of cardiac death ($p < 0.01$) and to have a close relationship with ejection fraction one year later ($R = -0.549$, $p < 0.01$). This score, therefore, was considered to be of use in predicting prognosis. When there were adequate collateral vessels or the occlusion was only subtotal, some blood supply to the infarct area was rather deemed to influence the improvement of regional wall motion one year later. Time to coronary thrombolysis did not coincide with regional wall motion.

5 patients who had reoccluded or subtotally occluded one year later, had well developed collateral vessels and tended improve their regional wall motion from 33.7 to 38.4%. It suggests that the patient with well developed collateral vessels can maintain his wall motion in case of reocclusion.

-313- (Omitted)

-314- CORONARY THROMBOLYSIS WITH TISSUE- TYPE PLASMINOGEN ACTIVATOR (t-PA) -COMPARISON WITH UROKINASE-

Takashi Susawa, Ryuichi Hattori, Keiji Sakaguchi, Mamoru Takahashi, Takeshi Aoyama, Yoshiki Takatsu, Natsuko Yui, Yoshiki Yui, Chuichi Kawai.

Third Division, Department of Internal Medicine, Faculty of Medicine, Kyoto University.

The requirement of heparin in experimental coronary thrombolysis by tissue-type plasminogen activator (t-PA) was studied in dogs with 1-hr-old coronary thrombi. Coronary thrombus was induced by advancing a copper coil in the anterior descending coronary artery, and thrombolysis was confirmed by repeat coronary angiography.

t-PA (1,000 IU/kg/min) was administered by intracoronary route for 44 ± 4 min ($n=5$; group 1), but thrombolysis could not be induced. Then, heparin (300 U/kg) was injected intravenously and successful thrombolysis was obtained 8 ± 4 minutes later in five dogs.

Heparin (300 U/kg) alone was administered intravenously to evaluate its thrombolytic effect in this model ($n=11$). Heparin could not induce thrombolysis, but ten minutes later, dogs received t-PA (1,000 IU/kg/min) by intracoronary route ($n=5$; group 2) or intravenous route ($n=6$; group 3). Successful reperfusion was obtained in 16 ± 5 min (group 2) and in 23 ± 6 min (group 3), each, respectively.

t-PA did not lead to the significant depletion of plasma fibrinogen (group 1; from 305 ± 27 mg/dl to 280 ± 28 mg/dl, group 2; from 315 ± 35 mg/dl to 285 ± 36 mg/dl, and group 3 305 ± 30 mg/dl to 293 ± 20 mg/dl), but it lowered alpha2-antiplasmin significantly (group 1; from 112.4 ± 4.7% to 79.0 ± 10.8%, $p < 0.001$, group 2; from 114.2 ± 5.5% to 96.4 ± 14.2%, $p < 0.05$, and group 3; from 108.7 ± 8.7% to 95.2 ± 14.0%, $p < 0.05$).

Intracoronary infusion of urokinase (1,000 IU/kg/min) for 27 ± 4 min elicited successful reperfusion without heparin pretreatment. Urokinase decreased plasma fibrinogen significantly (from 314 ± 29 mg/dl to 90 ± 35 mg/dl, $p < 0.001$), and plasma alpha2-antiplasmin significantly (from 113.2 ± 3.8% to the lowest detectable limit, $p < 0.001$).

The above results suggest that in experimental t-PA-induced coronary thrombolysis, preadministration of heparin seems to be absolutely necessary. However, in urokinase-induced thrombolysis, due to depletion of serum fibrinogen, heparin is not necessarily required for the coronary reperfusion.

-315- CORONARY THROMBOLYSIS WITH TISSUE- TYPE PLASMINOGEN ACTIVATOR (TPA) IN EXPERIMENTAL MODEL

Katsuo Kanmatsuse, Nagao Kajiwarra, Yohichi Satoh, Hiromi Seto, Masayuki Iwasaki, Tomiya Ohba, Kohji Yumi and Ikuyoshi Watanabe.

Dept. of Cardiology, Surugadai Nihon University Hospital, Tokyo.

Ten dogs were carried out to investigate whether two hours old coronary thrombi produced by cooper wire technique were lysed or not by intravenous TPA isolated from melanoma-tissue-culture.

TPA at a rate of 20,000 Unit/kg for 30 min were continuously infused intravenously in 5 dogs.

In only 1 of 5 dogs, complete recanalization and, in 1 dog, incomplete recanalization were achieved angiographically. In remaining 3 dogs, recanalization was not achieved in experimental period (during 60 min).

In contrast, administration of TPA 30,000 Unit/kg for 30 min reperused completely in 3 of 5 dogs, and incompletely in 1 of 5 dogs. In remaining 1 dog, reperfusion was not successful. Autopsy study recognized complete lysis in 3 of 5 dogs and incomplete lysis in 2 of 5 dogs in infusion of TPA 30,000 Unit/kg. Blood samples for ELT, Fibrinogen, FDP, α_2 Plasmin Inhibitor (α_2 PI), PT, APTT, were obtained from coronary sinus and femoral vein at before and post myocardial infarction, 15, 30, 60 min after TPA infusion. ELT was remarkably shortened after 20,000 Unit and 30,000 Unit/kg of TPA.

Fibrinogen at 30 min after TPA, decreased to 65 ± 12% before TPA infusion.

FDP from coronary sinus blood increased to two times greater values compared to no changed peripheral vein.

α_2 PI reduced to 39 ± 20% of control level at 30 min in 30,000 Unit/kg infusion. But, it returned to 90 ± 16% at 60 min after TPA.

PT, APTT were not changed significantly.

Thus, TPA 30,000 Unit/kg for 30 min infusion is effective to lyse 2 hours old thrombi without concomitant induction of a systemic lytic state.

-42- WALL MOTION ABNORMALITIES AND COMPENSATORY MECHANISM IN ACUTE TRANS-MURAL MYOCARDIAL INFARCTION -SERIAL DIGITAL SUBTRACTION ANGIOGRAPHIC (DSA) ASSESSMENT-

Kazuya Hayasaki, Hiroshi Kugimiya, Hirofumi Matsuda, Akira Maki, Tatsuya Kawaguchi, Masamichi Nakajima

Saiseikai Kumamoto Hospital

Serial changes in contraction abnormalities and compensatory mechanism were studied in patients with acute transmural myocardial infarction. Left ventricular function and regional wall motion were assessed by DSA performed in the RAO 30° and LAO 60° projection on admission, after one week and after one month.

The global ejection fraction and the extent of akinetic or dyskinetic, abnormally contracting segments (%ACS) were larger in anterior (EF, 44.7 ± 10.4; %ACS, 32.3 ± 10.4) than in inferior MIs (EF, 50.5 ± 7.4; %ACS, 20.8 ± 15.6). The LV endo-