A STATISTICAL MODEL TO PREDICT RISK FOR HEMODYNAMIC COMPROMISE DURING CORONARY ANGIOPLASTY

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In an effort to identify patients (pts) at risk for hemodynamic compromise (HC) during PTCA who would be candidates for circulatory support devices, we reviewed the angiograms of 157 pts undergoing PTCA of whom 12% suffered HC (defined as a fall in systolic blood pressure below 90 mmHg during balloon inflation). Multivariate analysis of 20 angiographic variables showed:

ANGIOGRAPHIC CHARACTERISTIC	UNIT	ODDS RATIO	P =
Multivessel disease (MVD)	Yes,No	4.3	0.03
Diffuse disease of segment (DD)	Yes,No	4.1	0.05
% myocardium at risk* (MAR)	10%	1.7	0.03
Pre-PTCA stenosis (PPS)	10%	0.6	0.04

(*MAR = % myocardium distal to the stenosis dilated)

Weighing the predictive value of these variables, a 13 point scoring system was constructed to express the patient's risk for HC. With MVD = 3, DD = 3, MAR = -2 to 4, and PPS = -3 to 3, a score of 1-3 was associated with a risk of <11%, 4-6 with 11-33%, 7-8 with 33-55%, 9-10 with 55-76%, and 11-13 with 76-93%.

In conclusion, these data identify angiographic characteristics associated with HC and assign a weighted value to them based on the strength of their relationship to HC. Therefore, this statistical model should improve the ability to prospectively predict HC and thus its validation seems warranted.

FLOW CYTOMETRIC ANALYSIS OF PLATELET PADGEM EXPRESSION DURING PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY.

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We studied the effect of percutaneous transluminal coronary angioplasty (PTCA) on platelets within the coronary and systemic circulations. PADGEM (also termed GMP 140), is a transmembrane alpha granule protein expressed on the platelet surface following activation and secretion. We used a fluorescence-activated flow cytometric assay to quantify PADGEM expression by circulating platelets. In 23 patients receiving aspirin, heparin, and a calcium channel blocker, coronary sinus (CS) and peripheral venous (PV) blood samples were collected before and immediately after left coronary PTCA. Binding of fluorescent monoclonal AC1.2 anti-PADGEM antibody to the platelet surface was used to determine the % of platelets that expressed PADGEM. In all patients, the % of CS platelets expressing PADGEM increased post-PTCA from 15.1 ± 16 % to 22.1 % ± 18 % (p = .014). Parallel changes were present in PV platelets (from 11.9 ± 12% to 17.5 ± 14%; p = .025). In 6 patients presenting with myocardial infarction or unstable angina, baseline levels were significantly higher compared to patients with stable angina (p < 0.001) and did not increase further post-PTCA. In 2 patients who developed vessel closure and/or myocardial infarction post-PTCA, PADGEM-positive platelets increased from 5.75 ± 3.3% to 42.7 ± 6.8% in CS and from 3.5 ± 2% to 37.2 ± 7.9% in PV.

PADGEM-POSITIVE PLATELETS (% of total; mean ± SD):

	Stable patients (n = 17)		Unstable patients (n = 6)	
	CS	PV	CS	PV
Baseline	7.3 ± 6.0%	7.1 ± 6.8%	37.3 ±16.2%	25.5 ± 14.3%
Post-PTCA	16.0 ± 14.9%	13.7 ± 11.8%	39.6 ±15.4%	28.3 ± 16.2%

Conclusions: 1) PTCA increases levels of PADGEM expression by circulating platelets despite aspirin therapy. 2) PADGEM expression is most elevated in patients with vessel closure, myocardial infarction or unstable angina. 3) Comparable PADGEM expression in CS and PV raises the possibility of extra-coronary platelet activation post-PTCA. 4) Fluorescence-activated flow cytometric assay of PADGEM expression permits the study of platelet activation and interventions designed to modify platelet function during PTCA.

