlated well with velocity of diastolic LV flow propagation ($r=-0.93$) ($p<0.001$) and could be estimated with the formula: T (ms) $= -0.10 \times$ velocity of flow propagation (mm/s) $\times 126$.

Velocity of diastolic LV flow propagation of controls correlated well with age ($r=-0.89$) ($p<0.005$) and was significantly higher (1050 ± 219 vs 614 ± 160 mm/s) ($p<0.0001$) than that of diabetic patients which showed no correlation with age ($r=-0.04$).

Thus, velocity of diastolic LV flow propagation measured by color-coded M-mode Doppler echo reflects time constant T of LV pressure decay, and is useful in identifying impaired LV relaxation in diabetic patients.

Clinical Cardiology: Electrocardiography Wednesday Afternoon

3229

Electrical Alternans of The Intracoronary Electrocardiography During Coronary Angioplasty

Tak Kwan, Alan Feit, Hal Chadow, Francisco Chio, Peter Elcock, and Michael S. Huber. SUNY, Health Science Center at Brooklyn, Brooklyn, NY

Electrical alternans (EA) is a serious cardiac event and predisposes to ventricular arrhythmias. To evaluate EA during PTCA, intracoronary (ic) electrocardiography (EKG) was performed in 65 consecutive lesions. Left anterior descending (LAD) lesions were found in 25/65 (9 proximal, 11 mid, 2 distal and 3 proximal diagonal branch). Left circumflex lesions were found in 14/65 (3 proximal, 2 mid, 6 proximal obtuse marginal (OM), and 3 mid OM). Right coronary lesions were found in 26/65 (10 proximal, 6 mid, and 10 distal). ST-T alternans was detected in 5 lesions in the ic EKG, all of them proximal LAD lesions. The EA was seen at least 130 seconds (mean 174 ± 57) into balloon inflation in these lesions. Only 2 cases showed ST-T alternans in both the surface EKG and ic EKG and only 1 had PVC's following the ST-T alternans. Three lesions required a second balloon inflation with duration of 300 seconds; there was no ST-T alternans despite ST segment elevation. Most of the patients with EA (4/5) were taking a calcium blocker. There was no difference in the duration of balloon inflation between the lesions with EA and without EA (241 ± 66 s. vs. 212 ± 71 s.). **We conclude:** 1) The detection of EA during PTCA is enhanced by using ic EKG. 2) EA during PTCA is only seen in proximal LAD lesions; this implies a large amount of myocardium must be involved for the EA to occur. 3) EA is not seen during the second balloon inflation indicating ischemic preconditioning may be present. 4) EA is not an immediate phenomenon but rather requires a long duration of balloon occlusion. 5) Ventricular arrhythmias with EA is only demonstrated in 1/5 lesions with alternans. 6) Calcium channel blockers do not abolish the EA.

3230

T Wave Normalization in the Long Term Evolution of Anterior MI: its Relation to Residual Perfusion and Recovery of Systolic Function.

Giuseppina Belotti, Giuseppe Pizzetti, Matteo Montorfano, Alberto Margonato, Sergio L. Chierchia. Istituto Scientifico H S Raffaele, Milano, Italy.

The significance of late normalization of negative T wave after myocardial infarction (MI) is still unknown. We studied 51 consecutive patients (pts, 47 males, mean age 53 years) presenting with anterior MI and angiographically documented disease of the left anterior descending (LAD) coronary artery. All underwent 2D-echocardiography and elective coronary angiography within 2 weeks from MI. The 12-lead EKG along with a 2D-echocardiogram, were recorded at discharge and repeated after an average of 18 months. Regional wall motion was assessed in 18 segments and an asinergy score (hypokinesia=1, akinesia=2, dyskinesia=3) was obtained. At discharge 42 pts had negative T waves; among these, 28 (Group A) exhibited late normalization, while 14 (Group B) did not. Nine pts had positive or flat T waves both at discharge and on follow up (Group C). Thrombolytic therapy (TT), perfusion (P) to the infarct area by a patent LAD (TIMI grade 3) or by collaterals (Cohen & Rentrop grade 2-3) are summarized in the table along with the changes in the asinergy SCORE observed over the 18 months follow-up:

	Group A	Group B	Group C
TT	18/28	9/14	5/9
P	28/28	10/14	5/9
SCORE	-5.2 ± 4.8	$+0.3 \pm 6.4$	-1 ± 10.7

Therefore, in pts with transmural anterior MI, late normalization of initially negative T waves, is significantly related both to residual perfusion to the infarct area and to the late improvement of regional function.