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	p
Stenosis before PTCA (%)	74 ± 16	72 ± 15	ns
Stenosis after PTCA (%)	23 ± 11	23 ± 10	ns
MLD before PTCA mm	0.9 ± 0.5	0.8 ± 0.4	ns
Reference Diameter mm	2.8 ± 0.7	2.9 ± 0.8	ns
MLD after PTCA mm	2.3 ± 0.5	2.4 ± 0.6	ns
Stents	33 (36%)	33 (33%)	ns
Myocardial infarction	4%	3%	ns
CABG	0	0	ns
Death	1%	0	ns
Vascular complications	1%	3%	ns

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 ± 18% from 234 ± 66 K/μL to 88 ± 40 K/μL at 3.9 ± 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 mesenteric infarction, 1 pt; recurrent intracoronary thrombus formation with Q-wave MI, 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 ± 2.1 days), dose of heparin (1050 ± 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 thrombotic 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 IVUS criteria were similar between groups (clinical success 98.9 vs 98.3% patients in the ic/control and intramural 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 InfusaSleeve 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).

Results:

	base	SH	pre LH	post LH
CS AT III (%)	71 ± 16	69 ± 16	75 ± 27	97 ± 23[a]
SA AT III (%)	67 ± 19	64 ± 25	79 ± 20	80 ± 19[b]
CS FpA (ng/ml)	10.0 ± 2.1	5.9 ± 3.8	2.2 ± 3.2	1.5 ± 2.8[c]
SA FpA (ng/ml)	9.7 ± 1.9	5.8 ± 3.8	4.8 ± 3.4	4.6 ± 2.6[d]

[a] = p < 0.001, [b] = 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 ± 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 angio 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