aspirin (Control MDA - MDA after incubation of aspirin / Control MDA) was significantly decreased in OMI patients compared with that in normal subjects ($p < 0.001$).

These results suggest that the increase of MDA formation in OMI patients may contribute to the pathogenesis and progress of ischemic heart disease, and that it may be difficult to prevent ischemic heart disease by aspirin.

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DYNAMICS AND ROLES OF THROMBOXANE A_2 (TXA_2) AND PROSTACYCLIN (PGI_2) IN ISCHEMIC HEART DISEASE

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Purpose: Dynamics and roles of TXA_2 and PGI_2 during myocardial ischemia were investigated.

Method: Thromboxane B and 6-KetoPGF, α , which are stable metabolites of TXA_2 and PGI_2 , respectively, in aorta and great cardiac vein were measured in 45 patients with effort angina pectoris using atrial pacing, and in 20 patients with acute myocardial infarction during intracoronary thrombolysis.

Result: A Positive transmural TXA_2 gradient, suggesting intracoronary TXA_2 production, occurred in 19 patients during pacing-induced myocardial ischemia, and the amount of TXA_2 production correlated to the severity of myocardial ischemia.

In contrast, PGI_2 were not significantly changed during pacing-induced myocardial ischemia in patients with effort angina pectoris.

The Production of TXA_2 decreased with improvement of myocardial ischemia by PTCA.

In Patients with AMI, intracoronary TXA_2 production were observed in the early stage.

Conclusion: It was suspected that, in ischemic heart disease, intracoronary TXA_2 production resulted from myocardial ischemia and correlated to the severity of myocardial ischemia, and that no significant PGI_2 increase was observed during myocardial ischemia.

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DECREASED SERUM LEVELS OF PGI_2 STABILIZING FACTOR AND PGI_2 SYNTHESIS STIMULATING FACTOR IN ACUTE MYOCARDIAL INFARCTION

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Human serum contains two factors which regulate PGI_2 metabolism. This investigation was performed to study the role of these factors in the initiation or the pathogenesis of acute myocardial infarction (AMI). The one is PGI_2 stimulating factor which stimulates PGI_2 synthesis in the vessel wall. The aortic ring (2mm length) of rat aorta was incubated for one hour with 200 microliter serum. The activity of this stimulat-

ing factor was expressed as 6-keto- $PGF_{1\alpha}$ ng/mg wet weight of the ring/hr. The other is stabilizing factor that binds to PGI_2 . After incubation with (3H) PGI_2 at 22°C for 3 minutes, one ml of serum was mounted on Sephadex G-25 column. PGI_2 stabilizing factor complex appeared at the voiding fraction as an increased radioactivity. The percentage of this activity for total activity was obtained and defined as the activity of stabilizing factor. The activities of the two factors were determined in seven patients with AMI 2.5 ± 0.8 (I) and 80 ± 17 (II) hrs after the onset of chest pain, and compared with those obtained from 7 normal volunteers.

	SF	STF
volunteer	117 ± 48	72 ± 5.5
AMI (I)	15 ± 15	52 ± 6.9
AMI (II)	68 ± 20	63 ± 5.5

$P < 0.05$

SF, PGI_2 stimulating factor, STF, PGI_2 stabilizing factor. Multiple comparison test was used for statistics.

In the very early phase of AMI, the serum levels of these two factors were significantly reduced, which may be related to the thrombus formation in the coronary artery in AMI.

Chairman

- 25-28 T. Iwasaki, *Nishinomiya*
29-33 K. Kuramoto, *Tokyo*
34-38 M. Saito, *Suita*
39-43 T. Takano, *Tokyo*

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EFFECTS OF CORONARY VENOUS ARTERIALIZATION ON ACUTE MYOCARDIAL ISCHEMIA

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The efficacy and safety of synchronized coronary venous retroperfusion (SRP) of arterial blood were studied in 18 closed chest dogs with 6 hr LAD coronary artery occlusion (O). Eight were untreated controls and 10 were treated with SRP from 30 min after O until 6 hrs. Regional systolic fractional area change (FAC) and wall thickening were measured consecutively by two dimensional echocardiography in a low left ventricular short-axis cross section, and formed blood element counts were obtained prior to O and at 6 hrs post O. Postmortem study included myocardial water content (index of myocardial edema) and infarct size.

FAC in the ischemic zone at 30 min of O exhibited marked depression in both groups, after which the dysfunction persisted in control dogs, but was largely reversed with SRP from $6.0 \pm 6.5\%$ at 30 min O to $34.0 \pm 14.7\%$ at 6 hrs O ($p < 0.001$). Segmental wall thickening followed a similar trend. Measurement of infarct size expressed as % of the left ventricle was significantly less in treated dogs (3.2 ± 3.9 vs 8.5 ± 5.5 , $p < 0.05$). There was no significant difference as to damage to blood cells or platelet between two groups. Measurement of postmortem myocardial water content showed that SRP did not induce tissue edema or hemorrhage.

Conclusion: Experimental evaluation over a 6 hr period of coronary artery occlusion suggested that SRP is an effective and safe treatment for acute myocardial ischemia.