The biopsied myocardium was examined by HE staining, and in 23 pts (M 9, HM 14) frozen section were prepared for immunoperoxidase staining (ABC method). Monoclonal antibodies; common leukocyte (DAKO-LC), T-cell (Leu4), B-cell (Leu12), helper/inducer (OKT4), suppressor/cytotoxic (OKT8), monocyte/macrophage (LeuM3) and NK/K cell (Leu7). The severity of M was classified by the number of inflammatory infiltrates; Grade I 5-10/high power field, Grade II 10-20/HPF, Grade III > 20/HPF. The majority of 61 pts revealed neither preceding common cold nor other inflammatory signs. Clinical diagnosis; AV block above 2nd degree 12 (M 2, HM 10), SSS 10 (M 8, HM 2), PSVT 7 (M 1, HM 6) and others. In M and HM, cardiac functions were relatively well preserved. Immunohistological studies: The ratio of Leu4 and Leu12 were 76.3% and 23.7% in M, 80.6% and 19.4% in HM. Subset of inflammatory infiltrates; OKT4 24.4%, OKT8 32.2%, LeuM3 22.6%, Leu7 13.0% and Leu12 7.8% in M. OKT4 8.0%, OKT8 36.3%, LeuM3 33.0%, Leu7 14.8% and Leu12 8.0% in HM. OKT 4/8 was 0.75 in M and 0.22 in HM. In peripheral blood, OKT4/8 was 2.08 ± 0.80 in 6 pts of M.

Conclusion; The majority of 61 pts was latent myocarditis that seemed to be pathogenesis of SSS or PSVT etc. Immunohistological studies; OKT8 and LeuM3 were found predominantly in M and HM. OKT4/8 in myocardium and peripheral blood was reversed. In HM, LeuM3 was increased and OKT4 was decreased. Endomyocardial biopsy and immunohistological studies seemed to be useful for diagnosis of myocarditis.

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IN SITU IDENTIFICATION OF T LYMPHOCYTE SUBSETS IN THE MYOCARDIAL BIOPSIES FROM PATIENTS WITH DILATED CARDIOMYOPATHY

-IMMUNOHISTOCHEMICAL STUDY USING MONOCLONAL ANTIBODIES-

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The present light (LM) and electron microscopic (EM) study provides an immunohistochemical demonstration of T lymphocyte (Lym) subsets in the myocardial biopsies (Bx) from 11 patients (pts) with dilated cardiomyopathy (DCM), 3 pts with viral or idiopathic myocarditis (MYO) and 2 pts with arrhythmias (ART). Immunohistochemistry. Frozen sections of each Bx were incubated with anti Leu 2a or Leu 3a+b antibodies and peroxidase conjugated anti-mouse immunoglobulin antibody. In the LM section, the percentages of cytotoxic/-suppressor (Tc/s) or helper/inducer T Lym (Th/i) in the total interstitial cells was calculated. The percentages of Tc/s of total interstitial cells were $8.7 \pm 3.5\%$, $9.1 \pm 5.4\%$, $1.8 \pm 0.4\%$ and the percentages of Th/i was $5.5 \pm 3.6\%$, $7.4 \pm 8.5\%$, $0.9 \pm 0.3\%$, in DCM, MYO and ART, respectively. Leu 3⁺/Leu 2a⁺ was 0.7 ± 0.6 in DCM, and in 10 pts of DCM the ratios were less than 1.0. In contrast, there were no consistent pattern of T cell subset distribution in MYO. In immuno-EM some Th/i were in close contact with macrophages in MYO. And conventional EM revealed that some Lym were in close contact

with plasma membrane of cardiocytes. In vitro study. In all pts, the percentages of T, B cells and T cell subsets were in normal range in the peripheral blood (PB). Conclusion. In our DCM pts, T cell subsets remained normal in the PB, while in the Bx there was aberration in frequency of T cell subsets. A decreased ratio of Leu 3⁺/Leu 2a⁺ suggests predominant infiltration of Tc/s in the Bx. Predominant infiltrates of Tc/s indicate the possibility that these Lym involve in cardiocyte injury in the myocardium in DCM.

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THE EFFECT OF PANCREATIC TRANSPLANTATION ON EXPERIMENTAL DIABETIC MYOCARDIOPATHY

-A QUANTITATIVE ULTRASTRUCTURAL STUDY-

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We have observed the ultrastructural changes of the myocardium in diabetic rats and tried to evaluate the effect of pancreatic transplantation on diabetic cardiomyopathy. Male Lewis rats were divided into three groups (4 rats each): 1) normal, 2) diabetic rats induced by intravenous administration of streptozotocin (STZ) 60 mg/kg, and 3) STZ induced diabetic rats with pancreatic transplantation. All animals were fed for 2 months, and were then examined. Twelve to fifteen blood capillaries from each tissue block were photographed in the electron microscope, and thickness of the basement membrane of the vessels was estimated by Williamson's method. Electron Microscopy: In the control group, the myocytes and capillaries appeared to be normal in the myocardium. In the diabetic group, various morphological changes were seen, including a thickening in the basal lamina of capillaries and that of unmyelinated nerve processes, and an accumulation of lipid droplets, vacuolar formation and an increased number of myelin figures in many cardiocytes. In the group with transplants, the above changes were very much smaller in size and degree (Table 1). These findings show that pancreatic transplantation decreases the incidence and severity of cardiac ultrastructural changes in diabetic rats.

Table 1

	Normal	Diabetic	Transplanted
Fasting blood sugar (mg/dl)	116 ± 11	$439 \pm 67^{**}$	$124 \pm 11^{**}$
Body weight (Bw) (g)	386 ± 15	$270 \pm 16^{**}$	$366 \pm 19^{**}$
Heart weight (Hw) (g)	0.953 ± 0.093	$0.782 \pm 0.067^*$	0.867 ± 0.050
Hw/Bw $\times 10^{-3}$	2.46 ± 0.20	2.90 ± 0.26	$2.37 \pm 0.11^*$
Diameter of cardiocytes (μm)	21.58 ± 3.58	$18.77 \pm 2.99^{**}$	$21.40 \pm 3.41^{***}$
Basement membrane width ($\text{\AA}$)	590 ± 152	$882 \pm 185^*$	$601 \pm 156^*$

*p<0.05 **p<0.001 statistically different from normal group

p<0.05 *p<0.001 statistically different from diabetic group

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EFFECT OF ONO-3114, A FREE RADICAL SCAVENGER ON THE REOXYGENATION INJURY IN THE ANOXIC MYOCARDIUM

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