The seven donor hearts, in which ASD and TR had been produced beforehand, were heterotopically transplanted into the recipient's chest cavity. Two to three mCi of ^{111}In -antimyosin (AM) was injected intravenously 6-12 hours before excised. ^{111}In -AM myocardial imaging was performed using scinticamera. T_2 relaxation time (T_2) was calculated using NMR-CT. Then, histopathologic rejection score was also calculated and the severity of rejection was divided into mild, moderate and severe. ^{111}In -AM uptake was correlated with T_2 and pathologic score. ^{111}In -AM uptake was higher in moderate and severe rejected myocardium, while T_2 was prolonged from mild to severe rejected myocardium. Therefore, ^{111}In -antimyosin uptake was specific for myocyte necrosis with edema, or mononuclear infiltration compared with T_2 . This procedure allows to evaluate the therapeutic intervention upon cardiac rejection, leading to replacement of right ventricular endomyocardial biopsy.

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**A NEW METHOD FOR INFARCT SIZING
-IMAGING OF ACUTE MYOCARDIAL INFARCTION WITH ^{111}In LABELED ANTIMYOSIN
HEAVY CHAIN MONOCLONAL ANTIBODY-**

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In order to visualize and quantify the size of myocardial infarction (MI) noninvasively, we used monoclonal antibody (HMC50) specific for cardiac myosin heavy chain. HMC50 Fab fragment was labeled with indium(^{111}In)-111, via a bifunctional chelating agent, diethylene triamine penta-acetic acid. MI was produced by ligation of either left anterior descending (7 dogs) or circumflex coronary artery (21 dogs) in 28 dogs, weighing 7-8 kg. In another dog right ventricular infarction was produced by right coronary ligation. At various intervals during 28 days after MI, single-photon emission computed tomography (SPECT) was performed using ^{111}In -HMC50 Fab (0.4 mCi/kg intravenously 22-24 hours before imaging) together with thallium (^{201}Tl)-201 (0.1 mCi/kg, 0.5 hour before imaging). In 8 cases technetium-99m pyrophosphate (^{99m}Tc -PYP, 3 mCi/kg, 3 hours before imaging) scintigrams were also taken 2 days before ^{111}In scintigraphy. At necropsy the hearts were cut into 3 mm-slices. Pathologic infarct size (P-IS) determined by planimetry was compared with the size of hot spot of ^{111}In (^{111}In -IS).

Infarct zone was clearly visualized by the ^{111}In hot spot throughout 16 days after MI in all 25 cases. The location of the hot spot approximated to the actual infarct site and the cold spot of ^{201}Tl . In 3 cases 17-28 days after MI, no hot spot of ^{111}In was detectable in 3 cases. Uptake of ^{99m}Tc -PYP was scarcely visible beyond 7 days after coronary ligation. Unlike ^{99m}Tc -PYP no bone uptake was noted in the images by ^{111}In . Subendocardial (7 cases) and right ventricular infarction (1 case) were also visualized by ^{111}In . During 8 days after left coronary ligation, ^{111}In -IS correlated well with P-IS ($r = 0.92$; $p < 0.001$ in 8 cases).

In conclusion SPECT with labeled HMC50 Fab was useful for infarct sizing as long as two weeks after acute myocardial infarction.

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THE STUDY OF THE PROCESS OF HEART FAILURE AND THE RESTORATION IN EXPERIMENTAL ACUTE COR PULMONALE

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Many investigator have reported the hemodynamics of acute cor pulmonale, clinically and experimentally. However, the quantitative evaluation of the effect of coronary perfusion pressure and hypoxia on right ventricular contractility is still unknown. We simulated acute cor pulmonale in nine isolated canine hearts and investigated the process of heart failure and restoration. Afterload, preload of right ventricle was controlled with a computer-assisted volume control servosystem. And at the same time, coronary perfusion pressure (COPP) was also controlled with a computer by measured cardiac output. Decrease in contractility and right ventricular failure was defined as decrease in E_{max} and peak systolic pressure despite of increased afterload respectively. Within the normal range of PaO_2 , the contractility of right ventricle decreased at COPP 64 ± 7.1 mmHg and right ventricle failed at COPP 51 ± 5.1 mmHg. Within the range of PaO_2 , 51 ± 2.3 mmHg, the contractility of right ventricle decreased at COPP 72 ± 3.8 mmHg and right ventricle failed at COPP 59 ± 8.8 mmHg. As long as the COPP was normal, 90 mmHg, contractility of right ventricle did not decrease despite of hypoxia (PaO_2 , 51 ± 2.3 mmHg). The increase in systemic vascular resistance or administration of β -stimulant in the heart failure state restored the contractility of right ventricle, but increase in preload decreased the contractility of right ventricle.

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CLINICAL SIGNIFICANCE OF LOW T_3 SYNDROME IN ACUTE MYOCARDIAL INFARCTION

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To assess the effect of thyroid hormone on left ventricular performance in acute myocardial infarction, serum T_4 and T_3 levels were serially measured by RIA