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Abstracts of Original Contributions: 45th Annual Scientific Session

The American College of Cardiology is pleased to announce that nearly 7,000 abstracts of original contributions were submitted to the Program Committee of the 45th Annual Scientific Session. Space and time considerations this year allowed the selection of 1,755.

Each abstract was peer reviewed by a panel of graders; chosen abstracts are presented in either oral or poster format.

The American College of Cardiology thanks the

thousands of abstract participants and the hundreds of category graders and chairs for their efforts.

James L. Ritchie, MD, FACC Chair 1996 Annual Scientific Session Program Committee

W. Douglas Weaver, MD, FACC Co-Chair

1996 Annual Scientific Session Program Committee



Key Contributions

Sunday, March 24, 1996, 6:30 p.m.-8:30 p.m. Orange County Convention Center, Hall E Presentation Hour: 6:30 p.m.-8:30 p.m.

ACUTE MYOCARDIAL INFARCTION – THERAPY

901-1

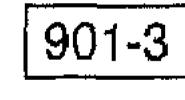
Megadose Bolus Heparin as Reperfusion Therapy for Acute Myocardial Infarction: Results of the **HEAP Pilot Study**

Freek W.A. Verheugt, Randall C. Marsh, Gerrit Veen, Jean G.F. Bronzwaer, Felix Zijlstra. North Colorado Med Ctr, Greeley, CO; Free University Hospital, Amsterdam; University Hospital, Nijmegen; Weezenlanden Hospital, Zwolle, The Netherlands

Angiography after thrombolysis for acute myocardial infarction (AMI) shows better patency when adjunct intravenous (iv) heparin is used. We wondered, if iv heparin alone also induces reperfusion.

In the HEAP (Heparin in Early Patency) pilot study 50 patients (pts) with <6 hours (h) signs; ($\geq 2 \text{ mm ST} \uparrow \text{ in } \geq 2 \text{ leads}$) and symptoms of AMI received a single iv bolus of 300 U/kg heparin, a dose usually given by cardiac surgeons prior to cardiopulmonary bypass. Doses of bolus heparin given varied from 10,000 to 40,000 U. Aspirin (160 mg chewed), but no thrombolytic agent was given. Patency was assessed by coronary angiography at 90 minutes (min) after the heparin bolus. In 28/50 (56%) pts TIMI flow 2-3 was seen at 90 min: TIMI flow 3 in 18 (36%) pts and TIMI flow 2 in 10 (20%) pts. Pts with < 2 h symptoms (n = 24) had 67% TIMI flow 2–3 versus 46% in pts with \geq 2 h symptoms (p = 0.09). Pts with TIMI flow 0–1 underwent direct angioplasty. At 90 min aPTT exceeded 120 sec in all pts. No significant bleeding was seen. Aspirin 80 mg daily was given, as was heparin (aPTT 2.0-2.5) for 48 h. Predischarge angio showed TIMI flow 3 in 15/18 (83%) pts. Thus, early therapy with high-dose front-loaded heparin alone can induce full coronary reperfusion in pts with AMI, especially in early (<2 h) pts. This simple, inexpensive and easily antagonizable AMI treatment seems to be an attractive alternative to regular thrombolysis, both in-hospital and pre-hospital, and can also be given prior to direct PTCA.

out over an eighteen month period by front line ambulance crews based at 15 London Ambulance Stations, 1850 patients with suspected acute myocardial infarction aged over 30 years were screened for eligibility. 1652 were randomised by the ambulance crews. 999 were allocated to a strategy of pre-hospital aspirin and 653 allocated to control (no aspirin). 980 (98%) of those allocated to the treatment group received the medication. At 35 days there were 91 deaths in the aspirin group (9.1%) compared with 56 (8.6%) in the control group. The absolute increased risk 0.5% (95% confidence interval -2.26% to 3.3%) was not significant. The majority of deaths were due to cardiac causes 77 (85%) in the aspirin group and 47 (84%) in control. There were no major side effects attributable to pre-hospital aspirin. During long term 24-38 months follow up, there were 248 deaths (24.8%) in the aspirin group and 163 (25%) in the control group. This 0.2% reduction in the risk of death (95% confidence interval -4.4% to 4.1%) was not significant. We conclude that a policy of pre-hospital administration of aspirin to patients with suspected acute myocardial infarction is safe but does not improve outcome.