IMPAIRED PRODUCTION OF CYCLIC AMP IN RESPONSE TO PROSTACYCLIN BY LASER-EXPOSED HUMAN BLOOD PLATELETS.

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Since platelets may play a critical role in thrombosis and reocclusion during laser angioplasty, we studied the effect of laser radiation (LR) on prostacyclin induced cyclic AMP (cAMP) formation in these cells using protein kinase binding method. The exposure of platelet rich plasma (PRP) to LR (3.5 W, 448 nM, 100 uM spot size, 1 sec pulse duration, 0.5 ml PRP) decreased the production of cyclic AMP formation induced by 10 nM PGI_2 from 10.35 ± 3.69 pmol/10^8 cells to 3.11 ± 0.28 pmol/10^8 cells ($n = 12$, $p < 0.05$). Formation of cyclic AMP in control PRP was 2.50 ± 0.71 pmol/10^8 cells. Exposure of PRP to increasing pulses of LR linearly decreased the PGI_2 induced production of cAMP and at 300 pulses 80% of the total cAMP formation was inhibited when compared with the controls. The laser exposed platelets (28 pulses) also showed significant ($p < 0.001$) resistance to the inhibitory effect of PGI_2 (IC_{50} 14.0 ± 1.15 nM) when compared to the controls (IC_{50} 7.01 ± 1.16 nM) using ADP and 1-epinephrine ($n = 9$) as the aggregating agents. However, the laser exposed platelets did not show any change in ^3H-prostaglandin E_1 activity (kd_1 9.5 nM; n_1 450 ± 30 sites/cell; kd_2 1.1 uM; du 1450 ± 150 sites/cell) when compared to control (kd_1 7.5 nM; n_1, 420 ± 50; kd_1 1.2 uM; n_2, 1420 ± 200 sites/cell). Conclusion - these results indicate that exposure of platelets to LR renders these cells less responsive to the inhibitory effect of PGI_2 through decreased cAMP formation, and the decreased cAMP production is due to post-receptor impairment. The impaired production of cAMP would lower the aggregation threshold of laser exposed platelet thus enhancing their prothrombotic potential during laser angioplasty.

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Vascular Stents: Technical Developments and Clinical Outcomes

DIMENSIONAL STABILITY OF THE GIANTURCO-ROUBIN BALLOON EXPANDABLE STENT ASSESSED BY QUANTITATIVE CORONARY ANGIOGRAPHY

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Recoil of balloon expandable intracoronary stents shortly after deployment could have serious consequences, such as potential for downstream migration, narrowing of lumen diameter and impeding formation of neoendothelium. In order to see if recoiling occurs with such stents, we utilized quantitative coronary angiography (Cardiovascular Angiography Analysis System) to measure stent diameter (mean diameter of the entire stent lumen) immediately after deployment, 30 minutes, and 1 to 4 days (mean 1.8) later in 10 pts undergoing Gianturco-Roubin stent implantation. Nominal stent size was 2.5 in 4 (2 RCA, 2 CFX), 3.0 in 4 (2 LAD, 1 RCA, 1 CFX), 3.5 in 1 saphenous vein graft and 4.0 mm in 1 saphenous vein graft. Mean normal arterial diameters 0.5 cm proximal and distal to the stent were 3.02 ± 0.43 and 2.71 ± 0.59 mm, respectively. Mean diameter of the fully expanded balloons used to deploy the stent was 3.34 mm. Mean stent diameters and ratios of stent to normal arterial (S:A) diameter for the 3 time intervals (mean ± SD) were:

	Stent diameter (mm)	S:A ratio
Immediately	2.91 ± 0.29	0.96
30 minutes	2.87 ± 0.32	0.95
1.8 days	3.00 ± 0.42	0.98

There were no significant differences (p=ns) between any two time periods.

Thus, stent diameter of the Gianturco-Roubin balloon expandable stent achieves the anticipated dimension of the normal segment of the artery and maintains dimensional stability after deployment.