the data using a multivariate logistic regression model to control for differences in patients' clinical characteristics for the occurrence of the composite endpoint, consisting of death, myocardial infarction, abrupt vessel closure, and coronary artery bypass surgery. The mean age was 60 (SD ± 11) years, with 72% men, and 46.2% received reused catheters. Composite endpoint occurred in 57 patients, with ACC/AHA lesion type, angina class, procedure time, and residual stenosis as predictors for outcome.

	Odds Ratio	95% Confidence Interval
Catheter Reuse	0.59	0.31–1.14
Age	1.00	0.97–1.03
Male	0.57	0.29–1.14
Angina class	1.95	1.23–3.11
ACC/AHA lesion type	2.30	1.03–5.11
Residual stenosis	1.03	1.01–1.04
Procedure Time	1.02	1.02–1.03

We conclude that, after controlling for baseline clinical and angiographic characteristics, reuse of PTCA catheters did not appear to be associated with an increase in in-hospital complications.

1010-112 Variability in Balloon Sizing During Routine PTCA — Quantitative Coronary and Ultrasound Analysis

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A balloon:artery (b:a) ratio of 1.0–1.1 is considered optimal for the performance of PTCA. To assess variability of balloon sizing, coronary angiography and ultrasound images from 48 lesions in 47 pts undergoing PTCA by 3 experienced operators were analyzed by an independent core lab. QCA measured b:a ratios at peak pressure inflation were determined.

Results: The maximal b:a ratio was 1.0–1.1 in only 14 lsns (29%); the b:a ratio was < 1.0 in 18 lsns (38%) and > 1.1 in 16 lsns (33%), ranging from 0.76 to 1.44. Implications of the variability in balloon sizing appear below:

Balloon:artery ratio	< 1.0	1.0–1.1	> 1.1	p
Peak pressure (atm.)	7.2 ± 1.4	7.3 ± 1.9	6.4 ± 1.2	NS
QCA ref. seg. dia. (mm)	2.9 ± 0.3	2.5 ± 0.4	2.2 ± 0.3	0.001
QCA dia. %stenosis pre	67 ± 14%	63 ± 13%	65 ± 10%	NS
QCA dia. %stenosis post	31 ± 13%	35 ± 11%	19 ± 23%	0.03
Δdia. %stenosis pre-post	−35 ± 17%	−28 ± 15%	−45 ± 24%	0.07
lisn MLD(mm) pre-post	1.0 ± 0.5	0.9 ± 0.4	1.2 ± 0.7	NS
Dissection score ≥ type B	1 (7%)	3 (17%)	4 (25%)	NS
ICUS area %stenosis post	48 ± 19%	46 ± 17%	43 ± 14%	NS
ICUS plaque burden post	77 ± 7%	80 ± 10%	76 ± 9%	NS

Conclusions: Contrary to expectations, the balloons that experienced operators choose result in markedly variable b:a ratios during PTCA, with relatively larger balloons used in smaller vessels. When safely applied, a b:a ratio of > 1.1 results in a lower angiographic residual stenosis. By ICUS, however, a large residual plaque burden is present after PTCA independent of b:a ratio.

1010-113 Rapid Assessment of Platelet Inhibition Using a Modified Whole Blood Aggregometer (Aggrestat™) in PTCA Patients Receiving ReoPro™

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ReoPro™ potently blocks GPIIb/IIIa, inhibits platelet aggregation and reduces ischemic complications in high risk PTCA patients. Currently, no bedside tests are available for rapid and accurate measurement of platelet inhibition during and following ReoPro™ treatment. To address this need, a bedside device (Aggrestat™) that measures platelet aggregation in whole blood using electrical impedance was developed and compared in 12 patients to standard measurement using light transmittance through platelet rich plasma (PRP). All patients received ReoPro™; 0.25 mg/kg bolus plus a 10 μg/min infusion for 12 hr. Inhibition of platelet aggregation in whole blood assessed by the Aggrestat™ yielded similar results to aggregation in PRP using light transmittance and showed the same close correlation with GPIIb/IIIa blockade (results are mean ± SD):

	%GPIIb/IIIa Blockade	% Inhibition of Aggregation	
		Aggrestat™	PRP Aggreg.
15 min post-ReoPro™ bolus	96 ± 1.6	96 ± 4.2	97 ± 2.4
2 h post-ReoPro™ bolus	92 ± 2.7	88 ± 7.5	97 ± 2.2
12 h post-ReoPro™ infusion	66 ± 7.5	69 ± 13	63 ± 20

These results indicate that the Aggrestat™ is a practical method for rapid measurement (within 10 minutes of blood draw) of platelet function at the bedside during and following anti-platelet therapy.

