SHOZHOO4> -POOHER

microscopy technique. The microscopist was blinded to the rotational speed of the samples. The numbers of aggregates 20–80 μ m diameter/ml blood are shown below as a function of the rotational speed with or without ReoPro* .

Number of Platelet		Rotational Sp	eed	
Aggregates	180k rpm	180k rpm + ReoPro	140k rpm	Control (0 rpm)
20-60 µm dia- mater/mi blood ³ Range	4131 ± 3336 444-13889	934 ± 715 133-2811	420 ± 435 111-1722	246 ± 293 83-1222

n = 20; ± Std Dev.; p < 0.002 for all comparisons.

Not all blood samples responded to ReoPro. In 4/20 samples. ReoPro decreased the number of platelet aggregates caused by the rotating burr by less than 20%. Platelet aggregates in all samples subjected to lower speed were decreased by greater than 75%. (Ratio of # of aggregates: 140k/180 + ReoPro 0.99).

Conclusion: This in vitro testing suggests that the use of the Rotablator system at 140,000 rpm (its minimum approved speed) may be more effective in reducing platelet aggregation than ReoPro. Whether reduced speed will provide clinical benefit in reducing slow flow and post procedure creatine kinase elevation is being investigated.

1189-61

Rotational Atherectomy Increases Circulating Platelet-Monocyte Complexes

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Rotational Atherectomy (RA) facilitates the debulking of various lesion types but is associated with an increase in NGMI. Proposed mechanisms include plugging by debris and platelet (pit) activation. Using whole blood flow cytometry we compared several markers of pit activation in peripheral blood: [plt-monocyte complexes, pt-neutrophil (PMN) complexes, P-selectin (CD82), and activated GP lib-Illa expression (PAC-1)] in patients undergoing RA with those undergoing coronary angioplasty (PTCA), peripheral angioplasty (PA), or cononary angiography (CA). No consistent increase after the procedure could be detected for pit-PMN, CD-62, or PAC-1. However, pit-monocyte complexes increased following RA:

	RA	PA	PTCA	CA
Pre	140 ± 2	13.0 ± 1.5	57±6	12.4 ± 2
1 Hr Post	32.2° ± 6.4	16.5 ± 2	23 9 ± 8.3	19.3 ± 5.9
24 Hr Post	15.2 ± 2.6	9.8 ± 4.8	8.5 ± 5.3	14.3 ± 3.4

p < 0.05 vs Pre

Pit/monocyte complexes represent a stable measure of pit activation in peripheral blood while direct assessment of peripheral pit activation are less conclusive. Measurement of pit/monocyte complexes suggests that RA activates pit more than PA, PTCA or CA. Increased pit activation may represent a mechanism for the higher incidence of NQMI following RA.

1189-62

Intracoronary Adenosine Administered During Rotational Atherectomy of Complex Lesions in Native Coronary Arteries Reduce the Incidence of 'no Reflow' Phenomenon

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Background: Rotational atherectomy (RA) of complex, highly calcified lesions has been associated with a high incidence of 'no reflow' ranging from 6–15% and concomitant myocardial necrosis with adverse prognostic implications. There are no uniform strategies for preventing this complication. The role of intracoronary adenosine for the prevention of this phenomenon during RA has not been fully evaluated.

Methods: We studied the procedural outcome of 122 patients who underwent RA of complex native coronary artery lesions. Fifty two patients received no adenosine, but a variety of other agents. Seventy patients received intracoronary adenosine boluses (24 mcg to 48 mcg prior to and after each RA run). There was no difference in the type of tesion studied, run time, or Burr to artery ratio (0.6–0.7) between the two groups.

Results: Six patients without adenosine experienced 'no reflow' (11.6%) with resultant infarction in the target artery territory, while only one of seventy patients (1.4%, p=0.023) in the adenosine group experienced no reflow. No untoward complications were observed during adenosine infusion.

Conclusion: Intracoronary adenosine bolus administered during rotational atherectomy is easy, safe and may significantly reduce the incidence of 'no reflow' which may improve the 30 day outcome of this procedure.

1189-63

Coronary Stenting After Rotational Atherectomy Versus Coronary Stenting Alone: An Anglographic Comparison

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Purpose: To retrospectively compare the immediate and six month angiographic angiographic outcome for these two different approaches.

