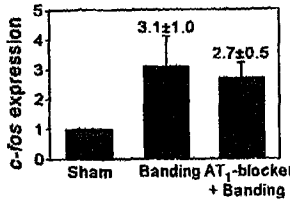
1 (AT_1) receptor. The purpose of this study was to test whether pressure overload induced IEG expression is also mediated by AT_1 receptor stimulation in *adult* (3 month old) *rat hearts in vivo.* In one group of rats (n = 7), pressure overload was created by ascending aortic constriction to increase left ventricular pressure by ≈ 70 mmHg for 90 mins. In a second group (n = 5), the same degree of pressure overload was induced 1 hour after administration of the AT_1 receptor antagonist GR138950X (4 mg/kg), at twice the dose which abolished hemodynamic responses to a 0.4 μg bolus of AII. Finally, a third group was comprised of sham operated animals (n = 5). RNA was isolated from the left ventricles and c-*fos* expression was analyzed by Northern blot; band intensities were normalized to those of GAPDH or β-actin.



c-*fos* expression increased after 90 minutes of pressure overload (3.1 times greater than sham). In AT_1-antagonist pretreated animals, c-*fos* expression increased to a similar degree as in the non-treated, aortic constricted animals (2.7 times greater than sham). There was no statistical difference between c-*fos* expression in these two groups.

Thus, in contrast to *neonatal myocytes in vitro,* AII stimulation of AT_1 receptor does not appear to be involved in pressure overload induced c-*fos* expression in *adult rat hearts in vivo.* Accordingly, the mediator of IEG expression during pressure overload *in vivo* remains to be elucidated.

MYOCARDIAL AND PERICARDIAL FUNCTION AND DISEASE – BASIC

901-95 Time-Dependent Interactions Between Epicardial and Endocardial Fibers Determine Left Ventricular Torsion

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Torsion begins in a clockwise direction in early systole, reverses during ejection, then recoils rapidly in early diastole. We hypothesized that this pattern results from time-dependent interactions between counterclockwise epicardial (epi) and clockwise endocardial (endo) fibers. We used cool pericardial lavage to selectively delay the onset and reduce the rate of epi fiber contraction and relaxation, and measured the effect on the monophasic action potential (MAP), endo early systolic clockwise torsion (ESCT), and recoil rate. Nine open chest dogs underwent continuous pericardial lavage at baseline (BL) and during cooling (CL) of lavage fluid, (36.7 ± 0.9° C vs. 30.5 ± 1.0° C, $p < 0.0001$), while blood and endo temperatures were held constant (35.4 ± 1.2° C vs. 35.6 ± 1.3° C). Tagged MRI was performed at BL and CL. From basal and apical short-axis images, torsion was measured as the apex to base difference in rotation about the cavity centroid. Recoil rate was expressed as the slope of linear regression of torsion versus time during first 65 ms after peak systolic torsion. Epi activation time (AT), and 90% repolarization time (90% RT) were measured using a MAP catheter.

	AT (ms)	90% RT (ms)	$ESCT_{epi}$ (°)	$ESCT_{endo}$ (°)	$Recoil_{epi}$ (°/ms)	$Recoil_{endo}$ (°/ms)
BL	33 ± 5	214 ± 16	0.5 ± 0.5	0.9 ± 1.3	0.17	0.07
CL	39 ± 4*	231 ± 13*	1.7 ± 1.3*	2.8 ± 2.3*	0.07*	0.01*

*$p < 0.05$, as compared to BL.

Conclusions: Selective delay in epi contraction and relaxation results in: (1) an increase in ESCT, indicating that this motion is due to unopposed contraction of clockwise endo fibers, and (2) a marked decrease in endo recoil rate, indicating that this transmural deformation is dominated by epi relaxation.

