9:15

THE INFLUENCE OF CORONARY COLLATERALS ON THE LONG-TERM OUTCOME OF PATIENTS AFTER UNSUCCESSFUL PTCA A REPORT FROM THE 1985-86 NHLBI PTCA REGISTRY

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The long-term outcome of patients (pts) after unsuccessful (U) but uncomplicated (no death, MI or emergency CABG) PTCA has not been previously reported, and may be influenced by the presence or absence of coronary collaterals. Accordingly, we assessed 3 year outcome after uncomplicated single lesion PTCA in 105 U and 852 successful (S) pts. Compared to S pts, U pts were more likely to have multi-vessel disease (50% vs 34%, p<.01), prior MI (44% vs 30%, p<.05), LV ejection fraction <50% (24% vs 15%, p<.05) and attempted PTCA of a total occlusion (45% vs 10%, p<.001). U pts were also more likely to undergo early elective CABG (19% vs 1%, p<.001) and have angina at hospital discharge (30% vs 2%, p<.001). At 3 year follow-up, death (5% vs 4%) and MI (6% vs 6%) rates were similar (p=NS, for U and S pts. U pts required more CABG (40% vs 10%, p<.001) whereas repeat PTCA was more common in S pts (22% vs 5% p<.001). At 3 years, 75% of U pts and 81% of S pts were angina free (p=NS). In U pts in whom coronary collateral flow to the target lesion was identified on the pre-PTCA angiograms (n=57), MI did not occur (0% vs 13% in U pts without collaterals (n=48), p<.01).

Conclusions:
1) Coronary collaterals provide protection against future MI after U PTCA.
2) U pts frequently require CABG, but despite more severe coronary disease and LV dysfunction acheive a similiar long-term outcome to pts after S PTCA.

9:30

OUTCOME WITH MULTILESION AND MULTIVESSEL CORONARY ANGIOPLASTY OF 3 OR MORE SITES

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Data describing multilesion and multivessel percutaneous transluminal coronary angioplasty (PTCA) is limited, and most series include patients who had only 2 sites dilated. We describe a large series of patients who had PTCA of ≥3 lesions. 272 patients had either 1 vessel multilesion (Group A, n=62) or multivessel/multilesion (Group B, n=210) PTCA. An overall mean of 3.5 lesions/patient (A=3.1, B=3.7 P<.01) were dilated with a success rate of 94.8% (A=96.3, B=94.5, P=NS). Between groups P=NS for age (64±10 overall), sex (73% male overall), prior MI (31% A, 34% B), prior coronary artery bypass grafting (CABG) (16% A, 24% B), ejection fraction (mean 54% vs 52%).

Over mean follow-up of 58 weeks the need for re-cath was the same (43% both groups) with documented restenosis (% of patients) similar (27% A, 32% B). Need for CABG was similar (15% vs 19% P=NS) as was death (9.7% vs 8.1% P=NS).

We conclude: "true" multilesion and multivessel PTCA is possible with success and restenosis rates similar to 1 and 2 lesion results. Results for several lesions in 1 vs ≥ 2 vessels is remarkably similar. However, need for re-cath and CABG is high, as is mortality over relatively short follow up periods. These results may compare unfavorably with those of CABG.

9:45

LONG-TERM OUTCOME OF PATIENTS WITH DEPRESSED LEFT VENTRICULAR FUNCTION UNDERGOING PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY-THE NHLBI PTCA REGISTRY

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An increasing number of pts with depressed LV function (EF<40%) are undergoing coronary angioplasty (PTCA); to evaluate their initial and longer-term outcome, the 1985-1986 NHLBI PTCA Registry experience was assessed. There were 126 pts with EF<40% (average 35%) (Group 1) and 1329 pts with EF>40% (average 61%) (Group 2) undergoing elective PTCA. Pts undergoing PTCA for acute myocardial infarction (MI) were excluded. Gr 1 pts had a high prevalence of prior MI (83 vs 33%), prior bypass surgery (33 vs 10%), and 3-vessel disease (37 vs 20%) (p<0.001). At least 1 lesion was successfully dilated (lesion improvement ≥20%) in 89% Gr 1 and 91% Gr 2. Complication rates were similar for Gr 1 vs Gr 2 death (0.8 vs 0.8%), nonfatal MI (4.8 vs 4.6%), and emergency surgery (4.8 vs 3.3%). Freedom from events and angina rate at 3 year follow-up were:

	No Event			No Event and No or Improved Angina		
Event (%)	Gr 1	Gr 2		Gr 1	Gr 2	
Death	84	95	<.001	77	89	<.001
Death/MI	72	87	<.001	67	81	<.001
Death/MI/surgery	63	75	<.01	59	70	<.01

1) Pts with depressed LV function have similar success and complication rates compared to pts with presence of normal LV function undergoing PTCA. 2) Pts with depressed LV function have increased cardiac events during follow-up although the majority are improved.

Tuesday, March 5, 1991
8:30AM–10:00AM, Room 254, West Concourse
Cardiac Function: Molecular and Basic Mechanisms

8:30

AN ABNORMALITY IN THE CATALYTIC SUBUNIT OF ADENYLATE CYCLASE CHARACTERIZES PRESSURE-OVERLOADED, FAILING HUMAN VENTRICULAR MYOCARDIUM. Michael R. Bristow, Lisa Skerl, Patti Larrabee, Wayne Minobe, Randy Rasmussen, Robert Ginsburg. University of Utah, Salt Lake City, UT

Most information on the adrenergic neuroeffector mechanism (ANM) in the failing human heart (FHH) has been derived from studies with idiopathic dilated cardiomyopathy (IDC). Recent data in ischemic and valvular cardiomyopathy indicate that model-dependent differences in ANM abnormalities may exist in the FHH. In the current study right ventricular (RV) myocardium failing due to marked pressure overload (PA pressure=57±4 mmHg, RA=9±1.4 mmHg) derived from 13 subjects of average age 34.9±2.2 yrs undergoing heart-lung transplantation for primary pulmonary hypertension (PPH) was compared to RVs with a similar degree of failure (RA=9.7±1.1 mmHg) taken from 50 subjects with IDC, age 38.9±2.3 yrs. Adrenergic receptors, the adrenergic neurotransmitters (NTS) norepinephrine (NE) and neuropeptide Y (NPY), adenylate cyclase (AC) stimulation and muscle contraction in isolated trabeculae (tissue M. Cntxn) were measured. Results ± SEM were: (*=p<.05)

Group	Receptors fmol/mg		Tissue NTS,ng/g		AC stimulation pmol/cAMP/mg/min		Tissue M. Cntxn	
	β_1	β_2	NE	NPY	Gpp(NH)p	Mn^{2+}	ISO	Ca^{2+}
PPH	*23.9 ±2.5	19.1 ±3.4	*190 ±64	*30 ±11	8.2 ±0.6	*23.0 ±3.6	*831 ±107	1061 ±233
IDC	34.0 ±2.5	17.8 ±1.4	756 ±108	66 ±10	9.1 ±1.3	40.0 ±2.9	1366 ±109	1222 ±100

Conclusion: PPH RV differs from IDC RV by exhibiting 1) greater adrenergic neurotransmitter depletion; 2) greater β_1 receptor down-regulation; and 3) an abnormality of the catalytic subunit of adenylate cyclase, which has not been previously described in FFH.