mid-slice was immediately frozen with liquid N₂ and the remaining slices were stained with TTC and giant mount slides for light microscopy were prepared.

A method of NADH-F was based on the evidence that NAD⁺ + NADH decreases in the necrotic tissue though it remains well preserved in only ischemic tissue. Therefore, the non-necrotic area fluoresces in 460nm when 365 nm ultraviolet light illuminates the cross-sectioned slice immersed in liquid N₂ since NAD⁺ reduces to NADH while the heart is removed and cross-sectioned. Thus the necrotic area can be distinguished from the non-necrotic area with loss of 460 nm fluorescence of NADH.

The area of necrosis was clearly delineated in all slices when TTC and microscopic changes showed compatible findings to necrosis. The areas determined with NADH-F correlated to those obtained with TTC and light microscopy.

NAD⁺ + NADH in the non-fluorescent area, measured by enzymatic method, reduced markedly.

In conclusion, NADH-F can be applied easily for the identification of necrotic area.

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CORONARY ANGIOGRAPHIC AND HEMODYNAMIC EFFECTS OF INTRACORONARY INJECTION OF ACETYLCHOLINE IN HUMANS

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The coronary angiographic and hemodynamic effects of intracoronary injection of acetylcholine (ACh) were measured in 29 patients (22 men and 7 women; mean age 51.3, range 15-65) with normal or near-normal coronary arteriograms. Patient with variant angina was excluded. ACh was given into the left coronary artery in sequential doses of 20, 50, 100, and 200 µg. Systemic hemodynamic variables, coronary sinus blood flow were measured and coronary arterial luminal diameters were measured from 35 mm cine-angiograms before and 1-1.5 min after each injection of ACh. Significant increase in coronary sinus blood flow (control vs ACh 20, 50, 100, 200 µg; 98.8 ± 35.8 , 118.9 ± 34.0 , 139.3 ± 47.7 , 170.0 ± 67.7 , 212.0 ± 130.0 ml/min, respectively) and significant decrease in coronary vascular resistance (control vs ACh 20, 50, 100, 200 µg; 1096.9 ± 369.4 , 848.5 ± 276.9 , 784.0 ± 333.0 , 660.7 ± 274.3 , 427.6 ± 176.2 mmHg min/L respectively) were recorded in all patients after ACh. In contrast to coronary hemodynamic changes, coronary arterial luminal diameters were decreased in many patients after ACh. Those effects of ACh also appeared even under stable hemodynamic condition maintained by right ventricular pacing, but were blocked by a prior

intravenous administration of atropine sulfate 1.0 mg. These results indicate that intracoronary injection of ACh induces vasoconstriction in major epicardial coronary arteries and vasodilatation in coronary arteriolar bed through activation of muscarinic receptors in humans.

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MECHANISM OF CORONARY ARTERIAL CONTRACTION INDUCED BY AGGREGATING PLATELETS

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To determine the mechanism of coronary arterial contraction induced by aggregating platelets, isolated coronary arteries from rabbits (n=43) were suspended for isometric tension recording in oxygenated Krebs solution. Suspensions of washed rabbit platelets were prepared by the method of Ardlie et al. Cumulative addition of 300 µl rabbit platelet suspensions ($10^6 - 3 \times 10^6$ /µl) activated with thrombin (0.2U/ml) into the 30ml organ bath resulted in platelet concentration-related contractions. Treatment of platelets with aspirin (0.5mM) caused little inhibition in platelet-evoked contractions. But the responses were significantly inhibited by pretreatment of coronary arteries with methysergide (0.3µM). And the responses to high concentrations of platelets were also inhibited by pretreatment with diphenhydramine (0.1µM). The concentrations of serotonin, thromboxane B₂ and histamine in the organ bath were 148 ± 11 ng/ml, 24 ± 2 ng/ml and 16 ± 2 µg/dl, respectively. Norepinephrine was not detected. These results indicate that platelet-evoked contractions of rabbit coronary arteries were mainly mediated by serotonin and partly mediated by histamine only at high concentrations of platelets.

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APPEARANCE OF CYCLIC FLOW CHANGE CAUSING MYOCARDIAL ISCHEMIA UNDER PHYSIOLOGICAL PERFUSION PRESSURE IN ISOLATED HUMAN CORONARY ARTERIES

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The effect of vasoactive drugs on tension development of isolated human coronary arteries has been studied. But it is unclear whether results from these studies can be applied to coronary spasm in vivo. It seems important to examine the relationship between cyclic tension development and cyclic ST elevation in patient with variant angina. To answer these questions, we evaluated changes in the resistance of isolated human coronary arteries perfused at a physiological pressure of 80mmHg. Coronary arteries were obtained from 11 patients without angina and one with variant angina. Cyclic flow changes were induced by PGF_{2α} in 5 of 6 cases (83%), by histamine in 5 of 10 (50%), by acetylcholine in 1 of 10 (10%),