and reperfusion, there were no differences in hemodynamic parameters or RMBF among three groups.

	15 min occl			120 min R		120 min R
Groups	ATP	CP	pH	ATP	CP	%SS
C (n = 10)	64 ± 7%	7 ± 6%	6.3 ± 0.2	71 ± 7%	105 ± 12%	39 ± 16%
ADO (n = 10)	76 ± 6%*	9 ± 6%#	6.5 ± 0.1*	90 ± 8%*	101 ± 6%#	29 ± 6%
IP (n = 10)	74 ± 9%*	18 ± 6%*	6.6 ± 0.1*	91 ± 9%*	126 ± 7%*	32 ± 16%

ATP, CP and %SS values; % of baseline, *p < 0.05 vs C, #p < 0.05 vs IP

[Conclusion] ADO pretreatment and IP had similar effects on ATP and pH, but not on CP. In spite of the protective effects on myocardial metabolism during ischemia and reperfusion, neither of the interventions improved %SS after ischemia.

M O N D A Y P M

932-91 Ventricular Interdependence in Explanted Human Hearts

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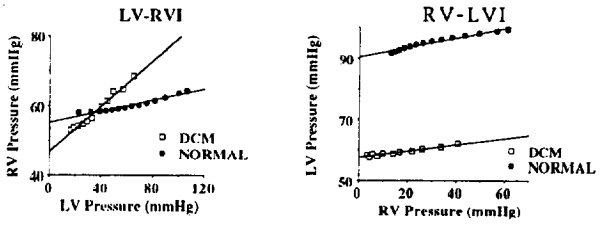
Left ventricular (LV) contraction contributes importantly to right ventricular (RV) performance via left to right ventricular interaction (LV-RVI). To determine the magnitude of LV-RVI in human heart failure, 5 explanted hearts with dilated cardiomyopathy (DCM) were obtained at the time of transplant and restored to a beating condition using a blood perfusion system. One normal human heart, unsuitable for transplantation, was also obtained and served as control. Balloons were placed in both RV and LV. With RV volume fixed at an end diastolic pressure (EDP) of 20 mmHg, LV volume was ramped from low (LV EDP = 0) to high (LV EDP = 30) and resulting changes in RV systolic pressures were plotted vs. changes in LV pressure. The slope of this line defines the LV-RVI pressure gain. An analogous procedure was used to determine RV-LVI. The figures depict representative data.



In the DCM hearts mean (±SD) LV-RVI gain was 0.26 ± 0.06 and RV-LVI was 0.16 ± 0.12. In the control heart LV-RVI = 0.08 and RV-LVI = 0.15. LV-RVI is markedly increased in heart failure when compared to the control. This is consistent with previous reports using animal models. Conversely, RV-LVI remains unaffected by heart failure. These data, not previously available from humans, suggest that LV contribution to RV performance is significantly enhanced in chronic heart failure.

933 Left Ventricular Hypertrophy and Left Ventricular Mass

Monday, March 20, 1995, 3:00 p.m.–5:00 p.m.
Ernest N. Morial Convention Center, Hall E
Presentation Hour: 3:00 p.m.–4:00 p.m.

933-92 Carotid Thickening Precedes Ventricular Remodeling in Early Essential Hypertension and is Significantly Related to Early Wave Reflection

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Ventricular and vascular remodeling occur early in essential hypertension, develop in parallel and may be influenced by early arterial wave reflection. To further study these relationship we examined 20 newly diagnosed, untreated mild hypertensives, 18 previously treated hypertensives and to 35 age and sex-matched normotensive normal volunteers (N), with both high frequency B-mode imaging of the carotid artery to determine carotid intima-medial thickness (IMT), diameter (CD) and relative thickness (CRWT = 2* IMT/CD) and 2-D guided M-mode echocardiography for determination of LV posterior wall thickness (PWT), end-diastolic diameter (LVEDD), LV relative wall thickness (LVRWT = 2* PWT/LVEDD) and LV mass index (LVMI). Using high-fidelity arterial tonometry the right carotid artery waveform was recorded and classified by measurement of augmentation index as having either a normal early systolic peak (Type C) or abnormal late systolic peak (Type A-indicating early wave reflection). Compared to N, the treated hypertensives had significantly higher LVMI (98 ± 22 vs 82 ± 22 g/m^2, p < 0.05) and carotid IMT (0.74 ± 0.17 vs 0.61 ± 0.15, p < 0.05). There were no significant differences in LV wall thickness, relative wall thickness or LV mass index between subjects with Type A and Type C arterial waveforms. In contrast, carotid IMT was significantly increased in patients with Type A arterial waveforms.

