INADEQUACY OF CURRENT CRITERIA FOR PREDICTING HIGH RISK CORONARY ANGIOPLASTY

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The ability to predict patients (pts) at risk for complications (C) associated with PTCA is of increased importance with the availability of prophylactic bypass assist devices. Accordingly, we prospectively classified 170 consecutive pts based on accepted criteria for risk including angiographic (ostial LAD, angled, sequential, bifurcation, thrombus, jeopardized collaterals) and clinical parameters (elderly, concomitent illness, reduced ejection fraction) into pts at high risk (HR) and low risk (LR) for C. There were no deaths in either group and the overall PTCA success rate vas 86% for HR and 93% for LR pts.

Complications:	HR(n=51)	LR(n=119)	p value
abrupt closure	19.6%	7.5%	.042
hemodynamic compromise	13.7%	3.4%	.031
CABG	5.9%	0.8%	NS
myocardial infarction	15.7%	6.7%	NS
all complications	41.1%	24.2%	.041

Though significant differences were found in the rate of C between the HR and LR pts, the ability to predict C or hemodynamic compromise had a sensitivity of only 42% and 64%, a specificity of 75% and 72%, and a mean predictive accuracy of 65% and 71%, respectively. These data suggest that present criteria for

identifying pts at HR for C are insufficient and that new criteria need to be investigated.

Wednesday, March 21, 1990

4:00PM-5:00PM, Room 41

Myocardial Ischemia and Infarction: Effects on Ventricular Function

Regional End-Systolic Wall Stress - A Detector of Wild Ischemia

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Regional end-systolic wall stress (ESNS), whose components are intracavitary pressure, endocardial radii and wall thickness, increases during acute ischemia. However, the severity of ischemia that produces this effect is unknown. To determine the relation between mild ischemia and ESNS, 2D-echo was used to measure ESNS derived from Janz's formula in 9 open-chest pigs during graded occlusion of the left anterior descending artery (LAD). Ischemic zones were determined by microspheres at each stage and Trypan Blue. The ESNS was measured in normal and ischemic zones at 0, 20, 30 and 40% LAD flow reduction.



During ischemia there was no change in ESWS in normal zones but ischemic zone ESWS progressively increased compared to normal and to baseline.

Conclusion: Regional ESWS increases significantly in ischemic Dyocardium during mild (20% flow reduction) ischemia and increases progressively during more severe ischemia. Measuring ESWS is a promising new approach in the detection of mild regional LV ischemia. EARLY INFARCT EXPANSION AND COLLAGEN DAMAGE: STRUCTURAL CHANGES AND TEMPORAL RELATIONSHIPS <u>Peter Whittaker. Ph.D.</u>, Derek R. Boughner, M.D., Ph.D., Robert A. Kloner, M.D., Ph.D. F.A.C.C. Heart Institute, Hospital of the Good Samaritan and University of Southern California, Los Angeles. CA.

The collagen framework of the heart provides its strength and stiffness. Collagen is damaged after myocardial infarction (MI), but infarct expansion does not occur for several days. To examine the temporal relation between collagen damage and expansion we occluded the left coronary artery of 21 rats and studied histologic sections from the hearts at 1-4 days post-MI; 5 non-infarcted rats served as controls. We measured 4 parameters: (1) infarct expansion, assessed by measurement of left ventricular cavity (LVC) cross-sectional area, (2) volume fraction of interstitial space (IS), measured using point counting, (3) collagen birefringence, a marker of structural integrity examined using polarized light and a silver stain (we noted the % of normal collagen (NC) fibers), and (4) collagen orientation in the infarct, measured using polarized light and picrosirius red staining (we calculated the angular deviation (AD) of the measured collagen fiber orientations, which indicates the degree of fiber alignment). Data from controls and from 3 & 4 days post-MI are shown below.

	CONTROL	[3]	[4]	
LVC (mm ²)	54 <u>+</u> 4	57 ± 6	67 ± 10*	
IS (%)	11 ± 1	10 ± 1	6 ± 1*	[] = days post-MI
NC (%)	70 ± 6	$48 \pm 8^{\circ}$	45 ± 8°	p<.05 vs. control
AD (°)	14 ± 1	13 ± 1	10 ± 1°	

We saw a reduction in the number of normal collagen fibers after 1 and 2 days, but the reduction was not significant until day 3. Expansion, however, was not observed until day 4. This was concurrent with a decrease in interstitial space and realignment of collagen fibers in the infarct, produced in part by the loss of obliquely aligned collagen struts. The results suggest that a decrease in % normal collagen alone is not sufficient to produce expansion. The loss of both interstitial space and collagen struts appear to provide the necessary additional damage for infarct expansion to occur.

EVALUATION OF VOLUME-CATACHOLAMINE COMBINATION THERAPY FOR RIGHT VENTRICULAR MYOCARDIAL INFARCTION.

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To evaluate the beneficial effect of volume loading combined with catecholamine administration for right ventricular myocardial infarction (RVI), we measured pulmonary blood volume (PBV) and cardiac output (CO) by means of intravascular dual dye-densitometry using optic fiber catheter system, which we developed, in 8 mengrel dogs with experimental RVI. RVI was produced with right coronary occlusion by injection of sliced own muscle cubes via catheter. Six hours later, 150ml of saline was infused at a rate of 15ml/min followed by 3ug/kg/min of dobutamine. PBV and CO were measured before producing RVI (CNT), before (B) and after (S) saline infusion, and after DOB administration (D). Changes in PBV and CO were;

	CNT	В	S	D
PBV	100%	64 <u>+</u> 11%	69 <u>+</u> 13%	82 <u>+</u> 16%*
CO	100%	57 <u>+</u> 13%	65 <u>+</u> 10%	72 <u>+</u> 14%*

* = p<0.05 vs S by ANOVA

Thus, volume loading alone failed to increase PEV and CO. However, the volume loading combined with catecholamine administration benefited deteriorated right cardiac function and increased PBV even with RVI. Accordingly, an enhancement of left ventricular contraction by catecholamine is suggested to affect the right ventricle directly and mechanically.