Methods: A total of 178 patients (270 lesions) who had stents implanted in de novo, non occluded vessels and had six month angiographic follow up were classified into two groups: stent alone (146 patients, 226 lesions) and rotostent (32 patients, 44 lesions). Acute and follow-up (FU) results are shown in the table:

	Rotostent	Stent	p value
Lesion Type (B2+C)	97.68%	66.07%	< 0.001
Calcium	84.09%	8.93%	~ 0.001
Pre reference (mm)	2.92 ± 0.45	3.0 ± 0.51	
Lesion length (mm)	12.27 ± 7.37	11.77 ± 6.54	กร
Post MLD (mm)	3.05 ± 0.56	3.0 ± 0.54	ns
Post %DS	3.65 ± 10.37	8.10 ± 10.42	ns
FU MLD (mm)	1.72 ± 0.82	1.91 ± 0.79	ns
FU %DS	40.80 ± 22.62	38.02 ± 21.20	ns
Acute gain (mm)	2.1 ± 0.59	1.98 ± 0.53	ns,
Late loss (mm)	1.33 ± 0.71	1.1 ± 0.72	ns,
Loss index	0.67 ± 0.37	0.59 ± 0.44	ns
Restenosis	40.91%	31.42%	ns.

Conclusion: Despite the presence of more unfavorable angiographic characteristics in the lesions treated with rotablation and stenting, the immediate and long-term angiographic results were similar to those lesions treated with stenting alone.

1189-64

The Role of Adjunctive Balloon Dilatation in Directional Coronary Atherectomy Without Subintimal Resection

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ABACAS (Adjunctive Balloon Angioplasty Following Coronary Atherectomy Study) is a prospective randomized multicenter trial to study whether aggressive debulking with IVUS-guided DCA followed by adjunctive balloon dilatation reduces restenosis. Restenosis rates were 23.6% for adjunctive balloon and 19.6% for DCA alone (n.s.). To elucidate the influence of deep wall resection and adjunctive balloon on the restenosis rates, histological and angiographic study were performed in the same cohort. According to the presence of subintimal resection and adjunctive balloon, eligible 194 pts were divided into four subgroups. DCA with intimal resection (D/I) group consisted of 38 pts, DCA with subintimal resection (D/S) group 57 pts, DCA/balloon with intimal resection (D/S/B) group 53 pts.

Results: QCA analysis revealed more (n.s.) late loss (D/I: 0.7 ± 0.5 , D/S: 0.9 ± 0.6 , D//B: 1.2 ± 0.7 , D/S/B: 1.0 ± 0.6 mm) and higher (n.s.) loss index (D/I: 49%, D/S: 59%, D//B: 64%, D/S/B: 50%) in D/I/B group than the other groups. Restenosis rate of D/I/B group was significantly higher than the other groups (D/I: 13.9%, D/S: 17.6%, D//B: 34.6%, D/S/B: 12.0%)(p < 0.05).

Conclusion: Adjunctive balloon tollowing DCA without subintimal resection increased restenosis rate. IVUS-guided complete removal of atheroma without adjunctive balloon may be the best strategy on DCA.

1190

Considerations in Use of Heparin, Adenosine, K+ Channel Opener, and GPIIa/IIIb Inhibition During Interventional Procedures

Wednesday, April 1, 1998, Noon–2:00 p.m. Georgia World Congress Center, West Exhibit Hall Level Presentation Hour: Noon–1:00 p.m.

1190-98

Prolonged Heparin After Uncomplicated Coronary Interventions: A Prospective Randomized Trial

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The aim of this trial was to evaluate if heparin infusion after uncomplicated coronary interventions reduced the incidence of acute cardiac complications.

A total of 191 consecutive patients who underwent a successful PTCA were randomly assigned to receive either prolonged (heparin group, 100 pts.) or no postprocedure heparin (control group, 91 pts). The two treatment groups were comparable with respect to clinical and angiographic characteristics.