901-96 Simultaneous Determination of Regional Left Ventricular Wall Stresses in Intact Canine Hearts

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LV wall stress plays an important role in determining myocardial O_2 consumption, modulating LV hypertrophy, and regulating LV mechanics. To determine the instantaneous, simultaneous average LV isotropic wall stress at different locations, myocardial markers were implanted into the LV walls of 14 dogs to calculate 3-D volume and wall thicknesses (using computer-assisted analysis of biplane videofluorographic images). A total of 26 markers were placed; 18 of these markers were positioned on the subepi- and subendocardial surfaces to measure wall thickness at nine locations: 3 each on the anterior (*ANT*), lateral (*LAT*), and posterior (*POS*) walls. One to two weeks following marker placement the dogs were studied both before and after inotropic stimulation with Ca^{++} (10 mg/kg IV bolus). End systolic wall stresses (σ, kdynes/cm^2) were then calculated from the instantaneous LV pressure, regional wall thicknesses, and local geometry.

Wall		Apical	Equatorial	Basal
ANT	σ	174 ± 51	255 ± 175	274 ± 44*
	σ,Ca^{++}	194 ± 52	269 ± 161	315 ± 81*
LAT	σ	116 ± 44	201 ± 74*	266 ± 115*
	σ,Ca^{++}	133 ± 64	224 ± 92*	304 ± 153*
POS	σ	147 ± 62	191 ± 98	206 ± 113*
	σ,Ca^{++}	167 ± 78	212 ± 126	229 ± 125*

mean ± 1 SD; *$p < 0.05$ vs. Apical level, ANOVA

Multivariate ANOVA demonstrated significant regional heterogeneity (level of marker insertion, $p < 0.001$; regional LV wall, $p = 0.002$) in end-systolic σ inotropic state had less of an influence ($p = 0.056$). Univariate ANOVA revealed a gradient of wall stress increasing from apex to base and decreasing from the anterior to posterior wall as well as significant increases in σ with Ca^{++} (with the exception of the equatorial sites). Thus, end systolic wall stress is heterogeneous, and may be influenced by myocardial fiber orientation as well as the insertion of the intact papillary muscles and chordae tendineae.

MYOCARDIAL AND PERICARDIAL FUNCTION AND DISEASE – CLINICAL

901-97 Midwall Left Ventricular Performance in Normotensive Normal-Weight Children and Adults

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Purpose: To study the relation of LV midwall shortening (mS) to LV geometry in a large normal population across a wide spectrum of age.

Method: mS and circumferential end-systolic stress (cESS) were assessed in 159 normal adults (AD, 79 women and 80 men, 18 to 71 years) and 438 infants to adolescents (CH, 198 females, 240 males, 1 day to 17 years) by echocardiography and cuff blood pressure (BP).

Results: mS was negatively related to cESS in a number of regression models (linear, inverse, allometric, various exponential models; all $p < 0.001$). The *inverse* model maximized the R-square and minimized the error (SEE), in AD (mS = 15 ± 400/cESS, $r = -0.33$, $p < 0.0001$) and in CH (mS = 18 ± 184/cESS, $r = -0.13$, $p < 0.008$), as well as in the whole population (mS = 17 + 261/cESS, $r = -0.18$, SEE = 2.6%, $p < 0.0001$). mS as a % of predicted from cESS by the group-specific equations (%PmS) was compared in 4 age-groups (I = birth to 10 years; II = 10 to 17 years, puberty and adolescence; III = 17 to 50 years; IV = over 50 years).

Age	%PmS (%)	Relat. Wall Thickn.	LVIDd (cm/m)
< 10 years	106 ± 14*	0.26 ± 0.05*	3.22 ± 0.24*
10–17 years	97 ± 17	0.31 ± 0.06	2.83 ± 0.24
17–50 years	99 ± 12	0.32 ± 0.06	2.80 ± 22
> 50 years	100 ± 10	0.34 ± 0.05	2.87 ± 0.28

*$p < 0.001$ by Scheffe's test vs the other groups

Conclusions: Thus, mS is related to cESS by inverse regression equations in a wide range of age. Midwall LV performance and LV size/height are enhanced during infancy and childhood and stabilize after age 10 even as LV geometry becomes more concentric.

901-98 Clinical Profile of Constrictive Pericarditis in the Modern Era: A Survey of 135 Cases

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Although the clinical features of constrictive pericarditis (CP) are well known, there is limited knowledge of its profile in the current era. Between January 1985 to June 1995, 135 pts (76% males, mean age 56 ± 16 yrs) had a diagnosis of CP established at the Mayo Clinic (133 by surgery, 2 by