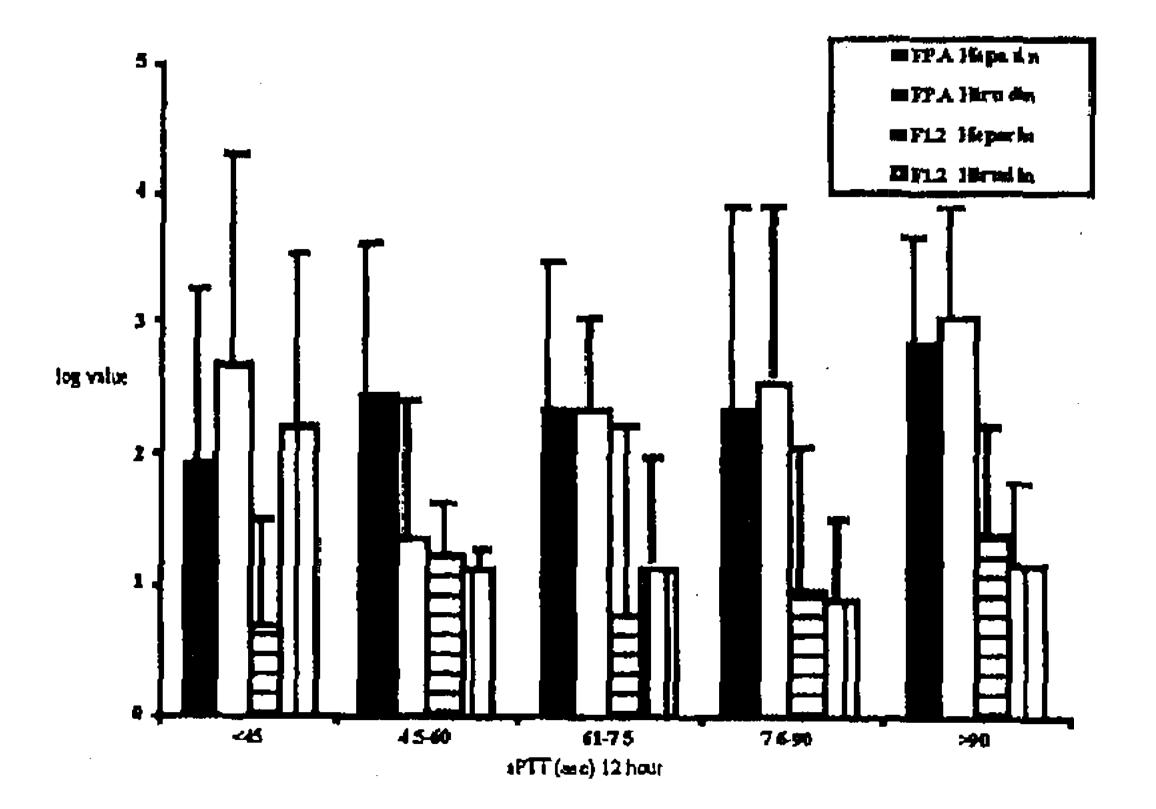
Early aPTT Measurements Are Not a Surrogate for in Vivo Thrombin Inhibition Among Patients Receiving **Thrombolytic Therapy and Adjunctive** Anticoagulation

Failure to Improve Outcome in Suspected Acute 901-2 Myocardial Infarction by Pre-Hospital Administration of Aspirin

Robert Greenbaum, Martin Flaherty, Koon Lan Chan, Dan Shanit. Cardiovascular Research Unit Edgware General Hospital and The London

Richard C. Becker, James Hebert, Thomas Hurley, Yunsheng Ma, Christopher P. Cannon, for the TIMI 5 Hemostasis and Thrombosis Study Group. Thrombosis Research Center, University of Massachusetts Medical School, Worcester, MA

The immediate goal of thrombolytic therapy is to rapidly establish and maintain physiologic coronary arterial blood flow. Adjunctive anticoagulant therapy may be required to achieve optimal reperfusion; however, the target aPTT value has been difficult to establish. In TIMI 5, measures of thrombin activity (FPA) and thrombin generation (F1.2) were obtained serially in 246 patients receiving accelerated tPA and either heparin or hirudin. FPA and F1.2 levels at 12 and 24 h did not correlate with aPTT at these time points in either heparin or hirudin-treated patients. An aPTT > 90 sec (commonly) considered high-intensity anticoagulation), was not consistently associated with decreased thrombin activity or generation. In fact, the lowest combined FPA and F1.2 value was seen with a 12 h aPTT of ≈ 60 sec.



Ambulance Accident and Emergency Service, London, UK

The ISIS-2 trial has shown that in-hospital treatment of suspected acute myocardial infarction (AMI) patients with aspirin is almost as effective as treatment with streptokinase in reducing 35 day mortality. We carried out a prospective, randomised, non-blind, controlled study of the effect of administration of a single oral dose of 300 mg aspirin to patients with suspected AMI prior to transportation to hospital on 35 day mortality. The study was carried