	Control group	Heparin group	ρ
Stenosis before PTCA (%)	74 ± 16	72 ± 15	na
Stenosis after PTCA (%)	23 1 11	23 ± 10	กร
MLD before PTCA mm	0.9 ± 0.5	0.8 ± 0.4	ns
Reference Diameter mm	2.8 ± 0.7	2.9 t 0.8	ns
MLD after PTCA mm	2.3 ± 0.5	24:06	กร
Stents	33 (36%)	33 (33%)	ne
Myocardial infarction	4%	3%	ns
CABG	0	0	ns
Death	1%	ō	ns
Vascular complications	1%	3%	กร

Four patients in the control group (4%) and 3 patients in the heparin group (3%) suffered a myocardial infarction. One patient in the control group died three days after the intervention. Peripheral vascular complications in the control and heparin group occurred in 1% and 3% of the patients, respectively.

Conclusions: Omission of heparin after successful PTCA with or without stent implantation in patients with stable and unstable angina did not significantly increase the incidence of acute cardiac complications. It allows for early sheath removal and patient discharge and saves costs. It should be the policy for routine patients.

1190-99

Heparin-induced Thrombocytopenia Syndrome **Complicating Percutaneous Coronary Intervention**

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Heparin-induced thrombocytopenia, a severe prothrombotic syndrome mediated by a heparin-associated platelet-activating IgG, has been little studied in the context of percutaneous coronary intervention (PTCA). Between 11/92 and 10/96, of 9486 patients (pts) who underwent PTCA, 69 (0.73%) pts (46 male; age 68 ± 9 (SD) y) demonstrated heparin-induced platelet aggregation associated with absolute (94% of pts) or relative (5.6%) thrombocytopenia. Platelet counts fell by 60 \pm 18% from 234 \pm 66 K/ μ L to 88 \pm 40 K/ μ L at 3.9 \pm 2.8 days after commencing i.v. unfractionated heparin. Thrombotic events occurred in 18.8%; lower limb arterial thrombosis, 4 pts (1 requiring amputation); lower limb venous thrombosis, 5 pts; dialysis fistula thrombosis, 1 pt; fatal mesentenc infarction, 1 pt; recurrent intracoronary thrombus formation with Q-wave Mt, 1 pt; and acute ≤24 h) thrombosis of 1 of the 24 coronary stents deployed (4.2%), with Q-wave MI in this pt. Pretreatment with aspirin (62.3%) or aspirin plus ticlopidine (28.9%), duration of heparin treatment (2.3 \pm 2.1 days), dose of heparin (1050 \pm 150 μ /h) or prior heparin exposure (75.4%) where not predictors of degree of fall in platelet count or occurrence of thrombotic events. Thrombotic events occurred in 3 of 19 pts (15.8%) who received abciximab and in 9 of 50 pts (18%) who did not (p = NS). No additional thrombotic events occurred among pts in whom unfractionated heparin was substituted with low molecular weight heparin (after excluding crossreactivity: n = 7), argatroban (n = 1) or ancrod (n = 1). Skin necrosis

occurred in 1 of 14 pts commenced on coumadin. Conclusion: Heparin-induced thrombocytopenia complicating coronary intervention results in a high frequency of major arterial and venous thrombctic events, irrespective of dose and duration of heparin, and despite optimal antiplatelet therapy.

1190-100

The Heparin Infusion Prior to Stenting (HIPS) Trial: Procedural, In-hospital, 30 Day, and six Month Clinical, Angiographic and IVUS Results

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Local delivery of heparin is a promising approach to reduce in-stent restenosis. Stent-lumen volume as measured by intravascular ultrasound (IVUS) provides a means to efficiently evaluate the impact of new therapies on in-stent restenosis. We studied the impact of locally delivered heparin on in-stent restenosis as measured by IVUS in the HIPS study, a multi-center, randomized conrolled trial. A total of 179 patients were randomized to receive heparin (5000 U in 5 ml) either intracoronary (ic/control) via the guide catheter or intramural via the InfusaSleeve (LocalMed, Inc.) prior to single Palmaz-Schatz stent placement. Baseline demographic, clinical and angiographic parameters were evenly distributed with the exception of initial Minimal Luminal Diameter, 1.12 ± 0.34 vs 0.99 ± 0.37 mm, in the ic/control and intramural groups respectively (p = 0.03). Core angiographic laboratory evaluation of the intramural group revealed 18 NHLBI Grade B/C dissections following initial PTCA of which 1 progressed following local heparin therapy. there were no significant differences in the B/C dissections in the two groups prior to stent placement, 33% (ic/control) vs 29% (intramural). Procedural outcomes as measured by clinical, angiographic and I-/US criteria were similar between groups (clinical success 98.9 vs 98.3% patients in the ic/control and inframural groups respectively). In-hospital event rates, 1.1% (ic/control) vs 1.2% (intramural) and 30 day complications (none reported) were similar. Six month clinical, angiographic and IVUS follow-up will be complete in January 1998.