	n	LVRWT	LVMI	CIMT (mm)	CRWT	MAP (mmHg)
Type A	(47)	0.38 ± 0.09	85 ± 25	0.69 ± 0.17*	0.22 ± 0.04*	105 ± 14*
Type C	(22)	0.38 ± 0.10	87 ± 18	0.59 ± 0.09	0.19 ± 0.03	96 ± 14

*p < 0.01 vs. Type C

In multivariate analysis, only age (p < 0.05), systolic blood pressure (p < 0.001) and augmentation index (p < 0.05) were independently associated with CIMT. We conclude that the Type A arterial waveform indicating abnormal early wave reflection coincides with increased mean arterial pressures and with intima-medial thickening in large conduit arteries. These changes precede left ventricular remodeling and left ventricular hypertrophy in mild, essential hypertension.

933-93 The Regression of Left Ventricular Myofibrillary Proteins by ACE-inhibition (Lisinopril) is Associated with an Increase in Protein Synthetic Rates in Vivo

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One of the most serious complications of systemic hypertension is myocardial tissue damage, including left ventricular hypertrophy, as a consequence of increased protein synthesis. However, the modulating role of translational events in the *hypertrophy-regression transition* is poorly understood, especially where therapeutic regimes have been employed. These events were investigated *in vivo* in a genetic model of hypertension, namely in the spontaneously hypertensive rat (SHR); comparative responses were investigated in normotensive Wistar Kyoto rats (WKY). Rats were used at 4 months of age and treated with either the ACE-inhibitor lisinopril (5 mg/kg/day) in tap water or plain tap water (controls). The groups were assigned as follows: WKY-CON, normotensive controls; WKY-LIS, normotensives plus lisinopril; SHR-CON, hypertensive controls; SHR-LIS, hypertensives plus lisinopril. Fractional rates of protein synthesis (k_s defined as the percentage of the myofibrillary protein pool renewed each day; %/day) were measured *in vivo* with the flooding dose technique using L-[4-^{3}H]phenylalanine. Left ventricular myofibrillary proteins were extracted by differential solubility and high-speed centrifugation techniques; purity was assessed with SDS-PAGE. After 8 weeks treatment the myofibrillary protein contents (mg per region) in normotensive rats were as follows (all data as mean ± SEMS, n = 6–9): WKY-CON, 45 ± 1 mg; WKY-LIS, 36 ± 1 (p < 0.001). In the hypertensive group regression of contractile protein content occurred; i.e., SHR-CON, 52 ± 3 mg; SHR-LIS, 38 ± 1 mg (p < 0.001). Corresponding k_s values were: WKY-CON, 8.3 ± 0.2 %/d; WKY-LIS, 8.2 ± 0.3 %/d (NS). In the SHR-group k_s values were: SHR-CON, 8.2 ± 0.3 %/d; SHR-LIS, 9.2 ± 0.3 mg (p < 0.025). *Conclusion; ACE-induced regression of contractile protein composition in hypertension is associated with an increase in rates of translation.*

933-94 Left Ventricular Hypertrophy in Hypertensives is Associated with an Increased QT Dispersion

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Hypertensive patients who develop left ventricular hypertrophy (LVH) are at much greater cardiovascular risk than those without and in particular their incidence of sudden death is several times higher. This may be mediated by an increase in ventricular arrhythmias. It has recently been suggested that QT dispersion may indicate arrhythmia risk by reflecting dispersion of recovery of ventricular excitability.

In order to test the hypothesis that left ventricular hypertrophy may be associated with an increase in QT dispersion 100 previously untreated subjects were studied. These consisted of 52 subjects with essential hypertension (BP > 160/90), 21 subjects with borderline hypertension (BP > 140/85) and 27 normotensives. Each underwent 2 dimensional and Doppler echocardiography to determine left ventricular mass index (LVMI), E/A ratio and isovolumic relaxation time (IVRT). Additionally from a 12-lead ECG examination QT length was measured for each lead and corrected for heart rate (QTc). QTc dispersion was determined as the difference between the maximum and minimum QTc interval.