1010-114 Eight Year, Long Term Outcome After PTCA: Factors Associated With Adverse Events. The NHLBI PTCA Registry

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Risk factors associated with poor outcome during long term follow up after percutaneous transluminal coronary angioplasty (PTCA) remain poorly defined. In the NHLBI PTCA Registry, there is now 8 year follow up of 1759 pts who had initially successful PTCA. A multivariate model was used and 24 clinical and angiographic factors including baseline demographics, presenting symptoms, left ventricular function and extent of coronary artery disease were evaluated to determine those factors associated with a hierarchical 8 year adverse endpoint of death (D), death and myocardial infarction (MI), and death, myocardial infarction and coronary surgery (CABG). Only factors with $p < 0.001$ were considered.

Factor	D		D/MI		D/MI/CABG	
	RR**	CI‡ RR CI	RR CI	RR CI	RR CI	
Hx †CHF	2.65	(1.78, 3.94)	2.22	(1.57, 3.13)	2.17	(1.53, 3.08)
Severe non cardiac disease	2.56	(1.70, 3.85)	1.91	(1.34, 2.74)	–	
Diabetes	2.04	(1.48, 2.81)	1.78	(1.38, 2.31)	1.90	(1.46, 2.47)
≥ 65 yrs	1.98	(1.50, 2.62)	–		–	
MVD*	1.76	(1.32, 2.36)	1.58	(1.25, 1.98)	1.68	(1.34, 2.16)

**RR = relative risk, ‡CI = 95% confidence interval
*MVD = multivessel disease, †CHF = congestive heart failure

Easily recognizable baseline risk factors are the factors most closely associated with long term (8 year) adverse prognosis following PTCA irrespective of whether death, death and MI, or death MI and CABG are used as endpoints; of these factors, a history of CHF at baseline is the most powerful.

1010-115 Periprocedure Myocardial Infarction Rates in the Bypass Angioplasty Revascularization Investigation (BARI)

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The Bypass Angioplasty Revascularization Investigation (BARI) examined the hypothesis that an initial revascularization strategy of percutaneous transluminal coronary angioplasty (PTCA) compared to coronary artery bypass graft (CABG) in pts with multivessel coronary disease does not compromise clinical outcome during 5 year follow-up. We report the incident rate of procedure-related myocardial infarction (MI), an event with a potential negative impact on long-term survival, in each treatment group. Between August, 1988 and August, 1991, 914 and 915 pts were randomly assigned to PTCA or CABG. The MI event rates were examined in a priori determined subgroups of unstable angina (69% of the population), severe chronic stable angina (16%), and mild or no angina with severe ischemia on noninvasive testing (15%); and in patient subsets stratified by number of diseased vessels and left ventricular ejection fraction; and in presence or absence of proximal left anterior descending stenosis ≥ 50%, hypertension, diabetes, and congestive heart failure. Ascertainment of MI was based on serial ECG analysis, cardiac enzyme data, and clinical history, and events were classified at St. Louis University Core laboratory. The new appearance of a 2 grade Minnesota code Q-wave progression defined a Q-wave MI event when it occurred < 96 hours post procedure. If the event occurred > 96 hours postprocedure, MI was defined if a 2 grade Minnesota code Q-wave progression occurred; or if cardiac enzymes were abnormal in the presence of a new left bundle branch block; or if cardiac enzymes were abnormal in the presence of chest pain or worsening ST-T wave abnormalities. The incident rate of Q-wave MI was compared within 96 hours post procedure and 3 months post procedure. The periprocedure MI rates associated with the index randomization and subsequent revascularization procedures will be presented for both treatment strategies.