We Conclude: Local delivery of heparin via the trifusaSleeve is feasible and safe. The impact of this therapy on restenosis following stent placement as determined by angiographic and IVUS parameters will be presented.

1190-101

Effects of Local Heparin Delivery on Coronary Thrombin and Anti-Thrombin Activity During Percutaneous Transluminal Coronary Angioplasty

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Background: The inhibitory effects of heparin (H) on the coagulation system are mediated by the activation of anti-thrombin III (AT III). Little is known of the affects of systemic H on trans-cardiac AT III activity and no information is available on trans-cardiac AT III following local H delivery during PTCA.

Methods: We analyzed paired systemic arterial (SA) and coronary sinus (CS) blood samples in 9 patients undergoing PTCA. AT III and FpA, a marker of thrombin activity, was measured prior to coronary instrumentation (base), 5 minutes following 10,000 units of intravenous H (SH), 5 minutes after completion of PTCA (pre LH), and 5 minutes after 4,000 units of H delivered to the PTCA site via a LocalMed infusion catheter (post LH).

Resuts:

	base	SH	pre LH	post LH
CS AT III (%)	71 ± 16	69 ± 16	75 ± 27	97 ± 23ª
SA AT III (%)	67 ± 19	64 ± 25	79 ± 20	80 ± 19 ^b
CS FpA (ng/ml)	10.0 ± 2.1	5.9 ± 3.6	22 ± 3.2	1.5 ± 2.8°
SA FpA (ng/mt)	9.7 ± 1.9	5.8 ± 3.6	4.8 ± 3.4	4.6 ± 2.6 ^d

 $t^{a} \sim p = 0.001^{-b} \approx p = 0.004$, c = p < 0.0001, d = p < 0.001 by ANOVA)

Conclusions: 1) SH during PTCA results in detectable increases in both SA & CS AT III activity and decreases in SA & CS FpA and 2) LH results in further increased local (CS) AT III activity and diminished FpA release. These results support enhanced and site-specific heparin activity when administered locally.

1190-102 Does Pre-treatment With Intravenous Heparin Produces any Angiographic Improvement in Patients Admitted With Unstable Angina?

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The presence of intracoronary thrombus is a common finding in patients (pts) undergoing cardiac catheterization during unstable angina (UA). Coronary angioplasty (PTCA) during this setting has been associated with a significant increment of complications. Temporary treatment with intravenous (IV) heparin prior to proceed with the PTCA has been postulated as a safer way in this pts. The purpose of this study was to evaluate the angiographic changes after the treatment with heparin during 48 to 96 hours (hrs) in pts admitted with UA. From 08/96 to 08/97, we prospectively evaluate 23 pts with UA who had the first coronary angiogram (angio) within 24 hrs (mean of 17.8 \pm 6.9 hrs) of the last episode of class IV angina and the second angio between 48 to 96 hrs (mean of 53.3 ± 17.4 hrs) after the treatment of IV heparin. The presence of thrombus, thrombus score, the TiMi flow, the frame count measurement, percentage of stenosis, ACC score and all the morphologic characteristics were evaluated in both angio by two blinded observers. (Angiographic thrombus was classified as 0 = no thrombus, 1 = haziness, 2-4 = definitive thrombus with size < 0.5, 0.5-1.5, > 1.5 the reference diameter, respectively). Both angro did not show any significant changes.

	First angiogram	Second angiogram	p value
Thrombus (+)	56.5%	56.5%	NS
Thrombus Score	2.23 ± 1.3	2.07 ± 1.04	0.52
TIMI flow	2.65 ± 0.57	2.65 ± 0.64	NS
Frame Count	50.3 ± 33	40.4 ± 29	0.29
Thrombus Size	3.63 ± 1.7 mm	2.6 ± 1.7 mm	0.27
% Stenosis	69 ± 7.8%	67.9 ± 10.8%	0.69